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Loiasis • Loiasis is a filarial infection caused by Loa Loa. • It is transmitted by the Chrysops deerfly and tends to occur in rainforest regions of Western and Central Africa. • It has less pathological features than other the microfilarial infections Onchocerciasis and Lymphatic Filariasis. Clinical features • pruritus • urticaria • Calabar swellings: transient, non-erythematous, hot swelling of soft-tissue around joints • 'eye worm' - the dramatic presentation of subconjuctival migration of the adult worm. Treatment • Ivermectin is currently the drug of choice for control of both Onchocerciasis and Lymphatic Filariasis in Africa. • high loa loa microfilaraemia is associated with encephalopathy following treatment with either Ivermectin or DEC. This occurs due to the death of vast numbers of blood microfilaria. Both of these drugs are contraindicated if loa loa microfilaraemia exceeds 2500 mf/ml. Adult Loa loa parasite. Loa loa is the filarial nematode (roundworm) species that causes loa loa filariasis. It is commonly known as the 'eye worm.' Its geographic distribution includes Africa and India. Credit: NIAID

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Infectious diseases

Animal bites • The majority of bites seen in everyday practice involve dogs and cats. • Dog bites become infected in 10% of cases. • the most common isolated organism is Pasteurella multocida. Management • cleanse wound • current BNF recommendation is co-amoxiclav • if penicillin-allergic then doxycycline + metronidazole is recommended Previous exams MRCPUK-part-1-January- 2019: H/O a dog bite to right hand. What is the most appropriate antibiotic therapy? Co-amoxiclav MRCPUK-part-1-January- 2018: A patient has been bitten by his dog that morning. the wound looks clean as he has washed it well. He is penicillin allergic. Which antibiotic therapy is suitable? Metronidazole and doxycycline in combination

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Rocky Mountain spotted fever • Rocky Mountain spotted fever (RMSF) is a systemic vasculitis caused by infection with Rickettsia rickettsii, a tick-borne, gram-negative, intracellular bacterium, that primarily infects vascular endothelial cells. • It is the most common fatal tick-borne infection in the USA • Transmitted by bites of the dog or wood tick, which predominantly occur in spring and

summer throughout much of the United States. Feature • Fever, headache, myalgia, rash, vomiting, and history of tick bite are commonly reported; however, the absence of any of these does not exclude diagnosis. A history of tick bite may not be elicited in up to 45% of cases. • The rash usually sparing the face and may involve palms and soles. • Signs and symptoms may be difficult to distinguish from those of common viral illnesses, leading to delayed diagnosis. • Diagnosis should be considered in any person with a compatible clinical presentation and recent outdoor exposure. • Late-stage manifestations, such as noncardiogenic pulmonary edema (acute respiratory distress syndrome [ARDS]) and cerebral edema, are consequences of microvascular leakage. Investigation • PCR (polymerase chain reaction) is the most appropriate test Treatment • Doxycycline is the drug of choice for adults and children and is almost always curative, especially if given in the first 5 days of illness. Tetracyclines acts on 30S ribosomes to prevent protein synthesis in the infecting organism.

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 Aluminium hydroxide can complex with this antibiotic in the gastrointestinal tract, preventing absorption. Dairy products ingested at the same time can also cause this. Aluminium hydroxide medication should be stoped till the antibiotic course is finished • Because the risk of death rises if appropriate therapy is not started before the fifth day of illness, doxycycline should be prescribed for suspected Rocky Mountain spotted fever before confirmatory diagnostic test results are available. Typhus (Rickettsial infection) • caused by Rickettsia typhi (endemic typhus) or Rickettsia prowazekii (epidemic typhus). T • Rickettsia prowazekii (epidemic typhus) is transmitted via human-to-human contact through body lice. • Arthropod vectors transmit the etiologic agents to humans. • Presented with fever and rash • Both forms of typhus consist of a rash that classically begins centrally, and spreads outwardly sparing the palms and soles (unlike Rocky Mountain spotted fever) • Rocky Mountain Spotted Fever can be distinguished from typhus because its rash begins peripherally, and spreads centrally to the palms, soles, and trunk. • Doxycycline is the drug of choice for treatment in patients of all ages.

