DERMATOLOGY
FINAL PAST PAPERS

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Explanations and notes are added depending on salsabela slides as a main source and other trusted sources

SPECIAL THANKS ☺

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Notes before we start.

1. This file contains all the available P.Ps regarding Dermatology rotation all up to 020 Batch sectioned by topics.

2. Some Answers has "?" next it, means not sure or the answer actually differs on the new guidelines

الدعاء بالرحمة للزميل عمر عطية المرابي

اللَّهُمَّ، اغْفِرْ له وَارْحَمْهُ، وَاعْفُ عنْه وَعَافِهِ، وَأَكْرِمْ نُزُلَهُ، وَوَسِّعْ مُدْخَلَهُ، وَاغْسِلْهُ بِمَاءٍ وَثَلْجٍ وَبَرَدٍ، وَنَقِّهِ مِنَ الخَطَايَا كما يُنَقَّى الثَّوْبُ الأبْيَضُ مِنَ الدَّنَسِ

Outlines

دعاء قبل المذاكرة

اللَّهُمَّ إِنِّي أَسْأَلُكَ فَهْمَ النَّبِيِّينَ، وَحِفْظَ الْمُرْسَلِينَ وَالْمَلَائِكَةِ الْمُقَرَّبِينَ،
اللَّهُمَّ اجْعَلْ أَلْسِنَتَنَا عَامِرَةً بِذِكْرِكَ، وَقُلُوبَنَا بِخَشْيَتِكَ، وَأَسْرَارَنَا بِطَاعَتِكَ،
إِنَّكَ عَلَى كُلِّ شَيْءٍ قَدِيرٌ، وَحَسْبُنَا اللَّهُ وَنِعْمَ الْوَكِيلُ..

Normal Skin & Skin Lesions

Q1

Which of the following is false about normal skin?

A.Sebaceous glands originate from ectoderm
B. Merkel cells are dendritic cells that are present near nerve endings
C. Mitotic cells only seen at the basal layer
D. Melanocytes connect to 36 surrounding keratinocytes
E. Melanocytes appear clear and big relative to surrounding cells under the microscope

Answer: B
Explanation:
Normal skin consists of three main layers: epidermis, dermis, and subcutaneous tissue. The epidermis contains key cell types: keratinocytes (majority; barrier/keratinization), melanocytes (basal layer; melanin production for UV protection), Langerhans cells (dendritic antigen-presenting immune cells), and Merkel cells (basal layer mechanoreceptors associated with sensory nerve endings for light touch). Merkel cells are not dendritic immune cells; Langerhans cells are the dendritic cells. Epidermal mitosis is limited mainly to basal keratinocytes (stem cells) in the stratum basale. Melanocytes form an epidermal melanin unit, transferring melanin to surrounding keratinocytes (commonly around 30–40, often cited as ~36). On routine histology, melanocytes can appear clearer and larger relative to surrounding keratinocytes.
[A] Sebaceous glands develop from epidermal (ectodermal) downgrowths.
[B] Merkel cells function as mechanoreceptors; dendritic antigen-presenting cells are Langerhans cells.
[C] Epidermal regeneration/mitosis occurs in the basal layer.
[D] One melanocyte supplies melanin to ~30–40 keratinocytes (often cited ~36).
[E] Melanocytes may appear pale/clear and relatively larger on microscopy.
Home takeaway:
Merkel = light touch receptor
Langerhans = dendritic immune cell
Mitosis = basal layer
Melanocyte connects to ~36 keratinocytes

Q2

Sebaceous glands are normally not found in:

A. Face
B. Vermilion of lip
C. Upper back
D. Buccal mucosa
E. Areola of nipple
Answer: D

Explanation:
Sebaceous glands are holocrine glands usually associated with hair follicles and produce sebum to lubricate skin and hair. They are abundant on face, scalp, chest, and upper back. Modified sebaceous glands occur in specific sites such as areola (Montgomery glands) and eyelids (Meibomian glands). Sebaceous glands are absent in glabrous skin (palms/soles) and are not normally present on true mucosal surfaces. Buccal mucosa is a mucous membrane lining the cheek and does not normally contain cutaneous adnexal structures like sebaceous glands (rare ectopic Fordyce spots may occur as a variant).
[A] Face is sebum-rich with many sebaceous glands.
[B] Vermilion border is modified skin and may show sebaceous structures.
[C] Upper back is a classic sebaceous-rich region.
[D] Buccal mucosa is mucosa, not typical skin; sebaceous glands are not normally present.
[E] Areola contains modified sebaceous glands (Montgomery glands).
Home takeaway:
Sebaceous glands → hair-bearing skin/sebum-rich areas
Absent in mucosa and in palms/soles

Q3

Melanin of Caucasians & Negros differs in all except:

A. Size of melanosomes
B. No. of melanosomes in melanocytes
C. Degree of dispersal of melanosomes
D. No. of melanin producing cells
E. No. of melanosomes in keratinocytes
Answer: D

Explanation:
Human skin color differences are primarily due to melanosome characteristics and melanin production, not the number of melanocytes. Across races, melanocyte number per unit area is generally similar; differences arise from melanosome size, number, distribution/dispersion, rate of melanin synthesis, and persistence of melanosomes within keratinocytes.
[A] Darker skin typically has larger melanosomes.
[B] Melanosome number/packaging differs functionally.
[C] Melanosomes are more dispersed in keratinocytes in darker skin.
[D] Melanocyte number is similar across races (this is the "does not differ" factor).
[E] Keratinocyte melanosome load/persistence differs.
Home takeaway:
Race differences = melanosome size/number/distribution + melanin synthesis
NOT melanocyte number

Q4

Sense of touch mediated by:

A. Free nerve endings
B. Meissner corpuscles
C. Pacini corpuscles
D. Muco-cutaneous endings
E. Superficial nerve plexus
Answer: B

Explanation:
Cutaneous sensation is mediated by specialized receptors. Meissner corpuscles (superficial dermal papillae) mediate fine/light touch and tactile discrimination, especially in glabrous skin (fingertips). Pacinian corpuscles (deep dermis/subcutis) mediate deep pressure and vibration. Free nerve endings mediate pain and temperature. Merkel cell-neurite complexes also contribute to light touch and sustained pressure.
[A] Free nerve endings mainly detect pain and temperature.
[B] Meissner corpuscles detect light touch and fine tactile discrimination.
[C] Pacinian corpuscles detect deep pressure and vibration.
[D] Not the primary standard classification for touch receptors here.
[E] A nerve plexus is structural innervation, not a specialized touch receptor.
Home takeaway:
Meissner = light touch
Pacini = deep pressure/vibration
Free nerve endings = pain/temp

Q5

Meibomian gland is an:

A. Eccrine gland
B. Apocrine gland
C. Sebaceous gland
D. Holocrine gland
E. Lacrimal gland
Answer: C

Explanation:
Meibomian glands are modified sebaceous glands located in the tarsal plates of the eyelids. They secrete lipids (meibum) that stabilize the tear film and reduce tear evaporation. Sebaceous glands use holocrine secretion (cells disintegrate to release contents). Eccrine glands produce watery sweat; apocrine glands secrete via decapitation; lacrimal glands produce aqueous tears.
[A] Eccrine glands produce watery sweat for thermoregulation.
[B] Apocrine glands use decapitation secretion (not Meibomian).
[C] Meibomian glands are modified sebaceous glands.

[D] Holocrine describes the secretion mechanism (sebaceous), not a separate gland category here.
[E] Lacrimal glands produce aqueous tears, not meibum.
Home takeaway:
Meibomian = modified sebaceous gland (lipid tear film layer)

Q6

Different color of people caused by differences in all except:

A. Size of melanosomes
B. Number of melanosomes
C. Concentration of melanocytes
D. Rate of melanocyte consumption
E. Rate of melanin synthesis
Answer: D

Explanation:
Skin color variation depends on melanin biology rather than melanocyte "consumption." Major determinants include melanosome size, melanosome number/packaging, melanosome dispersion within keratinocytes, rate of melanin synthesis, and melanosome persistence/degradation. Melanocyte density is broadly similar across races and is not the major driver compared with melanosome factors. "Rate of melanocyte consumption" is not a recognized physiological determinant of skin color.
[A] Melanosome size influences pigmentation intensity.
[B] Melanosome number/packaging contributes to darker pigmentation.
[C] Melanocyte density is not the main driver, but it is a biological variable; race differences are mainly melanosomal.
[D] "Melanocyte consumption" is not a standard determinant of pigmentation.
[E] Higher melanin synthesis rate increases pigmentation.
Home takeaway:
Pigmentation differences = melanosomes + melanin synthesis
Not "melanocyte consumption"

Q7

Which is associated with atopy:

A. Ichthyosis simplex
B. X-linked ichthyosis
C. Lamellar ichthyosis
D. Bullous ichthyosiform hyperkeratosis
E. Ichthyosis hystrix
Answer: A

Explanation:
Ichthyosis vulgaris (often referred to as ichthyosis simplex) is the most common inherited ichthyosis. It presents with dry skin and fine scaling, mainly on extensor surfaces, often with flexural sparing. It is frequently associated with keratosis pilaris and is commonly linked to atopic conditions (atopic dermatitis, allergic rhinitis, asthma).
[A] Ichthyosis vulgaris is commonly associated with atopy.
[B] X-linked ichthyosis is a steroid sulfatase deficiency; not classically atopy-associated.
[C] Lamellar ichthyosis is congenital AR ichthyosis; not classically linked to atopy.
[D] Bullous ichthyosiform hyperkeratosis is due to keratin mutations; not a typical atopy association.
[E] Ichthyosis hystrix is a severe keratinization disorder without classic atopy association.
Home takeaway:
Ichthyosis vulgaris/simplex = most common + associated with atopy

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Q8

Keratinization process is defective in all except:

A. Lichen sclerosus atrophicans
B. Ichthyosis hystrix
C. Psoriasis
D. Epidermolytic hyperkeratosis
E. Ichthyosis lamellaris
Answer: A

Explanation:
Keratinization disorders involve abnormal keratinocyte differentiation and stratum corneum formation, producing hyperkeratosis and scaling (e.g., ichthyoses, psoriasis, epidermolytic hyperkeratosis). Lichen sclerosus is primarily an inflammatory atrophic dermatosis characterized by epidermal thinning and dermal sclerosis rather than a primary disorder of keratinization.

[A] Lichen sclerosus is mainly atrophic/inflammatory, not a primary keratinization defect.
[B] Ichthyosis hystrix is a keratinization disorder.
[C] Psoriasis involves abnormal keratinocyte proliferation/differentiation and parakeratosis.
[D] Epidermolytic hyperkeratosis is a keratin mutation disorder.
[E] Lamellar ichthyosis is a congenital keratinization disorder.
Home takeaway:
Keratinization disorders → psoriasis + ichthyosis
Lichen sclerosus → atrophy/sclerosis

Q9

Glabrous skin characterized by all of the following except:

A. Dermatoglyphics
B. Thick epidermis
C. Presence of encapsulated organs
D. Presence of sebaceous gland
Answer: D

Explanation:
Glabrous skin (palms and soles) is specialized thick skin. It has a thick epidermis with prominent stratum corneum and a well-developed stratum granulosum. It lacks hair follicles and sebaceous glands, and it is rich in specialized sensory receptors. Dermatoglyphics (fingerprints) are characteristic.
[A] Dermatoglyphics are typical of palms/soles.
[B] Thick epidermis is characteristic.
[C] Rich in encapsulated sensory receptors.
[D] Sebaceous glands are absent.
Home takeaway:
Glabrous skin = thick epidermis, rich receptors, no hair, no sebaceous glands

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Q10

Sensation of pressure in skin is mediated by:

A. Autonomic n.
B. Mucocutaneous end organs
C. Vater Pacini corpuscles
D. Meissner corpuscles
Answer: C

Explanation:
Mechanoreceptors include Meissner corpuscles (light touch) and Pacinian (Vater-Pacini) corpuscles (deep pressure and vibration). Pacinian corpuscles are located deep in the dermis/subcutaneous tissue and detect deep pressure/vibration. Autonomic nerves regulate vessels and glands rather than mediating pressure sensation.
[A] Autonomic nerves regulate glands/vessels, not pressure sensation.
[B] Not a specific named pressure receptor.
[C] Pacinian corpuscles detect deep pressure and vibration.
[D] Meissner corpuscles detect light touch.
Home takeaway:
Pacini = deep pressure/vibration
Meissner = light touch

Q11

Mitotic division in epidermis is limited to:

A. Basal cells
B. Melanocyte
C. Granular cells
D. Prickle cells
Answer: A

Explanation:
The epidermis is organized into layers, with proliferative capacity mainly in the stratum basale. Basal keratinocytes act as stem cells that divide to regenerate the epidermis. As keratinocytes migrate upward into spinous and granular layers, they differentiate and lose proliferative capacity.
[A] Basal keratinocytes are the mitotically active stem-cell layer.
[B] Melanocytes synthesize melanin and are not the main proliferative cell population.
[C] Granular cells are differentiated keratinocytes with keratohyalin granules.
[D] Prickle (spinous) cells are differentiated and not primarily mitotic.
Home takeaway:

Epidermal mitosis occurs in stratum basale (basal keratinocytes)

Q12

One of the following cells is dendritic:

A. Langerhans
B. Histiocytes
C. T cells
D. B cells
Answer: A

Explanation:
Langerhans cells are dendritic antigen-presenting cells within the epidermis (mainly stratum spinosum). They originate from bone marrow lineage and are involved in recognizing, processing, and presenting antigens to T cells.
[A] Langerhans cells are dendritic antigen-presenting cells in epidermis.
[B] Histiocytes are tissue macrophages; not the classic epidermal dendritic APC here.
[C] T cells are lymphocytes, not epidermal dendritic cells.
[D] B cells are antibody-producing lymphocytes.
Home takeaway:
Langerhans = dendritic antigen-presenting immune cell in epidermis

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Q13

Not true about normal skin:

A. Stratum corneum is devoided from nuclei
B. Pacinian corpuscles are for touch
C. Sweat glands controlled by neurons
Answer: B

Explanation:
The stratum corneum consists of dead, anucleated keratinized cells. Sweat glands (especially eccrine) are primarily under neural control via sympathetic cholinergic fibers. Pacinian corpuscles are deep mechanoreceptors for pressure and vibration, not light touch.
[A] Stratum corneum cells are anucleated.
[B] Pacinian corpuscles detect deep pressure/vibration, not touch discrimination.
[C] Sweat glands are controlled mainly by autonomic (neural) pathways.
Home takeaway:
Stratum corneum = anucleated
Pacini = pressure/vibration
Sweat glands = neural control

Q14

Function of Meissner corpuscle:

A. Sense of touch
B. Erector pili
C. Pressure
D. Innervates smooth muscles of vessels
Answer: A

Explanation:
Meissner corpuscles are superficial encapsulated receptors in dermal papillae that detect fine/light touch and tactile discrimination, especially in glabrous skin. Pressure and vibration are mainly detected by Pacinian corpuscles. Erector pili and vascular smooth muscle are controlled by autonomic nerves.
[A] Meissner corpuscles mediate fine light touch.
[B] Erector pili is a smooth muscle associated with hair follicles.
[C] Deep pressure is mainly Pacinian-mediated.
[D] Vascular smooth muscle innervation is autonomic.
Home takeaway:
Meissner = fine light touch receptor

Q15

One of the following is correct regarding herald patch:

A. It is a well demarcated plaque over the knee
B. It is a scaly lesion associated with pityriasis rosea
C. It is a polygonal purple lesion
D. Vesicular lesion
Answer: B

Explanation:
Herald patch is the initial lesion of pityriasis rosea: a single, well-defined oval/round scaly plaque (often with collarette scale) that precedes the generalized eruption along cleavage lines. It is not typically vesicular. Polygonal purple lesions describe lichen planus. It is not specifically localized to the knee.
[A] Not characteristic or specific.
[B] Herald patch is associated with pityriasis rosea and is scaly.
[C] Polygonal purple lesions suggest lichen planus.
[D] Herald patch is not typically vesicular.
Home takeaway:
Herald patch = first scaly plaque of pityriasis rosea

Q16

Wrong statement:

A. Apocrine sweat glands are characterized by decapitation secretion
B. Eccrine sweat glands have cholinergic innervation
C. Sebaceous glands are controlled by androgens
D. None of the above
Answer: D

Explanation:
Apocrine glands secrete via decapitation. Eccrine glands are innervated by sympathetic cholinergic fibers and produce watery sweat for thermoregulation. Sebaceous glands are androgen-sensitive and increase activity at puberty. Since A–C are correct, the incorrect option is "None of the above."
[A] Apocrine secretion is decapitation type.
[B] Eccrine glands have cholinergic innervation.
[C] Sebaceous glands are androgen-responsive.
[D] Incorrect because A–C are correct.
Home takeaway:
Apocrine = decapitation
Eccrine = cholinergic
Sebaceous = androgen-dependent

Q17

Wrong in ichthyosis vulgaris:

A. Most common type
B. Usually associated with keratosis pilaris
C. Present at birth
D. Sparing flexures
Answer: C

Explanation:
Ichthyosis vulgaris is the most common inherited ichthyosis. It presents with fine scaling, mainly on extensor surfaces, often sparing flexures, and is commonly associated with keratosis pilaris and atopy. It usually presents in early childhood rather than at birth.
[A] It is the most common ichthyosis.
[B] Commonly associated with keratosis pilaris.
[C] Incorrect because it usually presents after birth in early childhood.
[D] Flexures are typically spared.
Home takeaway:
Ichthyosis vulgaris = common, childhood onset, extensor scaling with flexural sparing

Q18

Congenital ichthyosis associated with renal agenesis and hernia:

A. X-linked
B. Vulgaris
Answer: A

Explanation:
X-linked ichthyosis is due to steroid sulfatase deficiency and can have systemic associations (classically cryptorchidism and corneal opacities; renal associations may be reported). Ichthyosis vulgaris is usually limited to skin and associated with atopy rather than congenital systemic malformations.
[A] X-linked ichthyosis has recognized systemic associations.
[B] Ichthyosis vulgaris is not typically linked to congenital malformations.
Home takeaway:
X-linked ichthyosis may have systemic associations; ichthyosis vulgaris is usually skin-limited

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Q19

Dermatoscope, what is WRONG:

A. Used for seeing hyphae and spores
B. Used for pigmented lesions
C. Used for alopecia areata
D. Hand-held tool
Answer: A

Explanation:
A dermatoscope is a non-invasive hand-held tool used to visualize subsurface structures and patterns in skin lesions not visible to the naked eye. It is widely used for pigmented lesion assessment and can assist in hair disorders such as alopecia areata by revealing characteristic patterns. Fungal hyphae/spores require microscopic examination (e.g., KOH prep), not dermoscopy.
[A] Hyphae/spores require microscopy (KOH), not dermoscopy.
[B] Dermoscopy is standard for pigmented lesion evaluation.
[C] Dermoscopy can assist in alopecia areata assessment.
[D] Dermatoscopes are hand-held clinical tools.
Home takeaway:
Dermatoscope = patterns/subsurface structures; fungi = KOH microscopy

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Q20

One of the following condition leads to thickening of all skin layers:

A. Ichthyosis simplex
B. Ichthyosis hystrix
C. Ichthyosis nigricans
D. Ichthyosis congenital
Answer: C

Explanation:
Acanthosis nigricans (often loosely referenced in such questions) presents with velvety hyperpigmented plaques in intertriginous areas. Histologically it shows epidermal hyperplasia (acanthosis) and hyperkeratosis, giving a clinically thickened appearance. It is commonly associated with insulin resistance and can rarely be a paraneoplastic sign (especially gastric adenocarcinoma).
[A] Ichthyosis simplex mainly causes scaling.
[B] Hystrix is a keratinization disorder with hyperkeratosis but not classically "all layers" thickening in this framing.
[C] Acanthosis nigricans causes epidermal hyperplasia and clinically thickened velvety plaques.
[D] Congenital ichthyoses primarily cause scaling/cornification abnormalities.
Home takeaway:
Acanthosis nigricans = velvety thickened hyperpigmented plaques, insulin resistance association

Q21
Ichthyosis vulgaris wrong:
A. Usually involves extensors
B. Involves flexors
Answer: B

Explanation:
Ichthyosis vulgaris typically involves extensor surfaces with fine scaling and shows flexural sparing. Flexural involvement is not typical and helps distinguish it from other eczematous conditions.
[A] Extensor involvement is typical.
[B] Flexures are usually spared.
Home takeaway:
Ichthyosis vulgaris = extensor scaling + flexural sparing

Q22
Bullous ichthyosis erythroderma is inherited as:
Answer: Autosomal dominant

Explanation:
Bullous ichthyosiform erythroderma (epidermolytic hyperkeratosis) is due to keratin mutations (commonly KRT1/KRT10) and typically shows erythema with blistering early in life followed by hyperkeratosis. Inheritance is classically autosomal dominant.
Home takeaway:
Bullous ichthyosiform erythroderma = keratin mutation = autosomal dominant

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Q23
True about non-bullous ichthyosiform erythroderma:
Answer: Autosomal recessive inheritance

Explanation:
Non-bullous congenital ichthyosiform erythroderma is a congenital ichthyosis presenting with erythroderma and scaling without blistering and is classically inherited in an autosomal recessive pattern.
Home takeaway:
Non-bullous congenital ichthyosis = autosomal recessive

Q24
Which of the following is false about normal skin:
Answer: The Meissner's corpuscles is responsible for pressure sensations

Explanation:
Meissner corpuscles detect fine/light touch, especially in glabrous skin. Deep pressure and vibration are detected mainly by Pacinian corpuscles located deeper in dermis/subcutis. Therefore attributing pressure sensation to Meissner corpuscles is false.
Home takeaway:
Meissner = light touch
Pacini = deep pressure/vibration

Q25
Wrong statement:
Answer: Sweat glands are controlled by hormones

Explanation:
Eccrine sweat glands are primarily controlled by autonomic nervous system input (sympathetic cholinergic). Hormones may influence sweating indirectly, but primary control is neural rather than hormonal.
Home takeaway:
Sweat glands = primarily neural control (sympathetic cholinergic)

Q26

Macule is:

Answer: < 1 cm flat.

Explanation:

A macule is a flat lesion (not raised) representing a change in skin color only and measuring less than 1 cm. A patch is the same concept but larger than 1 cm.

Home takeaway:

Macule = flat color change < 1 cm

Patch = flat color change > 1 cm

Eczema

Q1
Seborrheic dermatitis, all are true except:

A. Occurs in children and adults
B. Most common in the extensors
C. Occurs in age less than 3 months
D. Scalp cradle cap in babies
E. Post-auricular and nasolabial folds are common sites
Answer: B

Explanation:
Seborrheic dermatitis is a chronic, relapsing, usually mild endogenous eczema that affects sebum-rich areas. It has a biphasic age pattern (infants and adults). Malassezia (lipid-dependent yeast) plays a role in many cases. Typical sites include scalp (dandruff/cradle cap), eyebrows, eyelids, nasolabial folds, retro/post-auricular areas, upper trunk, and flexures. Management is long-term control: antifungal shampoos/creams (e.g., ketoconazole), short courses of low-potency topical steroids for flares, and maintenance therapy.
[A] Fits biphasic pattern (infants + adults).
[B] Wrong because distribution is sebum-rich areas and folds, not extensor predominance.
[C] Fits infant onset (can start from early infancy).
[D] Classic infant presentation (cradle cap).
[E] Typical seborrheic sites.
Home takeaway:
Seborrheic dermatitis = chronic relapsing mild eczema on sebum-rich areas (scalp/face/folds); treat with antifungals + short steroid courses.

Q2
All about seborrheic dermatitis are true except:

A. May occur earlier than atopic dermatitis
B. Self-limiting
C. Itching is mild
D. Chronic
E. Prognosis poorer than atopic dermatitis
Answer: E

Explanation:
Seborrheic dermatitis can begin very early in infancy and also occurs in adults. It is usually mild, often with mild itch, and tends to be chronic/relapsing, requiring maintenance treatment. Atopic dermatitis is typically intensely itchy and many patients improve/remit by puberty. Saying seborrheic dermatitis has a poorer prognosis than atopic dermatitis is the incorrect statement in this set.
[A] Can start very early in infancy.
[B] Not reliably true as a rule; tends to relapse/chronic course.
[C] Usually mild itch compared with atopic dermatitis.
[D] Chronic/relapsing course.
[E] Incorrect comparative statement.
Home takeaway:
Seborrheic dermatitis = early onset possible + chronic/relapsing; atopic dermatitis often improves by puberty.

Q3
A case about a 5-year-old with features of atopic dermatitis, what do you do?

A. CBC
B. Chest x-ray
C. Renal function
D. IgG
E. IgE
Answer: E

Explanation:
Atopic dermatitis is a chronic pruritic inflammatory eczema due to barrier dysfunction (e.g., filaggrin defect) and immune dysregulation (Th2 skew). It is associated with atopy and often elevated serum IgE (and sometimes eosinophilia). Routine labs are usually unnecessary unless a specific indication exists; if any lab is requested to

support an atopic picture, IgE is the most relevant.
[A] Not first-line; routine testing usually not needed.
[B] No role unless respiratory indication.
[C] No routine role.
[D] Not characteristic.
[E] Most relevant supportive lab test.
Home takeaway:
Atopic dermatitis often has elevated IgE; labs are supportive only and not routinely required.

Q4
Lymphocytes from patient with atopic dermatitis bear greater than normal amounts of:

A. IgG
B. IgA
C. IgE
D. IgM
E. IgD
Answer: C

Explanation:
Atopic dermatitis is part of atopy and is linked to IgE-mediated allergic tendency with Th2-driven immune responses. Increased IgE is a hallmark immunologic feature in atopic disease.
[A] Not the hallmark immunoglobulin in atopy.
[B] Not the hallmark immunoglobulin in atopy.
[C] Fits IgE-driven atopy.
[D] Not the hallmark immunoglobulin in atopy.
[E] Not the hallmark immunoglobulin in atopy.
Home takeaway:
Atopy/atopic dermatitis is associated with elevated IgE.

Q5
All of the following statements are true except:

A. Infant's atopic eczema mostly affect the flexural sites as popliteal fossa and the wrists
B. Juvenile plantar dermatosis is caused mainly due to socks and shoes that are impermeable
C. Lichenification may be seen in chronic eczema
D. Seborrheic eczema is linked to Malassezia
E. Eczema may be induced by both external and internal factors
Answer: A

Explanation:
Atopic dermatitis distribution changes with age: infants/young children mainly cheeks/face and extensor surfaces; older children/adults mainly flexures. Chronic eczema leads to lichenification (thickened skin with exaggerated markings) and hyperpigmentation from repeated scratching/rubbing. Seborrheic dermatitis is often linked to Malassezia. Eczema can be triggered by internal (endogenous) and external (exogenous) factors. Juvenile plantar dermatosis is considered a variant within the atopic spectrum affecting the forefoot with dry cracking in children.
[A] Wrong: infants typically have face/cheeks and extensor involvement; flexures dominate later.
[B] Juvenile plantar dermatosis is linked to atopic spectrum; "impermeable socks/shoes" is not a core defining cause.
[C] Chronic eczema can cause lichenification.
[D] Malassezia association is typical for seborrheic eczema.
[E] Eczema can have internal and external triggers.
Home takeaway:
Infant AD = cheeks/extensors; older AD = flexures. Chronic eczema → lichenification.

Q6
Which one cause allergic contact dermatitis:

A. Cobalt
B. Nickel
C. Cement
D. Rubber
E. Silver
Answer: B

Explanation:
Allergic contact dermatitis is a type IV (delayed) hypersensitivity reaction that typically appears 48–96 hours after exposure. Nickel is the classic and most common allergen (e.g., jewelry, watches). Diagnosis is confirmed by patch testing. Management is avoidance of the allergen plus eczema treatment (emollients, topical steroids; treat secondary infection if present).
[A] Can cause allergy but not the most typical single answer here.
[B] Classic common allergen (nickel).
[C] Can contain sensitizers (e.g., chromates), but nickel is the classic commonest.
[D] Rubber chemicals can cause dermatitis, often hand-related.
[E] Not a common classic allergen compared with nickel.
Home takeaway:
Allergic contact dermatitis = type IV delayed; nickel is the classic most common trigger.

Q7
Which one commonly cause severe even bullous contact dermatitis:

A. Cement
B. Primula
C. Cobalt
D. Leather
E. Rubber
Answer: B

Explanation:
Allergic contact dermatitis can be severe with vesicles and even bullae in strongly sensitized individuals. Primula (plant allergen "primin") is a classic cause of severe vesicular/bullous allergic contact dermatitis. Management: strict avoidance and topical corticosteroids for inflammation; treat infection if present.
[A] Possible sensitizer (chromates) but not classic severe bullous answer.
[B] Classic trigger for severe vesicular/bullous ACD (Primula/primin).
[C] Less typical as a classic "severe bullous" teaching point.
[D] Leather chemicals can cause dermatitis but not the classic bullous example.
[E] Rubber allergens can cause dermatitis, typically hand-related.
Home takeaway:
Primula is a classic cause of severe vesicular/bullous allergic contact dermatitis.

Q8
Which one of the following agents cause pigmented contact dermatitis:

A. Lipsticks
B. Nail varnish
C. Deodorants
D. Perfumes
E. Hair dyes
Answer: D

Explanation:
Pigmented contact dermatitis (Riehl melanosis) is a contact dermatitis pattern where hyperpigmentation is prominent, commonly linked to fragrances/perfumes and cosmetic products. Patch testing can help identify the responsible allergen. Management is avoidance of the trigger and anti-inflammatory treatment for any active dermatitis.
[A] Can cause cosmetic dermatitis, but fragrances are the classic association.
[B] Usually causes dermatitis at touched sites (often neck/eyelids), not the classic pigmented form.
[C] Can cause dermatitis, but perfumes/fragrances are the classic pigmented-contact trigger.
[D] Fragrances/perfumes are classic causes.
[E] Hair dyes often cause acute severe dermatitis (e.g., PPD), not classically "pigmented contact dermatitis."
Home takeaway:
Pigmented contact dermatitis is classically linked to fragrances/perfumes in cosmetics.

Q9
Adult atopic dermatitis may be associated with the following except:

A. Pruritus ani
B. Pruritus vulvae photodermatitis
C. Asthma
D. Hair fall
Answer: D

Explanation:
Atopic dermatitis is associated with atopy (asthma, allergic rhinitis) and can present with several eczema variants (e.g., pityriasis alba, keratosis pilaris, hand eczema, nipple eczema, juvenile plantar dermatosis). Anogenital itching can occur in eczema patterns. Hair fall is not a typical association of atopic dermatitis as a disease feature.
[A] Can occur as an eczema-related symptom in sensitive areas.
[B] Photodermatitis can coexist as an eczema pattern, but not a defining AD feature.
[C] Asthma is part of the atopic triad.
[D] Not a typical association of AD.
Home takeaway:
AD is linked to atopy (including asthma) and multiple eczema variants; hair fall is not a typical AD association.

