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DIAGNOSTIC PLANNER

Factors to consider when formulating a differential diagnosis

Symptoms (eg fever, pain, pruritis)
Duration and temporal pattern (acute, sub-acute, chronic, intermittent)
Primary lesions: macules, patches, papules, pustules, nodules, plaques, vesicles, bullae
Secondary morphology: scale, crust, erosions, ulcers, scars, purpura, pallor, cyanosis
Arrangement: annular, linear, solitary, generalised,
Anatomical location: palmar plantar, acral, truncal etc
Colour: black, blue, brown, red, flesh-coloured, cream, xanthotic

Laboratory findings
Histopathologic findings

Is the skin condition/lesion

Epidermal, dermal, subcutaneous or mixed level?
Inflammatory (infectious/non infectious) or papulo-squamous?
Neoplastic (benign or malignant)?
Solitary or multiple lesions/characteristic distribution?

Is there

Any past personal, family or contact history?
Any relevant medication/drug/occupational history?
Any injury/self harm?
Dysplastic nevus

Compound nevus

Blue nevus

BCC

Melanoma in situ

MM
Dermoscopy

3 point check list
- asymmetry
- atypical network
- blue white structures

5 melanoma specific local criteria
- atypical network
- irregular streaks
- irregular dots/globules
- irregular blotches
- blue white structures

6 criteria for non melanocytic lesions
- blue gray blotches
- arborizing vessels
- milia like cysts
- comedo like openings
- red blue lacunae
- central white patch
FACIAL PIGMENTATION

• Depth (epidermal or dermal)
• Type (melanocytic or not)
• Skin type
• Ethnic background

Drugs:
- Minocycline
- Clofazimine
- Amioderone
- Zidovudine (AZT) – blue lunulae
- Diltiazem (Skin types IV or V)
- Dioxins
- Hydroquinone (ochronosis)
- Psychotropic drugs
- Psoralens
- Hormones
- Chemotherapeutic agents – B, C, D, F, H, MTX
- Antimalarials
- Heavy metals – arsenic, gold, iron, silver, lead, mercury

MELASMA/CHLOASMA – HYPERPIGMENTATION

• Erythema dyschromicum perstans (Ashy dermatitis)
• Lichen planus pigmentosum (actinic)
• Primary cutaneous amyloidosis
• Cafe au lait pigmentation
• Haemosiderin
• Nevus of Ota
• Pot traumatic
• Post inflammatory (numerous causes)
• Poikiloderma of Civatte
• Phytotoxic and phytophototoxicity
Skin Cancer – Pigmented Lesions Differential Diagnosis

- Tattoos – Foreign bodies – calciphylaxis
- Argyria – minocycline dyspigmentation (other drugs)
- Angiokeratoma – angiosoroma – venous lake
- Open comedone – cyst – blue naevus – hidrocystoma
- Post inflammatory – pigmented purpuric dermatoses (haemosiderin)
- Lichen planus – naevs of Ota – ochronosis
- Talon noir – Terra fima dermatosis – dermatosis papulosa nigra
- Lentigo simplex – stellate lentigo – solar lentigo – chloasma
- Warfarin necrosis – purpura – gangrene – sub ungual haemorrhage
- Seborrhoeic lentigo – pigmented actinic keratosis – Bowen’s disease
- Many other dermatoses esp. Pigmented skin types
- Deep dermal blue/black discolouration from any particulate matter.
## Tender/Painful Nodules

<table>
<thead>
<tr>
<th>Not inflamed</th>
<th>Inflamed</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>E</strong> Eccrine spiradenoma/erythematous nodosum</td>
<td>Keratoacanthoma</td>
</tr>
<tr>
<td><strong>N</strong> Neurilemmoma</td>
<td>Acne nodules/hydradenitis</td>
</tr>
<tr>
<td><strong>G</strong> Glomus tumour</td>
<td>Epidermal cyst (inflamed)</td>
</tr>
<tr>
<td><strong>L</strong> Leiomyoma</td>
<td>Staph lesions (boils, furuncles)</td>
</tr>
<tr>
<td><strong>A</strong> Angiolipoma/arthropod sting</td>
<td>Vasculitis/paniculitis</td>
</tr>
<tr>
<td><strong>N</strong> Neuroma/neurofibroma</td>
<td>Sweets neutrophillic dermatosis</td>
</tr>
<tr>
<td><strong>D</strong> Dercum’s/dermatofibroma</td>
<td>Chondrodermatitis helicis</td>
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</table>
ACNE