Histoplasmosis Overview • Histoplasmosis is one of the most common systemic fungal infections in the United States. It is endemic to the Ohio and Mississippi river valleys • often associated with spelunkers (cave divers) or patients recently exposed to bird and bat droppings. Feature • The majority are asymptomatic. • can closely mimic tuberculosis in symptomatology and imaging. dry cough, shortness of breath, fatigue, and fever • Disseminated infection causes bilateral adrenal enlargement in 80% of cases and it can result in adrenal insufficiency. Diagnosis: Adrenal biopsy or FNA with Groccott stain (Groccott-stained adrenal biopsy). Investigation • Chest X-ray often reveals a solitary lung lesion. • Disseminated histoplasmosis can cause systemic granulomatous inflammation and cavitation, which may be fatal. • The organisms can be visualized using methenamine silver or periodic acid-Schiff staining. • On histology Macrophages containing yeast Histoplasma capsulatum is a small intracellular yeast that is phagocytosed by alveolar macrophages. Treatment • Itraconazole for 3-6 months

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Actinomycosis Predisposing conditions include: • tooth extractions, • fractures of the jaw, • periodontal abscesses, • foreign bodies penetrating the mucosal barrier (bone splinters, fish bones) or • suppurating tonsillar crypts. • impaired immunity Features • cervicofacial actinomycosis the most common manifestation of infection with Actinomyces spp. • Initially, cervicofacial actinomycosis presents either as an acute, usually odontogenic, abscess or cellulitis of the floor of the mouth, or as a slowly developing hard, painless, reddish or livid swelling. • Small, acute actinomycotic abscesses may heal after surgical drainage alone. More often, however, the acute initial stage is followed by a subacute to chronic course if no specific antimicrobial treatment is administered. • Chronic disease is characterised by regression of central suppurative foci while the infection progresses peripherally; it can spread to involve other parts of the head and neck, including the meninges. • A quick and comparatively reliable diagnosis is possible microscopically, when sulphur granules are present; this is not conclusive, however, as nocardiosis may present similarly and has a similar appearance on microscopy. • One way to differentiate Actinomyces spp. from Nocardia spp. is through culture: the former grow in anaerobic conditions and the latter do not.

Malignant otitis externa Causes • Malignant otitis externa is a necrotizing infection of the ear that is commonly caused by Pseudomonas aeruginosa. Pseudomonas species are often found swimming pools and hot tubs, and can also cause “hot tub folliculitis”. Risk factors • Susceptible individuals include diabetics and other immunosuppressed patients. Feature • Physical exam may reveal discharge from the ear • severe pain, out of proportion to physical findings, on manipulation of the ear. • The disease can affect surrounding bony architecture and cause cranial nerve palsies. Such involvement suggests poor prognosis. Treatment • Treatment for suspected Pseudomonas infections anti-pseudomonal penicillin such as piperacillin-tazobactam, which is a penicillin paired with a beta-lactamase inhibitor.

Third edition Notes & Notes For MRCP part 1 & 2 By Dr. Yousif Abdallah Hamad Dermatology Updated

Notes & Notes for MRCP
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Dermatology

Epidermis • The epidermis is the outermost layer of the skin and is composed of a stratified squamous epithelium with an underlying basal lamina • It may be divided in to five layers: Layer Description Stratum corneum Flat, dead, scale-like cells filled with keratin Continually shed Stratum lucidum Clear layer - present in thick skin only Stratum granulosum Cells form links with neighbours Stratum spinosum Squamous cells begin keratin synthesis Thickest layer of epidermis Stratum germinativum The basement membrane - single layer of columnar epithelial cells Gives rise to keratinocytes Contains melanocytes

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Definitions • Plaque is a descriptive term for a skin lesion that is raised and greater than 1 cm in diameter. • Macule is an area of altered skin colour is irrespective of the size. • Papule is a raised lesion less than 1 cm in diameter. • Ulcer is a discontinuity of the skin with complete loss of the epidermis and often portions of the dermis and subcutaneous fat. • Vesicle is a fluid-filled, well-circumscribed raised lesion. • Pustule are small elevation of the skin containing cloudy or purulent material, usually consisting of necrotic inflammatory cells. • Bulla are large vesicle containing serous fluid. • Fissure are cracks in the skin that are narrow but deep. • Telangiectasia are collection of enlarged capillaries visible on the skin or mucous membranes. • Lichenification of the skin is due to epidermal thickening characterised by visible and palpable thickening of the skin with accentuation of skin markings. • Atrophy of the skin may be due to loss of epidermis, dermis or subcutaneous tissue. Thinning of the epidermis presents as skin that appears thin and translucent. Thinning of the dermis and subcutaneous tissue leads to a depression in the skin.