Q10
Atopic dermatitis vs seborrheic dermatitis all true except:

A. Seborrheic start at earlier age
B. Seborrheic has worse prognosis
C. Atopic severe pruritis
D. Atopic is associated with +ve family Hx
Answer: B

Explanation:
Seborrheic dermatitis can start very early in infancy and is usually mild with less itch, affecting sebum-rich areas (scalp, eyebrows, nasolabial folds). Atopic dermatitis is intensely pruritic, commonly associated with personal/family history of atopy, and many cases improve by puberty. "Seborrheic has worse prognosis" is not correct in this comparison.
[A] Seborrheic can start earlier (very early infancy).
[B] Incorrect comparative statement.
[C] AD is characterized by severe pruritus.
[D] AD commonly has positive personal/family atopy history.
Home takeaway:
AD = severe itch + family atopy; seborrheic = early infant onset + sebum-rich distribution.

Q11
Contact dermatitis is clinical manifestation of:

A. Cytotoxic reaction
B. Arthus reaction
C. Cell mediated reaction
D. Anaphylactic reaction
Answer: C

Explanation:
Allergic contact dermatitis is a type IV delayed hypersensitivity reaction mediated by sensitized T lymphocytes. It typically appears 48–96 hours after exposure.
[A] Type II reaction, not ACD.
[B] Type III immune complex reaction.
[C] Type IV T-cell mediated delayed reaction.
[D] Type I immediate hypersensitivity.
Home takeaway:
Allergic contact dermatitis = type IV (cell-mediated) delayed hypersensitivity.

Q12
Pathognomonic test used in diagnosis of contact dermatitis:

A. Intradermal tests
B. Patch test
C. Prick test
D. Skin biopsy
Answer: B

Explanation:
Patch testing is the key diagnostic test for allergic contact dermatitis. Allergens are applied under occlusion and readings are taken after removal to identify delayed hypersensitivity reactions.
[A] Not standard for ACD diagnosis.
[B] Correct diagnostic test for ACD.
[C] Used for immediate (type I) allergy, not ACD.
[D] Biopsy is not pathognomonic; used when diagnosis is unclear.
Home takeaway:
Patch test identifies allergens in allergic contact dermatitis.

Q13
Histology of spongiosis & parakeratosis with normal granular layer suggests:

A. Eczema
B. Psoriasis
C. Lichen planus
Answer: A

Explanation:
Eczema (dermatitis) histology is characterized by spongiosis (intercellular epidermal edema), which can lead to vesicle formation. Inflammatory infiltrate is typically present in the dermis. Parakeratosis can be seen in inflammatory dermatoses. Overall, spongiosis supports eczema rather than psoriasis or lichen planus.
[A] Spongiosis is the hallmark of eczema.
[B] Psoriasis has a different characteristic histologic pattern (not dominated by spongiosis).
[C] Lichen planus has a different interface dermatitis pattern.
Home takeaway:
Spongiosis on histology strongly suggests eczema/dermatitis.

Q14
Eruption of an erythematous lesion on the face particularly the nasolabial folds, eyebrows, scalp, what's the diagnosis?

A. Seborrheic dermatitis
B. Eczema
C. Atopic dermatitis
D. Psoriasis
Answer: A

Explanation:
Seborrheic dermatitis favors sebum-rich areas: scalp and central face including eyebrows and nasolabial folds. It often presents with erythema and greasy scale. This distribution is classic and helps distinguish it from atopic dermatitis (which in children often favors cheeks/extensors and in older ages flexures).
[A] Classic distribution (nasolabial folds, eyebrows, scalp).
[B] Too nonspecific; the distribution pattern is typical for seborrheic.
[C] AD distribution differs by age and is usually more intensely itchy.
[D] Psoriasis can involve scalp but nasolabial fold pattern with greasy scale is more typical for seborrheic.
Home takeaway:
Nasolabial folds + eyebrows + scalp scaling/erythema = seborrheic dermatitis.

Q15
Commonest site of contact dermatitis produced by nail varnish is:

A. Neck
B. Nail folds
C. Nail
D. Back of hands
Answer: A

Explanation:
Nail varnish allergy often produces dermatitis at "touched sites" rather than only around the nails. Commonly affected areas include the eyelids and sides of the neck due to hand-to-skin transfer of allergens. Patch testing confirms the allergen. Treatment is avoidance plus topical steroids for flares.
[A] Classic touched-site dermatitis (neck).
[B] Can be involved, but touched sites are classic common presentation.
[C] Nail plate may change, but dermatitis is often elsewhere.
[D] Hands can be involved, but neck/eyelids are classic common sites.
Home takeaway:
Nail varnish allergy often shows dermatitis on touched sites (neck/eyelids).

Q16
Commonest site of contact dermatitis produced by clothes:

A. Body flexures
B. Scalp
C. Arms
D. Legs
Answer: A

Explanation:
Clothing-related contact dermatitis commonly affects areas of occlusion, friction, and sweating where textiles/dyes/finishes contact skin most intensely. Body flexures and areas under tight clothing (axilla, groin, waistband) are typical.
[A] Common due to occlusion/friction/sweat in flexures.
[B] Not typical for clothing contact dermatitis.
[C] Can occur but less typical than flexures/occluded areas.
[D] Can occur (e.g., stockings) but flexures are most typical teaching point.
Home takeaway:
Clothing contact dermatitis → flexures/occluded friction areas.

Q17
Unilateral hand eczema, best next step:

A. Scrap and do KOH
B. Potent topical steroids
C. Make him wear gloves
D. Give emollients
Answer: A

Explanation:
Unilateral hand "eczema" raises suspicion for fungal infection (tinea manuum), which can mimic eczema. The best next step is to confirm/exclude fungus with skin scrapings for KOH microscopy (and culture if needed). Steroids can worsen or mask fungal infections. Once fungus is excluded or treated, standard eczema therapy (emollients, topical steroids) can be used appropriately.
[A] Best step to rule out tinea with KOH scraping.
[B] Risks worsening tinea if misdiagnosed.
[C] Protective but not diagnostic or definitive.
[D] Helpful but does not address possible fungal cause.
Home takeaway:
Unilateral hand eczema → rule out tinea (KOH scraping) before steroids.

Q18
One false about infantile atopic dermatitis:

A. Increase incidence of contact eczema
B. Present at birth
C. Severe pruritus
Answer: B

Explanation:
Atopic dermatitis typically begins in infancy/early childhood, not at birth. It is intensely itchy and often affects cheeks/face and extensor surfaces in infants/young children. Barrier dysfunction increases susceptibility to irritants and allergens, raising the risk of contact dermatitis.
[A] Barrier defect increases susceptibility to contact dermatitis.
[B] Incorrect: AD is not typically present at birth.
[C] Severe pruritus is a hallmark of AD.
Home takeaway:
Infant AD = cheeks/extensors + severe itching; not usually present at birth.

Q19
True about atopic dermatitis:

A. T helper cells have the major role in pathophysiology
B. Most common site in children is extensor areas
C. Always associated with asthma
Answer: A

Explanation:
Atopic dermatitis pathophysiology involves impaired skin barrier and immune dysregulation with Th2 predominance. Distribution changes with age: infants/young children often have cheeks and extensor involvement; older children/adults typically have flexural disease. AD is associated with asthma and allergic rhinitis but not in every patient.
[A] Th2/T helper-driven immune imbalance is central.
[B] Extensors are common in younger children, but distribution is age-dependent.
[C] Not always associated with asthma.
Home takeaway:
AD = barrier defect + Th2 predominance; distribution depends on age; asthma association is common but not universal.

Q20
Wrong about atopic dermatitis:

A. Begin before 2 months
B. Itching
C. Steroids
Answer: A

Explanation:
Atopic dermatitis is characterized by intense itching. Topical corticosteroids are a cornerstone of treatment for inflammatory flares, alongside emollients and trigger avoidance. Typical onset is infancy/early childhood, but "before 2 months" is not a universal rule and is the incorrect statement in this question.
[A] Incorrect as a general rule.
[B] Hallmark feature.
[C] Mainstay treatment for flares.
Home takeaway:
AD = intense itch; treat flares with topical steroids + emollients.

Q21
Thickening and hardening of the skin, with exaggeration of its normal markings:

A. Lichenification
B. Spongiosis
C. Hyperkeratosis
Answer: A

Explanation:
Lichenification is thickening and hardening of the skin with accentuated markings due to chronic scratching/rubbing, commonly in chronic eczema. Spongiosis is intercellular edema seen microscopically in acute eczema. Hyperkeratosis is thickening of the stratum corneum (scaling), not specifically "exaggerated markings."
[A] Matches definition of lichenification in chronic eczema.
[B] Microscopic edema, not the gross thickened-markings change.
[C] Thick stratum corneum; not the classic accentuated markings pattern.
Home takeaway:
Lichenification = chronic rubbing/scratching → thickened skin with exaggerated markings.

Q22
All can cause blisters except:

A. Chronic eczema
B. Impetigo
C. Pemphigoid
Answer: A

Explanation:
Blisters (vesicles/bullae) are seen in acute eczema, bullous impetigo, and pemphigoid. Chronic eczema is dominated by lichenification, scaling, and xerosis rather than blistering.
[A] Chronic eczema mainly causes lichenification/scaling, not blisters.
[B] Bullous impetigo causes blisters.
[C] Pemphigoid is a blistering disease.
Home takeaway:
Blisters → acute eczema/impetigo/pemphigoid; chronic eczema → lichenification.

Q23
One is correct regarding seborrheic dermatitis:

A. Scaly lesion on anterior knee
B. Greasy scales on sebum rich areas
Answer: B

Explanation:
Seborrheic dermatitis typically presents with erythema and greasy scale on sebum-rich areas such as the scalp and central face (including eyebrows and nasolabial folds).
[A] Anterior knee scaling suggests other dermatoses more than seborrheic distribution.
[B] Classic description of seborrheic dermatitis.
Home takeaway:
Seborrheic dermatitis = greasy scales on sebum-rich areas.

Q24
Wrong about eczema:

Answer: Contact dermatitis develops 12 hours from exposure

Explanation:
Allergic contact dermatitis is a delayed (type IV) hypersensitivity reaction that typically develops 48–96 hours after exposure. Irritant contact dermatitis can occur sooner, but the statement as written is wrong for allergic contact dermatitis timing.
Home takeaway:
Allergic contact dermatitis appears after 48–96 hours, not 12 hours.

Q25
2–4 year old child with red, itchy weeping rash on his cheeks and extensor surfaces, most likely diagnosis:

A. Seborrheic dermatitis
B. Atopic eczema
Answer: B

Explanation:
Atopic eczema in young children commonly affects cheeks and extensor surfaces and is intensely itchy. Acute flares can ooze/weeping due to vesiculation and exudation. Seborrheic dermatitis typically favors scalp and central face folds with greasy scale and often less intense itch.
[A] Distribution and symptom pattern fit AD more than seborrheic.
[B] Best match: cheeks + extensors + itch + weeping.
Home takeaway:
Young child with itchy weeping cheeks + extensor rash = atopic eczema.

Q26
Wrong statement:

Answer: Pityriasis alba appears depigmented on Wood's light

Explanation:
Pityriasis alba is a mild eczema-related hypopigmented condition (often in children) that appears lighter but not truly depigmented. Under Wood lamp it is typically nonfluorescent and does not show the bright depigmentation seen in vitiligo.
Home takeaway:
Pityriasis alba = hypopigmented (not depigmented) and nonfluorescent on Wood lamp.

Q27
Type IV hypersensitivity for:

Answer: Allergic contact dermatitis

Explanation:
Allergic contact dermatitis is mediated by T cells (type IV delayed hypersensitivity) and typically appears 48–96 hours after exposure.
Home takeaway:
Allergic contact dermatitis = type IV delayed (T-cell mediated).

Q28
Choose the mismatch:

Answer: Patch test – irritant contact dermatitis

Explanation:
Patch testing identifies allergens responsible for allergic contact dermatitis. Irritant contact dermatitis is not an immune sensitization process and is diagnosed clinically based on exposure and distribution.
Home takeaway:
Patch test is for allergic contact dermatitis, not irritant dermatitis

Acne & Rosacea

Q1
In acne vulgaris the precursor of large inflammatory lesions is:

A. Black head
B. White head
C. Papules
D. Pustules
E. None of the above
Answer: C

Explanation:
Acne is a disorder of the pilosebaceous unit characterized by follicular hyperkeratinization, increased sebum production, proliferation of Propionibacterium acnes, and inflammation. The earliest lesion is the comedone (open or closed). Inflammatory acne progresses from comedones to papules, then pustules, nodules, and cystic lesions. Large inflammatory nodules and cysts are preceded by inflammatory papules.
[A] Blackhead is an open comedone and is non-inflammatory.
[B] Whitehead is a closed comedone and is non-inflammatory.
[C] Papules are early inflammatory lesions that can progress to larger nodulocystic lesions.
[D] Pustules represent a later inflammatory stage.
[E] Incorrect because papules are the precursor of larger inflammatory lesions.
Home takeaway:
Comedone → Papule → Pustule/Nodule (progressive inflammation).

Q2
A neighbor asks your advice about oral isotretinoin for her severe acne. One of the following is incorrect:

A. Increased triglycerides is a common side effect
B. The cumulative therapeutic dose varies from one person to another usually depending on their weight
C. Blood test must be done prior to initialization of treatment
D. All patients will experience some degree of lip dryness
E. She should not get pregnant for one year after treatment as it is teratogenic
Answer: E

Explanation:
Isotretinoin is indicated for severe or resistant acne. It reduces sebum production, follicular plugging, inflammation, and P. acnes. Common side effects include hypertriglyceridemia, elevated liver enzymes, and mucocutaneous dryness (especially lips). Baseline and follow-up blood tests are required. The cumulative dose is calculated according to body weight (approximately 120–150 mg/kg). Isotretinoin is highly teratogenic; contraception is required during treatment and for one month after stopping therapy, not one year.
[A] Hypertriglyceridemia is a known common side effect.
[B] Total cumulative dose is weight-based.
[C] Baseline blood tests are mandatory before starting therapy.
[D] Lip dryness occurs in nearly all patients.
[E] Pregnancy must be avoided during therapy and for one month after cessation, not one year.

Home takeaway:

Isotretinoin = teratogenic + monitor lipids/LFTs + contraception for 1 month after stopping.

Q3
Not a side effect of retinoic acid:

A. Thrombocytopenia

B. Elevating liver enzymes

C. Dryness of mucosal membranes

D. Diffuse hair loss

E. Increased intracranial pressure

Answer: A

Explanation:

Systemic retinoids commonly cause mucocutaneous dryness, elevated liver enzymes, hyperlipidemia, hair thinning, and rarely pseudotumor cerebri (increased intracranial pressure). Thrombocytopenia is not a typical adverse effect.

[A] Thrombocytopenia is not a recognized common side effect.

[B] Elevation of liver enzymes can occur.

[C] Dryness of lips and mucosa is very common.

[D] Diffuse hair thinning may occur.

[E] Increased intracranial pressure is a rare but reported complication.

Home takeaway:

Retinoids → dryness + ↑LFTs + ↑lipids + rare pseudotumor cerebri.

Q4
All occur in acne vulgaris except:

A. Pustules

B. Nodules

C. Comedones

D. Papules

E. Vesicles

Answer: E

Explanation:

Acne lesions include comedones (open and closed), papules, pustules, nodules, and cysts. Vesicles are not part of acne morphology.

[A] Pustules are inflammatory acne lesions.

[B] Nodules occur in severe acne.

[C] Comedones are diagnostic for acne.

[D] Papules are inflammatory lesions.

[E] Vesicles are not characteristic of acne vulgaris.

Home takeaway:

Acne lesions = comedones + papules + pustules + nodules (not vesicles).

Q5
Difference between acne vulgaris and rosacea:

A. Comedones
B. Pustules
C. Papules
D. Telangiectasia
E. Erythema
Answer: A + D

Explanation:
Acne vulgaris is characterized by comedones and inflammatory lesions. Rosacea presents with flushing, persistent erythema, telangiectasia, and papulopustular lesions but lacks comedones. Telangiectasia is typical of rosacea, not acne.
[A] Comedones are present in acne and absent in rosacea.
[B] Pustules may occur in both conditions.
[C] Papules may occur in both.
[D] Telangiectasia is characteristic of rosacea.
[E] Erythema can be seen in both.
Home takeaway:
Comedones = acne
Telangiectasia + flushing = rosacea.

Q6
All true about acne vulgaris except:

A. Isotretinoin is very effective in cystic form
B. Patients with acne usually have much higher titers to staph. albus than normal adults
C. Application of CO_2 slush is useful in reducing acne pit scars on the face
D. Greasy cosmetics may cause acne
E. Comedones predominate the picture in chlor-acne
Answer: B

Explanation:
Acne pathogenesis involves follicular plugging, increased sebum production, and proliferation of Propionibacterium acnes. It is not caused by Staphylococcus albus. Isotretinoin is effective in nodulocystic acne. Greasy cosmetics can be comedogenic. Chlor-acne presents predominantly with comedones.
[A] Isotretinoin is highly effective in cystic acne.
[B] Acne is associated with P. acnes, not Staphylococcus albus.
[C] Certain procedures may help reduce acne scars.
[D] Comedogenic cosmetics may exacerbate acne.
[E] Chlor-acne shows prominent comedones.
Home takeaway:
Acne bacterium = Propionibacterium (Cutibacterium) acnes.

Q7
All are side effects of isotretinoin except:

A. Teratogenicity
B. Hair loss
C. Elevated liver enzymes
D. Infertility
Answer: D

Explanation:
Isotretinoin causes teratogenicity, elevated liver enzymes, hyperlipidemia, mucocutaneous dryness, and sometimes hair thinning. It does not cause infertility.
[A] Highly teratogenic.
[B] Hair thinning may occur.
[C] Liver enzyme elevation is common.
[D] Infertility is not a recognized side effect.
Home takeaway:
Isotretinoin affects fetus, lips, liver, lipids—not fertility.

Q8
All of the following result in flare up of acne except:

A. Estrogen
B. Steroid
C. Antimalarial drugs
D. Vitamin B12
Answer: A

Explanation:
Certain medications such as steroids and vitamin B12 may exacerbate acne. Estrogen-containing oral contraceptives may improve acne by reducing androgen effect on sebaceous glands.
[A] Estrogen may improve acne.
[B] Steroids can induce acne.
[C] Some drugs may trigger acneiform eruptions.
[D] Vitamin B12 may worsen acne.
Home takeaway:
Estrogen improves acne; steroids may worsen it.

Q9
Acne, all true except:

A. Propionibacterium acne is incriminated
B. Isotretinoin is group D in pregnancy
C. Clindamycin is not given to children
D. Follicular plugging is the first step in pathogenesis
Answer: B

Explanation:

Acne pathogenesis begins with follicular hyperkeratinization and plugging. Propionibacterium acnes contributes to inflammation. Isotretinoin is highly teratogenic and classified as pregnancy category X, not D. Clindamycin is used topically in acne treatment.

[A] P. acnes plays a role in inflammation.

[B] Isotretinoin is pregnancy category X.

[C] Topical clindamycin is used in acne.

[D] Follicular plugging is the initiating step.

Home takeaway:

First step in acne = follicular hyperkeratinization.

Q10

Patient with moderate acne not responding to tetracycline since 6 months, you give:

A. Isotretinoin

B. Antiandrogen

C. Benzyl peroxide

D. Salicylic acid

Answer: A

Explanation:

In moderate to severe acne unresponsive to adequate oral antibiotics, isotretinoin is indicated because it targets all major pathogenic factors.

[A] Appropriate for resistant moderate-to-severe acne.

[B] Antiandrogens may be used in selected females.

[C] Topical agents alone are insufficient in resistant disease.

[D] Salicylic acid is for mild cases.

Home takeaway:

Failure of antibiotics → consider isotretinoin.

Q11

One of the following is first line treatment for mild acne:

A. Topical steroids

B. Tar coal

C. Topical benzoyl peroxide

D. Topical permethrin

Answer: C

Explanation:

First-line treatment for mild acne includes topical benzoyl peroxide and/or topical retinoids. Benzoyl peroxide has antibacterial and comedolytic effects.

[A] Steroids can worsen acne.

[B] Coal tar is not used for acne.

[C] Benzoyl peroxide is first-line therapy.

[D] Permethrin is used for scabies.

Home takeaway:

Mild acne → topical benzoyl peroxide ± retinoid.

Q12
Wrong about acne vulgaris:

A. Epidermal edema
B. Increase in sebum production
C. Stagnation of sebum
D. Proliferation of Propionibacterium
Answer: A

Explanation:
Acne pathogenesis includes increased sebum production, follicular plugging with sebum stagnation, and proliferation of P. acnes leading to inflammation. Epidermal edema is not a primary pathogenic feature.
[A] Not a central mechanism in acne.
[B] Increased sebum production is essential.
[C] Plugging causes sebum stagnation.
[D] Bacterial proliferation contributes to inflammation.
Home takeaway:
Acne = plugging + sebum + bacteria + inflammation.

Q13
Not side effect of retinoids:

A. Paronychia
B. Distal lamellar splitting
C. Nail thinning
Answer: B

Explanation:
Retinoids may cause mucocutaneous dryness, nail fragility, paronychia, and thinning. Distal lamellar splitting is not a typical classic adverse effect.
[A] Paronychia may occur with retinoids.
[B] Not a characteristic side effect.
[C] Nail thinning may occur.
Home takeaway:
Retinoids may affect nails and mucosa.

Q14
One is not side effect of Isotretinoin:

A. Increase lipids
B. Scarring alopecia
C. Dryness of mouth
D. Increase liver enzyme
Answer: B

Explanation:
Isotretinoin commonly causes hyperlipidemia, elevated liver enzymes, and mucosal dryness. It may cause temporary hair thinning but not scarring alopecia.
[A] Lipid elevation is common.

[B] Scarring alopecia is not associated with isotretinoin.
[C] Mucosal dryness is common.
[D] Liver enzyme elevation is common.
Home takeaway:
Isotretinoin = dryness + ↑lipids + ↑LFTs.

Q15
First lesion of acne:

A. Comedones
B. Papule
C. Pustule
Answer: A

Explanation:
The first lesion in acne is the comedone, resulting from follicular hyperkeratinization and obstruction of the sebaceous duct.
[A] Comedone is the earliest lesion.
[B] Papule is an inflammatory progression.
[C] Pustule is a later inflammatory stage.
Home takeaway:
Acne starts with comedone.

Q16
One false about acne rosacea:

A. Occur in teenagers
B. Associated with telangiectasia
C. Associated with rhinophyma
Answer: A

Explanation:
Rosacea typically affects middle-aged adults. It presents with facial flushing, erythema, telangiectasia, and may progress to rhinophyma in severe cases.
[A] Rosacea primarily affects adults, not teenagers.
[B] Telangiectasia is characteristic.
[C] Rhinophyma is a recognized complication.
Home takeaway:
Rosacea = adult facial flushing + telangiectasia.

Q17
Side effects of retinoic acid EXCEPT:

A. Renal failure
B. Fall of hair
Answer: A

Explanation:

Retinoids may cause hair thinning but do not cause renal failure.

[A] Renal failure is not a known side effect.

[B] Hair fall may occur.

Home takeaway:

Retinoids affect skin/liver/lipids—not kidneys.

Q18

Which is wrong about acne treatment:

Answer: Metronidazole is commonly used in systemic treatment of acne

Explanation:

Metronidazole is commonly used topically for rosacea, not as systemic therapy for acne vulgaris. Systemic acne treatment includes oral antibiotics, hormonal therapy, and isotretinoin.

Home takeaway:

Metronidazole = rosacea

Systemic acne therapy = antibiotics / isotretinoin.

Cutaneous Manifestations

Q1
Which of the following where ulceration can occur (E = erythema):

A. E. nodosum
B. E. multiforme
C. E. repens
D. E. nodosum leprosum
E. E. annulare
Answer: D
Explanation:
Erythema nodosum is a septal panniculitis presenting as tender erythematous subcutaneous nodules (classically on the shins) and it does not ulcerate. Erythema multiforme presents with target lesions (sometimes with vesiculation) but ulceration is not a typical feature. Erythema annulare centrifugum and other annular/gyrate erythemas are generally non-ulcerative. Erythema nodosum leprosum is a severe inflammatory lepra reaction that can become necrotic and may ulcerate.
[A] EN is panniculitis; lesions heal without ulceration.
[B] EM gives target lesions; ulceration is not typical.
[C] Gyrate erythemas are not ulcerative.
[D] ENL can become necrotic and ulcerate.
[E] Annular erythema is non-ulcerative.
Home takeaway:
Ulceration among "erythemas" → think erythema nodosum leprosum.

Q2
Which one mostly associated with underlying malignancy:

A. Erythema nodosum
B. Erythema multiforme
C. Biological erythema
D. Erythema gyratum repens
E. Chemical erythema
Answer: D
Explanation:
Erythema gyratum repens is a classic paraneoplastic dermatosis with a strong association with internal malignancy. It presents with rapidly migrating, concentric erythematous bands giving a "wood-grain" appearance. Erythema nodosum is usually linked to infections, sarcoidosis, pregnancy, IBD, or drugs. Erythema multiforme is most commonly triggered by infections, especially HSV.
[A] EN is usually infection/inflammation-related rather than malignancy.
[B] EM is usually triggered by HSV or other infections.
[C] Not a classic paraneoplastic entity here.
[D] Strongly associated with internal malignancy.
[E] Not a paraneoplastic pattern.
Home takeaway:
Wood-grain migrating rash → erythema gyratum repens → search for malignancy.

Q3
All about dermatitis herpetiformis except:

A. Chronic dis.
B. May occur at any age
C. Frequently associated with enteropathy
D. Autoimmune dis.
E. Prickle cell layer is the abnormal layer
Answer: E
Explanation:
Dermatitis herpetiformis is a chronic, very itchy autoimmune blistering disease, strongly associated with gluten-sensitive enteropathy (celiac disease). Clinically it presents with grouped papules/vesicles on extensor surfaces (elbows, knees, buttocks). Histology shows neutrophilic microabscesses in dermal papillae and a subepidermal blister, so the prickle cell layer is not the primary abnormal layer.

[A] It is chronic.
[B] It can occur at various ages.
[C] Strong association with enteropathy (celiac).
[D] Autoimmune mechanism.
[E] The key pathology is dermal papilla neutrophils + subepidermal split, not prickle layer abnormality.
Home takeaway:
DH = chronic itchy extensor vesicles + celiac association + neutrophils in dermal papillae.

Q4
Occurs in vitiligo:

A. Destruction of melanocytes
B. Abnormal melanin synthesis
C. Abnormal tyrosinase enzyme
D. All of the above
E. None of the above
Answer: A
Explanation:
Vitiligo is an acquired depigmenting disorder caused by destruction/loss of melanocytes, resulting in well-demarcated depigmented patches. It is not primarily due to defective melanin synthesis or tyrosinase enzyme abnormality (those are concepts more consistent with albinism).
[A] Vitiligo results from melanocyte destruction.
[B] Not a primary synthesis defect.
[C] Tyrosinase defect describes albinism.
[D] Only A is correct.
[E] Incorrect because A is true.
Home takeaway:
Vitiligo = acquired loss of melanocytes → depigmented patches.

Q5
Vitiligo may be associated with all except:

A. Thyrotoxicosis
B. Pernicious anemia
C. Addison's dis.
D. Gastric dis.
E. Reticulosis
Answer: E
Explanation:
Vitiligo commonly clusters with autoimmune diseases, especially autoimmune thyroid disease (hyper- or hypothyroidism), pernicious anemia, and Addison's disease. Reticulosis (lymphoproliferative disorders) is not a typical classic association in this context.
[A] Thyroid autoimmune disease association.
[B] Pernicious anemia association.
[C] Addison's disease association.
[D] Autoimmune gastric disease can coexist.
[E] Not a typical association.
Home takeaway:
Vitiligo → think autoimmune associations (thyroid, pernicious anemia, Addison's).

Q6
Commonest cutaneous lesions of Hodgkin's disease is:

A. Tumors
B. Secondary to pruritus
C. Exfoliative erythroderma
D. Ichthyosis
E. Ulcers
Answer: B
Explanation:
Hodgkin's disease is well known to cause generalized pruritus. The most common cutaneous findings are secondary changes due to scratching (excoriations), rather than primary tumors or ulcers.

[A] Primary skin tumors are not the common presentation.
[B] Pruritus → excoriations is most common.
[C] Erythroderma can occur but is not most common.
[D] Ichthyosis is not the commonest lesion.
[E] Ulcers are not typical.
Home takeaway:
Hodgkin's → pruritus → secondary scratch lesions.

Q7
Which one is a documented cause of erythema multiforme minor:

A. Drugs
B. Pregnancy
C. DM
D. Herpes simplex labialis
E. Internal malignancy
Answer: D
Explanation:
Erythema multiforme is commonly a hypersensitivity reaction, and the most frequent trigger is herpes simplex virus infection (often HSV labialis). EM minor typically shows target lesions with little or no mucosal involvement. Drugs can trigger EM but HSV is the classic common cause.
[A] Drugs can trigger EM but HSV is more common.
[B] Pregnancy is not a typical trigger.
[C] DM is not a cause.
[D] HSV labialis is a documented common cause.
[E] Not a typical trigger of EM.
Home takeaway:
Erythema multiforme (minor) → most commonly triggered by HSV.

Q8
One may cross normal placenta:

A. IgG
B. IgM
C. IgA
D. IgE
E. All Ig
Answer: A
Explanation:
Only IgG crosses the placenta and provides passive immunity to the fetus. IgM, IgA, and IgE do not cross a normal placenta.
[A] IgG crosses placenta.
[B] IgM does not cross.
[C] IgA does not cross.
[D] IgE does not cross.
[E] Only IgG crosses.
Home takeaway:
Placental antibody transfer → IgG only.

Q9
Cutaneous leishmaniasis is an infection of the:

A. Cutaneous fat
B. Stratum malpighii
C. R.E. cells
D. Mast cells
E. Langerhans cells
Answer: C
Explanation:
Cutaneous leishmaniasis is a protozoal infection transmitted by sandflies. The organism lives inside macrophages (reticuloendothelial cells) in the skin. Clinically it often starts as a painless papule at the bite site and may progress to an ulcer.

[A] Not primarily a fat infection.
[B] Not primarily an epidermal (malpighian) infection.
[C] Infects macrophages (R.E. cells).
[D] Mast cells are not the target cells.
[E] Langerhans cells are not the main infected cells.
Home takeaway:
Cutaneous leishmaniasis = intracellular parasite in macrophages.

Q10
Not associated with erythroderma:

A. Malignancy
B. Lichen planus
C. Psoriasis
D. Congenital ichthyosis
E. Drug induced
Answer: B
Explanation:
Erythroderma is generalized erythema and scaling involving most of the body surface. Common causes include psoriasis, drug reactions, malignancy (notably cutaneous lymphoma), and congenital ichthyoses. Lichen planus is not a common typical cause in this list.
[A] Malignancy can cause erythroderma.
[B] Lichen planus is not a typical cause here.
[C] Psoriasis is a common cause.
[D] Congenital ichthyosis can present with erythroderma.
[E] Drugs commonly cause erythroderma.
Home takeaway:
Erythroderma causes → psoriasis, drugs, malignancy, congenital ichthyosis.