• Multifactorial disorder of pilosebaceous units
• Significant psychologic and economic impacts
• Comedones, papules, pustules, cysts, scarring

Increased risk -
- xyy genotype
- PCOD
- hypercortisolism
- precocious puberty

Pathogenesis -
- genetic predisposition (sebaceous glands)
- hormonal responsiveness – sebum excretion
- increased cellular cohesion and proliferation
- comedone formation and rupture
- propionibacterium acnes (coproporphyrin III)
- immune response
- DHEA → testosterone → DHT

Clinical -
- non-inflammatory (comedones, open and closed, micro and macro) scars; ice pick erythematous papules; sterile pustules indurated nodules and cysts scars, hypertrophic, atrophic depressed, aggregated pitted, bridged, tethered

Acne Variants -
- acne fulminans (haemorrhagic plaques, fever, osteolytic lesions)
- acne conglobata (eruptive nodulo-cystic acne)
- solid facial oedema
- acne mechanica
- acne excoriée des jeunes filles
- drug induced acne (steroids, azathioprine, PUVA etc)
- occupational acne (follicular occlusion tetrad)
- choracne (halogenated hydrocarbons)
- EGFR inhibitors
- neonatal and infantile
- radiation
- endocrine
- tropical
Isotretinoin (13-cis-retinoic acid) since 1971

• Severe inflammatory nodulo-cystic acne
• Persistent inflammatory acne with scarring potential
• Other inflammatory acne resulting in significant emotional stress (dysmorphophobia)

Isotretinoin induced (1) sebaceous gland atrophy by prohibiting maturation of basal cells (2) normalisation of follicular keratinisation (3) reduction of p. acnes.

Dosing varies (0.5 – 1.0mg/kg/day for 16-20 weeks). Lower dose regimens may be equally effective. Repeat treatments in 40% of patients. Intermittent and long term treatments experimental.

**Lab studies on all patients:**
- complete blood count
- Liver function
- Creatinine
- Fasting lipids
- HCG
- Repeat lab studies

**Poor responders**
- macrocomedones
- dry skin types
- persistent inflammation females
- scarred nodules and sinus tracts
- endocrine disorders

**Combined treatment**
- prednisone
- erythromycin (tetracyclines contraindicated)
Side effects

• numerous (retinoic acid receptors ubiquitous)
• skin and mucous membranes (dose related) dryness, cheilitis
• alopecia, facial hair, dermatitis may occur
• xerophthalmia, conjunctivitis
• photophobia, night vision impairment, keratitis
• neuromuscular – myalgia, headache, fatigue, blurred vision
• superinfection (impetigo)
• GI irritation, nausea, vomiting, anorexia
• hepatitis rare and reversible
• benign intracranial hypertension (tetracyclines)
• psychiatric effects – suicide, suicidal ideation, depression ?a ‘real’ phenomenon but NB careful monitoring and support systems in place probable idiosyncratic and unrelated to previous or family history
• central effects – tiredness, lethargy, anxiety
• skeletal effects

Issue of teratogenicity
- individualise each situation
- menstrual cycle may be disrupted

Issue of consenting
- written consent
- initial against each point
- sign and witness

Other medication
- OCP, antidepressants, antibiotics
PHYSICAL TREATMENTS

• comedo extraction/peels (TCA, glycolic acid)
• electrosurgery/curettage
• blue light acne therapy
• photodynamic therapy
• near infrared lasers
• subscision/excision/punch grafting
• dermabrasion/laser resurfacing
• deep fractional CO² laser resurfacing
• soft tissue augmentation/fat transfer
• Common, disfiguring, male and female, fair skinned, 3rd and 4th decade

• **Pathogenesis multifactoral** - vascular hyper reactivity – vasodilation  
  - Neuro cutaneous component – bacterial overgrowth – dermatotex  
  - Corticosteroid association – U.V. exposure – ‘sensitive skin’

• **Variants** – granulomatous/periorificial/steroid/pyoderma faciale/lymphoedema/lupus miliaris disseminatus