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Acanthosis nigricans Overview • Describes symmetrical, brown, velvety plaques that are often found on the neck, axilla and groin • presents as a dark thickened area of the skin of the back of the neck or the armpit, • Obesity is the most common cause • Classically acanthosis nigricans associated with malignancy appears abruptly, and it can precede diagnosis of malignancy. • Diabetes causes acanthosis nigricans due to stimulation of insulin-like growth factor receptor-1. Causes • paraneoplastic phenomenon (usually tumours of the GI tract, especially adenocarcinoma of the stomach) and Endometrial carcinoma • diabetes mellitus • obesity • polycystic ovarian syndrome • acromegaly • Cushing's disease • hypothyroidism • familial (autosomal dominant) • Prader-Willi syndrome • drugs: oral contraceptive pill, nicotinic acid (Niacin) Management • first line is treatment of the underlying cause. • In persistent acanthosis nigricans despite treatment of the underlying cause, topical retinoids can be tried.

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Acne rosacea is a chronic skin disease of unknown aetiology Features • typically affects nose, cheeks and forehead • flushing is often first symptom • telangiectasia are common • later develops into persistent erythema with papules and pustules • rhinophyma • ocular involvement: blepharitis Management • topical metronidazole may be used for mild symptoms (i.e. Limited number of papules and pustules, no plaques) • more severe disease is treated with systemic antibiotics e.g. Oxytetracycline • recommend daily application of a high-factor sunscreen • camouflage creams may help conceal redness • laser therapy may be appropriate for patients with prominent telangiectasia

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_Acne vulgaris • Acne vulgaris is a common skin disorder which usually occurs in adolescence. • It typically affects the face, neck and upper trunk • characterised by the obstruction of the

pilosebaceous follicle with keratin plugs which results in comedones, inflammation and pustules. Epidemiology • Affects around 80-90% of teenagers • Age of onset: typically by 11–12 years, with symptoms usually disappearing around 20–30 years of age Acne presenting at beyond aged 20 years should always prompt investigation of a possible secondary cause. • Sex: more common in males during adolescence, but more common in women during adulthood Aetiology & Pathophysiology • Hormonal factors ↑ Androgens during puberty → increased production of sebum by sebaceous glands In women: menstrual cycle • Follicular hyperkeratosis: Follicular epidermal hyperproliferation →formation of a keratin plug →obstruction of pilosebaceous follicle. Higher keratinocyte activity and decreased keratinocyte shedding in pilosebaceous units leads to the formation of comedones. • Bacterial colonisation with Cutibacterium acnes; inflammatory reactions with formation of papules, nodules, pustules, and/or cysts Features • Localisation: common in areas with sebaceous glands (predilection sites: face, shoulders, upper chest, and back) • Primary lesions

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 Non-inflammatory: comedonal acne Closed comedones (“whiteheads”): closed small round lesions that contain whitish material Open comedones (“blackheads”): dark, open portion of sebaceous material Inflammatory: affected areas are red and can be painful papules, pustules that arise from comedones Nodular acne (> 5 mm in diameter): Commonly the back and neck • Secondary lesions: hyperpigmentation, and scarring Management • A simple step-up management scheme often used in the treatment of acne is as follows: Single topical therapy (topical retinoids, benzyl peroxide) Topical combination therapy (topical antibiotic, benzoyl peroxide, topical retinoid) Oral antibiotics: e.g. Oxytetracycline, doxycycline. Improvement may not be seen for 3-4 months. Minocycline is now considered less appropriate due to the possibility of irreversible pigmentation. Gram negative folliculitis may occur as a complication of long-term antibiotic use . high-dose oral trimethoprim is effective if this occurs Oral erythromycin may be used for acne in pregnancy. The other drugs are contraindicated • Oral isotretinoin: only under specialist supervision • Ethinylestradiol with cyproterone acetate (Dianette) is useful in some female patients with acne unresponsive to standard treatment. • There is no role for dietary modification in patients with acne Weight loss is the most important intervention.