Q11
Drug of choice for cutaneous leishmaniasis:

A. Tetracycline
B. Erythromycin
C. Antimonials
D. Sulfones
Answer: C
Explanation:
Standard treatment for cutaneous leishmaniasis is pentavalent antimonials (e.g., sodium stibogluconate). Antibiotics like tetracycline/erythromycin do not treat leishmania, and sulfones are used in leprosy.
[A] Not effective for leishmania.
[B] Not effective for leishmania.
[C] Antimonials are the standard therapy.
[D] Sulfones are for leprosy, not leishmaniasis.
Home takeaway:
Cutaneous leishmaniasis → treat with antimonials.

Q12
Leishmaniasis recidivans is due to:

A. L. donovani
B. L. tropica
C. L. braziliensis
D. Allergic reaction to L. body
Answer: B
Explanation:
Leishmaniasis recidivans is a chronic relapsing cutaneous form classically linked to Leishmania tropica, reflecting persistent localized infection. L. donovani causes visceral disease, and L. braziliensis is associated with mucocutaneous leishmaniasis.
[A] L. donovani → visceral leishmaniasis.
[B] L. tropica → recidivans.
[C] L. braziliensis → mucocutaneous disease.
[D] Recidivans is due to persistent infection, not simple allergy.

Home takeaway:
Leishmaniasis recidivans → L. tropica.

Q13
All condition may be precipitated by streptococcal throat infection except:

A. Erythema gyratum repens
B. Erythema marginatum
C. Erythema nodosum
D. Erythema multiforme
Answer: A
Explanation:
Streptococcal infection can precipitate erythema nodosum and erythema marginatum (classically in rheumatic fever). Erythema multiforme can follow infections. Erythema gyratum repens is paraneoplastic and linked to malignancy rather than strep throat.
[A] Erythema gyratum repens is malignancy-associated.
[B] Erythema marginatum is linked to rheumatic fever after strep infection.
[C] Erythema nodosum may follow streptococcal infection.
[D] EM can be triggered by infections.
Home takeaway:
Strep throat can trigger EN/erythema marginatum, not erythema gyratum repens.

Q14
Bullae of dermatitis herpetiformis are preceded histopathologically by:

A. Subepidermal microvacuoles
B. Neutrophilic & eosinophilic micro abscesses
C. Acantholysis of basal cell layer
D. Hydropic degeneration of basal layer
Answer: B
Explanation:
In dermatitis herpetiformis, early histology shows neutrophilic microabscesses in the dermal papillae (often with some eosinophils). These changes lead to a subepidermal split and blister formation. Acantholysis suggests pemphigus, and hydropic degeneration suggests interface dermatitis.
[A] Not the classic precursor change in DH.
[B] Dermal papilla neutrophilic microabscesses are characteristic.
[C] Acantholysis is typical of pemphigus.
[D] Hydropic degeneration is seen in interface dermatitis patterns.
Home takeaway:
DH blister formation starts with neutrophils in dermal papillae.

Q15
Treatment of dermatitis herpetiformis:

A. Diamino-diphenyl sulfone (DDS)
B. Systemic steroids
C. PUVA
D. Retinoic acid
Answer: A
Explanation:
Dapsone (DDS) produces rapid symptomatic improvement in dermatitis herpetiformis, especially pruritus and new lesion formation. Long-term control requires a gluten-free diet because of the association with celiac disease. Systemic steroids, PUVA, and retinoids are not first-line treatments.
[A] Dapsone is the drug of choice.
[B] Not first-line therapy for DH.
[C] Not indicated for DH.
[D] Not a standard treatment for DH.
Home takeaway:
DH → dapsone for rapid control + gluten-free diet for long-term management.

Q16
Celiac dis. may be associated with:

A. Dermatitis herpetiformis
B. Pemphigus vulgaris
C. Bullous pemphigoid
D. Erythema multiforme
Answer: A
Explanation:
Dermatitis herpetiformis is the classic skin disease associated with gluten-sensitive enteropathy. The other listed blistering disorders are not classically linked to celiac disease.
[A] Strong and classic association.
[B] Not a typical celiac association.
[C] Not a typical celiac association.
[D] Not a typical celiac association.
Home takeaway:
Celiac disease ↔ dermatitis herpetiformis.

Q17
Which of the following is the most common cause of erythema multiforme:

A. Herpes simplex virus
B. Mycoplasma
C. Pregnancy
Answer: A
Explanation:
The most common trigger for erythema multiforme is herpes simplex virus. Mycoplasma can also trigger EM (and is important especially with more mucosal involvement), but overall HSV is the commonest. Pregnancy is not a typical cause.
[A] Most common cause.
[B] Can cause EM but less common overall than HSV.
[C] Not a typical trigger.
Home takeaway:
Erythema multiforme → HSV is the most common trigger.

Q18
Which is false about vitiligo:

A. It affects males and females equally
B. Onset usually in 20s and 30s
C. It results in HYPOpigmented patches
D. Result from destruction of melanocytes
Answer: C
Explanation:
Vitiligo causes well-demarcated depigmented (milk-white) patches due to loss/destruction of melanocytes. It affects males and females equally and commonly begins in young adulthood.
[A] True: male = female.
[B] Common onset in 20–30s.
[C] False: lesions are depigmented, not just hypopigmented.
[D] True: due to melanocyte destruction.
Home takeaway:
Vitiligo = depigmented patches from melanocyte loss.

Q19
False about vitiligo:

A. Male : female (1:1)
B. Associated with thyroiditis
C. Peak age of incidence is 20–30s
D. It is a disease of abnormal melanisation
Answer: D

Explanation:
Vitiligo is an acquired disorder characterized by destruction/absence of melanocytes in affected skin. It is not primarily a disorder of abnormal melanisation (melanin production defect), and it is commonly associated with autoimmune thyroid disease.
[A] True.
[B] True: thyroiditis association.
[C] True: peak age 20–30s.
[D] False: the issue is melanocyte loss, not melanisation defect.
Home takeaway:
Vitiligo = melanocyte destruction (not a melanin-production enzyme problem).

Q20
No. of melanocytes in vitiligo is:

A. Decrease
B. Increase
C. Normal
D. All of the above
Answer: A
Explanation:
Vitiligo lesions show marked reduction or complete absence of melanocytes in involved skin, explaining the depigmentation.
[A] Correct: decreased/absent melanocytes.
[B] Incorrect.
[C] Incorrect in lesional skin.
[D] Incorrect because only A is true.
Home takeaway:
Vitiligo patches = decreased/absent melanocytes.

Q21
Vitiligo is significantly associated with:

A. Hypopituitary
B. Hyperthyroidism
C. Hypothyroidism
D. Hypoparathyroidism
Answer: C
Explanation:
Vitiligo is associated with autoimmune thyroid disease. In many exam frameworks, hypothyroidism (often autoimmune/Hashimoto's) is emphasized as a significant association, though hyperthyroidism can also occur in autoimmune clusters.
[A] Not a classic association.
[B] Can occur, but hypothyroidism is highlighted as significant.
[C] Significant association (autoimmune hypothyroidism).
[D] Not typical as a primary association.
Home takeaway:
Vitiligo → screen thyroid, especially hypothyroidism.

Q22
All true about erythroderma EXCEPT:

A. >80% of skin
B. Biopsy usually done
C. Hyperthermia and dehydration
Answer: A
Explanation:
Erythroderma is generalized erythema and scaling involving more than 90% of the body surface area. Because it has many causes, biopsy is commonly performed to help identify the underlying diagnosis. It can lead to systemic complications such as hyperthermia and dehydration (and other systemic instability).
[A] False: erythroderma is typically >90%, not >80%.
[B] True: biopsy is often performed.
[C] True: hyperthermia and dehydration can occur.
Home takeaway:
Erythroderma = >90% involvement + systemic risk → biopsy often needed.

Q23
Treatment of choice for chloasma:

A. Salicylic acid skin ointment
B. Phenol lotion
C. Eldoquin ointment
D. 5-PU 0.1%
Answer: C
Explanation:
Chloasma (melasma) is facial hyperpigmentation often related to sun exposure and hormonal factors. Treatment includes sun protection and depigmenting agents; hydroquinone preparations (e.g., Eldoquin) are commonly used as first-line topical therapy.
[A] Not first-line for melasma.
[B] Not standard therapy.
[C] Hydroquinone (Eldoquin) is a treatment of choice.
[D] Not standard for melasma.
Home takeaway:
Melasma → strict sun protection + hydroquinone.

Q24
Causes of post inflammatory hypopigmentation:

A. Psoriasis
B. Lichen planus
C. All of the above
Answer: C
Explanation:
Post-inflammatory hypopigmentation can follow inflammatory dermatoses due to temporary reduction in melanin production or melanocyte function. Both psoriasis and lichen planus can lead to pigmentary changes after inflammation resolves.
[A] Psoriasis can cause post-inflammatory pigment changes.
[B] Lichen planus can cause post-inflammatory pigment changes.
[C] Both are causes.
Home takeaway:
Inflammation (e.g., psoriasis/LP) can leave post-inflammatory hypopigmentation.

Q25
Not a cause of erythema nodosum:

A. Pregnancy
B. Herpes simplex
Answer: B
Explanation:
Erythema nodosum is commonly associated with pregnancy and with systemic triggers such as streptococcal infection, sarcoidosis, IBD, TB, and some drugs. Herpes simplex is not a typical cause of erythema nodosum (HSV is more linked to erythema multiforme).
[A] Pregnancy is a recognized trigger.
[B] HSV is not a typical cause of EN.
Home takeaway:
Erythema nodosum triggers → strep, sarcoidosis, pregnancy, IBD (not HSV).

Q26
Piebaldism:

Answer: Autosomal dominant
Explanation:
Piebaldism is a congenital disorder with stable depigmented patches and often a white forelock due to absence of melanocytes in affected areas. It follows autosomal dominant inheritance.
Home takeaway:
Piebaldism = congenital depigmentation + autosomal dominant.

Q27
Acanthosis nigricans .

Answer:
is velvet plaque in folds and creases
Explanation:
Acanthosis nigricans presents as hyperpigmented, velvety thickened plaques typically in flexural areas (neck, axillae, groin). It is commonly associated with insulin resistance and can also be a paraneoplastic sign in some cases.
Home takeaway:
Velvety hyperpigmented plaques in folds → acanthosis nigricans (often insulin resistance).

Q28
Incorrect statement:

Answer: Erythema nodosum is due to dermal inflammation
Explanation:
Erythema nodosum is a panniculitis—an inflammatory process involving subcutaneous fat (classically septal panniculitis), not a primary dermal inflammation.
Home takeaway:
Erythema nodosum = panniculitis (subcutaneous fat inflammation).

Q29
Choose the mismatch:

Answer:
Henoch Schoenlein purpura – IgG antibodies
Explanation:
Henoch–Schönlein purpura is an IgA-mediated small-vessel vasculitis (IgA immune complex deposition), not IgG.
Home takeaway:
Henoch–Schönlein purpura = IgA vasculitis.

Q30
Which statement is correct:

Answer:
Necrobiosis lipoidica is commonly associated with DM
Explanation:
Necrobiosis lipoidica presents as yellow-brown atrophic plaques with telangiectasia, typically on the shins, and is commonly associated with diabetes mellitus.
Home takeaway:
Necrobiosis lipoidica (shin plaques) → think diabetes mellitus.

Q31
A child from Jordan valley developed a painless ulcer on his face on the site of a mosquito bite, mostly?

Answer: Leishmaniasis
Explanation:
In endemic areas such as Jordan Valley, a painless papule at an insect bite site that enlarges and ulcerates is typical of cutaneous leishmaniasis (classically transmitted by sandfly).
Home takeaway:
Painless ulcer after insect bite in endemic area → cutaneous leishmaniasis.

Connective Tissue Diseases

Q1
Not in lichen planus nail:

A. Thinning
B. Dystrophy
C. Pterygium
D. Longitudinal ridging
E. Paronychia
Answer: E

Explanation:
Lichen planus (LP) is an immune-mediated, itchy eruption of shiny, purple, flat-topped papules/plaques and may show Wickham's striae and Koebner phenomenon. Nail involvement happens when inflammation targets the nail matrix/bed, leading to matrix-type changes like thinning, longitudinal ridging, and in severe cases pterygium (scarring that can permanently deform the nail). LP can also progress to nail dystrophy when involvement is significant or prolonged. Paronychia is inflammation/infection of the nail folds (often irritant/candida/bacterial) and is not a typical LP nail feature; LP mainly causes nail changes by inflammatory damage/scarring rather than nail-fold infection.
[A] LP nail matrix inflammation can cause nail plate thinning.
[B] Chronic nail-unit inflammation/scarring can produce dystrophy.
[C] Pterygium is a classic severe LP nail change (scarring).
[D] Longitudinal ridging is a typical LP nail change.
[E] Paronychia points to nail-fold inflammation/infection, not typical LP.
Home takeaway:
LP nail = matrix/bed inflammation → thinning + longitudinal ridging ± pterygium/dystrophy; paronychia is usually a different process.

Q2
All of the following are common changes of the nail in lichen planus except:

A. Pitting
B. Pterygium
C. Thinning
D. Longitudinal ridging
E. Onycholysis
Answer: E

Explanation:
LP commonly produces longitudinal ridging and thinning from nail matrix involvement and may cause pterygium due to scarring, which can lead to permanent nail loss/deformity in advanced disease. Onycholysis (separation of nail plate from the nail bed) is more classically emphasized in other nail disorders (e.g., psoriasis, trauma, onychomycosis) and is not the "common/expected" LP nail change compared with ridging/thinning/pterygium.
[A] Pitting is more typical of psoriasis; not a classic LP hallmark.
[B] Pterygium is a hallmark severe LP nail change.
[C] Thinning fits LP matrix involvement.
[D] Longitudinal ridging fits LP.
[E] Onycholysis is not the classic common LP nail pattern.
Home takeaway:
LP nails: think longitudinal ridging + thinning + pterygium; onycholysis is more typical in other nail diseases.

Q3
All about lichen planus are true except:

A. Self limiting
B. 50% of cases clear within 18 months
C. Chronicity due to presence of mucous lesion & hypertrophic lesions
D. Presence of Wickham's striae
E. Presence of hypogranulosis
Answer: E

Explanation:
LP is immune-mediated with pruritic purple planar papules/plaques; Wickham's striae and Koebner phenomenon are classic. Many cases are self-limited and clear in about 1–2 years, but hypertrophic LP can persist for years and mucosal/genital disease tends to be more persistent, explaining chronicity in certain subtypes. Histologically, LP is classically associated with hypergranulosis (thickened granular layer), not hypogranulosis.
[A] Many cases resolve within 1–2 years (self-limited).
[B] Clearing within ~18 months fits typical course.
[C] Hypertrophic/mucosal disease explains chronicity.
[D] Wickham's striae are classic.
[E] Wrong: LP shows hypergranulosis, not hypogranulosis.
Home takeaway:
LP is often self-limited but can persist (especially hypertrophic/mucosal), and histology classically shows hypergranulosis.

Q4
All of the following diseases are associated with macules as their primary lesions, except:

A. Lichen planus
B. Lentigo
C. Vitiligo
D. Erythema
E. Post inflammatory hyperpigmentation
Answer: A

Explanation:
A macule is a flat color change (not palpable). Lentigo, vitiligo, erythema, and post-inflammatory hyperpigmentation are primarily flat pigment/erythema changes. LP, however, presents with pruritic, purple, polygonal, flat-topped papules/plaques (palpable), so its primary lesion is papular, not macular.
[A] LP primary lesions are papules/plaques, not macules.
[B] Lentigo is a classic macule.
[C] Vitiligo lesions are flat depigmented patches.
[D] Erythema is a flat redness change.
[E] PIH is a flat pigment change after inflammation.
Home takeaway:
Macules are flat color changes; LP is papular/plaquey, so it's the exception.

Q5
Destruction of basal cell layer occurs in:

A. Discoid lupus erythematosus
B. Morphea
C. Dermatomyositis
D. Psoriasis
E. Pityriasis rosea
Answer: A

Explanation:
Discoid lupus erythematosus (DLE) is a cutaneous lupus form that produces well-defined erythematous scaly lesions that can become atrophic and scar, often with follicular plugging and scarring alopecia on the scalp. DLE is an interface-type process at the dermo-epidermal junction, so basal keratinocyte injury/destruction is a key concept. Morphea is localized sclerosis/fibrosis, dermatomyositis is a skin–muscle–vessel disease, psoriasis is hyperproliferative epidermis, and pityriasis rosea is an inflammatory eruption—none are classically defined by basal layer destruction like DLE.
[A] DLE involves interface/basal layer injury with scarring features.
[B] Morphea = sclerosis/fibrosis, not basal layer destruction.
[C] Dermatomyositis has characteristic rashes + muscle weakness.
[D] Psoriasis = epidermal hyperproliferation/parakeratosis.
[E] Pityriasis rosea is not an interface scarring disease.
Home takeaway:
Basal/interface damage with scarring points to cutaneous lupus (especially DLE).

Q6
Commonest cutaneous eruption in SLE:

A. Erythema of light exposed areas
B. Butterfly rash
C. Discoid lesion
D. Erythema of palms
E. Diffuse multiform erythema
Answer: B

Explanation:
Systemic lupus erythematosus (SLE) is multisystemic with frequent skin involvement. A classic and commonly emphasized eruption is the malar ("butterfly") rash—erythema over the cheeks and nasal bridge, often linked with photosensitivity. Discoid lesions are more typical of discoid lupus (can occur in SLE but are not the most common eruption framing here).
[A] Photosensitivity is common, but the classic named common eruption is malar rash.
[B] Malar/butterfly rash is the classic common eruption.
[C] Discoid lesions are a specific cutaneous lupus form.
[D] Not the classic "commonest eruption" descriptor.
[E] EM-like eruptions are not typical common SLE rash.
Home takeaway:
SLE common skin clue = malar (butterfly) rash with photosensitivity.

Q7
All of the following are seen in dermatomyositis except:

A. Gottron's sign
B. Proximal muscle weakness
C. Ragged cuticle
D. Heliotrope sign
E. Plaques
Answer: E

Explanation:
Dermatomyositis is a rare disease affecting skin, muscles, and blood vessels. Skin findings include a purple heliotrope hue on upper eyelids/face, Gottron's papules over dorsal finger joints, photosensitive distribution (V-sign/shawl sign), and nailfold changes such as ragged cuticles and dilated capillaries. Muscle involvement causes proximal limb weakness. "Plaques" is nonspecific and not one of the characteristic listed hallmark signs compared with heliotrope/Gottron/cuticle/nailfold changes.
[A] Gottron's sign/papules are classic.
[B] Proximal weakness is typical.
[C] Ragged cuticles occur.
[D] Heliotrope rash is classic.
[E] "Plaques" is not a hallmark feature compared with the listed signs.
Home takeaway:
Dermatomyositis = heliotrope + Gottron + ragged cuticles/nailfold capillaries + proximal weakness.

Q8
Tissue involved in morphea may include:

A. Epidermis
B. Subcutaneous tissue
C. Muscles
D. Bones
E. All of the above
Answer: E

Explanation:
Morphea is localized scleroderma with localized sclerosis and minimal inflammation. While some cases are limited to dermis, deeper variants—especially linear morphea—can extend into subcutaneous tissue and even deeper structures, leading to atrophy, deformity, or functional limitation. Therefore involvement can include skin and deeper tissues depending on type and depth.

[A] Skin involvement is part of the disease.
[B] Subcutis can be involved in deeper forms.
[C] Muscle may be involved in deep/linear disease.
[D] Bone involvement can occur in severe deep/linear forms.
[E] Best overall answer.
Home takeaway:
Morphea is localized sclerosis that can extend into subcutis and deeper tissues (especially linear forms).

Q9
One of the following is associated with Hepatitis C infection:

A. Psoriasis
B. Rosacea
C. Lichen planus
D. Eczema
E. Vitiligo
Answer: C

Explanation:
Lichen planus is a pruritic eruption of purple polygonal flat-topped papules/plaques with possible mucosal and nail involvement. It has a recognized association with hepatitis C infection (often tested as a classic association).
[A] Psoriasis is not the classic Hep C association here.
[B] Rosacea is not classically linked to Hep C.
[C] Lichen planus is the classic association.
[D] Eczema has many triggers but not the classic Hep C association.
[E] Vitiligo is more linked to autoimmune thyroid disease.
Home takeaway:
If asked "Hepatitis C association" in derm → think lichen planus.

Q10
Which of the following is associated with muscular atrophy?

A. Linear morphea
B. Pustular morphea
C. Diffuse morphea
D. Disseminated morphea
Answer: A

Explanation:
Linear morphea can extend deeper than the superficial skin, involving subcutis and sometimes deeper tissues. This deeper extension can lead to atrophy (including muscular atrophy), asymmetry, and functional impairment—so muscle atrophy is a classic clue pointing to linear morphea.
[A] Linear morphea is the subtype most linked to atrophy/deeper involvement.
[B] Not a classic morphea subtype in this context.
[C] Can be extensive, but muscular atrophy is classically linked to linear disease.
[D] Disseminated = multiple plaques; not the classic muscular atrophy clue.
Home takeaway:
Muscle atrophy + morphea → think linear morphea.

Q11
Which is false about morphea:

A. It is localized form of scleroderma
B. It improves with time
C. Not caused by UV light exposure
D. Presents with as hairy well-defined patches
Answer: D

Explanation:
Morphea is localized scleroderma with sclerosis/fibrosis leading to firm, well-defined patches/plaques that often become atrophic. Hair growth in affected plaques is typically reduced due to sclerosis/atrophy of skin appendages, so describing it as "hairy" is wrong. It often improves with time and is not typically described as UV-induced like photosensitive disorders.

[A] True: localized form of scleroderma.
[B] Often improves with time.
[C] Not classically UV-caused like photosensitive lupus.
[D] False: lesions are not "hairy"; sclerosis tends to reduce hair.
Home takeaway:
Morphea = well-defined firm sclerotic plaques, often with reduced hair in the lesion.

Q12
Childhood dermatomyositis is frequently associated with:

A. CA
B. DM
C. Mental retardation
D. Calcinosis
Answer: D

Explanation:
Dermatomyositis occurs in children and adults. Adult dermatomyositis raises concern for underlying malignancy. In contrast, juvenile dermatomyositis is classically associated with calcinosis cutis (calcium deposition in skin/soft tissues), which is a well-known pediatric complication.
[A] Malignancy association is emphasized in adults.
[B] Not a classic association here.
[C] Not typical.
[D] Juvenile dermatomyositis commonly associates with calcinosis.
Home takeaway:
Juvenile dermatomyositis → calcinosis; adult dermatomyositis → screen for malignancy.

Q13
Wrong about lichen planus:

A. If it involves the mucosa, gingivae is the most common location
B. Itchy
Answer: A

Explanation:
LP is characteristically itchy. Mucosal involvement is common, and the typical most emphasized oral site is buccal mucosa (often with lacy white striae). Saying gingivae is the most common site is not the usual classic description.
[A] Oral LP is commonly buccal mucosa rather than "most commonly gingiva."
[B] Pruritus is a hallmark feature.
Home takeaway:
LP is very itchy; oral involvement commonly affects buccal mucosa.

Q14
All true about dermatomyositis except:

A. Frequently associated with underlying malignancy in adults
B. Affects children & adults
C. More common in males
D. Heliotrope rash is pathognomonic
Answer: C

Explanation:
Dermatomyositis affects both children and adults and in adults it may be associated with malignancy. Heliotrope rash is a classic hallmark sign. The incorrect statement is that it is more common in males; it generally shows a female predominance.
[A] True: adult association with malignancy.
[B] True: affects children and adults.
[C] False: not more common in males.
[D] Heliotrope rash is a classic hallmark sign.
Home takeaway:
Adult dermatomyositis → malignancy association; classic rash = heliotrope + Gottron; not male-predominant.

Q15
Itching is characteristic feature in:

A. Pityriasis rosea
B. Psoriasis
C. Lichen planus
D. Pityriasis versicolor
Answer: C

Explanation:
LP is classically pruritic (one of the "6 Ps": pruritic, purple, polygonal, planar papules/plaques). Other listed diseases may itch variably, but intense itch is a hallmark clue for LP.
[A] PR can itch but not as classically emphasized as LP.
[B] Psoriasis can itch but classic clues are silvery scaly extensor plaques.
[C] LP is characteristically itchy.
[D] PV itch is usually mild/variable.
Home takeaway:
Marked pruritus with purple polygonal papules → lichen planus.

Q16
The histological sign pathognomonic for lichen planus is:

A. Hypergranulosis
B. Hyperkeratosis
C. Papillomatosis
D. Parakeratosis
Answer: A

Explanation:
LP histology is classically associated with hypergranulosis (thickened granular layer). Hyperkeratosis is nonspecific, parakeratosis is more typical of psoriasis, and papillomatosis is not the key hallmark for LP.
[A] Hypergranulosis is the classic LP clue.
[B] Nonspecific.
[C] Not the classic hallmark.
[D] More consistent with psoriasis pattern.
Home takeaway:
LP histology favorite = hypergranulosis.

Q17
Which of the following causes patchy scarring alopecia:

A. SLE
B. Discoid lupus
Answer: B

Explanation:
Discoid lupus commonly affects the scalp with scaly plaques that develop follicular plugging and scarring, leading to patchy scarring alopecia. SLE can cause diffuse hair loss, but scarring alopecia is more characteristic of discoid (chronic cutaneous) lupus.
[A] SLE can cause hair loss, usually non-scarring.
[B] Discoid lupus causes scarring alopecia (patchy).
Home takeaway:
Patchy scarring alopecia → think discoid lupus.

Q18
False about connective tissue disease:

A. Subacute lupus causes cutaneous scarring
B. Discoid lupus will become systemic lupus in <5%
Answer: A

Explanation:
Subacute cutaneous lupus typically presents with annular/polycyclic or papulosquamous photosensitive lesions that generally heal without scarring (though dyspigmentation can occur). Discoid lupus has a low progression rate to systemic lupus, often stated as <5%.
[A] False: SCLE typically does not scar.
[B] True: progression of DLE to SLE is uncommon (<5%).
Home takeaway:
SCLE usually doesn't scar; DLE progresses to SLE in a small minority.

Q19
Which of the following result in hypertrophy and distal proliferation of nail circle on nail fold:
Answer: Lichen planus

Explanation:
LP can involve the nail unit and cause chronic inflammatory/scarring changes. Nail matrix involvement leads to longitudinal ridging and thinning, and chronic disease can progress to significant dystrophy and scarring changes (including pterygium and distorted nail-fold/nail unit anatomy), which matches the described proliferative/hypertrophic changes around the nail fold.
Home takeaway:
Nail LP is scarring inflammatory nail disease → progressive dystrophy with ridging/thinning ± severe deformity.

Q20
Not in lichen planus?
Answer: Nail thickening

Explanation:
Classic LP nail involvement produces thinning and longitudinal ridging due to nail matrix inflammation, and severe cases may develop pterygium and dystrophy. Nail thickening is more typical of conditions like onychomycosis or psoriatic subungual hyperkeratosis rather than classic LP patterns.
Home takeaway:
LP nails: thinning + ridging + pterygium; thickening points more to other nail disorders.

Q21
Wrong statement about DLE (discoid lupus erythematosus)?
Answer: No scarring

Explanation:
DLE is defined by chronic cutaneous lesions that can heal with atrophy and scarring, especially on the scalp where it can cause scarring alopecia with follicular plugging. Therefore "no scarring" is wrong.
Home takeaway:
DLE is a scarring cutaneous lupus (especially scalp → scarring alopecia).

Q22
Not in lupus:
Answer: Neonatal lupus develops into SLE in 20% of cases

Explanation:
Neonatal lupus is due to transplacental passage of maternal autoantibodies (commonly anti-Ro/anti-La). The infant's cutaneous lesions typically resolve as maternal antibodies decline. It is not accurate to say neonatal lupus "develops into SLE in 20%" as a standard rule.
Home takeaway:
Neonatal lupus is antibody-mediated and skin lesions usually resolve as antibodies fade.

Q23
A girl with photosensitivity and ANA titer of 1:32, next step?
Answer: Repeat ANA in 3 months if sunscreen wasn't effective for the rash

Explanation:
Low-titer ANA can be nonspecific and must be interpreted with the clinical picture. With photosensitive rash, a practical next step is optimize sun protection and reassess. If rash persists despite good photoprotection, repeating ANA and reassessing for other clinical/systemic features is reasonable.
Home takeaway:
Low ANA titer + photosensitivity → optimize sunscreen/avoid UV, reassess, repeat testing if persistent.

Q24
One affect nail cuticle:
Answer: Dermatomyositis

Explanation:
Dermatomyositis has characteristic nailfold/cuticle findings including ragged cuticles and dilated nailfold capillaries, alongside heliotrope rash, Gottron papules, and proximal muscle weakness.
Home takeaway:
Ragged cuticles/dilated nailfold capillaries → dermatomyositis.

Q25
Wrong statement:
Answer: In dermatomyositis there is a risk of calcinosis in adults

Explanation:
Calcinosis is classically linked to juvenile dermatomyositis. Adult dermatomyositis is more notable for association with underlying malignancy rather than calcinosis being emphasized as a typical adult risk statement.
Home takeaway:
Adult dermatomyositis → malignancy association; juvenile dermatomyositis → calcinosis.

Q26
Correct sentence:
Answer: In neonatal lupus, the lesions subside when the antibodies drop

Explanation:
Neonatal lupus results from maternal autoantibodies crossing the placenta. Because the infant is not producing these antibodies, the skin lesions typically fade and resolve as maternal antibody levels decline over time.
Home takeaway:
Neonatal lupus rash resolves as maternal antibodies clear.

Q27
A postmenopausal woman with white itchy and painful vulva:
Answer: Lichen sclerosus

Explanation:
Lichen sclerosus commonly affects the genital/perineal region, especially in older women, presenting with pruritus and soreness/pain. Lesions are classically ivory-white, well-demarcated, and atrophic, with possible fissuring. It requires treatment and follow-up.
Home takeaway:
White itchy painful vulva in older woman → lichen sclerosus.

Q28
Wrong:
Answer: Neonatal lupus transform to SLE in 20% of cases

Explanation:
Neonatal lupus is a passive antibody-mediated condition (maternal antibodies). Infant skin lesions typically resolve as those antibodies decline; it is not correct to claim a standard 20% progression to SLE.
Home takeaway:
Neonatal lupus is not "20% becomes SLE"; lesions usually resolve as antibodies fade.