• **1° features:**  
  - flushing (transient erythema)  
  - non-transient erythema  
  - papules and pustules  
  - telangiectasia

• **2° features:**  
  - burning/stinging sensation central face  
  - pustular plaques/confluence of papules  
  - dryness and flaking of central facial skin  
  - soft or solid facial/forehead oedema  
  - ocular/rhinophyma/extra facial

• **Differential diagnosis**  
  - seborrheic dermatitis, acne vulgaris  
  - erythromelanosis, keratosis pilaris rubra  
  - lupus erythematosus  
  - Haber syndrome – demodex folliculitis (HIV)
**ROSACEA TREATMENT**

**Topical**
- metronidazole cream or gel (0.75%)
- Sulphur and salicylic acid (<2%)
- Azelaic acid (15% - 20%)
- Benzoyl peroxide 5%, clindamycin 1% (DUAC)
- Tretinoin <0.1% cream or gel
- Vitamin C

**Oral**
- Tetracyclines
- Erythromycins
- Cotrimoxazole
- Isotretinoin

**Physical**
- Intense Pulsed Light
  - KTP (532nm) laser
- Pulsed dye laser
- CO² laser
- Surgical
- Electrosurgical
FOLLCULITIS

Superficial and deep

- Disorder of follicular keratinisation
- Follicular occlusion (acne conglobata, hidradenitis suppurativa)
- Dissecting cellulitis, pilonidal sinus)

Superficial

- Staph
- Pseudomonas

Deep

- Acne keloidalis
- Pseudofolliculitis barbae
- Pustules and erythema
- Terminal hairs

Follicular keratinisation

- Lichen planopilaris
- Lichen spinulosus
- Pityriasis rubra pilaris
- Keratosis pilaris atrophicans
- Erythromelanositis
- Follicularis faciei
• Eosinophilic (lymphoma, AIDS, neonatal) – pustular (Ofuji’s)
• Irritant folliculitis (tars, ointments, etc)
• Gram negative folliculitis (pseudomonas, klebsiella, enterobacter, proteus spp)
  - - long term antibiotics RX/hot tbb “spa” folliculitis - -)
• Dermatophyte folliculitis (T. metagrophytes/verrucosum/rubrum ‘Majocchi’s’)
• Pityrosporum folliculitis (young adults/warm weather/occlusion/↑sebum)
• Candida folliculitis (diabetics)
• Phrynoderma (vit A deficiency)
• Herpes simplex folliculitis (males shaving, HIV positive)
• Dermodex folliculitis (immune suppression)
• Drug induced folliculitis (steroids, androgens, iodides, lithium (AGEP), cotrimoxazole)
• Actinic folliculitis (hours x sun exposure or x phototherapy)
## NON MELANOMA SKIN CANCER (NMSC)

### Evaluation and risk management:

- **History**
  - Duration
  - Prior therapy
  - Rate of growth

- **Physical exam**
  - Location and size
  - Palpation/tethered
  - Definition

- **Biopsy**
  - Tumour type
  - Depth
  - Prognosis

### Neurologic symptoms
- Past history
- Family history
- Lymphadenopathy

### Surgical Management (tumour size is important)
- BCC invasive: 4mm excision margins or Mohs
- SCC (high risk): 6mm excision margins or Mohs
## RISK FACTORS FOR RECURRENCE OF NON MELANOMA SKIN CANCER

<table>
<thead>
<tr>
<th>CLINICAL RISK FACTORS</th>
<th>LOW RISK</th>
<th>HIGH RISK</th>
</tr>
</thead>
<tbody>
<tr>
<td>Location/size</td>
<td>Area L &lt;20mm</td>
<td>Area L &gt;20mm</td>
</tr>
<tr>
<td></td>
<td>Area M &lt;10mm</td>
<td>Area M &gt;10mm</td>
</tr>
<tr>
<td></td>
<td>Area H &lt;6mm</td>
<td>Area H &gt;6mm</td>
</tr>
<tr>
<td>Borders</td>
<td>Well defined</td>
<td>Poorly defined</td>
</tr>
<tr>
<td>Primary vs recurrent</td>
<td>Primary</td>
<td>Recurrent</td>
</tr>
<tr>
<td>Immunosuppression</td>
<td>Negative</td>
<td>Positive</td>
</tr>
<tr>
<td>Tumour at site of prior radiation therapy</td>
<td>Negative</td>
<td>Positive</td>
</tr>
<tr>
<td>Rapidly growing tumour (SCC only)</td>
<td>Negative</td>
<td>Positive</td>
</tr>
<tr>
<td>Neurologic symptoms: pain, paresthesia, paralysis (SCC only)</td>
<td>Negative</td>
<td>Positive</td>
</tr>
</tbody>
</table>