Isotretinoin Overview • Isotretinoin is an oral retinoid used in the treatment of severe acne. Two-thirds of patients have a long-term remission or cure following a course of oral isotretinoin Indication • Moderate to severe acne Contraindications • Pregnancy, women of childbearing age without contraception: strong teratogenic effects • Liver disease • Precautions (in all females of childbearing potential) • A serum/urine pregnancy test Side effects • Teratogenicity females should ideally be using two forms of contraception (e.g. Combined oral contraceptive pill and condoms) should be discontinued at the latest one month before • Dry skin, eyes and lips: the most common side-effect of isotretinoin • Low mood • Raised triglycerides • Hair thinning Retinoid (isotretinoin) therapy should be discontinued at the latest one month before

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• Nose bleeds (caused by dryness of the nasal mucosa) • Benign intracranial hypertension: isotretinoin treatment should not be combined with tetracyclines for this reason • Photosensitivity • Laboratory test abnormalities: ↑ Triglycerides, ↓ HDL, ↑ glucose

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Alopecia Divided into scarring (destruction of hair follicle) and non-scarring (preservation of hair follicle) Scarring alopecia • trauma, burns • radiotherapy • lichen planus • discoid lupus • tinea capitis (scarring may develop in untreated tinea capitis if a kerion develops) Non-scarring alopecia • male-pattern baldness • drugs: cytotoxic drugs, carbimazole, heparin, oral contraceptive pill, colchicine • nutritional: iron and zinc deficiency • autoimmune: alopecia areata • telogen effluvium (hair loss following stressful period e.g. surgery) • trichotillomania psychological disorder where patients are compelled to pull their own hair, resulting in alopecia. It is typically encountered in teenage females and children Cicatricial alopecia (also known as scarring alopecia) • inflammation injures hair follicles resulting in permanent bald patches with no visible follicles. inflammation can be seen as redness, scaling and crusting. • Common causes include: discoid lupus erythematosus, and lichen planopilaris (a variant of lichen planus). • Treatment is dependent on the underlying causes but often requires topical corticosteroids.

Alopecia areata • Alopecia areata is a presumed autoimmune condition causing localised, well demarcated patches of hair loss. Feature • localised patches of non-scarring hair loss. • Remaining hairs have a characteristic 'exclamation mark' appearance, and are tapered towards the base. small, broken hairs at the edge of the hair loss • More severe involvement may present as alopecia totalis (total loss of scalp hair) or alopecia universalis (total loss of all body hair). Treatment • Hair will regrow in 50% of patients by 1 year, and in 80-90% eventually. Careful explanation is therefore sufficient in many patients. • Other treatment options include:

 topical or intralesional corticosteroids the most appropriate treatment for area of hair loss Intra-lesional triamcinolone topical minoxidil phototherapy dithranol contact immunotherapy wigs Differential diagnosis • Androgenetic alopecia presents after puberty as a more diffuse slow hair loss with characteristic loss over the temporal regions and vertex in males. • Discoid lupus erythematosus (DLE) presents as scarring alopecia. Areas of alopecia are usually atrophic with visible loss of hair follicles. Patients may have DLE lesions elsewhere. If not treated early, hair loss is usually irreversible. • Telogen effluvium presents with diffuse hair loss and usually presents one to three months after a stressful episode, for example, viral illness, surgery, childbirth, emotional stress. Hair loss is never complete and usually stops after three to five months. Subsequent hair regrowth is usually complete. • Trichotillomania more commonly seen in children compared to adults. Patients also present with localised hair loss but in a bizarre pattern. Hairs of differing lengths are usually seen within and at the edges of the patches. Patients may or may not volunteer a history of hair pulling. Pemphigus vulgaris Overview • Pemphigus vulgaris is an autoimmune disease caused by antibodies (IgG) directed against desmoglein 3, a cadherin-type epithelial cell adhesion molecule. • The binding of autoantibodies

results in a loss of cell-to-cell adhesion, a process termed acantholysis. • It is more common in the Ashkenazi Jewish population • seen predominantly in patients ages 50-60, but can affect many ages. Features • mucosal ulceration is common and often the presenting symptom. Oral involvement is seen in 50-70% of patients • skin blistering - flaccid, easily ruptured vesicles and bullae. Blisters are thin-walled and rupture easily (intact blisters are rarely seen). • Lesions are typically painful but not itchy. These may develop months after the initial mucosal symptoms. • Nikolsky's describes the spread of bullae following application of horizontal, tangential pressure to the skin