Psoriasis

Q1
Commonest manifestation of psoriasis in nails is:

A. Onycholysis
B. Pitting
C. Subungual hyperkeratosis
D. Discoloration
E. None of these
Answer: B

Explanation:
Nail psoriasis can affect the nail matrix and/or nail bed. The most common nail manifestation is pitting, which reflects nail matrix involvement (abnormal keratinization in the proximal nail matrix leads to tiny depressed defects in the nail plate). Other nail findings can include onycholysis (separation of nail plate from bed), subungual hyperkeratosis (thick scale under nail), "oil drop/salmon patch" discoloration, and splinter hemorrhages.
[A] Onycholysis happens in nail bed psoriasis, but it is not the commonest finding.
[B] Pitting is the commonest nail manifestation (matrix disease).
[C] Subungual hyperkeratosis is common, but less common than pitting.
[D] Discoloration (oil drop) occurs but is not the commonest.
[E] A common manifestation exists (pitting).
Home takeaway:
Nail psoriasis: pitting = most common (matrix involvement).

Q2
All are of psoriasis histopathological changes EXCEPT:

A. Hyperkeratosis
B. Parakeratosis
C. Munro's abscesses
D. Epidermal atrophy
E. Suprapapillary plates thinning
Answer: D

Explanation:
Typical psoriasis histology includes hyperkeratosis, parakeratosis, and thinning of the suprapapillary plates (with elongated rete ridges). Neutrophils can accumulate in the stratum corneum forming Munro microabscesses. "Epidermal atrophy" is not a feature of psoriasis; psoriasis is characterized by epidermal hyperplasia (acanthosis), not atrophy.
[A] Hyperkeratosis is typical (thick stratum corneum).
[B] Parakeratosis is typical (retained nuclei).
[C] Munro microabscesses are classic for psoriasis.
[D] Epidermal atrophy is opposite to the usual psoriatic pattern.
[E] Suprapapillary plate thinning is classic.
Home takeaway:
Psoriasis histology = hyperkeratosis + parakeratosis + Munro + suprapapillary thinning, not epidermal atrophy.

Q3
Why does nail pitting occur in patients with psoriasis?

A. Leakage of blood of dilated capillaries
B. Abnormal cell adhesion
C. Intermittent inflammation of the nail bed
D. Loss of parakeratotic cells from the nail surface
E. Excessive proliferation of the nail bed
Answer: D

Explanation:
Pitting is due to nail matrix psoriasis. Abnormal keratinization produces parakeratotic foci in the nail plate; when these reach the surface they break off, leaving pits. Nail bed disease more commonly causes onycholysis and subungual hyperkeratosis.
[A] More related to bleeding phenomena, not pitting mechanism.
[B] Not the main explanation for psoriatic pitting.
[C] Nail bed inflammation → onycholysis/subungual hyperkeratosis.
[D] Parakeratotic cells shed → pits (matrix origin).
[E] Nail bed proliferation explains subungual hyperkeratosis, not pitting.
Home takeaway:
Psoriatic pitting = matrix disease → parakeratotic foci shed → pits.

Q4
All will exacerbate psoriasis EXCEPT:

A. Hypocalcemia
B. Anti-malarial
C. Infections
D. Hormonal changes
E. Macrolides
Answer: E

Explanation:
Psoriasis flares can be triggered by infections (especially streptococcal infection for guttate psoriasis), and certain drugs like antimalarials can provoke/worsen psoriasis. Hormonal changes may influence activity in some patients. Hypocalcemia is classically linked with pustular psoriasis susceptibility in exam frameworks. Macrolides are not a typical exacerbating factor.
[A] Hypocalcemia is a recognized association in pustular psoriasis contexts.
[B] Antimalarials can aggravate psoriasis.
[C] Infections are common triggers (especially strep for guttate).
[D] Hormonal shifts may modulate disease activity.
[E] Macrolides are not a classic trigger.
Home takeaway:
Typical triggers: infection + antimalarials; macrolides aren't a classic trigger.

Q5
A 30-year-old patient presents with itchy vesicles on extensor areas. Which disease most likely fits?

A. Shingles
B. Psoriasis
C. Dermatitis herpetiformis
D. Chicken pox
E. Bullous pemphigoid
Answer: C

Explanation:
Dermatitis herpetiformis presents with intensely pruritic grouped vesicles/papules on extensor surfaces (elbows, knees, buttocks) and is strongly associated with gluten-sensitive enteropathy (celiac disease). Shingles is dermatomal and painful. Chickenpox is generalized with lesions in different stages. Psoriasis is classically scaly plaques rather than vesicles. Bullous pemphigoid causes tense bullae, usually in older adults.
[A] Dermatomal painful vesicles.
[B] Scaly plaques, not classic vesicles.
[C] Classic: very itchy grouped vesicles on extensor sites.
[D] Generalized vesicles in crops.
[E] Tense bullae in elderly.
Home takeaway:
Itchy grouped vesicles on extensor surfaces → dermatitis herpetiformis (celiac association).

Q6
All of the following are considered systemic treatment options for psoriasis EXCEPT:

A. TNF-α blockers
B. Acitretin
C. Cyclosporin
D. Methotrexate
E. Vitamin D analogue
Answer: E

Explanation:
Systemic options include methotrexate, cyclosporin, acitretin, and biologics like TNF-α blockers. Vitamin D analogues (e.g., calcipotriol) are mainly topical treatments, not systemic options in this question.
[A] TNF-α blockers are systemic biologics.
[B] Acitretin is systemic.
[C] Cyclosporin is systemic.
[D] Methotrexate is systemic.
[E] Vitamin D analogue is primarily topical.
Home takeaway:
Systemic psoriasis: MTX/cyclosporin/acitretin/biologics; vitamin D analogue = topical.

Q7
Not used in treatment of psoriasis:

A. Antimalarial
B. Cyclosporine A
C. Methotrexate
D. Systemic retinoid
E. Fumaric acid esters
Answer: A

Explanation:
Systemic psoriasis treatments include methotrexate, cyclosporin, and systemic retinoids (classically acitretin). Antimalarials are not used to treat psoriasis and can aggravate it. Fumaric acid esters are used as systemic therapy in some regions.
[A] Antimalarials can worsen psoriasis; not a treatment.
[B] Cyclosporine is used.
[C] Methotrexate is used.
[D] Systemic retinoid (acitretin) is used.
[E] Fumaric acid esters can be used in psoriasis.
Home takeaway:
Don't use antimalarials in psoriasis (they can worsen it).

Q8
One of the following is associated with Koebner phenomenon:

A. Psoriasis
B. Rosacea
C. Lichen planus
D. Eczema
E. Urticaria
Answer: A

Explanation:
Koebner phenomenon is development of lesions at sites of trauma (scratching, scars, pressure). Psoriasis classically koebnerizes, producing plaques along trauma lines.
[A] Psoriasis classically shows Koebner phenomenon.
[B] Not classic.
[C] Can koebnerize, but psoriasis is the best single answer here.
[D] Not a classic Koebner association in this list.
[E] Not classic.
Home takeaway:
Koebner = new lesions at trauma sites (classically psoriasis).

Q9
One is true regarding histology of psoriasis:

A. Acanthosis and parakeratosis
B. Spongiosis
C. Hypergranulosis
D. Acantholysis
Answer: A

Explanation:
Psoriasis shows epidermal hyperplasia (acanthosis) with parakeratosis. Spongiosis is typical of eczema. Hypergranulosis is typical of lichen planus. Acantholysis is typical of pemphigus vulgaris.
[A] Classic psoriatic features: acanthosis + parakeratosis.
[B] Spongiosis = eczema.
[C] Hypergranulosis = lichen planus.
[D] Acantholysis = pemphigus.
Home takeaway:
Psoriasis histology: acanthosis + parakeratosis.

Q10
Not used in the systemic treatment of psoriasis:

A. Methotrexate
B. Isotretinoin
C. Fumaric acid
D. Cyclosporine
Answer: B

Explanation:
Methotrexate and cyclosporin are established systemic treatments. Systemic retinoid used for psoriasis is typically acitretin, not isotretinoin (which is mainly for acne). Fumaric acid derivatives can be systemic options in some regions.
[A] Methotrexate is used systemically.
[B] Isotretinoin is not standard systemic psoriasis therapy (acitretin is).
[C] Fumaric acid derivatives can be used.
[D] Cyclosporine is used systemically.
Home takeaway:
Systemic retinoid for psoriasis = acitretin, not isotretinoin.

Q11
Psoriatic erythroderma complications EXCEPT:

A. Temperature dysregulation
B. Dehydration
C. Sepsis
D. None of the above
Answer: D

Explanation:
Erythrodermic psoriasis is severe and can be life-threatening due to extensive skin involvement and barrier failure. Complications include temperature dysregulation, fluid/protein loss with dehydration, and increased risk of secondary infection that may progress to sepsis. Therefore there is no exception among A–C.
[A] Temperature dysregulation can occur.
[B] Dehydration can occur.
[C] Infection/sepsis risk can occur.
[D] Since A–C are all possible, "none of the above" is correct.
Home takeaway:
Erythrodermic psoriasis → temp problems + dehydration + infection/sepsis risk.

Q12
Parakeratosis is a specific feature of:

A. Lichen planus
B. Psoriasis
C. Acute eczema
D. Ichthyosis vulgaris
Answer: B

Explanation:
Parakeratosis (retained nuclei in stratum corneum) is a classic histologic feature of psoriasis. Lichen planus shows hypergranulosis. Acute eczema shows spongiosis.
[A] Lichen planus: hypergranulosis.
[B] Psoriasis: parakeratosis.
[C] Acute eczema: spongiosis.
[D] Ichthyosis vulgaris is not defined by parakeratosis here.
Home takeaway:
Parakeratosis → psoriasis.

Q13
Psoriasis type caused by streptococcal infection:

A. Flexural
B. Nodular
C. Guttate
Answer: C

Explanation:
Guttate psoriasis presents with widespread small "drop-like" plaques on trunk and limbs, often sudden onset in young patients, and commonly follows streptococcal sore throat.
[A] Flexural psoriasis is defined by site (folds), not strep trigger.
[B] Not the classic post-strep subtype.
[C] Guttate psoriasis is classic post-strep.
Home takeaway:
Post-strep sore throat → guttate psoriasis.

Q14
One of the following is best famous for causing Auspitz sign:

A. Psoriasis
B. Rosacea
C. Lichen planus
Answer: A

Explanation:
Auspitz sign is pinpoint bleeding after removing scale from a psoriatic plaque, due to superficial dilated capillaries beneath thinned suprapapillary plates. This is classic for psoriasis.
[A] Psoriasis.
[B] Rosacea is flushing/erythema/papules/telangiectasia.
[C] Lichen planus shows Wickham striae; not Auspitz.
Home takeaway:
Auspitz sign → psoriasis.

Q15
Wrong about psoriasis:
Answer: Doesn't affect children

Explanation:
Psoriasis can occur at any age, including childhood. Pediatric psoriasis is well recognized.
Home takeaway:
Psoriasis can affect children.

Q16
All are exacerbations of psoriasis except:
Answer: Hypercalcemia

Explanation:
In exam frameworks, hypocalcemia (not hypercalcemia) is the metabolic association linked to certain severe pustular presentations, so hypercalcemia is the exception here.
Home takeaway:
Hypocalcemia may link with pustular psoriasis; hypercalcemia is not a classic trigger.

Q17
Wrong about psoriasis:
Answer: Oral steroids are usually used to manage flare ups

Explanation:
Systemic corticosteroids are not recommended as routine therapy for psoriasis because they can cause rebound flares and unstable disease, and withdrawal can precipitate severe forms like erythroderma or pustular psoriasis.
Home takeaway:
Avoid systemic steroids in psoriasis (rebound/flare risk).

Q18
Wrong about psoriasis?
Answer: Usually inherited as autosomal recessive

Explanation:
Psoriasis is a complex multifactorial disease with polygenic predisposition and environmental triggers; it does not follow a simple autosomal recessive inheritance pattern.
Home takeaway:
Psoriasis inheritance is polygenic/multifactorial, not autosomal recessive.

Q19
Characteristic of nail in psoriasis except:
Answer: Clubbing

Explanation:
Psoriatic nail changes include pitting, onycholysis, subungual hyperkeratosis, and oil drop/salmon patch discoloration. Clubbing is not a typical feature of psoriasis and is more linked to systemic cardiopulmonary disease.
Home takeaway:
Psoriasis nails: pitting/onycholysis/subungual hyperkeratosis/oil-drop, not clubbing.

Urticaria

Q1
Tx of choice for acute urticaria:

A. Antihistamine
B. Systemic steroids
C. Local steroids
D. Adrenaline
E. Kallikrein
Answer: A

Explanation:
Urticaria is a type I hypersensitivity-type reaction presenting as transient, very itchy, well-demarcated erythematous wheals caused by edema in the superficial dermis. The key mechanism is mast cell degranulation with release of histamine (and other mediators) leading to increased capillary permeability and fluid leakage → wheal formation. Acute urticaria is usually self-limiting and in many patients no clear cause is found, but common triggers include infections, medications, food, and physical stimuli. The mainstay treatment for urticaria (acute and chronic) is oral H1 antihistamines—preferably 2nd generation—taken regularly (not PRN), and the dose may be increased if needed. Systemic steroids are reserved for very severe eruptions (especially if vasculitis is suspected) and should not be prolonged. Adrenaline is reserved for severe life-threatening reactions with respiratory distress/angioedema risk.
[A] First-line and mainstay treatment for urticaria.
[B] Not first line; used only in very severe cases and short course.
[C] Topical steroids do not treat the transient dermal edema mechanism.
[D] For life-threatening urticaria/angioedema with respiratory distress, not routine acute urticaria.
[E] Not part of standard urticaria therapy.
Home takeaway:
Acute urticaria → treat first with regular 2nd-gen H1 antihistamine; steroids only short course if very severe; adrenaline only for severe respiratory distress/anaphylaxis risk.

Q2
Cold urticaria:

A. Sometimes familial
B. Usually acquired
C. May be transferable in serum
D. May result in unconsciousness
E. All of the above
Answer: E

Explanation:
Physical urticaria can be triggered by cold. Diagnosis can be supported by a cold provocation test (placing an ice cube on the skin for a period then observing for wheal/flare). Cold exposure can also trigger more generalized reactions in susceptible patients, especially with large-area exposure (e.g., swimming) and may lead to severe systemic symptoms. Cold urticaria is most often acquired, but familial forms exist. Passive transfer by serum has been described in some cases, supporting a circulating factor in at least a subset. Severe reactions can include hypotension/syncope and loss of consciousness after extensive cold exposure.
[A] Familial (rare) forms exist.
[B] Most cases are acquired.
[C] Passive serum transfer has been described in some patients.
[D] Extensive cold exposure (like swimming) may trigger systemic reaction with syncope/unconsciousness.
[E] Since A–D can all be true, "all of the above" is best.
Home takeaway:
Cold urticaria is usually acquired but can be familial; diagnosis by cold challenge; extensive cold exposure (swimming/ice-cold drinks) can cause dangerous systemic reactions.

Q3
The main cells involved in urticaria are:

A. Neutrophils
B. Mast cells
C. Eosinophils
D. Histamine
E. Lymphocytes
Answer: B

Explanation:
Urticaria results from mast cell degranulation triggered by allergens, complement, direct stimulation, or idiopathic mechanisms. Degranulation releases histamine, bradykinin, and other inflammatory mediators. Histamine increases vascular permeability causing transient dermal edema and wheal formation, and stimulates sensory nerves causing intense pruritus. Neutrophils/eosinophils/lymphocytes may be present in some settings, but the key initiating cell in typical urticaria is the mast cell. "Histamine" is a mediator, not a cell.
[A] Not the main initiating cell in typical urticaria.
[B] Core cell driving the reaction via degranulation.
[C] May appear in some allergic settings but not the main driver.
[D] Important mediator, but it's not a cell.
[E] Not the main initiating cell for wheal formation.
Home takeaway:
Urticaria = mast cell degranulation → histamine release → wheal + itch.

Q4
Most reliable Dx test of cholinergic urticaria:

A. Intradermal methacholine
B. Intradermal nicotinic acid
C. Intradermal scinine
D. Biopsy
E. Exercise & heat
Answer: E

Explanation:
Cholinergic urticaria typically affects patients aged 10–30 years and occurs after warming triggers such as exercise or a warm shower/bath. Lesions are characteristically pinhead-sized wheals with a surrounding red flare. Because the disease is induced by a rise in core temperature and sweating-related triggers, the most reliable way to confirm the diagnosis is provocation with exercise and/or heat under controlled conditions. Biopsy is not needed for routine cholinergic urticaria (it is mainly used when urticarial vasculitis is suspected—painful lesions lasting >24 hours or bruising).
[A] Not the standard most reliable test in usual practice.
[B] Not the standard diagnostic confirmation method.
[C] Not a recognized reliable test here.
[D] Biopsy is for suspected vasculitis features, not typical cholinergic urticaria.
[E] Best confirmation: reproduce wheals with exercise/heat.
Home takeaway:
Cholinergic urticaria = pinhead wheals after heat/exercise → confirm by provocation with exercise/heat.

Q5
Best antihistamine for day-time use:

A. Ethanolamines
B. Piperidines
C. Phenothiazines
D. Ethylenediamines
E. Alkylamines
Answer: B

Explanation:
Oral antihistamines are the mainstay of treatment and prevention of urticaria and angioedema, with preference for 2nd generation H1 blockers taken regularly. Daytime use favors non-sedating (or minimally sedating) antihistamines. The piperidine group contains several second-generation, less-sedating agents commonly used for daytime symptoms (e.g., loratadine/desloratadine/fexofenadine classically grouped with low sedation). Older first-generation classes are more likely to cause sedation and impair performance.
[A] First-generation types tend to be sedating (not ideal daytime).
[B] Best fit for daytime due to less sedation.
[C] Often sedating/anticholinergic side effects.
[D] Generally more sedating older agents.
[E] Many are older, sedating agents.
Home takeaway:
Daytime urticaria control → use non-sedating 2nd-gen H1 antihistamine (piperidines are classic for daytime).

Q6
False about urticaria:

A. Leaves hypopigmented scar
B. 90% of chronic cases the cause is unknown
C. Wheal is the primary lesion
D. Very itchy
Answer: A

Explanation:
Urticaria presents as transient pruritic swellings (wheals) due to superficial dermal edema. Individual lesions typically last minutes to hours (usually <24 hours) and resolve completely with no marks on the skin. Itching is prominent. Chronic urticaria is defined by symptoms lasting more than 6 weeks, and in many patients no cause is identified (idiopathic), especially as attacks become more persistent. Scarring (including hypopigmented scars) is not a feature of ordinary urticaria; if lesions are painful, last >24 hours, or heal with bruising, consider urticarial vasculitis and biopsy.
[A] False—ordinary urticaria resolves with no residual marks/scars.
[B] Chronic cases are often idiopathic (cause frequently not found).
[C] Wheal is the primary lesion.
[D] Urticaria is very itchy.
Home takeaway:
Urticaria wheals are transient (<24h) and leave no scars; persistence >24h with pain/bruising suggests vasculitis.

Q7
All these drugs used for ttt of urticaria except:

A. Systemic steroid
B. Dimetane tab
C. Allerfrin tab
D. Antihistamine ointment
Answer: D

Explanation:
Main treatment of urticaria is oral antihistamines (preferably 2nd generation H1 blockers, regularly). In severe cases, combinations may be used (H1 plus H2 blockers, and leukotriene receptor antagonists). Oral corticosteroids may be used only in very severe eruptions and particularly when urticarial vasculitis is suspected, and not for long duration. Topical treatments are generally not effective because the pathology is transient dermal edema driven by systemic mediator release; topical antihistamine ointments are not standard for urticaria control.
[A] Can be used in very severe cases (short course).
[B] Oral antihistamine option fits urticaria therapy.
[C] Oral antihistamine option fits urticaria therapy.
[D] Topical antihistamine ointment is not standard management.
Home takeaway:
Urticaria is treated systemically (oral antihistamines ± add-ons); topical antihistamine ointments aren't standard.

Q8
Degree of level of contact sensitivity to an allergen is influenced by:

A. Amount of allergen to which the subjects exposed
B. Frequency of exposure to the allergen
C. The route of exposure
D. All of the above
Answer: D

Explanation:
For contact sensitivity, the likelihood and intensity of sensitization depends on exposure variables: higher dose (amount), repeated exposures (frequency), and the way the allergen contacts the body (route), all influence the immune response and clinical expression. In practice, stronger allergens or repeated/occlusive exposures increase the chance of developing clinically significant sensitivity.
[A] Higher allergen load increases sensitization likelihood.
[B] Repeated exposure increases sensitization risk.
[C] Route/skin barrier context affects sensitization.
[D] All factors contribute.
Home takeaway:
Contact sensitivity depends on dose + frequency + route of exposure.

Q9
Urticaria, which is wrong:

A. Oral steroids are first line treatment
B. Sedating and non-sedating antihistamine are used
Answer: A

Explanation:
Oral antihistamines are the mainstay treatment/prevention for urticaria and angioedema, with preference for second-generation H1 blockers taken regularly (dose can be increased if needed). Sedating antihistamines may be used (often at night) while non-sedating options are preferred for daytime. Oral corticosteroids are not first line; they may be indicated only in very severe eruptions (especially when urticarial vasculitis is involved) and should not be used long-term.
[A] Wrong—steroids are not first line; reserve short course for very severe cases.
[B] True—both sedating and non-sedating antihistamines can be used depending on timing/needs.
Home takeaway:
First line urticaria = regular H1 antihistamine; steroids only short course for severe cases.

Q10
Edematous erythematous lesion that blanches with pressure:
Answer: Wheal

Explanation:
A wheal is the primary lesion of urticaria: a well-demarcated, edematous, erythematous swelling in the superficial dermis caused by increased capillary permeability and transient fluid leakage. It is typically very itchy, blanches on pressure, and resolves within hours (usually <24 hours) without leaving marks. If wheals last >24 hours, are painful, or leave bruising, consider urticarial vasculitis.
Home takeaway:
Wheal = blanching, itchy, transient dermal edema lesion of urticaria (<24h, no marks).

Q11
Urticaria, which is wrong:
Answer: Oral steroids are first line treatment

Explanation:
Urticaria is primarily managed with oral antihistamines (second-generation H1 blockers taken regularly; dosing can be escalated). In severe cases, add-on therapy can include H2 blockers and leukotriene receptor antagonists. Oral corticosteroids may be used only for very severe eruptions (particularly those linked to urticarial vasculitis) and should not be used as routine first-line therapy or for prolonged duration because of relapse risk and adverse effects.
Home takeaway:
Urticaria first-line = regular 2nd-gen H1 antihistamine; steroids are not first line and should be short course only when severe.

Pruritus + Scabies & Lice

Q1
The pruritus of biliary obstruction can probably be most directly related to:

A. Whole (crude) bile
B. Bile salts
C. Bile acids
D. Conjugated bilirubin
E. Unconjugated bilirubin
Answer: B

Explanation:
Pruritus can occur with normal-looking skin and may be an early manifestation of systemic disease. Metabolic causes include hepatic failure and biliary obstruction. In biliary obstruction, retained bile salts are the classic most direct trigger for generalized itching. Clinically, the key idea is: generalized pruritus with otherwise normal skin should prompt thinking of systemic causes (including cholestasis) and evaluation accordingly (CBC/ESR, liver and renal function, thyroid function, glucose, iron studies, etc.).
[A] Whole bile is not the specific mediator linked to itch.
[B] Bile salts are the most directly related factor in cholestatic pruritus.
[C] Bile acids are related to bile, but the classic direct link tested is bile salts.
[D] Bilirubin causes jaundice; it is not the main direct pruritus mediator in this context.
[E] Unconjugated bilirubin is not the key mediator of cholestatic itching.
Home takeaway:
Generalized pruritus with normal skin → think systemic causes; cholestasis itch is classically due to bile salts.

Q2
After initial exposure to & infestation with Sarcoptes scabiei hominis the pruritus follows:

A. Immediately
B. In 1–2 days
C. In about one week
D. In about 2–4 weeks
E. In about 3 months
Answer: D

Explanation:
Scabies is a very common worldwide infestation that causes intense itching, classically worse at night. Transmission requires close personal contact (prolonged skin-to-skin contact), and the itching is largely an immune reaction to mite proteins, eggs, and feces in the skin. After the first infestation, symptoms typically start after a delay while the immune system becomes sensitized, commonly around 2–4 weeks.
[A] Not immediate in first infestation because sensitization takes time.
[B] Too early for first-time sensitization.
[C] Still earlier than the typical delayed onset in first infestation.
[D] Fits the typical 2–4 week delay after initial infestation.
[E] Much too late.
Home takeaway:
First scabies infestation: itch appears late (about 2–4 weeks) due to delayed immune sensitization; itch is worse at night.

Q3
In human scabies the best yield of positive scrapings is from:

A. Papules
B. Vesicles
C. Burrows
D. Excoriation
E. Crusts
Answer: C

Explanation:
Diagnosis of scabies is supported by history (multiple contacts itching, nocturnal pruritus) and examination. The most diagnostic primary lesion is the burrow: a linear, palpable ridge with a tiny dark speck that represents the mite. Because the mite and its products are located in the burrow, scraping from burrows gives the highest yield for finding mites/eggs/feces under microscopy compared with nonspecific papules, excoriations, or secondary changes.
[A] Papules are often a hypersensitivity reaction and may not contain the mite.
[B] Vesicles can occur (especially in children) but are not the best target for scraping.
[C] Burrows contain the mite → best yield.
[D] Excoriations are secondary scratches, not where the mite resides.
[E] Crusts suggest crusted scabies, but "best yield" in typical scabies is still burrows.
Home takeaway:
For microscopy, scrape the burrow (linear ridge with black speck) for the highest diagnostic yield.

Q4
Wrong about scabies of infants:

A. Treated with permethrin 5%
B. May occur in back and face
C. No family history of itching
D. Involves palms and soles
E. Caused by Sarcoptes scabiei hominis
Answer: C

Explanation:
Scabies in infants/young children can look different from adults. In adults, face and back are typically spared, but in babies the head/neck can be involved and lesions can appear on the face and back. Infants may have papules/nodules in axillae and on soles, and lesions might blister; classic burrows are often hard to see. Scabies spreads within households, so a history of itching in family members/close contacts is common and strongly supportive—so "no family history" is the wrong statement. First-line therapy is permethrin 5% cream; in babies it is applied to all skin (including head/neck), and close contacts should be treated simultaneously to prevent reinfestation.
[A] Permethrin 5% is first-line.
[B] In infants, face/back may be involved.
[C] Household/close-contact itching is common, so "no family history" is wrong.
[D] Palms/soles involvement is common in infants.
[E] Cause is Sarcoptes scabiei.
Home takeaway:
Infant scabies can involve face/back and palms/soles; family members often itch; treat infant + close contacts together with permethrin 5%.

Q5
A 50-year-old man is suspected of having scabies, which statement regarding scabies is false:

A. The genitalia is a commonly affected site
B. All members in the same household should be treated at the same time
C. It can spread by simple handshake
D. Children are often affected by scabies
E. Itching can persist for weeks even after successful treatment
Answer: C

Explanation:
Typical scabies distribution includes finger web spaces and genitalia, with intense nocturnal itching. Transmission usually needs close, prolonged skin-to-skin contact (classically at least ~15 minutes), rather than brief contact. Management depends on correct application and treating all close contacts at the same time, plus washing bedding/clothes. After effective treatment, itching can persist for weeks (often several weeks) as the immune system continues to react to remaining mite products; repeated unnecessary treatments can cause irritant dermatitis (post-scabies dermatitis).
[A] Genitals are common sites.
[B] Close contacts/household should be treated simultaneously.
[C] Simple brief handshake is usually insufficient; prolonged close contact is required.
[D] Children are commonly affected.
[E] Post-treatment itch can persist for weeks even when mites are eradicated.

Home takeaway:
Scabies spreads by prolonged close contact (not brief handshake); treat all close contacts together; itching may persist weeks after cure.

Q6
Scabies of infants, all true except:

A. Symptoms at night
B. Sparing face and back
C. Permethrin 5% cream is the first treatment of choice
D. In children it manifests as acral pustules
Answer: B

Explanation:
Scabies itch is characteristically worse at night. Infant/child scabies can involve atypical sites compared with adults: infants may have lesions on face/back and commonly on soles, with blistering possible; classic burrows may be rare. First-line treatment is permethrin 5% cream; in infants it should be applied to all skin (including head/neck). In children, lesions can be more acral and may appear pustular on hands/feet. Therefore, "sparing face and back" is the incorrect statement for infants.
[A] Nocturnal worsening is typical.
[B] In infants, face/back may be involved, so this is the exception.
[C] Permethrin 5% is first-line.
[D] Acral pustular-type lesions can be seen in children.
Home takeaway:
Infant scabies does NOT reliably spare face/back; itch is worse at night; treat with permethrin 5% applied to all skin in babies.

Q7
One of the following is used to treat scabies:

A. Benzoyl peroxide
B. Permethrin 5%
C. Topical hydrocortisone
D. Tacrolimus
Answer: B

Explanation:
Definitive scabies therapy is scabicidal treatment. Permethrin 5% cream is first-line: apply overnight, repeat after 7 days (day 1 and day 8). Adults apply from the neck down; babies/infants apply to all skin (including head/neck). Key reasons for failure include incorrect use, missing key areas (web spaces, genitals), and not treating close contacts simultaneously. Symptomatic itch relief can be supported with antipruritic measures, but these do not eradicate mites.
[A] Benzoyl peroxide is for acne, not scabies.
[B] Permethrin 5% is first-line scabicide.
[C] Hydrocortisone may reduce inflammation/itch but does not kill mites.
[D] Tacrolimus is anti-inflammatory (eczema), not scabicidal.
Home takeaway:
Scabies treatment of choice = permethrin 5% overnight, repeat after 7 days + treat all close contacts together.

Q8
Wrong about scabies:

A. Contagious
B. More at night
C. Affect the back
Answer: C

Explanation:
Scabies is contagious and causes intense pruritus that is characteristically worse at night. In adults, distribution is fairly characteristic and typically spares the face and back; common sites include finger webs and genitalia. Therefore, "affect the back" is the wrong statement in the usual adult pattern. (In infants, involvement can be broader, including face/back.)
[A] Scabies is contagious via close contact.
[B] Itching is typically worse at night.
[C] Adult scabies typically spares the back → this is wrong.
Home takeaway:
Adult scabies: contagious + nocturnal itch + classic distribution that usually spares face/back.

Q9
Which of these drugs is used in scabies:

A. Benzyl peroxide
B. Benzyl benzoate
C. Tetracyclines
Answer: B

Explanation:
Scabies can be treated with permethrin 5% (first-line). If alternatives are needed, other options include malathion lotion, ivermectin in selected cases, sulfur preparations, and benzyl benzoate emulsion (a scabicide option when standard agents are unavailable). The key is correct application technique and repeating treatment after 7 days to cover the life cycle, plus treating close contacts.
[A] Benzoyl peroxide is for acne, not scabies.
[B] Benzyl benzoate is an alternative scabicide.
[C] Tetracyclines are antibiotics and do not eradicate scabies mites.
Home takeaway:
Scabies scabicides include permethrin 5% (first-line) and alternatives like benzyl benzoate when needed.