### PATHOLOGIC RISK FACTORS

<table>
<thead>
<tr>
<th>Perineural involvement</th>
<th>Negative</th>
<th>Positive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subtype (BCC only)</td>
<td>Nodular, superficial</td>
<td>Micronodular, infiltrating, sclerosing</td>
</tr>
<tr>
<td>Degree of differentiation</td>
<td>Well differentiated</td>
<td>Moderately or poorly differentiated</td>
</tr>
<tr>
<td>Adenoid, adenosquamous or desmoplastic (SCC only)</td>
<td>Negative</td>
<td>Positive</td>
</tr>
<tr>
<td>Depth: Clark level or thickness (SCC only)</td>
<td>I, II, III or &lt;4mm</td>
<td>IV, V or &gt;4mm</td>
</tr>
</tbody>
</table>

**Area L:** Low risk: trunk, extremities  
**Area M:** Medium risk: cheeks, forehead, neck, scalp  
**Area H:** High risk: central face, eyelids, eyebrows, periorbital, nose, lips, chin, mandible, periauricular, ears, genitalia, hands and feet
Hello.
ATOPIC DERMATITIS

Major Features (3 of 4 present)

Pruritus (polished nails/subungual debris)
Typical morphology/distribution of skin lesions
Chronic or chronically relapsing dermatitis
Personal or family history of atopy

Minor Features

Xerosis/asteatosis/dryness of skin
AD ichthyosis/palmar hyperlinearity
‘Type I’ skin test reactivity
Elevated IgE/positive RAST tests
Cheilitis/conjunctivitis/blepharitis
Infraorbital fold/’nasal’ salute
Facial pallor/erythema
White dermographism
ATOPIC DERMATITIS

**Therapeutic ladder**

- Emollients (maintenance and relapse prevention)
- Irritant avoidance and recognition of other trigger factors
- Treatment of associated bacterial, viral or fungal infections
- Oral antihistamines for antipruritic and sedative effects
- Topical corticosteroids/intralesional corticosteroids
- Topical calcineurin inhibitors
- Other topical including coal tar derivatives
- Narrowband UVB phototherapy
- Systemic corticosteroids (short term)
- Cyclosporine
- Azathioprine
- Methotrexate/mycophenolate
- Interferon/ intravenous immunoglobulin
- Biologic agents (efalizumab, infliximab, omalizumab)
ATOPIC DERMATITIS

Interaction between environmental and genetic factors
• Hygiene hypothesis – Auto allergy IgE x link
• Defective epidermal barrier and bacterial colonisation
• Acute stage: Th2 cells and cytokines. Chronic stage: Th1.
• Loss of function mutations in the filaggrin gene – keratin aggregation
Psoriasis/pityriasis rubrapilaris
Seborrhoeic dermatitis
Asteatotic eczema
Venous syndrome dermatitis
Nummular dermatitis
Pityriasis alba
Pityriasis rosea
Juvenile plantar dermatosis
Parapsoriasis/superficial scaly
dermatitis
Impetiginised eczema
Eczema herpeticum
Dermatophytosis/eczema herpeticum
Rosaceous dermatitis (POD)
Lichen planus/lichen planopilaris
Keratosis pilaris/follicular ichthyosis
Lichen simplex chronicus
Nodular prurigo
Contact irritant/allergic dermatitis
Photosensitivity dermatitis
Chronic actinic dermatitis
Drug eruption
Graft versus host disease
Systemic lupus erythematosus
Dermatomyositis
Pemphigus foliaceus
Cutaneous T-cell lymphoma
Histiocytosis (Langerhans cell)
Primary immune deficiencies
Metabolic and genetic disorders
HIV associated dermatosis
Scabies/cutaneous larva migrans
Sarcoid/TB/syphilis
Dermatitis artefacta