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• Immunofluorescent staining of a biopsy sample shows deposition of immunoglobulin (IgG) directed against to keratinocyte desmosomes and to desmosome-free areas of the keratinocyte cell membrane, resulting in a 'chicken wire' appearance. • acantholysis on biopsy Mucosal ulceration is common with pemphigus Management • steroids • immunosuppressants

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Bullous pemphigoid Overview • Bullous pemphigoid is an autoimmune condition causing sub-epidermal blistering of the skin. • This is secondary to the development of antibodies against hemidesmosomal proteins BP180 and BP230 caused by (IgG) autoantibodies against components of the basement membrane. Epidemiology • Pemphigoid, erythema multiforme, and herpes are the commonest causes of a blistering rash. • Bullous pemphigoid is more common in elderly patients (over 60 years). Remember, this is a disease of the elderly (uncommon under the age of 60). Features • Include • itchy, tense blisters typically around flexures • the blisters usually heal without scarring • mouth is usually spared* *in reality around 10-50% of patients have a degree of mucosal involvement. It would however be unusual for an exam question to mention mucosal involvement as it is seen as a classic differentiating feature between pemphigoid and pemphigus. Investigations • Skin biopsy: Perilesional skin biopsy for examination by direct immunofluorescence immunofluorescence shows IgG and C3 at the dermo-epidermal junction Differential diagnosis • Blistering in pemphigoid occurs at the sub-epidermal level - deeper than the blisters of pemphigus vulgaris (which occur at the dermal-epidermal junction); hence the tense blisters seen in pemphigoid. Blisters are thin-walled and fragile in pemphigus - few intact blisters are ever seen.

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 In pemphigus vulgaris, mucous membrane involvement is more common, and intact bullae are rare. Skin biopsy for routine and direct immunofluorescence is needed to differentiate from bullous pemphigoid. Management • referral to dermatologist for biopsy and confirmation of diagnosis • oral corticosteroids are the mainstay of treatment • topical corticosteroids, immunosuppressants and antibiotics are also used Topical corticosteroids may be attempted in patients with mild, localised bullous pemphigoid.

Dermatitis herpetiformis (DH) Overview • autoimmune blistering skin disorder associated with coeliac disease and gluten sensitivity. • caused by deposition of IgA in the dermis. • associated with HLA-DR3. • Virtually all patients with DH carry the HLA DQ2 or HLA DQ8 haplotype. Features • itchy, vesicular skin lesions on the extensor surfaces (e.g. elbows, knees, buttocks) Association • increased risk for the development of other autoimmune diseases. Thyroid disease is the most common autoimmune disorder associated with DH. • increased risk for lymphoma. Diagnosis • skin biopsy: direct immunofluorescence (The gold standard test for diagnosis) shows: Subepidermal deposition of IgA in a granular pattern in the upper dermis (in the dermal papillae)(Granular IgA deposits at the basement membrane zone) neutrophilic dermal infiltrates in the superficial dermis Neutrophils are the immune cell that is involved in the blistering skin lesion DH. • Serology blood test showing the presence of IgA antibodies against tissue transglutaminase. Management • gluten-free diet • dapsone Dermatitis herpetiformis Dermatitis herpetiformis is associated with HLA-DR3 Dermatitis herpetiformis - caused by IgA deposition in the dermis