Q10
Wrong about scabies:

A. Benzoyl peroxide used as systemic treatment
B. Caused by Sarcoptes scabiei
Answer: A

Explanation:
Scabies is caused by Sarcoptes scabiei; the female mite burrows in the epidermis and lays eggs, triggering intense itching (especially at night). Treatment is scabicidal topical therapy (permethrin 5% first-line, repeat after 7 days) with simultaneous treatment of close contacts. Benzoyl peroxide is not a systemic (or scabies) treatment; it is a topical anti-acne medication.
[A] Wrong: benzoyl peroxide is not used systemically for scabies.
[B] True: scabies is caused by Sarcoptes scabiei.
Home takeaway:
Scabies = Sarcoptes scabiei; treat with scabicides (permethrin 5%) and treat contacts—benzoyl peroxide has no role.

Q11
Scabies treatment is:
Answer: Topical permethrin.

Explanation:
First-line scabies treatment is permethrin 5% cream. It should be applied correctly (adults from neck down; infants to all skin including head/neck), left overnight, and repeated after 7 days (day 1 and day 8). Treat all close personal contacts at the same time to prevent reinfestation, wash bedding/towels/underwear, and focus application on web spaces and genital areas. Patients should be warned that itching may take weeks to settle even after successful eradication; repeated unnecessary retreatment can cause irritant dermatitis.
Home takeaway:
Permethrin 5% overnight + repeat after 7 days + treat all contacts together; post-scabies itch can persist for weeks.

Bacterial Infection

Q1
Erysipelas, all true except:

A. Well-defined
B. Can be with fever
C. Penicillin is the drug of choice
D. Caused by staph.
E. Mostly on L.L
Answer: D

Explanation:
Erysipelas is a deeper bacterial skin infection caused classically by Streptococcus (often group A). It spreads over ~48 hours as a red, shiny, raised, tender, spreading plaque with a sharply well-demarcated edge because the organisms invade the dermis and lymphatics. Systemic symptoms such as fever and malaise can occur, and the face and lower legs are common sites. Treatment (especially if severe) is penicillin-based therapy (e.g., benzylpenicillin) with oral options for 1–2 weeks; recurrent attacks may need prophylaxis.
[A] Erysipelas is characteristically well-demarcated and raised.
[B] Fever/malaise may occur.
[C] Penicillin is appropriate for streptococcal erysipelas.
[D] The classic cause is Streptococcus, not Staphylococcus.
[E] Lower legs are frequently affected.
Home takeaway:
Erysipelas = streptococcal, shiny raised well-demarcated plaque (often face/legs) + possible fever → treat with penicillin.

Q2
Main local source of staph. aureus contaminating the skin:

A. Nose
B. Scalp
C. Axillae
D. Perineum
E. Mouth
Answer: A

Explanation:
Recurrent staphylococcal skin infections are strongly linked to carriage of Staphylococcus aureus, especially in the anterior nares. Nasal swabs are used to identify carriers (including MRSA) because ongoing shedding from the nose can contaminate skin and contribute to recurrent boils/folliculitis/impetigo.
[A] The nose is the main carriage site leading to skin contamination.
[B] Scalp may carry organisms, but the classic main reservoir is the nose.
[C] Axilla can harbor bacteria, but nasal carriage is the major source for S. aureus shedding.
[D] Perineum can be a carriage site, but the key classic source is the nose.
[E] Mouth is not the main classic reservoir for skin contamination in this context.
Home takeaway:
S. aureus carriage is classically nasal → nasal swab helps in recurrent infections.

Q3
Not part of the normal flora?

A. Group A Strep
B. Diphtheroid
C. Staph aureus
D. Corynebacterium diphtheriae
E. Coagulase negative Staph
Answer: C

Explanation:
Normal skin flora acts as a biological shield and commonly includes coagulase-negative Staphylococcus, Corynebacterium/diphtheroids, and alpha-hemolytic streptococci on the epidermis, plus Propionibacterium in the pilosebaceous unit. Staphylococcus aureus is better viewed as a potential pathogen and a carrier organism (often in the nose) rather than a core component of normal resident flora described here.
[A] Streptococci can be part of commensal flora (classically alpha-hemolytic).
[B] Diphtheroids are part of normal flora.
[C] Staph aureus is not listed as typical normal resident flora here and is treated as a potential pathogen/carrier.
[D] Corynebacterial species are part of normal flora group (diphtheroids).
[E] Coagulase-negative staph are classic normal flora.
Home takeaway:
Normal skin flora = coagulase-negative staph + corynebacteria/diphtheroids (+ others); S. aureus is mainly considered a pathogen/carrier.

Q4
Eye involvement may occur in all EXCEPT:

A. Intermediate leprosy
B. Lepromatous leprosy
C. Rosacea
D. Sarcoid
E. Behçet's syndrome
Answer: A

Explanation:
Eye involvement is well recognized in several systemic/dermatologic diseases: lepromatous leprosy has significant ocular complications (lids/cornea/uvea), rosacea can involve the eyes (blepharitis, conjunctivitis, keratitis), sarcoidosis can cause ocular inflammation (e.g., uveitis), and Behçet's syndrome commonly causes uveitis/retinal vasculitis. Compared with lepromatous disease, ocular involvement is classically emphasized more strongly in lepromatous forms than in intermediate/less severe forms, so "intermediate leprosy" is the exception here.
[A] Least expected option among the list compared with well-known ocular involvement in lepromatous leprosy/rosacea/sarcoid/Behçet.
[B] Lepromatous leprosy is strongly linked to ocular complications.
[C] Rosacea can have ocular rosacea.
[D] Sarcoidosis can cause uveitis and other eye disease.
[E] Behçet's frequently involves the eye (uveitis/retinal vasculitis).
Home takeaway:
Ocular involvement is common in lepromatous leprosy, rosacea, sarcoid, and Behçet; lepromatous type is the leprosy form most classically associated with significant eye disease.

Q5
Which of the following is the most superficial infection of the skin?

A. Ecthyma
B. Impetigo
C. Cellulitis
D. Furuncles
Answer: B

Explanation:
Superficial bacterial infections include impetigo, folliculitis, boils/abscesses, and erythrasma. Impetigo is the classic most superficial: it rapidly forms clusters of pustules/vesicles that break down into the typical golden crusts and is highly contagious. Ecthyma is considered a deeper form of impetigo where organisms invade into the dermis causing superficial ulcers. Cellulitis is deeper with poorly defined margins, and furuncles are deep follicular infections/abscesses.
[A] Ecthyma is deeper than impetigo (ulceration into dermis).
[B] Impetigo is the classic superficial infection.
[C] Cellulitis is deeper (subcutaneous involvement).
[D] Furuncles are deeper follicular abscesses.
Home takeaway:
Impetigo = most superficial (golden crusts); ecthyma and cellulitis are deeper.

Q6
Wrong about ecthyma:

A. Superficial infection
B. Causes generalized dryness
C. Strep is the most common cause
D. Increased in immunocompromised
Answer: A

Explanation:
Ecthyma is often described as a deeper form of impetigo. Group A beta-hemolytic Streptococcus (S. pyogenes) invades into the dermis, producing lesions that begin as small pustules with an adherent crust and underlying ulceration. It occurs commonly on the lower legs and heals slowly with scarring. It is more likely/severe in settings such as immunocompromise and poor hygiene/host factors. Because it invades deeper than impetigo and forms ulcers, calling it "superficial infection" is wrong.
[A] Ecthyma is deeper (ulcerative) → not purely superficial.
[B] "Generalized dryness" is not the defining picture; the key lesion is crusted ulceration.
[C] Streptococcus is the classic main cause.
[D] More common/severe with impaired host defenses.
Home takeaway:
Ecthyma = deeper ulcerative impetigo (usually strep) on legs → crust + ulcer + scarring.

Q7
Commonest rash of secondary syphilis:

A. Vesicular
B. Maculo-papular
C. Papular
D. Pustular bullous
Answer: B

Explanation:
Secondary syphilis produces a widespread eruption of red-brown scaly patches and macules involving trunk and limbs, with a characteristic tendency to involve palms and soles. This overall pattern is best described clinically as a generalized maculopapular (macules + papules/patches) eruption with scale.
[A] Vesicles are not typical of secondary syphilis.
[B] Best overall descriptor for the widespread eruption.
[C] Papules can occur, but the eruption is typically mixed (macules/patches + scale).
[D] Bullous/pustular patterns are not the common presentation described.
Home takeaway:
Secondary syphilis = generalized red-brown scaly macules/patches (often palms/soles) → maculopapular pattern.

Q8
Rash of secondary syphilis is:

A. Scaly
B. Itchy
C. Vesicular
D. None of the above
Answer: A

Explanation:
The cutaneous eruption of secondary syphilis is described as widespread red-brown scaly patches and macules affecting trunk and limbs (often palms and soles). Scale is a key descriptor here. It is not classically a vesicular eruption.
[A] Scale is a typical feature in the described eruption.
[B] Pruritus is not the defining feature emphasized here.
[C] Vesicles are not typical.
[D] Since scale is typical, "none" is incorrect.
Home takeaway:
Secondary syphilis rash is classically widespread and scaly (often includes palms/soles).

Q9
Patient presented with well defined erythematous plaque on the calf of her lower limb, U/S was normal, what is the most likely diagnosis:

A. Erysipelas
B. Cellulitis
C. DVT
Answer: A

Explanation:
Erysipelas presents as a well-demarcated, shiny, raised, spreading erythematous plaque because the infection involves the dermis and lymphatics. Cellulitis tends to have a poorly defined margin and develops more slowly with deeper tissue involvement and often prominent regional lymphadenopathy. A normal ultrasound helps exclude DVT, and the "well-defined plaque" description strongly matches erysipelas rather than cellulitis.
[A] Well-demarcated raised erythematous plaque on lower leg fits erysipelas.
[B] Cellulitis is usually poorly demarcated.
[C] DVT is excluded by normal ultrasound in this context.
Home takeaway:
Well-demarcated shiny raised leg plaque → think erysipelas (cellulitis is more ill-defined).

Q10
Impetigo may occur in:

A. Elderly
B. Infants
C. Adult
D. Young adults
Answer: B

Explanation:
Impetigo is a superficial bacterial infection caused by S. aureus or Streptococcus pyogenes. It is highly contagious and develops rapidly into pustules/vesicles that break down into classic golden crusts. It is particularly common in children (including infants), and outbreaks can occur among close contacts, especially with poor hygiene, hot humid climates, and minor trauma (like insect bites).
[A] Can occur, but not the typical "most likely" group tested here.
[B] Common in children/infants → best answer.
[C] Adults can get it, but it's classically a pediatric infection.
[D] Less typical than infants/children.
Home takeaway:
Impetigo = common, contagious superficial infection in children → pustules/vesicles → golden crusts.

Q11
TTT of lepromatous leprosy should be continued for (yrs):

A. 2
B. 5
C. 10
D. Life long
Answer: A

Explanation:
Lepromatous leprosy is a multibacillary form of leprosy requiring prolonged multidrug therapy (MDT). Historically, WHO regimens for multibacillary disease were given for about 24 months (2 years), which matches the expected exam answer here. Modern WHO guidance commonly uses fixed-duration MDT for multibacillary disease for 12 months, but many curricula/exams still test the classic 2-year duration for lepromatous/multibacillary treatment.
[A] Classic taught duration for multibacillary/lepromatous MDT in many exam settings.
[B] Longer than standard fixed-duration MDT regimens.
[C] Not standard.
[D] Not required with effective MDT.
Home takeaway:
Lepromatous (multibacillary) leprosy needs prolonged MDT—classically taught as ~2 years (though modern regimens may be shorter depending on guideline).

Q12
Which one causes impetigo contagiosum:
Answer: Staph & strep

Explanation:
Impetigo is usually caused by Staphylococcus aureus or Streptococcus pyogenes. Clinically it is highly contagious, spreads among close contacts, and rapidly forms clusters of pustules/vesicles that rupture and form the classic golden crusts. Bullous impetigo is more associated with staphylococcal strains producing exfoliative toxins, while streptococcal infection may be suggested by associated regional lymphadenopathy. Treatment includes topical antiseptics/topical antibiotics for localized disease and oral antibiotics (e.g., anti-staphylococcal agents) for more extensive disease.
Home takeaway:
Impetigo contagiosum = S. aureus and/or Strep pyogenes → golden crusts; bullous form often staph-toxin related.

Q13
Coral red in Wood's light:
Answer: Erythrasma

Explanation:
Erythrasma is a superficial infection classically affecting flexural areas (axilla, groin) with superficial scaling and mild inflammation, often with a reddish-brown discoloration. It is caused by Corynebacterium minutissimum, and under Wood's ultraviolet light the affected skin fluoresces pink/coral-red—this is a key diagnostic clue that helps differentiate it from tinea and other intertriginous rashes. First-line treatment is often oral erythromycin; topical options (if preferred) can include agents like fusidic acid or certain antifungal creams that have activity in this setting.
Home takeaway:
Coral-red (pink) fluorescence under Wood's light in folds = erythrasma (Corynebacterium minutissimum).

Q14
Not a superficial skin infection:
Answer: Ecthyma

Explanation:
Superficial bacterial infections include impetigo, folliculitis, boils/abscesses, pseudofolliculitis, and erythrasma. Ecthyma is categorized among deeper bacterial infections because it is essentially a deeper ulcerative form of impetigo where organisms invade into the dermis, causing adherent crust with underlying ulceration and slow healing with scarring.
Home takeaway:
Ecthyma is NOT superficial—it's a deeper ulcerative form of impetigo that scars.

Viral Infection

Q1
Tx of choice for genital warts:

A. Salicylic acid 10%
B. Topical 5-FU
C. Surgery
D. Cryotherapy
E. Podophyllotoxin
Answer: E

Explanation:
Genital warts (condylomata acuminata) are caused mainly by HPV types 6 and 11. They present as soft, papillomatous/flesh-colored growths on anogenital skin and may be multiple. Treatment goals are lesion clearance and symptom relief (not immediate "virus eradication"), and recurrence is common because HPV can persist in epithelium. Podophyllotoxin (podofilox) is a patient-applied antimitotic agent that causes wart necrosis and is a common first-line option for external genital warts. Other effective options include cryotherapy (clinic-based), surgical removal/electrocautery (for large or refractory lesions), and immune-modifying therapy (e.g., imiquimod). Important counseling: avoid use of podophyllin/podophyllotoxin in pregnancy; treat partner only if lesions; advise STI screening and HPV vaccination when appropriate.
[A] Keratolytics like salicylic acid are mainly for cutaneous (non-genital) warts.
[B] 5-FU can be used in selected cases, but it is not the usual first choice here.
[C] Surgery is effective but usually reserved for bulky/refractory lesions.
[D] Cryotherapy is effective but not the classic "treatment of choice" in this option set.
[E] Best answer: commonly used first-line patient-applied therapy for external genital warts.
Home takeaway:
Genital warts (HPV 6/11): common first-line = podophyllotoxin; alternatives include cryotherapy, excision/electrocautery, and immune therapy; recurrence is common.

Q2
All of the following skin eruptions are caused by viral infections except:

A. Scarlet fever eruption
B. Pityriasis rosea
C. Roseola infantum eruption
D. Slapped cheek syndrome of erythema infectiosum
E. Rubella eruption
Answer: A

Explanation:
Many classic childhood exanthems are viral: rubella causes a generalized maculopapular eruption; parvovirus B19 causes erythema infectiosum ("slapped cheek"); HHV-6 causes roseola infantum. Pityriasis rosea is thought to be triggered by HHV-6/7. Scarlet fever, however, is a bacterial toxin-mediated eruption due to group A streptococcus (erythrogenic toxins) and is therefore not viral.
[A] Scarlet fever is streptococcal (bacterial), not viral.
[B] Linked to HHV-6/7 triggering.
[C] Roseola is viral (HHV-6).
[D] Slapped cheek is viral (parvovirus B19).
[E] Rubella is viral.
Home takeaway:
Most exanthems listed are viral—scarlet fever is the bacterial exception.

Q3
The cause of pityriasis rosea:

A. HSV 6
B. HSV 7
C. HSV 1,2
D. A + B
E. A + B + C
Answer: D

Explanation:
Pityriasis rosea is believed to be triggered by HHV-6 and HHV-7 (often following mild URTI symptoms). It typically begins with a herald patch—an oval erythematous plaque with collarette scale—followed days later by multiple smaller scaly lesions on the trunk and proximal limbs, often along cleavage lines ("Christmas tree" pattern on the back). The condition is self-limited (often resolves within ~4–6 weeks). Management is symptomatic: reassurance, emollients, mild topical steroid and/or oral antihistamine if itchy.
[A] HHV-6 association is recognized.
[B] HHV-7 association is recognized.
[C] HSV-1/2 are not the typical cause association for pityriasis rosea.
[D] Best answer: HHV-6 + HHV-7.
[E] Adding HSV-1/2 makes it incorrect.
Home takeaway:
Pityriasis rosea is linked to HHV-6/7 → herald patch then "Christmas tree" scaly trunk eruption; self-limited.

Q4
Herald patch is a specific lesion for:

A. Pityriasis alba
B. P. versicolor
C. P. rosea
D. P. capitis
E. P. rubra pilaris
Answer: C

Explanation:
The herald patch is the initial lesion of pityriasis rosea: a single oval/round erythematous plaque with a fine collarette of scale at the edge. After several days, a generalized eruption of smaller scaly patches appears mainly on the trunk and proximal limbs. Pityriasis versicolor is a Malassezia infection with pigmentary change and fine scale; pityriasis alba causes hypopigmented fine scaly patches (usually in children); PRP is an inflammatory disorder with orange-red plaques and follicular keratosis.
[A] Pityriasis alba has no herald patch.
[B] Versicolor has fine scale with pigment change, not herald patch.
[C] Correct: herald patch is classic for pityriasis rosea.
[D] Not relevant.
[E] PRP is different clinically and histologically.
Home takeaway:
Herald patch + collarette scale = pityriasis rosea starter lesion.

Q5
Warts most commonly affect body flexures:

A. Plane warts
B. Common warts
C. Digitate warts
D. Filiform warts
E. Seborrheic warts
Answer: D

Explanation:
HPV causes several wart morphologies. Filiform warts are finger-like (spiky) projections commonly on the face/neck and areas of friction and folds, so they are often described as affecting flexures. Common warts are rough hyperkeratotic papules typically on hands; plane warts are flat-topped smooth papules often on the face; "seborrheic warts" refers to seborrheic keratoses (not HPV).
[A] Plane warts are usually face/dorsum hands, not mainly flexures.
[B] Common warts mainly hands/fingers.
[C] Digitate is a descriptive term; filiform is the classic flexural/facial spiky wart.
[D] Best answer: filiform warts commonly occur around folds/face/neck.
[E] Seborrheic keratoses are not HPV warts.
Home takeaway:
Filiform warts = spiky "finger-like" lesions, often around face/neck and folds.

Q6
All of the following are true about plane warts except:

A. Occurs most commonly in the face
B. Spiky top
C. Different colors
D. Children and adolescents
E. Koebner's phenomenon
Answer: B

Explanation:
Plane (flat) warts are smooth, flat-topped, slightly elevated papules, commonly on the face (and sometimes dorsum of hands), especially in children/adolescents. They may be skin-colored or light brown/gray. Koebner phenomenon (linear spread at sites of trauma/scratching due to autoinoculation) can occur. A spiky top is typical of filiform/digitate warts, not plane warts.
[A] Common site is the face.
[B] Spiky projection suggests filiform, not plane warts.
[C] Color can vary (flesh to brownish).
[D] Typical age group.
[E] Can spread along scratch lines (Koebner).
Home takeaway:
Plane warts are flat-topped and smooth; spiky warts are filiform.

Q7
Commonest form of recurrent herpes simplex is:

A. Herpes labialis
B. Herpetic whitlow
C. Herpetic conjunctivitis
D. Eczema herpeticum
E. Marginal keratitis
Answer: A

Explanation:
HSV is a double-stranded DNA virus that becomes latent in sensory ganglia and reactivates with triggers such as fever, sunlight, stress, trauma, or immunosuppression. The most common recurrent presentation is herpes labialis (cold sores): prodrome of tingling/burning followed by grouped vesicles on an erythematous base, then crusting and healing. Other forms exist (whitlow on fingers, ocular disease, eczema herpeticum), but they are less common as recurrent patterns.
[A] Most common recurrent HSV presentation.
[B] Less common.
[C] Less common.
[D] Severe disseminated HSV in eczema—important but not commonest.
[E] Not the commonest recurrent form.
Home takeaway:
Recurrent HSV most often = herpes labialis with prodrome → grouped vesicles → crust.

Q8
In herpes zoster, all are true except:

A. Pain may precede the appearance of rash
B. The rash is vesicular
C. Commonly bilateral
D. Frequently associated with underlying malignancy
E. More serious in elderly pt
Answer: C

Explanation:
Herpes zoster (shingles) is reactivation of varicella-zoster virus from dorsal root ganglia. It commonly causes prodromal pain/paresthesia, then a vesicular eruption on an erythematous base in a dermatomal distribution. It is typically unilateral. Disease and complications are more severe in older patients, especially post-herpetic neuralgia. Multidermatomal/disseminated zoster suggests immunosuppression and may be associated with serious underlying disease.

[A] Prodromal pain is common.
[B] Vesicles are typical.
[C] Zoster is usually unilateral, not commonly bilateral.
[D] Can be associated with immunosuppression/serious disease.
[E] More serious in elderly (PHN risk).
Home takeaway:
Zoster = unilateral dermatomal pain then vesicles; elderly have higher PHN risk.

Q9
Ask about family history in all except:

A. Pityriasis rosea
B. Scabies
C. Psoriasis
D. Atopic dermatitis
E. Vitiligo
Answer: A

Explanation:
Family history is important for conditions with genetic/atopic/autoimmune predisposition such as psoriasis, atopic dermatitis, and vitiligo. For scabies, the "history" focus is whether household/close contacts are itchy (contact history). Pityriasis rosea is usually an acute, self-limited viral-triggered eruption, so family history is not a key diagnostic requirement.
[A] PR is viral-triggered and self-limited, not classically hereditary.
[B] Contact/household itching is important history for scabies.
[C] Psoriasis often has a family history.
[D] Atopic dermatitis often has family atopy.
[E] Vitiligo can have autoimmune/familial association.
Home takeaway:
Family history is key for psoriasis/atopy/vitiligo; PR usually doesn't need family history.

Q10
ttt of choice of condylomata acuminata is:

A. Trichloroacetic acid
B. Monochloroacetic acid
C. Cantharidin
D. Podophyllin
Answer: D

Explanation:
Condylomata acuminata are HPV anogenital warts. Several treatments work: destructive methods (cryotherapy, electrocautery, curettage) and topical agents. Podophyllin resin is a classic provider-applied antimitotic therapy that can clear lesions but must be used carefully (avoid pregnancy; avoid large surface areas; wash off after instructed time) because of toxicity/irritation risk. Patient-applied podophyllotoxin is related but distinct.
[A] Trichloroacetic acid is effective but the classic "choice" in this list is podophyllin.
[B] Not the classic standard answer here.
[C] Not standard for genital warts in this context.
[D] Correct choice among listed: classic provider-applied therapy (with safety restrictions).
Home takeaway:
Condylomata acuminata (HPV) can be treated with podophyllin (provider-applied; avoid pregnancy), or other destructive/immune therapies.

Q11
Not a treatment for viral warts:

A. Cryotherapy
B. 5-FU
C. Salicylic acid
D. Topical steroids
Answer: D

Explanation:
Cutaneous viral warts often resolve spontaneously, especially in children, but treatment is used if painful, spreading, persistent, or cosmetically troubling. Effective options include keratolytics (salicylic acid) and destructive therapy (cryotherapy). Other agents (including 5-FU) may be used in selected stubborn warts. Topical steroids do not treat HPV and may worsen viral persistence by local immunosuppression.
[A] Effective destructive option.
[B] Can be used in selected cases.
[C] Core first-line keratolytic option.
[D] Not used for warts; may worsen them.
Home takeaway:
Warts: use keratolytics/destruction; topical steroids have no role.

Q12
Wrong about systemic ttt of H. zoster:

A. Immunodeficiency
B. >50-year-old
C. Peripheral N involvement
D. More than one dermatome
Answer: C

Explanation:
Systemic antivirals are indicated in zoster particularly for higher-risk or severe disease: older age, immunodeficiency, extensive involvement (more than one dermatome), or complicated disease (e.g., ophthalmic involvement). Early systemic therapy reduces severity and risk of complications such as postherpetic neuralgia. "Peripheral nerve involvement" is not the standard phrasing/indication compared with the clear risk factors listed (age, immunodeficiency, multidermatomal).
[A] Immunodeficiency → systemic antivirals indicated.
[B] Older age → systemic antivirals recommended.
[C] Not a standard "indication" compared with the others.
[D] Multidermatomal involvement → systemic therapy needed.
Home takeaway:
Systemic antivirals in zoster are most important in elderly, immunocompromised, and extensive/multidermatomal disease.

Q13
Papule with rough, dry, hyperkeratotic surface represent which type of warts:

A. Common warts
B. Plantar warts
Answer: A

Explanation:
Common warts are rough, dry, hyperkeratotic papules caused by HPV, typically on hands/fingers. Plantar warts occur on soles; pressure flattens them and they often have pain on walking and visible black dots (thrombosed capillaries).
[A] Matches the typical rough hyperkeratotic wart.
[B] Plantar warts are on soles and often flattened/painful.
Home takeaway:
Rough dry hyperkeratotic papule = common wart; plantar wart = painful sole lesion often with black dots.

Q14
Plantar warts, all true except:

A. Most common in children
B. Smooth surface
C. The most common type of warts
D. Fleshy, pink and greyish
Answer: C

Explanation:
Plantar warts are common in children and adolescents. Because of pressure and callus on the sole, their surface may appear smoother than typical hand warts. Color can vary (fleshy/pink/gray) and they may show black dots (thrombosed capillaries). However, the most common overall wart type is generally the common wart (especially on hands), not plantar warts.
[A] Common in children.
[B] Can appear relatively smooth due to pressure/callus.
[C] False: plantar warts are not the most common wart type overall.
[D] Color variation is possible.
Home takeaway:
Plantar warts are common in kids and may look smoother due to pressure; common warts are usually the most common overall type.

Q15
Wrong about common wart:

A. If not treated majority will turn to skin cancer
B. Caused by dsDNA
C. Common in children
D. Majority will resolve spontaneously
Answer: A

Explanation:
Common warts are caused by HPV (a double-stranded DNA virus). They are frequent in children and often resolve spontaneously over months to years as immunity develops. Malignant transformation is not the expected outcome for ordinary common warts; instead, persistence/recurrence relates to HPV persistence and host immunity. Concerning changes (rapid growth, ulceration, persistent pain, atypical appearance) should prompt reassessment, but "majority become cancer" is false.
[A] False: most common warts do not become cancer.
[B] True: HPV is dsDNA.
[C] True: common in children.
[D] True: many resolve spontaneously.
Home takeaway:
Common warts (HPV dsDNA) are common in children and often self-resolve; routine warts don't usually become cancer.

Q16
Shingles, all true except:

A. Oral and topical steroids are frequently used
B. Postherpetic neuralgia can last for months
C. Its reactivation of varicella zoster virus
Answer: A

Explanation:
Shingles is reactivation of VZV causing unilateral dermatomal vesicles and pain. A major complication is postherpetic neuralgia that can persist for months, especially in older adults. The key therapy is systemic antivirals (e.g., oral acyclovir) started early. Steroids are not "frequently" used routinely; if used, it is in selected severe/complicated cases and usually alongside antivirals, not as standard therapy.
[A] False: steroids are not routinely/frequently used for shingles.
[B] True: PHN can last months.
[C] True: reactivation of VZV.
Home takeaway:
Zoster = VZV reactivation; antivirals early; PHN can last months; steroids only in selected cases.

Q17
Elevated papules with a smooth surface, flesh lesions, colored brownish grayish or pinkish:

A. Plane warts
B. Common warts
C. Filiform warts
Answer: A

Explanation:
Plane (flat) warts are smooth-surfaced, flat-topped or slightly elevated papules that are flesh-colored to pinkish or light brown/gray and commonly appear on the face. Common warts are rough and hyperkeratotic. Filiform warts are spiky, finger-like projections.
[A] Best match: smooth, flesh/pink/brown papules.
[B] Rough hyperkeratotic surface—doesn't fit.
[C] Spiky projections—doesn't fit.
Home takeaway:
Smooth flesh/pink/brown papules = plane warts; rough = common; spiky = filiform.

Q18
One is false about pityriasis rosea:

A. Cause herald patches
B. Very itchy rash
C. Self-limiting
Answer: B

Explanation:
Pityriasis rosea is typically self-limited and begins with a herald patch. It can be mildly itchy or even non-itchy in many patients; while itching can occur, it is not usually "very" itchy as a defining feature. The eruption is scaly and often follows cleavage lines on the trunk. Symptomatic treatment is used only if itch/inflammation is significant.
[A] True: herald patch is classic.
[B] False: itch is often mild–moderate if present, not typically "very itchy."
[C] True: self-limiting.
Home takeaway:
PR is self-limited and begins with a herald patch; itch is usually mild if present.

Q19
Pityriasis rosea wrong:

A. Mild non itchy usually
B. Rash before herald lesion
C. Self-limiting
Answer: B

Explanation:
The typical sequence is herald patch first, then a generalized eruption of smaller scaly lesions days later. The course is self-limiting over several weeks. Symptoms may be mild and can be non-itchy or mildly itchy.
[A] Generally mild; may be non-itchy or mildly itchy.
[B] Wrong: herald patch usually precedes the generalized rash.
[C] True: self-limiting.
Home takeaway:
Herald patch comes first → then PR rash; condition resolves spontaneously.

Q20
True about warts:

A. HPV is a double stranded DNA virus
B. Caused by HHV-6
Answer: A

Explanation:
Warts are caused by HPV, a double-stranded DNA virus that infects basal keratinocytes, leading to epidermal hyperplasia and hyperkeratosis. HHV-6 is associated with roseola infantum and is implicated (with HHV-7) in pityriasis rosea triggering—not in warts.
[A] True: HPV is dsDNA.
[B] False: HHV-6 is not a wart cause.
Home takeaway:
Warts are HPV (dsDNA) lesions; HHV-6 relates to roseola/PR, not warts.

Q21
Associated with HSV 6 & 7:

A. Pityriasis rosea
B. Lichen planus
Answer: A

Explanation:
Pityriasis rosea is associated with HHV-6 and HHV-7 triggering. Lichen planus is an inflammatory dermatosis with different associations (not HHV-6/7 as the classic link).
[A] Correct: PR ↔ HHV-6/7.
[B] Not the classic association here.
Home takeaway:
HHV-6/7 association points to pityriasis rosea.

Q22
Slapped cheek disease cause?
Answer: Erythema infectiosum (parvovirus)

Explanation:
Erythema infectiosum (fifth disease) is caused by parvovirus B19. It often occurs in children and classically shows bright red cheeks ("slapped cheek") followed by a rash on the limbs/trunk that fades over 1–2 weeks.
Home takeaway:
Slapped cheek syndrome = parvovirus B19 (erythema infectiosum).

Q23
Which is the causative of molluscum contagiosum?
Answer: Pox virus

Explanation:
Molluscum contagiosum is caused by a poxvirus and presents as multiple flesh-colored, dome-shaped, umbilicated papules. It spreads by direct contact (including autoinoculation) and is common in children. Most cases resolve spontaneously without scarring, so destructive treatments should be used carefully; options include observation, cryotherapy/curettage in selected cases, or topical approaches depending on setting.
Home takeaway:
Molluscum = poxvirus → umbilicated papules, often self-resolving.

Q24
Wrong about plain warts:
Answer: Should always be treated because they don't resolve by themselves

Explanation:
Many warts—especially in children—resolve spontaneously as immunity develops. Plane warts can also regress without treatment. Treatment is chosen when lesions are persistent, spreading, symptomatic, cosmetically troubling, or in special circumstances (immunosuppression).
Home takeaway:
Plane warts can self-resolve; treat only if needed (persistent/symptomatic/spreading).

Q25
False about herpes genitalia:
Answer: Patient should be symptomatic to be contagious

Explanation:
Genital herpes (commonly HSV-2) causes painful grouped vesicles/ulcers, but transmission can occur even when lesions are not visible due to asymptomatic viral shedding. Therefore, patients can be contagious without symptoms. Counseling includes safe sex practices, avoiding sex during outbreaks, and considering suppressive antiviral therapy in recurrent disease to reduce shedding and transmission risk.
Home takeaway:
Genital HSV can spread even without symptoms (asymptomatic shedding).

Q26
Wrong about shingles:
Answer: Treated with topical acyclovir

Explanation:
Shingles requires systemic antiviral therapy (e.g., oral acyclovir) started early (ideally within 72 hours) to reduce severity and complications. Topical acyclovir is used for localized HSV lesions (e.g., cold sores), not as appropriate primary therapy for zoster. Pain control is also important, and complicated cases (e.g., ophthalmic zoster, immunocompromised, disseminated disease) may need IV antivirals and specialist care.
Home takeaway:
Zoster is treated with systemic antivirals early—not topical acyclovir.

Q27
A rash that is not primarily macular?
Answer: Pityriasis rosea

Explanation:
Pityriasis rosea is mainly a scaly patch/plaque eruption. It begins with a herald patch and then multiple scaly lesions on the trunk and proximal limbs, often following cleavage lines. This is not a "primarily macular" exanthem.
Home takeaway:
PR is a scaly patch eruption (herald patch → multiple scaly lesions), not primarily macular.

Q28
Patient presented with mild itchy erythematous patches on the trunk, neck and upper limb with collaret scales, what is the diagnosis?
Answer: Pityriasis rosea

Explanation:
Erythematous patches with collarette scaling on trunk/proximal areas strongly suggest pityriasis rosea. Typical PR starts with a herald patch (oval plaque with collarette scale), followed by multiple smaller scaly lesions on trunk/upper limbs and sometimes neck, often arranged along cleavage lines ("Christmas tree" pattern on the back). It is self-limiting over weeks, so reassurance is key; treat symptoms (emollients, mild topical steroid, oral antihistamine) if itch is present.
Home takeaway:
Collarette scales on trunk/proximal erythematous patches = pityriasis rosea; self-limited, treat symptoms only.

Fungal Infection

Q1
Regarding cutaneous fungal infections one of the following statements is incorrect:

A. Tinea capitis is usually treated with systemic antifungals
B. Seborrheic dermatitis is a differential diagnosis for psoriasis
C. Tinea pedis is usually treated topically
D. Chronic paronychia is usually caused by mixed yeast and bacterial infection
E. Pityriasis versicolor rarely recurs
Answer: E

Explanation:
Superficial fungal infections involve epidermis, hair, nails, and mucosa. Tinea capitis requires systemic antifungals because topical agents do not penetrate the hair shaft sufficiently to cure infection. Tinea pedis is commonly managed with topical antifungals (e.g., topical terbinafine/azoles) unless extensive or refractory. Chronic paronychia is strongly linked to "wet work" and commonly involves Candida (often with secondary bacterial involvement), so management includes keeping hands dry plus topical antifungal/anti-inflammatory measures. Pityriasis (tinea) versicolor is a Malassezia infection that commonly relapses, especially in hot/humid climates or with sweating, so "rarely recurs" is incorrect.
[A] Hair-shaft disease needs systemic therapy.
[B] Both can present as scaly erythematous plaques; seborrheic dermatitis is an important differential for psoriasis.
[C] Typical tinea pedis responds well to topical therapy.
[D] Chronic paronychia is classically mixed (yeast ± bacteria) in a wet environment.
[E] Versicolor often recurs/relapses → this statement is wrong.
Home takeaway:
Tinea capitis = systemic; tinea pedis = usually topical; chronic paronychia = wet-work + Candida/mixed infection; versicolor commonly recurs.

Q2
All about Griseofulvin are true except:

A. Has better absorption after meal
B. Commonest side effect is headache
C. Contraindicated in pregnancy
D. Phenobarbitone may neutralize its effect
E. May be used in treatment of sycosis barbae
Answer: E

Explanation:
Griseofulvin is used mainly for dermatophyte infections, especially tinea capitis. Absorption increases with a fatty meal. Headache is a common adverse effect. It is contraindicated in pregnancy. Enzyme inducers (e.g., phenobarbital) can reduce its efficacy by increasing metabolism. "Sycosis barbae" is classically bacterial folliculitis of the beard area; griseofulvin is for dermatophytes (tinea), not bacterial sycosis barbae.
[A] True: take with meal (fatty meal improves absorption).
[B] True: headache is common.
[C] True: avoid in pregnancy.
[D] True: phenobarbital can reduce effect.
[E] Wrong: sycosis barbae is bacterial; antifungals are for tinea barbae, not sycosis barbae.
Home takeaway:
Griseofulvin treats dermatophytes (esp. tinea capitis); take with fatty meal; headache common; avoid pregnancy; phenobarbital reduces effect.

Q3
One of the following fungi induce inflammatory tinea capitis:

A. T. violaceum
B. T. sulphureum
C. T. tonsurans
D. T. verrucosum
E. T. mentagrophytes
Answer: D

Explanation:
Inflammatory tinea capitis (often presenting as kerion) is more likely with zoophilic species that trigger a strong inflammatory response. Trichophyton verrucosum is classically acquired from cattle and is well known for causing inflammatory scalp infection and kerion.
[A] Not the classic "zoophilic cattle inflammatory" organism.
[B] Not a typical best answer for inflammatory kerion-type infection.
[C] Common cause of tinea capitis (often endothrix), but the classic inflammatory cattle link is not this.
[D] Best answer: cattle-associated zoophilic organism → inflammatory tinea capitis/kerion.
[E] Can also cause kerion, but the single best classic answer here is T. verrucosum.
Home takeaway:
Zoophilic species (especially T. verrucosum from cattle) commonly cause inflammatory tinea capitis/kerion.

Q4
All true about tinea versicolor except:

A. Apple green color on Wood's lamp
B. Hypo or hyperpigmentation
C. Patches or plaques
D. Scaling
E. Most common causative agent is Malassezia
Answer: A

Explanation:
Pityriasis (tinea) versicolor is a superficial yeast infection caused by Malassezia. It presents with hypo- or hyperpigmented patches on the upper trunk/neck with fine scale and may become more obvious after sun exposure because affected areas don't tan normally. Under Wood's lamp it typically shows yellow/golden fluorescence, not apple-green.
[A] Wrong: versicolor is typically yellow/golden fluorescence, not apple-green.
[B] True: lesions can be hypo- or hyperpigmented.
[C] True: patches/plaques can be seen clinically.
[D] True: fine scaling is typical.
[E] True: Malassezia is the causative organism.
Home takeaway:
Versicolor = Malassezia + fine scale + pigment change; Wood's lamp tends to be yellow/golden (not green).

Q5
Tinea unguium is the infection of:

A. Lateral nail folds
B. Posterior nail folds
C. Nail plate
D. Nail bed
E. Nail matrix
Answer: C

Explanation:
Tinea unguium (onychomycosis) refers to fungal infection primarily involving the nail plate, leading to discoloration, thickening, brittleness, and sometimes onycholysis. Nail fold involvement points more toward paronychia; matrix involvement causes proximal dystrophy patterns.
[A] Nail fold inflammation suggests paronychia.
[B] Nail fold disease suggests paronychia.
[C] Best answer: nail plate infection.
[D] Nail bed can be involved, but the classic definition in this style targets the plate.
[E] Matrix disease causes proximal changes; not the classic definition.
Home takeaway:
Tinea unguium (onychomycosis) = nail plate infection → thick, brittle, discolored nails.

Q6
Not associated with candidal infection:

A. Occurs between the 2nd and 3rd fingers
B. Affects proximal lamella
C. Corner of the mouth
D. Tongue and oral mucosa
E. Genital area
Answer: A

Explanation:
Candida commonly involves mucous membranes (oral thrush), genital area, and skin flexures (intertrigo with satellite pustules). It can also be involved in chronic paronychia affecting the proximal nail fold/cuticle region. Angular cheilitis at the corners of the mouth can be Candida-related. "Between the 2nd and 3rd fingers" is not a typical hallmark association compared with classic flexural and mucosal sites.
[A] Not a typical classic Candida association site.
[B] Proximal nail fold/cuticle involvement fits chronic paronychia patterns.
[C] Angular cheilitis may be Candida-related.
[D] Oral mucosa involvement is common.
[E] Genital involvement is common.
Home takeaway:
Candida favors mucosa + genital + flexures (satellite pustules) + chronic paronychia patterns.

Q7
One of the following fungi is ectothrix:

A. T. schoenleinii
B. T. violaceum
C. T. mentagrophytes
D. T. tonsurans
E. T. sulphureum
Answer: C

Explanation:
Ectothrix means arthroconidia are on the outside of the hair shaft. Trichophyton mentagrophytes is a classic ectothrix dermatophyte and may cause inflammatory tinea capitis/kerion.
[A] More linked with favus-type patterns; not the classic ectothrix answer here.
[B] More often taught as endothrix in many exam sets.
[C] Correct: classic ectothrix organism.
[D] Often endothrix.
[E] Not the classic ectothrix answer.
Home takeaway:
Ectothrix (spores outside hair) → classic example: T. mentagrophytes.

Q8
Fungi doesn't fluoresce under Wood's light:

A. Microsporum audouinii
B. Microsporum canis
C. Microsporum gypseum
D. Trichophyton mentagrophytes
E. Trichophyton schoenleinii
Answer: D

Explanation:
Wood's lamp fluorescence is classically seen with many Microsporum infections (green/blue-green). Most Trichophyton species do not fluoresce. Therefore, Trichophyton mentagrophytes is the best "non-fluorescent" choice here.
[A] Microsporum often fluoresces.
[B] Microsporum can fluoresce.
[C] Microsporum species may fluoresce variably.
[D] Correct: Trichophyton mentagrophytes typically does not fluoresce.
[E] Not the best "non-fluorescent" choice compared with D in this question style.

Home takeaway:
Wood's lamp: Microsporum often fluoresces; most Trichophyton infections don't.

Q9
Which dermatophyte is likely to be acquired from cattle:

A. Trichophyton rubrum
B. Trichophyton schoenleinii
C. Trichophyton verrucosum
D. Trichophyton tonsurans
E. Microsporum gypseum
Answer: C

Explanation:
Trichophyton verrucosum is a zoophilic dermatophyte strongly associated with cattle and can cause inflammatory human infection after contact.
[A] T. rubrum is mainly human-associated (anthropophilic).
[B] Not the classic cattle organism.
[C] Correct: cattle-associated dermatophyte.
[D] Often human-to-human spread.
[E] More soil-associated (geophilic).
Home takeaway:
Cattle-associated dermatophyte = T. verrucosum.

Q10
Fungal responsible for epidemics of tinea capitis:

A. T. canis
B. T. verrucosum
C. T. mentagrophyte
D. M. audouinii
Answer: D

Explanation:
Microsporum audouinii is classically associated with epidemic/outbreak tinea capitis, especially in school settings due to efficient spread.
[A] Not the standard organism naming here (Microsporum canis is a recognized agent but more zoonotic).
[B] Cattle-linked and usually sporadic.
[C] Can cause disease but not the classic epidemic answer.
[D] Correct: classic outbreak organism.
Home takeaway:
Epidemic tinea capitis (schools) classically = Microsporum audouinii.

Q11
Doesn't cause tinea capitis:

A. Microsporum audouinii
B. Trichophyton schoenleinii
C. Trichophyton tonsurans
D. Trichophyton verrucosum
Answer: All the above are causes of tinea capitis

Explanation:
Tinea capitis is caused by dermatophytes, mainly Microsporum and Trichophyton species. The listed organisms are all recognized causes of tinea capitis (including outbreak-associated species and zoophilic inflammatory species).
[A] Recognized cause of tinea capitis.
[B] Recognized cause (favus-type patterns in classic teaching).
[C] Common cause in many communities.
[D] Zoophilic cause often leading to inflammatory disease.
Home takeaway:
Tinea capitis can be caused by both Microsporum and Trichophyton species—these options are all causes.

Q12
Wrong about tinea pedis:

A. Most common adult fungal infection
B. Can be caused by Epidermophyton floccosum
C. Zoophilic
D. Caused by T. violaceum
Answer: D

Explanation:
Tinea pedis is the commonest adult superficial dermatophyte infection, typically acquired from communal wet surfaces. It presents as itching, scaling, and maceration between toes (especially 4th web space) and sometimes a dry "moccasin" scale pattern. It may be caused by several dermatophytes including Epidermophyton floccosum. T. violaceum is classically linked more with scalp infection patterns than with tinea pedis in standard teaching.
[A] True: very common adult infection.
[B] True: E. floccosum can cause tinea pedis.
[C] Not the best general descriptor; many cases are anthropophilic/environmental rather than strictly zoophilic.
[D] Wrong: T. violaceum is not a typical cause of tinea pedis.
Home takeaway:
Tinea pedis = common adult interdigital scaling/maceration; treat topically; not classically due to T. violaceum.

Q13
Which of the following is the initial test in fungal skin infection:

A. KOH preparation and microscopy
B. Wood lamp
Answer: A

Explanation:
Initial evaluation is direct microscopy of skin/nail/hair samples (commonly KOH preparation) to detect hyphae/yeast. Culture can identify species but is slower. Wood's lamp is an adjunct for selected infections (e.g., some Microsporum tinea capitis, versicolor fluorescence), not the universal first test.
[A] Correct: KOH microscopy is the initial test.
[B] Adjunct only; not the first step in most cases.
Home takeaway:
First test for suspected fungal infection = KOH prep + microscopy.

Q14
Which is wrong about tinea unguium:

A. Change in nail color
B. Onycholysis
C. Prolonged treatment
D. Caused by T. verrucosum
Answer: D

Explanation:
Onychomycosis typically causes discoloration, thickening, brittleness, and sometimes onycholysis. Treatment is prolonged, especially for toenails. While dermatophytes are common causes, T. verrucosum is not the typical "standard" pathogen answer for tinea unguium in this exam style compared with more common dermatophytes.
[A] True: discoloration is typical.
[B] True: onycholysis can occur.
[C] True: treatment is prolonged (especially toenails).
[D] Wrong in this question style: not the typical onychomycosis cause.
Home takeaway:
Onychomycosis = discolored thick brittle nails ± onycholysis; needs long treatment.

Q15
Candidiasis may affect all of the following except:

A. Skin
B. Mucous membranes
C. Nail
D. Hair
Answer: D

Explanation:
Candida affects mucosa (oral/vaginal), skin flexures (intertrigo), and nail folds (chronic paronychia) and can affect nails. Hair is not a typical Candida infection site in routine candidiasis patterns.
[A] True: skin (flexures) can be affected.
[B] True: mucosa commonly affected.
[C] True: nail folds/nails can be involved.
[D] Correct exception: hair is not typical.
Home takeaway:
Candida affects skin, mucosa, and nails—hair is the exception.

Q16
All dermatophytes fluoresce under Wood's light except:

A. Microsporum canis
B. Microsporum audouinii
C. Trichophyton schoenleinii
D. Trichophyton rubrum
Answer: D

Explanation:
Wood's lamp fluorescence is classically associated with Microsporum infections (green/blue-green). Trichophyton rubrum typically does not fluoresce, so it is the best exception.
[A] Often fluoresces.
[B] Often fluoresces in classic teaching.
[C] Can show fluorescence in some settings/teaching sets.
[D] Correct: typically non-fluorescent.
Home takeaway:
Microsporum often fluoresces; T. rubrum is typically non-fluorescent.

Q17
Young male with scaly hypopigmented patches over the back, diagnosis:

A. Pityriasis versicolor
B. Vitiligo
C. Pityriasis rosea
D. Rosacea
Answer: A

Explanation:
Pityriasis versicolor causes hypo- or hyperpigmented patches with fine scale on upper back/neck/chest and becomes more obvious after sun exposure when affected areas fail to tan. Vitiligo is chalk-white depigmentation without scale. Pityriasis rosea usually has a herald patch and collarette scaling in a cleavage-line pattern. Rosacea is facial.
[A] Correct: scaly hypopigmented trunk patches fit versicolor.
[B] Vitiligo is non-scaly depigmentation.
[C] PR has herald patch and patterned trunk eruption.
[D] Rosacea is facial.
Home takeaway:
Scaly hypopigmented back patches that "don't tan" = pityriasis versicolor.

Q18
First line treatment of tinea capitis:

A. Griseofulvin
B. Topical miconazole
C. Steroids
Answer: A

Explanation:
Tinea capitis requires systemic antifungals because infection involves the hair shaft/follicle and topical treatments alone are inadequate. Griseofulvin is a classic first-line systemic agent in many curricula. Steroids do not treat the fungus and can worsen/mask infection.
[A] Correct: systemic griseofulvin.
[B] Topical alone is insufficient for hair involvement.
[C] Steroids can worsen/mask fungal infection.
Home takeaway:
Tinea capitis = systemic antifungal (griseofulvin classic first-line).

Q19
Tinea corporis lesion:

A. Multiple vesicles
B. Annular
C. Wheal
Answer: B

Explanation:
Tinea corporis classically forms annular erythematous plaques with a raised scaly active border and central clearing (ringworm). Wheals suggest urticaria; vesicles are not the defining primary description for corporis.
[A] Not the classic morphology.
[B] Correct: annular lesion with central clearing and scaly edge.
[C] Wheal = urticaria.
Home takeaway:
Tinea corporis = annular scaly border with central clearing.

Q20
Which one is not commonly colonize healthy skin:

A. Staph. albus
B. Staph. aureus
C. Candida albicans
Answer: C

Explanation:
Normal skin flora commonly includes coagulase-negative staphylococci (often called Staph. albus historically) and many individuals carry Staph. aureus (especially in the nose with skin colonization). Candida is more typically a commensal of mucous membranes (mouth/vagina) and becomes an opportunistic pathogen with risk factors, rather than a common healthy skin colonizer in this teaching context.
[A] Common skin commensal.
[B] Common colonizer in many people.
[C] Best answer: not a common healthy skin colonizer (more mucosal commensal/opportunist).
Home takeaway:
Skin flora: staph common; Candida is more typically mucosal and opportunistic.

Q21
Not seen in tinea capitis:

A. Exclamation mark sign
B. Scales
C. Focal alopecia
Answer: A

Explanation:
Tinea capitis commonly presents with scalp scaling, patchy alopecia, broken hairs/black dots, and sometimes inflammatory kerion. Exclamation mark hairs are characteristic of alopecia areata, not tinea capitis.
[A] Correct: exclamation-mark hairs point to alopecia areata.
[B] Scaling is common in tinea capitis.
[C] Patchy/focal alopecia is common in tinea capitis.
Home takeaway:
Tinea capitis = scaling + patchy alopecia; exclamation-mark hairs = alopecia areata.

Q22
Apple green fluorescence is seen in:

A. Tinea capitis
B. Tinea cruris
Answer: A

Explanation:
Wood's lamp can show green/blue-green fluorescence in some tinea capitis cases (especially Microsporum hair infections). Tinea cruris is not classically diagnosed by apple-green fluorescence; a key groin Wood's lamp clue is erythrasma (coral-red), not dermatophyte cruris.
[A] Correct: some tinea capitis fluoresces green/blue-green.
[B] Not typical for tinea cruris.
Home takeaway:
Green/blue-green Wood's lamp fluorescence suggests some tinea capitis (Microsporum), not tinea cruris.

Q23
Commonest fungal infection in adults:
Answer: Tinea pedis

Explanation:
Tinea pedis (athlete's foot) is the most common adult superficial dermatophyte infection, often presenting with interdigital scaling/maceration and itching, sometimes with plantar "moccasin" scale.
Home takeaway:
Most common adult fungal infection = tinea pedis.

Q24
Wrong about tinea versicolor:
Answer: Cherry red fluorescence under Wood's lamp

Explanation:
Versicolor typically shows yellow/golden fluorescence under Wood's lamp. Coral/cherry red fluorescence is typical of erythrasma (a bacterial infection), not versicolor.
Home takeaway:
Versicolor fluoresces yellow/golden; coral-red fluorescence suggests erythrasma.

Q25
Treatment of toenail onychomycosis (three yellow nails):
Answer: Oral antifungal

Explanation:
Multiple toenail onychomycosis usually requires systemic therapy because topical agents penetrate poorly and cure rates are low for multi-nail disease. Treatment duration is long for toenails (often several months).
Home takeaway:
Multiple toenail onychomycosis → systemic antifungal and prolonged course.

Q26
Which of the following causes erythema with satellite pustules over body flexors:
Answer: Candida cutaneous infection

Explanation:
Candida intertrigo occurs in flexures with bright erythema and characteristic "satellite" pustules/papules beyond the main rash margin—this satellite pattern is a classic clue.
Home takeaway:
Flexural erythema + satellite pustules = Candida intertrigo.

Q27
Kerion is caused by:
Answer: T. verrucosum & T. mentagrophytes

Explanation:
Kerion is a boggy, inflamed, pustular plaque on the scalp due to an intense inflammatory response to dermatophyte infection (a severe form of tinea capitis). Zoophilic species commonly cause kerion, especially T. verrucosum (cattle-associated) and T. mentagrophytes. It is treated with systemic antifungals; surgical drainage is not needed.
Home takeaway:
Kerion = inflammatory tinea capitis (often zoophilic) → treat with systemic antifungal; no incision/drainage.

Q28
Onychomycosis treatment:
Answer: Systemic antifungal

Explanation:
Onychomycosis often needs systemic antifungals (especially toenails) because infection is within/under the nail plate and treatment must continue long enough for healthy nail regrowth. Topicals are reserved for mild/single-nail disease or adjunct prevention.
Home takeaway:
Onychomycosis usually needs systemic therapy (especially toenails); topical is for mild cases.

Q29
Wrong:
Answer: Tinea versicolor rarely recurs

Explanation:
Pityriasis versicolor is well known to relapse/recurs, particularly with heat, humidity, sweating, and oily skin. Preventive/maintenance therapy may be needed in recurrent cases.
Home takeaway:
Versicolor is a relapsing condition—recurrence is common.

Q30
Slightly elevated scaling margins and halo central clearing:
Answer: Tinea corporis

Explanation:
Tinea corporis produces annular lesions with a slightly raised scaly active border and central clearing. It is often itchy and responds to topical antifungals; systemic therapy is reserved for extensive, recurrent, or refractory disease.
Home takeaway:
Raised scaly border + central clearing = tinea corporis; treat topically (systemic if extensive).

Bullous Diseases

Q1
Antibodies are directed in bullous pemphigoid towards:

A. Hemidesmosomes
B. Desmosomes
C. Dermal papilla
D. Granular cell layer
E. None of the above
Answer: A
Explanation:
Bullous pemphigoid is an autoimmune subepidermal blistering disease. The immune target is the basement membrane zone anchoring system (hemidesmosomes), mainly BP180 (collagen XVII) and BP230. Antibody binding activates complement (C3) and recruits inflammatory cells (often eosinophils), leading to separation at the dermo-epidermal junction. Clinically this produces tense, firm bullae and usually a negative Nikolsky sign because the epidermis remains intact as a roof.
[A] Correct: hemidesmosomes (BP180/BP230) are the targets in bullous pemphigoid.
[B] Desmosomes are targeted in pemphigus (intraepidermal acantholysis), not pemphigoid.
[C] Dermal papilla is not the target structure.
[D] Granular layer is not the level/target here.
[E] Incorrect because there is a clear target at the basement membrane zone.
Home takeaway:
Bullous pemphigoid = anti-hemidesmosomes (BP180/BP230) → subepidermal split → tense bullae.

Q2
Which of the following diagnostic aids is the most valuable in differentiating bullous pemphigoid from erythema multiforme:

A. Histology
B. Tzanck test
C. Immunofluorescence
D. Electron microscopy
E. Clinical features
Answer: C
Explanation:
Direct immunofluorescence (DIF) is the most valuable test for immunobullous diseases because it shows the characteristic pattern of immune deposition in skin. Bullous pemphigoid classically shows linear IgG and C3 along the basement membrane zone. Erythema multiforme is an interface dermatitis pattern and is not defined by linear basement-membrane IgG/C3 deposition. Histology and clinical picture can help, but DIF is the most specific discriminator.
[A] Helpful (subepidermal blister with eosinophils in BP), but less definitive than DIF.
[B] Tzanck helps for herpes/acantholysis screening, not best for BP vs EM.
[C] Correct: DIF pattern is most diagnostic.
[D] Not routinely required.
[E] Helpful but not as definitive as DIF.
Home takeaway:
Best way to confirm BP and distinguish from look-alikes = DIF (linear IgG/C3 at DEJ).

Q3
Pemphigus vulgaris characterized by all except:

A. More in elderly people
B. Rare disease
C. More in Jews
D. Fatal if untreated
E. Presence of acantholysis
Answer: A

Explanation:
Pemphigus vulgaris is a rare autoimmune blistering disease with autoantibodies against desmosomal proteins (mainly desmoglein 3 ± desmoglein 1). This causes loss of keratinocyte adhesion (acantholysis) leading to intraepidermal (classically suprabasal) blisters. Clinically the bullae are flaccid and rupture easily, leaving painful erosions; mucosal involvement (especially oral) is common and may precede skin lesions. Untreated disease can be fatal due to fluid loss, infection, and metabolic complications. The typical age is middle-aged adults, not mainly elderly.
[A] Incorrect: classic age is middle age (not "more in elderly").
[B] True: PV is rare.
[C] True: ethnic/racial predisposition is recognized in some populations.
[D] True: can be fatal if untreated.
[E] True: acantholysis is a hallmark.
Home takeaway:
Pemphigus vulgaris = middle age + acantholysis + flaccid bullae + mucosal erosions; fatal if untreated.

Q4
Which of the following disease showing racial prevalence:

A. Bullous pemphigoid
B. Pemphigus vulgaris
C. Chronic cicatricial pemphigoid
D. Juvenile pemphigoid
E. Dermatitis herpetiformis
Answer: B
Explanation:
Among common blistering disorders, pemphigus vulgaris is classically described as having notable ethnic/racial predilections in exam framing (with higher prevalence in certain groups). It is autoimmune (anti-desmogleins) and produces intraepidermal flaccid blisters with frequent mucosal erosions. Bullous pemphigoid is more strongly linked to older age and is the most common autoimmune blistering disease, but "racial prevalence" here points to pemphigus vulgaris.
[A] BP is mainly age-associated rather than "racial prevalence" in this style.
[B] Correct: PV has recognized ethnic predilection.
[C] Defined more by mucosal scarring presentation than racial prevalence.
[D] Defined by age group.
[E] More linked to gluten-sensitive enteropathy patterns than the "racial prevalence" wording here.
Home takeaway:
If asked "racial/ethnic prevalence" in immunobullous disease → think pemphigus vulgaris.

Q5
Pemphigus vulgaris is a:

A. Viral disease
B. Autoimmune disease
C. Bacterial disease
D. Hormonal disease
E. Unknown
Answer: B
Explanation:
Pemphigus vulgaris is an autoimmune disease mediated by IgG autoantibodies against desmosomal cadherins (desmoglein 3 ± 1). This causes acantholysis and intraepidermal blister formation, producing fragile flaccid bullae that rupture into erosions, commonly with painful oral mucosal involvement.
[A] Not viral.
[B] Correct: autoimmune.
[C] Not bacterial.
[D] Not a hormonal disease (though hormones can influence immune disease generally).
[E] Mechanism is known.
Home takeaway:
Pemphigus vulgaris = autoimmune anti-desmosomes → acantholysis → flaccid bullae + mucosal erosions.

Q6
All are healing diseases without scarring except:

A. Epidermolysis bullosa simplex
B. Rash of secondary syphilis
C. Impetigo
D. Dystrophic epidermolysis bullosa
E. Herpes zoster
Answer: D
Explanation:
Scarring depends on the depth of skin damage. Superficial processes limited to the epidermis typically heal without scarring, while deeper structural cleavage or dermal injury tends to scar. Dystrophic epidermolysis bullosa involves deeper structural fragility and classically heals with scarring and deformity. Epidermolysis bullosa simplex is usually more superficial and tends to heal without scarring. Impetigo is superficial and typically heals without scarring. Secondary syphilis rash is generally non-scarring. Herpes zoster can leave post-inflammatory pigmentation and occasionally scarring, but the classic scarring blistering disorder here is dystrophic EB.
[A] Usually non-scarring (superficial).
[B] Typically heals without scarring.
[C] Superficial infection, usually non-scarring.
[D] Correct: dystrophic EB → scarring.
[E] Can leave pigment change ± scars, but not the classic "scarring by definition" compared with dystrophic EB.
Home takeaway:
Dystrophic EB = scarring blistering; superficial epidermal conditions usually heal without scarring.

Q7
Bullous pemphigoid:

A. Linear IgG & C3
B. Granular IgG & C3
C. Linear IgA & C3
D. Granular IgA & C3
Answer: A
Explanation:
Bullous pemphigoid shows autoantibody/complement deposition along the basement membrane zone. On direct immunofluorescence, the classic pattern is linear IgG and C3 at the dermo-epidermal junction. This correlates with subepidermal tense bullae and usually a negative Nikolsky sign.
[A] Correct: linear IgG and C3 at DEJ.
[B] Granular IgG/C3 is not typical for BP.
[C] Linear IgA suggests IgA bullous dermatosis pattern.
[D] Granular IgA suggests dermatitis herpetiformis pattern.
Home takeaway:
BP DIF hallmark = linear IgG + C3 at basement membrane zone.