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Discoid lupus erythematous Pathology • it is a chronic type of Cutaneous lupus erythematosus (CLE) • characterised by follicular keratin plugs • characterised by a well-demarcated macular rash with erythema, scales, and plaques that often results in scarring and atrophy. Aetiology • thought to be autoimmune in aetiology Association • may occur in the absence or in association with systemic SLE. Approximately 10% of patients may have signs of SLE. Epidemiology • generally seen in younger females. • occurs 2-3 times more frequently in women than in men • more common in African–Caribbean female. Features • erythematous, raised rash, sometimes scaly • may be photosensitive • more common on face, neck, ears and scalp • lesions heal with atrophy, scarring (may cause scarring alopecia), and pigmentation Diagnosis • made by biopsy of the lesion. Management • 1st line: topical potent steroid cream • 2nd line: oral antimalarials e.g. hydroxychloroquine other options Topical calcineurin inhibitors Intralesional corticosteroids Oral corticosteroids. • Avoid sun exposure Prognosis • The risk of progression to SLE in patients with DLE was demonstrated to be higher than previously reported (16.7% progression within 3 years of diagnosis, as compared with previous data indicating that <5-10% of patients with DLE progress to SLE). • children with DLE seem to have a higher early rate of progression to SLE (up to 25%) indicating that the age at onset might influence disease severity According to a recent epidemiologic study, approximately 16% of patients with discoid lupus erythematosus (DLE) may develop systemic involvement within 3 years of diagnosis.

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Discoid lupus erythematous affecting the scalp Discoid lupus erythematous affecting the face Contact dermatitis Types • There are two main types of contact dermatitis Irritant contact dermatitis: common non-allergic reaction due to weak acids or alkalis (e.g. detergents). Often seen on the hands. Erythema is typical, crusting and vesicles are rare Allergic contact dermatitis: type IV hypersensitivity reaction. Uncommon often seen on the head following hair dyes. Presents as an acute weeping eczema, which predominately affects the margins of the hairline rather than the hairy scalp itself. Topical treatment with a potent steroid is indicated The main difference between allergic contact dermatitis and irritant contact dermatitis: The rash caused by allergic contact dermatitis confined to contacted area, whereas in irritant contact dermatitis, the rash is more widespread. In allergic contact dermatitis the rash usually appears after a day or two after exposure to the allergen, unlike irritant contact dermatitis that appears immediately after the contact with the trigger.

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Pruritus

Causes The table below lists the main characteristics of the most important causes of pruritus Liver disease History of alcohol excess Stigmata of chronic liver disease: spider naevi, bruising, palmar erythema, gynaecomastia etc Evidence of decompensation: ascites, jaundice, encephalopathy Iron deficiency anaemia Pallor Other signs: koilonychia, atrophic glossitis, post-cricoid webs, angular stomatitis Polycythaemia Pruritus particularly after warm bath 'Ruddy complexion' Gout Peptic ulcer disease Chronic kidney disease Lethargy & pallor Oedema & weight gain Hypertension Lymphoma Night sweats Lymphadenopathy Splenomegaly, hepatomegaly Fatigue Other causes • hyper- and hypothyroidism • diabetes • pregnancy • 'senile' pruritus • skin disorders: eczema, scabies, psoriasis, pityriasis rosea • Idiopathic urticaria: Up to 50% of cases are idiopathic

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Eczema herpeticum • Eczema herpeticum describes a severe primary infection of the skin by herpes simplex virus 1 or 2. Features • It is more commonly seen in children with atopic eczema. • Typically, the child has a high fever for seven days, and recurrent attacks can occur. • It may affect any site but is most often seen on face and neck. Treatment • Eczema herpeticum is considered as one of the few dermatological emergencies. • As it is potentially life threatening children should be admitted for IV acyclovir Complications • Death can result from physiological disturbances (loss of fluid electrolytes and protein through the skin) or dissemination of the virus to brain and other organs or from secondary bacterial sepsis. • may be further complicated by secondary staphylococcal infection. This is treated by adding oral antibiotics, for example, flucloxacillin 500 mg q.i.d.

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Eczema: topical steroids Use weakest steroid cream which controls patients symptoms The table below shows topical steroids by potency Mild Moderate Potent Very potent Hydrocortisone 0.5-

2.5% Betamethasone valerate 0.025% (Betnovate RD) Clobetasone butyrate 0.05% (Eumovate) Finger tip rule • 1 finger tip unit (FTU) = 0.5 g, sufficient to treat a skin area about twice that of the flat of an adult hand Topical steroid doses for eczema in adults Area of skin Fingertip units per dose Hand and fingers (front and back) 1.0 A foot (all over) 2.0 Front of chest and abdomen 7.0 Back and buttocks 7.0 Face and neck 2.5 An entire arm and hand 4.0 An entire leg and foot 8.0 The BNF makes recommendation on the quantity of topical steroids that should be prescribed for an adult for a single daily application for 2 weeks: Notes & Notes for MRCP
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Clobetasol propionate 0.05% (Dermovate) Fluticasone propionate 0.05% (Cutivate) Betamethasone valerate 0.1% (Betnovate)

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Area Amount Face and neck 15 to 30 g Both hands 15 to 30 g Scalp 15 to 30 g Both arms 30 to 60 g Both legs 100 g Trunk 100 g Groin and genitalia 15 to 30 g Pompholyx Pompholyx is a type of eczema which affects both the hands (cheiropompholyx) and the feet (pedopompholyx). It is also known as dyshidrotic eczema Features • small blisters on the palms and soles • pruritic, sometimes burning sensation • once blisters burst skin may become dry and crack Management • cool compresses • emollients • topical steroids

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Erythema ab igne • Erythema ab igne is a skin disorder caused by over exposure to infrared radiation. • It classically presents on the front of the legs due to the patient sitting too close to a fire or heater. It may also arise as a response to chronic hot water bottle use. • Characteristic features include reticulated, erythematous patches with hyperpigmentation and telangiectasia. • A typical history would be an elderly women who always sits next to an open fire. • Hypothyroidism can make patients feel cold and hence more likely to sit next a heater / fire. • If the cause is not treated then patients may go on to develop squamous cell skin cancer.

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Erythema ab igne Erythema ab igne

Erythema multiforme Pathophysiology • Type IV hypersensitivity reaction; triggered by the following Infections: herpes simplex virus (HSV - the most common cause), Mycoplasma pneumoniae. Drugs: phenytoin; beta-lactam antibiotics (e.g., penicillins); sulfonamides Classification • Erythema multiforme minor (typical targets or raised oedematous papules, with acral distribution, without involvement of mucosal sites, and involving <10% total body surface area) • Erythema multiforme major (typical targets or raised oedematous papules, with acral distribution, plus involvement of 1 or more mucosal sites, and involving <10% total body surface area) Features • Erythematous , maculopapular rash (many forms), hence the ‘multiforme’ in the

name. • Target lesions (also called iris lesion): an inner dark red/brown zone, surrounded by a pale zone, and an outer erythematous ring. • Distribution: Symmetrical and affects backs of hands and feet first → spreads proximally and can affect the entire body. Treatment • Supportive: treat the underlying infection or stop the offending drug . Prognosis • usually mild and self-limiting disease with the lesions healing within 2 to 3 weeks without scarring.

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Erythema multiforme Erythema multiforme Differential diagnosis • Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) are the same entity but differ in terms of disease severity (based on surface area of skin involved). □ < 10% – SJS □ 10–30% – SJS/TEN overlap □ 30% – Toxic epidermal necrolysis (severe SJS)

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Erythema multiforme VS Stevens-Johnson syndrome Erythema multiforme (EM) Stevens-Johnson syndrome (SJS) Causes usually triggered by infections, most commonly herpes simplex virus (HSV). Medications are uncommon cause (<10%) Most commonly triggered by drugs (~80%). Lesions distribution Lesions begin on the extremities Lesions typically begin on the face and trunk. Target lesions Typical target lesions No typical target lesions. Mucosal membranes Mucosal membranes may be involved, but usually not Mucosal membranes almost always involved Swelling No associated swelling of face, hands or feet Associated swelling of face, hands or feet Systemic symptoms Systemic symptoms such as fever and malaise, are absent or mild Systemic symptoms such as fever and malaise, are prominent Histology high density of cell infiltrate rich in Tlymphocytes. (more dermal inflammation and individual keratinocyte necrosis) poor infiltrate of macrophages and dendrocytes with tumor necrosis factor (TNF) (minimal inflammation and sheets of epidermal necrosis.) Severity Usually mild Shock may develop Treatment Treat underline cause need urgent supportive care , fluid resuscitation similar to that of burns and Wound management Prognosis Self-limiting High mortality rate (SJS: ~25%, TEN: ~50%) Erythema multiforme (EM): • a type IV hypersensitivity reaction of the skin. • can be triggered by certain infections (e.g., HSV, Mycoplasma pneumonia) and medications (e.g., beta-lactam antibiotics, sulfonamides, phenytoin). Erythema multiforme (EM): • EM is characterized by lesions of varying morphology (e.g., macules, papules, vesicles) that typically progress to target lesions and spread proximally from the backs of the hands and feet. Herpes simplex virus (HSV) infection is the commonest cause of Erythema multiforme