Q8
Wrong about pemphigus and pemphigoid:

A. Pemphigoid is associated with more morbidity and mortality
B. Abs against desmogleins in pemphigus and collagen 17 in pemphigoid
C. Intraepidermal blisters in pemphigus
D. Subepidermal blisters in pemphigoid
Answer: A
Explanation:
Pemphigus is due to antibodies against desmogleins (desmosomes) causing intraepidermal blistering and significant mucosal disease; it is generally the more severe condition if untreated. Pemphigoid is due to antibodies against basement membrane zone proteins such as BP180 (collagen XVII) and BP230 causing subepidermal blistering with tense bullae and typically less mucosal involvement. Therefore, saying pemphigoid has "more morbidity and mortality" is the incorrect statement in this comparison.
[A] Wrong: pemphigus is generally more severe overall.
[B] Correct: pemphigus = desmogleins; pemphigoid = BP180/collagen XVII (± BP230).
[C] Correct: pemphigus is intraepidermal.
[D] Correct: pemphigoid is subepidermal.
Home takeaway:
Pemphigus = intraepidermal flaccid bullae (often severe); Pemphigoid = subepidermal tense bullae.

Q9
The bullae of pemphigus vulgaris are:

A. Subcorneal
B. Supradermal
C. Dermal
D. None of the above
Answer: D
Explanation:
Pemphigus vulgaris produces intraepidermal blisters, classically suprabasal due to acantholysis. "Subcorneal" would fit pemphigus foliaceus rather than pemphigus vulgaris, and "dermal" is not correct for pemphigus. Since "intraepidermal/suprabasal" is not listed, the best answer is "none of the above."
[A] Subcorneal = more typical of pemphigus foliaceus.
[B] Not the standard way PV is described; PV is intraepidermal (suprabasal).
[C] Dermal bullae is incorrect.
[D] Correct: PV bullae are intraepidermal (suprabasal), which is not listed.
Home takeaway:
Pemphigus vulgaris blister level = intraepidermal (suprabasal).

Q10
One doesn't cause epidermal bullous:

A. Impetigo
B. Dermatitis herpetiformis
C. Eczema
D. Pemphigoid vulgaris
Answer: B + C
Explanation:
"Epidermal bullous" in this framing refers to blistering within the epidermis. Bullous impetigo is an intraepidermal blister due to toxin-mediated cleavage. Dermatitis herpetiformis produces subepidermal blisters (not epidermal). Eczema forms vesicles due to spongiosis (intercellular edema) rather than a classic epidermal cleavage blister category; it is not considered a primary epidermal bullous disease in this classification. Pemphigoid diseases are subepidermal as well, but the keyed answer here identifies DH and eczema as the "doesn't cause epidermal bullous" options.
[A] Can cause intraepidermal bullae (bullous impetigo).
[B] Subepidermal blistering → not epidermal.
[C] Spongiotic vesicles, not classic epidermal bullous disease in this framing.
[D] Pemphigoid is subepidermal, but the provided answer focuses on B and C.
Home takeaway:
Epidermal blistering ≠ subepidermal diseases (DH/pemphigoid); eczema vesicles are spongiosis-based.

Q11
Choose the correct statement regarding bullous pemphigoid:

A. Antibodies against hemidesmosomes
B. Positive nikolsky sign
C. The bullae are flaccid
D. Mucous membranes are commonly involved
Answer: A
Explanation:
Bullous pemphigoid is caused by antibodies against hemidesmosomes at the basement membrane zone, producing a subepidermal split. Clinically the bullae are tense and Nikolsky sign is usually negative. Mucosal involvement may occur but is not as common or as extensive as in pemphigus vulgaris.
[A] Correct: anti-hemidesmosomes.
[B] Usually negative in BP.
[C] BP bullae are tense, not flaccid.
[D] Mucosa may be involved but not "commonly" compared with pemphigus.
Home takeaway:
BP = tense bullae + Nikolsky negative + anti-hemidesmosomes.

Q12
45-year-old woman with tense bullae on trunk and extremities, negative nikolsky's sign, immunofluorescence showed subepidermal deposition of IgG and C3, diagnosis:

A. Pemphigus vulgaris
B. Bullous pemphigoid
C. Epidermolysis bullosa
Answer: B
Explanation:
Tense bullae plus negative Nikolsky strongly suggests subepidermal blistering. DIF showing IgG and C3 at the basement membrane zone is classic for bullous pemphigoid. Pemphigus vulgaris would show flaccid bullae/erosions with intercellular IgG and positive Nikolsky. Epidermolysis bullosa is usually inherited with onset earlier in life.
[A] PV: flaccid bullae + mucosal erosions + intercellular IgG.
[B] Correct: BP = tense bullae + linear IgG/C3 at DEJ.
[C] EB: inherited, often presents early, not this DIF pattern.
Home takeaway:
Tense bullae + linear IgG/C3 at DEJ = bullous pemphigoid.

Q13
Wrong about pemphigus vulgaris:

A. Middle age
B. Bad general condition
C. Tense bulla
Answer: C
Explanation:
Pemphigus vulgaris usually affects middle-aged adults and can cause poor general condition due to widespread painful erosions, fluid loss, and infection risk. The blisters are intraepidermal and fragile, producing flaccid bullae that rupture easily—so "tense bulla" is the wrong statement.
[A] Typical age = middle age.
[B] Severe disease can make the patient systemically unwell.
[C] Wrong: PV bullae are flaccid, not tense.
Home takeaway:
PV = flaccid bullae + erosions; tense bullae suggest subepidermal disease (BP).

Q14
Mucous membranes are extensively involved in epidermolysis bullosa:

A. Simplex
B. Hyperplastica
C. Polydysplastica
D. Cockayne
Answer: C
Explanation:
Epidermolysis bullosa is a group of inherited blistering disorders. Extensive mucosal involvement is seen in the more severe forms rather than epidermolysis bullosa simplex, which is typically more superficial and mainly cutaneous. In this classification set, "polydysplastica" corresponds to the severe type with extensive mucous membrane involvement.
[A] Simplex: usually less mucosal involvement.
[B] Not the best match for extensive mucosal involvement in this set.
[C] Correct: extensive mucosal involvement in the severe category.
[D] Not the typical best match here.
Home takeaway:
Extensive mucosal involvement happens in severe EB variants, not usually in EB simplex.

Q15
Patient presented with painful mouth ulcers and flaccid bullae, which of the following is the most likely diagnosis:

Answer: Pemphigus vulgaris

Explanation:
Painful oral erosions plus flaccid bullae strongly point to pemphigus vulgaris. PV is autoimmune with anti-desmoglein antibodies causing acantholysis and intraepidermal (suprabasal) blistering. Because the blister roof is thin, bullae rupture quickly, leaving painful erosions; Nikolsky sign is typically positive, and mucosal disease is common and often early.
Home takeaway:
Painful oral ulcers + flaccid bullae = pemphigus vulgaris.

Q16
Not a cause of generalized blistering:
Answer: Eczema
Explanation:
Generalized blistering is more typical of widespread immunobullous disorders (pemphigus/pemphigoid), severe drug reactions, infections, or inherited blistering diseases. Eczema primarily causes eczematous plaques with pruritus; vesicles can occur from spongiosis, but eczema is not classically categorized as a primary cause of generalized blistering in the same way as immunobullous diseases.
Home takeaway:
Generalized blistering → think immunobullous/drug/infectious/genetic; eczema is mainly spongiotic dermatitis, not a classic generalized bullous disease.

Sexually Transmitted Diseases

Q1
Commonest serological test used in follow up at syphilitic pt:

A. FTA
B. TPI
C. WR
D. RPCF
E. FTA-ABS
Answer: C
Explanation:
Syphilis tests are divided into:
• Non-treponemal tests (screening/follow-up): titers correlate with disease activity and usually fall after effective treatment, so they are used to monitor response and detect reinfection.
• Treponemal tests (confirmatory): usually remain positive for life, so they are not ideal for follow-up.
"WR" (Wassermann reaction) is an older non-treponemal-type test conceptually used for follow-up because it can be trended (like VDRL/RPR).
[A] Treponemal test → stays positive → not good for follow-up trend.
[B] Not routinely used for follow-up; not practical for titer monitoring.
[C] Best option here for follow-up monitoring among listed choices.
[D] Not a standard follow-up tool compared with non-treponemal titers.
[E] Confirmatory test; usually stays positive long-term.
Home takeaway:
Follow-up syphilis = use a non-treponemal titer you can trend (WR here; modern equivalents are VDRL/RPR).

Q2
All about 2ry syphilis true except:

A. Never itches
B. Contagious
C. STD (serologic test for syphilis) is +ve in 100% of cases
D. Most commonly vesicular
E. Presents with generalized rash
Answer: D
Explanation:
Secondary syphilis is a disseminated stage with widespread mucocutaneous disease. Typical features:
• Generalized rash (often maculopapular and can be scaly)
• Palms and soles may be involved
• Usually little or no itch
• Highly contagious (lesions contain organisms)
• Serology is typically positive by this stage
Vesicles are not the common morphology of secondary syphilis; vesicular eruptions suggest other diagnoses.
[A] Fits classic description: usually non-itchy.
[B] True: contagious in secondary stage.
[C] Serology is usually positive in secondary syphilis.
[D] Wrong statement: "most commonly vesicular."
[E] True: generalized rash is typical.
Home takeaway:
Secondary syphilis = generalized (often non-itchy) rash ± palms/soles + very contagious; not typically vesicular.

Q3
Which about secondary syphilis is incorrect:

A. Lesions usually appear 6–16 weeks after infection
B. Lesions usually involve palms & soles
C. Most lesions contain spirochetes
D. Lymphadenopathy is usually absent
E. Lesions seldom itch
Answer: D

Explanation:
Secondary syphilis commonly presents weeks after primary infection with systemic spread. Typical findings include:
• Generalized rash (often includes palms/soles)
• Generalized lymphadenopathy is common
• Lesions are infectious (organisms can be present)
• Rash usually has minimal itch
Therefore "lymphadenopathy is usually absent" is incorrect.
[A] Timing fits typical secondary stage window.
[B] Palms/soles involvement is classic.
[C] Secondary lesions are contagious and can contain organisms.
[D] Incorrect: lymphadenopathy is commonly present.
[E] Correct: lesions usually do not itch much.
Home takeaway:
Secondary syphilis often includes generalized LAD + palms/soles rash and minimal itch.

Q4
In classical syphilitic chancre all of the following statement are true except:

A. Occur at site of inoculation
B. Commonly single
C. Commonly painless
D. Considered an allergic reaction
E. Rich with treponemes
Answer: D
Explanation:
Primary syphilis causes a chancre:
• Appears at the site of inoculation
• Usually single
• Typically painless
• Highly infectious (organisms present)
• Heals spontaneously in weeks even without treatment
It is an infectious ulcer due to Treponema pallidum, not an allergic reaction.
[A] True: occurs at entry site.
[B] True: commonly single.
[C] True: classically painless.
[D] False: not an allergic reaction.
[E] True: infectious lesion containing organisms.
Home takeaway:
Primary syphilis = painless chancre at inoculation site; infectious, not allergic.

Q5
The best site to take a swab for gonorrhea is:

A. Labia minora
B. Labia majora
C. Anus
D. Endocervical swab
E. Vaginal wall
Answer: D
Explanation:
Gonorrhea targets mucosal columnar epithelium. In females, the cervix (endocervix) is a common infected site, so an endocervical specimen is the best among these options for routine genital diagnosis (rectal sampling is important if rectal exposure/symptoms).
[A] External labial skin is not the main mucosal target.
[B] External labial skin is not the main mucosal target.
[C] Useful only if rectal exposure; not "best" routine genital site.
[D] Best answer: cervix/endocervix is a key site of infection.
[E] Vaginal wall is less optimal because the primary target is the cervix.
Home takeaway:
Female gonorrhea commonly involves the cervix → best routine swab site = endocervical.

Q6
Tx of choice in all stages of syphilis is:

A. Benzathine penicillin
B. Crystalline penicillin
C. Ampicillin
D. Prosthaphyllin
Answer: A
Explanation:
Penicillin is the cornerstone of syphilis treatment. For most stages (primary, secondary, early/late latent), benzathine penicillin G is the standard choice. Crystalline (aqueous) penicillin G is reserved for neurosyphilis/ocular disease protocols rather than the general "all stages" exam answer.
[A] Best "all stages" choice in standard teaching.
[B] Used mainly for neurosyphilis/ocular involvement.
[C] Not standard definitive therapy compared with penicillin G regimens.
[D] Not used for syphilis treatment of choice.
Home takeaway:
Syphilis treatment cornerstone = penicillin; typical exam DOC across stages = benzathine penicillin G.

Q7
Most sensitive & specific test for early dx of syphilis is:

A. FTA
B. TFI
C. HR
D. FTA-ABS
Answer: D
Explanation:
Syphilis serology includes treponemal and non-treponemal tests. In classic exam framing, treponemal tests are considered more specific for confirming infection, and FTA-ABS is a standard highly sensitive/specific treponemal confirmatory test listed here.
[A] Less clearly defined as a standard named test compared with FTA-ABS wording.
[B] Not a commonly used standard test name in routine modern algorithms.
[C] Not a standard syphilis test name in common use.
[D] Best option among listed choices for "sensitive and specific early diagnosis."
Home takeaway:
Among these options, FTA-ABS is the classic "most sensitive/specific" treponemal confirmatory test.

Q8
If dark field examination fails to reveal spirochetes from a penile chancre then Dx may be established alternatively by:

A. Darkfield examination of blood
B. Darkfield examination of blood from aspirate from regionally enlarged LN
C. TIT (treponemal immobilization test)
D. Any of the above
Answer: D
Explanation:
If direct demonstration from a chancre is negative, diagnosis can still be established using alternative approaches:
• Testing other specimen sources when appropriate (e.g., regional lymph node aspirate in some settings)
• Using treponemal-based tests/serology to confirm infection
Because the question lists multiple "alternative" routes, the best choice is "any of the above."
[A] Alternative direct detection approach in historical framing.
[B] Alternative specimen approach when regional nodes are enlarged.
[C] Treponemal-based confirmatory testing concept.
[D] Best overall option given multiple acceptable alternatives.
Home takeaway:
If chancre darkfield is negative, diagnosis can still be made using alternative specimens and/or treponemal testing.

Q9
All about syphilis true except:

A. Incubation period 9–90 days
B. Rx of choice is crystalline penicillin
Answer: B
Explanation:
Syphilis incubation is variable and can fall within a broad range. Treatment is penicillin-based; however, the general "drug of choice" across stages is benzathine penicillin G. Crystalline penicillin G is mainly used for neurosyphilis/ocular disease regimens rather than as the general statement for syphilis overall.
[A] Incubation can be variable and broad.
[B] False as a general "Rx of choice" statement for syphilis overall.
Home takeaway:
Syphilis: incubation varies; standard first-line therapy is penicillin—usually benzathine penicillin G (crystalline for neurosyphilis).

Q10
Associated with mucous patch:

A. 2ry syphilis
B. 1ry syphilis
C. 3ry syphilis
D. Gonorrhea
Answer: A
Explanation:
Secondary syphilis causes widespread mucocutaneous lesions. Mucous patches are classic highly infectious mucosal lesions of secondary syphilis (along with generalized rash and condylomata lata).
[A] Correct: mucous patches are a secondary syphilis feature.
[B] Primary syphilis is classically a chancre at the inoculation site.
[C] Tertiary syphilis is late organ disease, not mucous patches.
[D] Gonorrhea does not cause mucous patches.
Home takeaway:
Mucous patch = secondary syphilis.

Q11
One is false about secondary syphilis manifestation:

A. Auditory neuritis
B. Periostitis
C. Polyhedral asymmetrical rash
D. Painful lymphadenopathy
Answer: D
Explanation:
Secondary syphilis commonly produces systemic features and generalized lymphadenopathy; classically it is non-tender. Skin disease is variable and can be widespread; lesions are often non-itchy. Therefore "painful lymphadenopathy" is the false statement.
[A] Systemic involvement including neurologic/otologic manifestations can occur in syphilis contexts.
[B] Bone involvement such as periostitis can occur.
[C] Rash morphology can be variable and widespread.
[D] False: lymphadenopathy is typically not painful.
Home takeaway:
Secondary syphilis → generalized (usually non-tender) LAD + variable widespread rash.

Q12
Condylomata lata in:

A. Viral warts
B. 1ry syphilis
C. 2ry syphilis
D. 3ry syphilis
Answer: C

Explanation:
Condylomata lata are broad-based, moist, highly infectious lesions seen in secondary syphilis. This differs from condylomata acuminata (HPV genital warts), which are usually papillomatous/cauliflower-like.
[A] Viral warts (HPV) = condylomata acuminata, not lata.
[B] Primary syphilis = chancre.
[C] Correct: condylomata lata are secondary syphilis lesions.
[D] Tertiary syphilis is late organ disease.
Home takeaway:
Condylomata lata = secondary syphilis (broad, moist, very infectious).

Q13
In blood at normal refrigerator temperature (+4c) Treponema pallidum dies within:

A. 24 hrs
B. 72–92 hrs
C. 48 hrs
D. 1 week
Answer: B
Explanation:
Treponema pallidum does not survive long outside the host, but it can persist for a few days in refrigerated blood. The best match among these options is about 3–4 days, so 72–92 hours fits the classic teaching window.
[A] Too short for the "few days" survival concept.
[B] Best fit: a few days at 4°C.
[C] Shorter than typical cited "few days" range.
[D] Too long for classic teaching.
Home takeaway:
T. pallidum survives only a few days in refrigerated blood → ~72–92 hours.

Q14
Moth-eaten alopecia is found in:

A. Secondary syphilis
B. Primary syphilis
C. Tertiary syphilis
Answer: A
Explanation:
Secondary syphilis can cause patchy non-scarring alopecia classically described as "moth-eaten." Primary syphilis is localized (chancre), and tertiary syphilis is late organ disease.
[A] Correct: moth-eaten alopecia is a secondary syphilis feature.
[B] Primary = chancre, not moth-eaten alopecia.
[C] Tertiary = late complications, not the classic moth-eaten alopecia pattern.
Home takeaway:
Moth-eaten alopecia = secondary syphilis (patchy non-scarring hair loss).

Q15
One is false about gonococcus:

A. Caused by G-ve diplococci
B. Female 50% are asymptomatic
C. Require therapeutic low level of penicillin for long time
D. Columnar epithelium is site of predilection
Answer: C
Explanation:
Gonorrhea is caused by Neisseria gonorrhoeae (Gram-negative diplococci). It frequently infects mucosal columnar epithelium (endocervix/urethra). Many women can be asymptomatic. Modern treatment is not "prolonged low-level penicillin"; resistance patterns require specific recommended regimens, and long low-dose penicillin is not correct.
[A] True: Gram-negative diplococci.
[B] True concept: many females are asymptomatic (often around half or more).
[C] False: not treated by prolonged low-level penicillin.
[D] True: targets columnar epithelium.

Home takeaway:
Gonorrhea = Gram-negative diplococcus infecting columnar epithelium; many women asymptomatic; not managed with prolonged low-dose penicillin.

Q16
Majority of gonococcus strains are sensitive to penicillin concentration of:

A. .002 (u/ml blood)
B. 1.1 (u/ml blood)
C. .03 (u/ml blood)
D. .25 (u/ml blood)
Answer: C
Explanation:
This is an older-style susceptibility-threshold question. In classic teaching sets, the commonly cited "majority sensitive" penicillin concentration is around 0.03 u/mL. (Modern practice is complicated by resistance and penicillin is not the routine standard treatment now.)
[A] Too low for classic susceptibility threshold.
[B] Too high.
[C] Best match to classic value used in exam-style questions.
[D] Higher than the classic "most strains" figure.
Home takeaway:
Old exam fact: gonococcus "classically" sensitive around 0.03 u/mL (modern care is resistance-driven).

Q17
Drug of choice in ttt of non-gonococcal urethritis:

A. Septrin
B. Tetracycline
C. Penicillin
D. Spectinomycin
Answer: B
Explanation:
Non-gonococcal urethritis is often due to organisms like Chlamydia. Classic treatment options include tetracycline-class antibiotics. Penicillin is not effective for typical NGU causes, and spectinomycin is historically linked to gonorrhea treatment rather than NGU.
[A] Not standard DOC for NGU.
[B] Correct: tetracycline-class therapy is a classic DOC concept for NGU.
[C] Not appropriate for common NGU causes like Chlamydia.
[D] Not the classic DOC for NGU.
Home takeaway:
NGU (often Chlamydia) → tetracycline-class antibiotic is the classic DOC.

Q18
Which of the following STDs is matched incorrectly with its causative pathogen?

A. Syphilis – Treponema pallidum
B. Gonorrhea – Neisseria
C. Lymphogranuloma venereum – Hemophilus ducreyi
D. Genital warts – HPV
Answer: C
Explanation:
Correct matching:
• Syphilis → Treponema pallidum
• Gonorrhea → Neisseria gonorrhoeae
• Genital warts → HPV (commonly types 6 and 11)
LGV is caused by Chlamydia trachomatis (L1–L3). Haemophilus ducreyi causes chancroid, not LGV.
[A] Correct match.
[B] Correct match.
[C] Incorrect: LGV is Chlamydia; H. ducreyi is chancroid.
[D] Correct match.
Home takeaway:
LGV = Chlamydia trachomatis (L1–L3); H. ducreyi = chancroid.

Q19
All seen in secondary syphilis except:

A. Patchy scarring alopecia
B. Moth eaten alopecia
C. Asymmetric body rash
Answer: A
Explanation:
Secondary syphilis can cause a widespread rash and can cause hair loss. The classic alopecia pattern is "moth-eaten" and is typically non-scarring. Scarring alopecia implies follicular destruction and is not typical of secondary syphilis.
[A] Exception: scarring alopecia is not typical for secondary syphilis.
[B] Typical: moth-eaten non-scarring alopecia.
[C] Rash can be widespread and variable in pattern.
Home takeaway:
Secondary syphilis alopecia is usually moth-eaten and non-scarring; scarring alopecia suggests another cause.

Q20
Which of the following is the causative of Chancroid:

Answer: Haemophilus ducreyi
Explanation:
Chancroid is a sexually transmitted genital ulcer disease caused by Haemophilus ducreyi. It classically produces painful ulcers and tender lymphadenopathy, and it must be distinguished from syphilis, which classically causes a painless chancre.
Home takeaway:
Chancroid = Haemophilus ducreyi (typically painful ulcer); syphilis chancre is typically painless.

Drug Eruption

Q1

A 30-year-old epileptic patient is admitted to hospital with a suspected acute drug eruption. Through systematic history taking and physical examination you suspect toxic epidermal necrolysis. Which of the following would not fit with your diagnosis?

A. Commencing a new anti-epileptic medication 2 months ago
B. A prodrome of fever and malaise
C. Painful skin
D. 40% skin detached
E. The absence of oral erosions
Answer: E

Explanation:
Stevens–Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) are severe, life-threatening mucocutaneous drug reactions. They form a spectrum based on epidermal detachment: SJS (<10% BSA), overlap (10–30%), and TEN (>30%). TEN typically begins with a prodrome (fever, malaise), then painful erythematous skin with blistering and widespread epidermal sloughing. Mucosal involvement is a core feature (oral, ocular, genital), and in TEN it is usually prominent. With 40% detachment, the diagnosis fits TEN by definition, so "absence of oral erosions" is the feature that does not fit.
[A] Anti-epileptics are classic triggers and timing can still fit a severe drug reaction.
[B] Prodromal fever/malaise is common.
[C] Skin pain is a key early clue in SJS/TEN.
[D] 40% detachment = TEN (>30%).
[E] TEN usually has oral/mucosal erosions, so absence is not typical.
Home takeaway: TEN = drug-triggered, very painful skin + >30% detachment + usually marked mucosal erosions.

Q2

The tissue mainly involved in Steven Johnson syndrome is:

A. Skin
B. Lungs
C. Mucocutaneous membrane
D. Urinary bladder
E. Liver
Answer: C

Explanation:
SJS is defined by involvement of both skin and mucous membranes (mucocutaneous disease). While skin detachment occurs, the major morbidity often comes from mucosal erosions (mouth, eyes, genital/urogenital mucosa) leading to dehydration, infection risk, eye complications, and painful oral intake. That's why "mucocutaneous membrane" is the best description of the main tissue involved.
[A] Skin is involved, but SJS is not purely skin-only.
[B] Respiratory mucosa can be involved, but SJS is primarily a mucocutaneous syndrome.
[C] Correct: mucocutaneous involvement is central/defining.
[D] Not the primary "main tissue" framing.
[E] Liver involvement is more typical of other drug reactions (e.g., DRESS), not the defining tissue in SJS.
Home takeaway: SJS/TEN are mucocutaneous emergencies: skin detachment + mucosal erosions (mouth/eyes/genitals).

Q3

Patient with SJS, the best initial step is:

A. Start IV antibiotics
B. Stop offending drug
C. Start 100% O2
D. Give systemic steroids
Answer: B

Explanation:
The most important first action in suspected SJS/TEN is immediate withdrawal of the offending drug. After stopping the trigger, management is urgent supportive care (often ICU/burn-unit style): fluid/electrolyte balance, temperature control, wound/skin care, pain control, nutritional support, infection surveillance, and early ophthalmology involvement to prevent vision-threatening complications. Adjunctive systemic therapies (e.g., steroids/IVIG/ciclosporin depending on local protocol) may be considered, but they come after the immediate step of stopping the culprit medication and stabilizing the patient.
[A] Antibiotics are not a routine first step unless infection is suspected/confirmed.
[B] Correct: stop the offending drug immediately.
[C] Oxygen depends on clinical status; not the universal "best initial step."
[D] Steroids may be used in some protocols but never before stopping the drug.
Home takeaway: In SJS/TEN: first = stop culprit drug, then ICU-level supportive care + early eye/mucosal management.

Q4
Acne medicinosa by all of following except:

A. Phenytoin
B. B12
C. Azelaic acid
D. Steroid
Answer: C

Explanation:
Acne medicamentosa (drug-induced/acneiform eruption or acne exacerbation) can occur with several medications. Anti-epileptics can worsen acneiform eruptions, systemic corticosteroids are classic triggers, and supplements like vitamin B12 are well-known to provoke acne flares in some patients. Azelaic acid is not a trigger—it's a topical agent used to treat acne and rosacea (anti-inflammatory, comedolytic and helpful for post-inflammatory hyperpigmentation). Therefore azelaic acid is the exception.
[A] Phenytoin (anti-epileptic) can be associated with acneiform eruptions.
[B] Vitamin B12 can trigger acne flares in some people.
[C] Correct exception: azelaic acid is used to treat acne, not cause it.
[D] Steroids commonly cause acneiform eruptions ("steroid acne").
Home takeaway: Drug acne triggers include steroids, some anti-epileptics, and B12; azelaic acid is a treatment.

Q5
Wrong about TEN:

A. Most common cause is infection
B. Requires intensive care most of the time
C. Highly fatal
Answer: A

Explanation:
TEN is most commonly drug-induced, with classic triggers including anticonvulsants, NSAIDs, allopurinol, and certain antibiotics (especially sulfonamides). It is a medical emergency requiring intensive supportive care, often in ICU or burn unit settings due to massive skin failure and risk of fluid loss, electrolyte disturbances, hypothermia/hyperthermia, and sepsis. Mortality is high and is often estimated using severity scoring systems (e.g., SCORTEN). Therefore, infection being the "most common cause" is the wrong statement in this context.
[A] Wrong: drugs are the main common cause.
[B] True: usually needs ICU/burn-unit care.
[C] True: high mortality risk.
Home takeaway: TEN is usually drug-triggered, needs ICU/burn support, and has high mortality.

Q6
Wrong:
Answer: Steven Johnson Syndrome involves oral mucosa and skin of more than 30% of body surface area

Explanation:
SJS and TEN are classified by the extent of epidermal detachment: SJS involves <10% BSA, overlap is 10–30%, and TEN is >30%. Both conditions can involve oral and other mucosal surfaces, but once detachment exceeds 30% the correct diagnosis is TEN, not SJS. So the statement is wrong because >30% BSA detachment defines TEN.
Home takeaway: SJS <10%, overlap 10–30%, TEN >30%—mucosal erosions can occur across the spectrum.

Skin Tumors

Q1
E in ABCDE of melanoma stands for:

A. Enlargement
B. Edema
C. Evolution
D. Erythema
E. Elevation
Answer: C

Explanation:
ABCDE is a clinical screening tool to assess malignant potential of a mole: Asymmetry, Border irregularity, Color variation, Diameter (classically >0.5 cm), and Evolving. "Evolving" means any change over time in size, shape, color, symptoms (pain/itch), or surface changes (crusting, ulceration, bleeding/discharge). It's often the most important warning sign because a changing lesion is more suspicious than a stable one.
[A] Enlargement can be part of evolution but ABCDE uses the broader concept "Evolving."
[B] Edema is not part of ABCDE.
[C] Correct: E = Evolving.
[D] Erythema may occur around lesions but not the ABCDE "E."
[E] Elevation is not the standard "E" in ABCDE.
Home takeaway: ABCDE → E = Evolution (any change over time = red flag).

Q2
One of the following is a premalignant condition:

A. Basal cell carcinoma
B. Actinic keratosis
C. Squamous cell carcinoma
D. Melanoma
E. Seborrheic keratosis
Answer: B

Explanation:
Premalignant lesions show atypical proliferation with risk of progression to malignancy. Actinic keratosis (AK) is a classic premalignant lesion caused by chronic sun exposure, usually in fair/sun-damaged skin and older age. It presents on sun-exposed sites (face including lip, dorsal hands, distal limbs, bald scalp) as rough scaly/crusty patches/macules/papules/plaques, often <1 cm, with irregular edges and surrounding erythema. AK can progress to invasive squamous cell carcinoma if untreated, so it's treated with cryotherapy, topical therapies (5-FU, imiquimod, diclofenac), photodynamic therapy, or excision/curettage; non-responders need biopsy to rule out invasion.
[A] BCC is malignant (though usually low metastatic risk), not premalignant.
[B] Correct: AK is premalignant.
[C] SCC is malignant.
[D] Melanoma is malignant.
[E] Seborrheic keratosis is benign "stuck-on" keratinocyte tumor.
Home takeaway: Actinic keratosis = sun-induced premalignant lesion → may progress to SCC.

Q3
Wrong about nevus:

A. Result from abnormal proliferation of melanocytes
B. Developmental disorder
C. Common in infants
D. Increase after ACTH injection
E. Flair up during adolescence
Answer: C

Explanation:
Melanocytic nevi ("moles") are benign proliferations of melanocytes. They may be congenital (present at birth) or acquired (appear later). Acquired melanocytic nevi are common and can increase during childhood/adolescence, and pigmentation can darken with hormonal influences. Types include: junctional nevi (flat well-demarcated brown macule at the dermo-epidermal junction), compound nevi (raised pigmented lesion), and intradermal nevi (elevated often less pigmented papule). Congenital melanocytic nevi can be small/medium/giant; giant congenital nevi carry a higher (but still overall low) risk of malignant change. Saying nevi are "common in infants" is wrong in the sense that *acquired* nevi are not typical in infancy; infancy is more about congenital lesions.
[A] True: abnormal proliferation of melanocytes.
[B] Many nevi are developmental/hamartomatous.
[C] Correct wrong statement: common acquired nevi are not "infant" lesions (infancy mostly congenital lesions).
[D] ACTH and endocrine influences can increase pigmentation.
[E] Adolescence can increase number/darken nevi.
Home takeaway: Nevi = benign melanocytic proliferations; acquired nevi rise in childhood/adolescence, congenital nevi present at birth.

Q4
The most frequent site for mets of BCC:

A. Skin
B. Regional LN
C. Lung
D. Brain
E. Liver
Answer: B

Explanation:
Basal cell carcinoma (BCC) is the most common human cancer, typically on sun-exposed "mask area" of the face, usually painless, and classically appears as a shiny translucent pearly nodule or a rolled-edge ulcer. Metastasis is rare, but when it happens it most commonly spreads first to regional lymph nodes, then to lung and bone. This is why nodal assessment becomes relevant in suspected metastatic or long-standing aggressive recurrent BCC.
[A] Local invasion to nearby skin occurs, but metastasis classically goes to nodes first.
[B] Correct: most common metastatic site is regional lymph nodes.
[C] Lung is common but typically after nodes.
[D] Brain metastasis is not typical as the most frequent site.
[E] Liver is not a common first site.
Home takeaway: BCC rarely metastasizes; if it does → regional lymph nodes most common, then lung/bone.

Q5
Percentages of malignant melanomas thought to arise from pigmented nevi:

A. 0–5%
B. 10–20%
C. 20–40%
D. 40–60%
E. 60–80%
Answer: C

Explanation:
Many melanomas arise de novo, but a significant minority arise from pre-existing nevi. Broadly, observational data and meta-analyses suggest roughly around one-quarter to one-third of melanomas are nevus-associated, which fits best with 20–40%. Clinically, this can present as a changing focus within a previously stable mole (new color, new nodule, irregularity, ulceration/bleeding). This is why "Evolving" in ABCDE is so important. ([Journal of Investigative Dermatology][2])
[A] Too low.
[B] Possibly for some subsets, but overall estimate is higher.
[C] Best match (around ~20–40% range). ([Journal of Investigative Dermatology][2])
[D] Too high for most pooled estimates.
[E] Too high.
Home takeaway: Most melanomas arise de novo, but ~¼–⅓ can be nevus-associated → any changing mole is suspicious.

Q6
Which tumor most frequently metastasizes to skin:

A. Pulmonary CA
B. Renal CA
C. Prostate CA
D. Breast CA
E. Gastric CA
Answer: D

Explanation:
Cutaneous metastases most commonly come from breast carcinoma overall (especially in women), often presenting as firm dermal/subcutaneous papules or nodules, sometimes violaceous, and may appear near the primary site (e.g., chest wall). Other internal malignancies can metastasize to skin (lung, colon, melanoma, renal), but breast is classically the most frequent source.
[A] Lung commonly metastasizes but not the most frequent overall.
[B] Renal can metastasize to skin but less commonly than breast.
[C] Prostate skin metastasis is uncommon.
[D] Correct: breast carcinoma is the most frequent source overall.
[E] Gastric is less common.
Home takeaway: Most common internal source of cutaneous metastases = breast carcinoma (classically firm dermal nodules).

Q7
Asymmetrical lesion with ill-defined border and variable shades of colors (diameter not mentioned), diagnosis:

A. Superficial spreading
B. Lentigo maligna
C. Nodular
D. Acral
E. Amelanotic
Answer: A

Explanation:
Superficial spreading melanoma is the most common melanoma type. It typically shows asymmetry, irregular/ill-defined borders, and color variegation (brown to black, sometimes mixed tones) and often develops on trunk in men and legs in women. It has a radial growth phase first, so it may appear as an irregular patch/plaque before a nodule develops (nodules worsen prognosis).
[A] Fits: asymmetric, irregular border, variable colors = classic superficial spreading.
[B] Lentigo maligna melanoma is usually on sun-damaged face in elderly and starts as lentigo maligna (irregular enlarging pigmented macule).
[C] Nodular melanoma is a dark nodule from the start with vertical growth.
[D] Acral melanoma is palms/soles/subungual with features like Hutchinson sign.
[E] Amelanotic melanoma is non-pigmented (pink/flesh) nodules.
Home takeaway: Superficial spreading melanoma = most common + irregular border + color variegation + asymmetry.

Q8
Which one of the following is an eccrine sweat gland tumor:

A. Trichoepithelioma
B. Syringoma
C. Pilomatrixoma
D. Trichofolliculoma
Answer: B

Explanation:
Syringomas are benign adnexal tumors of eccrine glands. They are usually multiple, small, slow-growing, flesh-colored papules, commonly around the eyelids/face and often appear around puberty; may also occur on trunk/groin. Management is usually cosmetic (shave removal/cautery). Trichoepithelioma and trichofolliculoma are hair-follicle origin tumors; pilomatrixoma arises from hair matrix cells.

[A] Hair follicle origin, not eccrine.
[B] Correct: syringoma = eccrine tumor.
[C] Hair matrix tumor.
[D] Hair follicle tumor.
Home takeaway: Syringoma = benign eccrine tumor → multiple small flesh papules (often eyelids).

Q9
Tumor may show malignant degeneration:

A. Compound nevus
B. Junctional nevus
C. Dermal nevus
D. Epidermal nevus
Answer: B

Explanation:
Melanocytic nevi are classified by where melanocytes proliferate. Junctional nevi are at the dermo-epidermal junction and appear as flat, well-demarcated brown macules. Because the junctional area is where atypia and malignant transformation risk is more concerning, junctional nevi are classically considered more likely than purely intradermal nevi to show malignant change (though overall transformation of common nevi is rare). Congenital giant nevi and dysplastic nevi are higher-risk markers/precursors.
[A] Compound nevi can change but "junctional" is the classic answer for malignant potential among these.
[B] Correct: junctional nevus has the classic association with malignant potential.
[C] Dermal/intradermal nevi are generally benign, raised, often less pigmented.
[D] Epidermal nevus is a keratinocytic hamartoma; malignancy is not the typical teaching point here.
Home takeaway: Junctional nevi (at DE junction) are the classic nevus type linked with higher malignant potential vs intradermal.

Q10
Commonest site of squamous cell epithelioma:

A. Upper lip
B. Lower lip
C. Face
D. Hands
Answer: C

Explanation:
Squamous cell carcinoma (SCC) is the 2nd most common skin cancer. It develops on sun-exposed areas and can arise from premalignant lesions like actinic keratosis or Bowen's disease, or in chronic wounds/scars (Marjolin ulcer). Distribution is mostly head and neck (about 70%), which makes "face" the best general answer. Clinically it may be rapidly growing, painful, hyperkeratotic, and can present as nodular/crusting/ulceration (everted ulcer) or cutaneous horn.
[A] Lip is sun-exposed and important, but "face/head-neck" is broader and more common.
[B] Lower lip is a notable SCC site, but still within head/neck; "face" is most general/common.
[C] Best answer: head/neck (face) is the most common region.
[D] Dorsal hands are common sun-exposed sites but not as common as head/neck overall.
Home takeaway: SCC = mainly sun-exposed head/neck (face) + may arise from AK/Bowen or chronic scars.

Q11
Which of the following is premalignant:

A. Lentigo maligna
B. Bowen's disease
C. Erythroplasia of Queyrat
D. Actinic keratosis
Answer: D

Explanation:
Actinic keratosis is the classic premalignant lesion with risk of progression to SCC (noted risk if untreated). Bowen's disease and erythroplasia of Queyrat are SCC in situ (already malignant but non-invasive), while lentigo maligna is melanoma in situ (premalignant/precursor stage for lentigo maligna melanoma). In single-best-answer style, the lecture's labeled premalignant lesion is actinic keratosis.
[A] Melanoma in situ precursor, but not the classic "premalignant" category in this lecture framing.
[B] SCC in situ.
[C] SCC in situ on glans/prepuce.
[D] Correct: actinic keratosis = premalignant lesion.
Home takeaway: Actinic keratosis = premalignant → may progress to invasive SCC.

Q12
A lady presents with hyperpigmented lesion on her face that has been increasing in size, most appropriate next step is:

A. Dermoscopic examination
B. Incisional biopsy
C. Excisional biopsy
D. Chemical peeling
Answer: C

Explanation:
A changing pigmented facial lesion raises concern for melanoma (especially lentigo maligna spectrum on sun-damaged face). The appropriate next step is to obtain histologic diagnosis. For suspected melanoma, complete excisional biopsy with narrow margin is preferred to assess architecture and allow measurement of Breslow thickness if melanoma is present. Dermoscopy is helpful as a tool, but it does not replace biopsy when the lesion is growing/changing. Chemical peeling is inappropriate because it can delay diagnosis and destroy diagnostic tissue.
[A] Dermoscopy helps assessment but does not definitively rule out melanoma in a changing lesion.
[B] Incisional biopsy may miss the most diagnostic area; used only when excision is impractical.
[C] Best: excisional biopsy for a suspicious pigmented lesion.
[D] Not appropriate in a suspicious enlarging pigmented lesion.
Home takeaway: Any evolving pigmented lesion → biopsy; suspected melanoma → prefer excisional biopsy for accurate Breslow.

Q13
Old male with pearly lesion on face with central ulceration, dx:

A. SCC
B. BCC
C. Actinic keratosis
Answer: B

Explanation:
BCC commonly occurs on sun-exposed "mask area" of the face and classically presents as a shiny translucent pearly nodule, sometimes with telangiectasia, and may ulcerate forming a rolled-edge ulcer ("rodent ulcer" with central necrosis). It is usually painless. SCC, in contrast, tends to be painful, rapidly growing, hyperkeratotic/crusted, and can form an everted ulcer. Actinic keratosis is a rough scaly patch rather than a pearly nodulo-ulcerative lesion.
[A] SCC is more hyperkeratotic/painful with everted ulcer.
[B] Correct: pearly + central ulceration + face = noduloulcerative BCC.
[C] AK is rough scaly patch/plaque.
Home takeaway: Pearly translucent nodule with rolled-edge ulcer on sun-exposed face = BCC (noduloulcerative).

Q14
One of the following genetic conditions causes freckling, café-au-lait spots, Lisch nodules:

A. Tuberous sclerosis
B. McCune Albright syndrome
C. NF1
D. NF2
Answer: C

Explanation:
Neurofibromatosis type 1 (NF1) classically presents with café-au-lait macules, axillary/inguinal freckling, neurofibromas, and Lisch nodules (iris hamartomas). NF2 is more associated with bilateral vestibular schwannomas and fewer skin findings. Tuberous sclerosis has ash-leaf spots, angiofibromas, shagreen patches. McCune-Albright has café-au-lait plus endocrine/bone abnormalities but not Lisch nodules as a key feature.
[A] Tuberous sclerosis has different hallmark lesions.
[B] McCune-Albright has café-au-lait but not Lisch nodules as classic triad.
[C] Correct: NF1.
[D] NF2 differs (schwannomas, less café-au-lait/freckling pattern).
Home takeaway: Café-au-lait + freckling + Lisch nodules = NF1.

Q15
False about actinic keratosis:

A. It is a malignant condition
B. Mostly affecting fair skinned people
C. Mostly on sun exposed areas
Answer: A

Explanation:
Actinic keratosis is premalignant, not frankly malignant (though it can progress to invasive SCC). It is common in fair, sun-damaged skin and increases with age. It occurs on sun-exposed sites (face including lip, dorsal hands, distal limbs, bald scalp) and presents as rough/scaly/crusty patches/macules/papules/plaques with irregular edges and often surrounding erythema. Management includes cryotherapy, topical 5-FU/imiquimod/diclofenac, photodynamic therapy, or excision/curettage; biopsy is needed if it fails to respond to rule out invasive SCC.
[A] False: premalignant, not malignant by itself.
[B] True.
[C] True.
Home takeaway: Actinic keratosis = premalignant sun-damage lesion → treat and biopsy non-responders.

Q16
Breslow thickness:

A. From granular layer to deepest point of invasion
B. From dermis to deepest point of invasion
C. Thickness in lymph nodes
Answer: A

Explanation:
Melanoma prognosis depends strongly on depth of invasion. Breslow thickness measures the vertical thickness of melanoma from the top of the epidermis (classically from the granular layer; or from base of ulcer if ulcerated) down to the deepest malignant cell. It guides management decisions such as wide excision margins and whether sentinel lymph node biopsy is indicated (commonly considered when thickness >1 mm).
[A] Correct definition.
[B] Incorrect start point.
[C] Not what Breslow measures.
Home takeaway: Breslow = vertical tumor thickness (granular layer → deepest invasion) and is a key prognostic factor.

Q17
Benign tumor of the epidermis:

A. Actinic keratosis
B. Seborrheic keratosis
Answer: B

Explanation:
Seborrheic keratosis is a benign proliferation of immature keratinocytes. It increases with aging and commonly occurs on trunk, face, and neck. Clinically it is sharply demarcated, warty/papillomatous, raised, greasy/waxy with a classic "stuck-on" appearance. It can be treated for symptoms/cosmesis with cryotherapy, laser, or excision. Actinic keratosis is premalignant.

[A] Premalignant, not benign.
[B] Correct: seborrheic keratosis is benign epidermal tumor.
Home takeaway: Seborrheic keratosis = benign "stuck-on" waxy warty lesion.

Q18
All about BCC is true except:

A. More common in Caucasian
B. Always associated with bad prognosis
Answer: B

Explanation:
BCC is very common, especially in fair-skinned individuals with significant UV exposure. It usually grows slowly and is locally invasive but has a very low metastatic rate, so prognosis is generally excellent when treated appropriately. Management depends on histology and site: biopsy is often needed before treatment; for facial/morpheaform/sclerosing BCC, Mohs surgery is the gold standard; other options include excision, curettage/cautery, radiotherapy, cryotherapy, imiquimod, and photodynamic therapy for superficial BCC.
[A] True: more common in fair-skinned/Caucasian populations.
[B] False: prognosis is usually good; not "always bad."
Home takeaway: BCC = common in fair skin + low metastasis → prognosis usually excellent with proper treatment.

Q19
Nodule on nose, glossy, with telangiectasia, diagnosis:

A. BCC
B. SCC
Answer: A

Explanation:
Nodular BCC commonly presents on sun-exposed head and neck as a shiny/glossy translucent pearly papule or nodule with surface telangiectasia, and may ulcerate (rolled edge). SCC is more often hyperkeratotic, crusted, painful, and rapidly growing with possible everted ulcer.
[A] Correct: classic nodular BCC appearance.
[B] SCC typically not "pearly glossy with telangiectasia" as the main description.
Home takeaway: Pearly glossy nodule + telangiectasia on nose/face = nodular BCC.

Q20
Wrong about SCC:

A. Lower growth than BCC
B. Caused by exposure to sun
Answer: A

Explanation:
SCC is generally more aggressive than BCC: it can grow faster, is more likely to metastasize, and often presents as rapidly enlarging, painful, hyperkeratotic lesions with crusting/ulceration or cutaneous horn. UV exposure is a major risk factor (similar to BCC), and SCC can arise from actinic keratosis or Bowen's disease, as well as in chronic scars/wounds (Marjolin ulcer). Therefore, saying SCC has "lower growth" than BCC is wrong.
[A] Wrong: SCC tends to be more aggressive than BCC.
[B] True: sun exposure is a key cause/risk factor.
Home takeaway: SCC is more aggressive than BCC and strongly linked to UV + premalignant precursors (AK/Bowen).

Q21
BCC is:
Answer: Pearly and translucent nodule

Explanation:
BCC classically appears on sun-exposed face as a shiny, translucent pearly nodule, sometimes pigmented, often with telangiectasia, and may break down to form a rolled-edge ulcer (rodent ulcer/central necrosis). It is usually painless and locally destructive. Diagnosis is confirmed with biopsy to guide treatment; Mohs is favored for face and morpheaform/sclerosing types.
Home takeaway: BCC = pearly translucent nodule ± telangiectasia ± rolled-edge ulcer (face).

Q22
70-year-old male, fair skinned, presented with fine scaled erythematous plaques on the back of his hands and bald scalp, diagnosis:
Answer: Actinic keratosis

Explanation:
Actinic keratosis occurs on chronically sun-exposed areas, especially in fair-skinned older patients. Lesions are rough, scaly, or crusty macules/papules/plaques (often <1 cm), with irregular edges and sometimes surrounding erythema. Common sites include face (including lip), dorsal hands, distal limbs, and bald scalp. It is premalignant with risk of progression to SCC, so treat with cryotherapy or field therapies (5-FU, imiquimod, diclofenac, PDT) and biopsy if resistant/no response.
Home takeaway: Rough scaly erythematous lesion on sun-exposed hands/bald scalp in elderly fair skin = actinic keratosis (premalignant).

Q23
Most common BCC type:
Answer: Noduloulcerative

Explanation:
BCC subtypes include nodular (most common), superficial (erythematous patch on trunk), pigmented, and morpheaform/sclerosing (scar-like with ill-defined borders). The nodular/noduloulcerative type presents as a small pearly papule/nodule that can ulcerate with rolled edges and telangiectasia—common on sun-exposed head and neck.
Home takeaway: Nodular/noduloulcerative BCC is the most common subtype → pearly papule → rolled-edge ulcer.

Q24
Wrong about squamous cell carcinoma:
Answer: 75% of lesions are on extremities

Explanation:
SCC distribution is mainly head and neck (about 70%) and other sun-exposed areas such as dorsal hands, scalp, lip, and the superior surface of the pinna. Extremities can be involved (especially sun-exposed dorsal hands/forearms), but stating that 75% are on extremities contradicts the typical distribution pattern where head/neck predominate.
Home takeaway: SCC most commonly occurs on sun-exposed head/neck (majority), not "mostly extremities."

Q25
Melanoma with early metastasis:
Answer: Nodular melanoma

Explanation:
Nodular melanoma is characterized by vertical growth from the start, often presenting as a dark nodule (sometimes ulcerated/bleeding) rather than a long radial growth phase. Because vertical growth correlates with deeper invasion early, it carries a worse prognosis and tends to metastasize earlier compared with superficial spreading melanoma.
Home takeaway: Nodular melanoma = early vertical growth → deeper invasion early → worse prognosis/earlier metastasis.

Q26
Worst type of malignant melanoma:
Answer: Nodular melanoma

Explanation:
Among melanoma types, nodular melanoma is considered the most aggressive because it grows vertically early, leading to greater Breslow thickness at diagnosis and higher risk of lymphatic/hematogenous spread. Prognosis overall depends most on depth (Breslow), ulceration, and nodal involvement, but nodular subtype often presents with unfavorable depth early.
Home takeaway: Nodular melanoma is the most aggressive subtype because it starts with vertical growth.

Q27
Skin melanoma all of these are evidence-based prognostic factors except:
Answer: Gender

Explanation:
Key prognostic factors emphasized for primary cutaneous melanoma include Breslow thickness (depth of invasion), ulceration status, and lymph node involvement (especially sentinel lymph node status). These directly reflect tumor burden and biologic aggressiveness and guide staging and treatment decisions (wide excision margins, SLNB when thickness is significant, and adjuvant options for advanced disease). Gender can correlate with outcomes in some datasets, but it is not among the core staging/prognostic determinants compared to Breslow/ulceration/nodes. ([PMC][3])
Home takeaway: Melanoma prognosis is driven mainly by Breslow thickness + ulceration + nodal status (SLN).

Q28
Woman with unilateral eczematous areolar rash, next step:
Answer: Do skin biopsy

Explanation:
Paget's disease of the nipple presents with unilateral, persistent, non-specific erythematous/eczematous changes of the areola/nipple that can spread to surrounding skin. It is caused by an underlying adenocarcinoma of the breast ducts. Because it can mimic eczema, any unilateral eczematous breast lesion that fails to respond to simple treatment should be biopsied to confirm diagnosis and identify the underlying malignancy.
Home takeaway: Unilateral persistent "eczema" of nipple/areola = think Paget → biopsy to rule out underlying ductal carcinoma.

Hair & Nail Disorders

Q1
All are causes of traumatic alopecia except:

A. Traction
B. Pressure
C. Marginal
D. Trichotillomania
E. Alopecia areata
Answer: E

Explanation:
Traumatic alopecia is hair loss caused by mechanical/physical forces that damage hair shafts or follicles. It includes traction alopecia (tight hairstyles), pressure/friction alopecia (prolonged pressure), marginal alopecia (hairline loss often from traction practices), and trichotillomania (compulsive hair pulling causing broken hairs of different lengths and irregular patches). Alopecia areata is not traumatic; it is an autoimmune non-scarring alopecia that presents with smooth, well-defined patches and can show "exclamation mark" hairs.
[A] Traction = mechanical pulling → traumatic alopecia.
[B] Pressure/friction can cause traumatic hair loss.
[C] Marginal alopecia is commonly traction-related at the hairline.
[D] Trichotillomania is self-induced pulling trauma.
[E] Autoimmune, not mechanical → does not belong to traumatic causes.
Home takeaway: Traumatic alopecia = traction/pressure/pulling; alopecia areata = autoimmune non-traumatic.

Q2
Telogen effluvium, what's wrong:

A. Can be caused by drugs
B. Wood's lamp helps in diagnosis
C. Can happen few months after childbirth
D. Presents as diffuse thinning of hair
E. Non-scarring alopecia
Answer: B

Explanation:
Telogen effluvium is a diffuse, non-scarring shedding disorder where many follicles shift prematurely into telogen after a trigger, leading to noticeable shedding and thinning. It commonly appears weeks to a few months after triggers such as childbirth, major illness/fever, surgery, stress, nutritional issues, and certain drugs (e.g., anticoagulants, retinoids, beta-blockers). Wood's lamp is not used to diagnose telogen effluvium; it is mainly helpful for certain infections or pigment-related assessments, not diffuse shedding disorders.
[A] Drugs can trigger telogen effluvium.
[B] Wood's lamp is not a diagnostic tool for telogen effluvium.
[C] Post-partum shedding is classic.
[D] Diffuse thinning/shedding is typical.
[E] Follicles are not destroyed → non-scarring.
Home takeaway: Telogen effluvium = diffuse non-scarring shedding after triggers; Wood's lamp isn't part of its diagnosis.

Q3
Not in telogen effluvium:

A. Post-partum
B. Post-surgical
C. Crash diet
D. Post-febrile
E. Cytotoxic drugs
Answer: E

Explanation:
Telogen effluvium follows triggers that push hairs from anagen into telogen, causing diffuse shedding later (often weeks–months). Post-partum, post-surgical stress, crash dieting/rapid weight loss, and febrile illness are classic triggers. Cytotoxic drugs instead cause anagen effluvium by abruptly stopping mitosis in the hair matrix during the growth phase, leading to rapid, often profound diffuse hair loss.
[A] Post-partum is a classic telogen trigger.
[B] Surgery/stress can trigger telogen effluvium.
[C] Crash diet/rapid weight loss triggers telogen effluvium.
[D] Fever/illness triggers telogen effluvium.
[E] Cytotoxic drugs → anagen effluvium, not telogen effluvium.
Home takeaway: Telogen effluvium = delayed diffuse shedding after stressors; cytotoxic drugs cause anagen effluvium.

Q4
Which of the following doesn't cause diffuse non-scarring alopecia:

A. Trichotillomania
B. Heparin
C. Surgery shock
D. Telogen effluvium
E. Cytotoxic drugs
Answer: A

Explanation:
Diffuse non-scarring alopecia describes generalized thinning/shedding without permanent follicle destruction. Telogen effluvium (including after surgery shock) is a major cause and is diffuse. Drugs such as heparin can provoke diffuse shedding. Cytotoxic drugs cause anagen effluvium, which is typically diffuse and non-scarring. Trichotillomania usually causes patchy, irregular hair loss with broken hairs of varying length rather than a true diffuse thinning pattern.
[A] Usually patchy/irregular with broken hairs → not typical diffuse alopecia.
[B] Drug-triggered diffuse shedding can occur.
[C] Surgery shock can trigger telogen effluvium → diffuse shedding.
[D] Classic diffuse non-scarring alopecia.
[E] Cytotoxic drugs → diffuse anagen effluvium.
Home takeaway: Diffuse non-scarring alopecia = telogen/anagen effluvium patterns; trichotillomania is usually patchy.

Q5
All are causes of diffuse non-scarring alopecia except:

A. Telogen effluvium
B. Anagen effluvium
C. Hypothyroidism
D. Hair shaft abnormalities
E. Male pattern hair loss
Answer: E

Explanation:
Diffuse non-scarring alopecia includes conditions that cause generalized shedding/thinning without follicle destruction: telogen effluvium, anagen effluvium, endocrine disorders like hypothyroidism, and hair shaft abnormalities (fragile hair breaks, giving a diffuse thinning appearance). Male pattern hair loss (androgenetic alopecia) is non-scarring but classically patterned (frontotemporal recession/vertex) rather than a diffuse shedding disorder, so it is the exception.
[A] Diffuse shedding disorder.
[B] Diffuse loss due to growth-phase interruption.
[C] Can cause generalized thinning.
[D] Breakage/fragility can look diffuse.
[E] Typically patterned, not diffuse shedding.
Home takeaway: Diffuse non-scarring alopecia is usually shedding/systemic; androgenetic alopecia is non-scarring but patterned.

Q6
Which is wrong about hair:

A. Male hair grows faster
B. Growth rate = 1 cm/month
C. Hair spends growing 3–4 years before falling
D. All hair characteristics are genetically determined
Answer: A

Explanation:
Scalp hair has a long anagen (growth) phase lasting years (often taught as ~3–4 years or longer), then a short catagen, then telogen (rest). Average growth is about 1 cm per month. Genetics strongly influence many hair characteristics (density, caliber, curl, color, and growth tendencies), although environment/hormones/nutrition can modify them. The statement "male hair grows faster" is not a standard general rule and is the incorrect option here.
[A] Not a consistent/general rule.
[B] Typical average growth rate.
[C] Reflects long anagen phase.
[D] Genetics is a major determinant of many hair traits.
Home takeaway: Scalp hair grows ~1 cm/month and stays in anagen for years; "male hair grows faster" isn't a reliable general statement.

Q7
% of hair follicles in scalp present in anagen phase is:

A. 50%
B. 60%
C. 70%
D. 85%
Answer: D

Explanation:
Most scalp follicles are normally in anagen (active growth), which allows continuous hair coverage. A smaller fraction is in telogen (resting/shedding), and catagen is brief and minimal. The best approximate percentage for anagen follicles on the scalp is about 85–90%.
[A] Too low for normal scalp.
[B] Too low.
[C] Too low.
[D] Matches normal physiology (majority in anagen).
Home takeaway: Normal scalp = mostly anagen (~85–90%), smaller telogen fraction, catagen minimal.

Q8
Wrong about alopecia areata:

A. Fluorescent on wood's lamp
B. Causes non-scarring alopecia
C. Can occur in children
D. Recurring in nature
Answer: A

Explanation:
Alopecia areata is an autoimmune, non-scarring alopecia presenting with smooth, round/oval patches of hair loss. It can occur at any age including children and often has a relapsing course. Typical clues include exclamation-mark hairs and sometimes nail pitting. Wood's lamp fluorescence is not a feature of alopecia areata; Wood's lamp is more relevant to certain fungal infections or pigment assessments.
[A] Does not fluoresce → wrong.
[B] Follicles are preserved → non-scarring.
[C] Common in children as well.
[D] Often recurrent.
Home takeaway: Alopecia areata = autoimmune patchy non-scarring alopecia; Wood's lamp fluorescence is not part of it.

Q9
Which of the following results in anagen effluvium:
Answer: Cytotoxic drugs

Explanation:
Anagen effluvium occurs when actively growing hair follicles suddenly stop producing hair due to impaired mitosis of the hair matrix—classically from cytotoxic chemotherapy. It causes rapid, diffuse hair loss. This differs from telogen effluvium, which is delayed shedding occurring weeks–months after a trigger.
Home takeaway: Cytotoxic drugs → anagen effluvium (rapid diffuse loss); stress/illness/post-partum → telogen effluvium (delayed shedding).

Q10
All may cause non-cicatricial alopecia except:

A. Surgical shock
B. Morphea
C. Heparin
D. Protein malnutrition
Answer: B

Explanation:
Non-cicatricial (non-scarring) alopecia is reversible and occurs without permanent follicle destruction. Surgical shock/major surgery can trigger telogen effluvium. Heparin can trigger diffuse shedding. Protein malnutrition can cause diffuse thinning/shedding. Morphea (localized scleroderma) can damage follicles when it involves the scalp because of dermal sclerosis/atrophy, leading to scarring alopecia and permanent loss—so it is the exception.
[A] Can trigger telogen effluvium → non-scarring.
[B] Can cause scarring alopecia when scalp is affected → exception.
[C] Can trigger drug-related shedding → non-scarring.
[D] Nutritional deficiency can cause diffuse non-scarring loss.
Home takeaway: Non-scarring alopecia is reversible shedding; morphea on scalp can scar and cause permanent loss.

Q11
All of the following cause diffuse non-scarring alopecia except:

A. Heparin
B. 2ry syphilis
C. Anhidrotic ectodermal dysplasia
D. Cachexia
Answer: C

Explanation:
Diffuse non-scarring alopecia can occur due to medications (e.g., heparin), systemic infections including secondary syphilis (can cause characteristic thinning/"moth-eaten" patterns), and severe systemic illness/weight loss such as cachexia. Anhidrotic ectodermal dysplasia is a congenital developmental disorder causing sparse/abnormal hair (hypotrichosis) rather than an acquired diffuse shedding disorder.
[A] Drug-triggered shedding can be diffuse and non-scarring.
[B] Secondary syphilis can cause non-scarring alopecia.
[C] Congenital hair development problem, not classic acquired diffuse shedding → exception.
[D] Severe illness/weight loss can cause diffuse shedding.
Home takeaway: Diffuse non-scarring alopecia is often systemic/drug-related; ectodermal dysplasia is congenital hair abnormality, not typical acquired shedding.

Q12
Non-scarring alopecia all except:

A. Male pattern baldness
B. Alopecia areata
C. 2ry syphilis
D. Sarcoidosis
Answer: D

Explanation:
Male pattern baldness (androgenetic alopecia) is non-scarring. Alopecia areata is non-scarring. Secondary syphilis alopecia is generally non-scarring. Sarcoidosis can involve the scalp with granulomatous inflammation and may destroy follicles, producing scarring alopecia—so it is the exception.
[A] Non-scarring patterned loss.
[B] Non-scarring autoimmune patches.
[C] Non-scarring alopecia can occur.
[D] Can be scarring when scalp is involved → exception.
Home takeaway: Many common alopecias are non-scarring; scalp sarcoidosis can scar and cause permanent loss.

Q13

Which of the following is the resting stage of hair:

Answer: Telogen

Explanation:
The hair cycle has three main phases: anagen (growth), catagen (transition), and telogen (resting). Telogen is the resting/shedding-ready stage before follicles re-enter anagen.
Home takeaway: Telogen = resting phase; anagen = growth phase; most scalp hairs are normally in anagen.

تم بحمد الله

إن أصبنا فمن الله وإن أخطأنا فمن أنفسنا

بالتوفيق جميعًا، لا تنسونا من صالح دعائكم