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By Dr. Yousif Abdallah Hamad

Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) Definition • Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) are severe mucocutaneous reactions, most commonly triggered by medications, characterized by extensive necrosis and detachment of the

epidermis • SJS and TEN are the same entity but differ in terms of disease severity (based on surface area of skin involved). < 10% – SJS 10–30% – SJS/TEN overlap ≥ 30% – Toxic epidermal necrolysis (severe SJS) Pathophysiology • Delayed hypersensitivity reaction (type IV) Causes • Most commonly triggered by medications, ~80% of cases Antibiotics: sulfonamides (e.g., TMP/SMX), aminopenicillins Antiepileptics: phenytoin, phenobarbital, lamotrigine, valproic acid, carbamazepine, Sulfasalazine Nonsteroidal anti-inflammatory drugs (NSAIDs) Features • Begins with a prodrome of fever and influenza-like symptoms one to three days before the development of mucocutaneous and skin lesions. • Extensive, full-thickness epidermal necrosis and sloughing (resembling large superficial burns) • Mucosal membranes: almost always involved ~90% of cases • Systemically unwell e.g. pyrexia, tachycardic, shock may develop • Positive Nikolsky's sign (the epidermis separates with mild lateral pressure) • Skin biopsy Keratinocyte necrosis with apparent subepidermal split Eosinophilic infiltration with minimal infiltration of lymphocytes and histiocytes around blood vessels Treatment • Stop precipitating factor is most likely to improve prognosis • Supportive care, often in intensive care unit • Wound management: similar to that of burns

Notes & Notes for MRCP
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Dermatology

Stevens-Johnson syndrome (SJS)

Erythema nodosum Always do a chest x-ray on a patient with erythema nodosum, to exclude sarcoidosis Overview • inflammation of subcutaneous fat • Histology of these lesions shows a vasculitis of small venules and panniculitis. • typically causes tender, erythematous, nodular lesions • usually occurs over shins, may also occur elsewhere (e.g. forearms, thighs) • usually resolves within 6 weeks • lesions heal without scarring Causes • infection: streptococci, TB, brucellosis The commonest cause is streptococcal infection. • systemic disease: sarcoidosis, inflammatory bowel disease (ulcerative colitis), Behcet's , SLE • malignancy/lymphoma • Drugs (oral contraceptive, sulfonamides, penicillins, antipyretics, montelukast, Hep B vaccination, omeprazole). • pregnancy

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Erythema induratum (EI) • EI is a form of panniculitis characterised by chronic, recurrent, tender, subcutaneous, and sometimes ulcerated nodules on the lower legs that may also appear elsewhere. (Erythema nodosum also commonly associated with TB but do not ulcerate) • Females are more frequently affected, with a female: male ratio of 7:1 and it is more frequent in younger females. • It is found in association with tuberculosis.

Erythrasma • Erythrasma is a generally asymptomatic, flat, slightly scaly, pink or brown rash usually found in the groin or axillae. • It is caused by an overgrowth of the diphtheroid Corynebacterium minutissimum • Examination with Wood's light reveals a coral-red fluorescence. •

Topical miconazole or antibacterial are usually effective. Oral erythromycin may be used for more extensive infection

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Erythroderma • Erythroderma is a term used when more than 95% of the skin is involved in a rash of any kind • Mechanism The mechanism behind erythroderma is most likely from cutaneous thermal dysregulation. Increased blood flow to the skin leads to heat and fluid loss, and increased rate of skin cell turnover and skin sloughing. • Causes of erythroderma Eczema (40%) Psoriasis (25%) drugs e.g. gold lymphoma, leukaemia pityriasis rubra pilaris idiopathic • often accompanied with fever, shivering and malaise. • Erythrodermic psoriasis may result from progression of chronic disease to an exfoliative phase with plaques covering most of the body. Associated with mild systemic upset more serious form is an acute deterioration. This may be triggered by a variety of factors such as withdrawal of systemic steroids. Patients need to be admitted to hospital for management