MELANOCYTIC NAEVI, VASCULAR NAEVI, AND OTHER BENIGN SKIN TUMOURS

DR L F WENTZEL

Importance

i) To distinguish from malignant or premalignant tumours
ii) Cosmetic considerations
iii) When troublesome, ie in areas of friction
iv) Indicative of underlying conditions

MELANOCYTIC NAEVI

Melanocytes migrate from the embryonal neural crest to the epidermis, dermis, leptomeninges, retina, mucous membranes, inner ear and vestibular system. Naevus cell precursers form clusters of cells in the epidermis, dermis, or subcutaneous tissue.

Epidermal melanocytic lesions

i) Ephelis (freckle)
ii) Naevus spilus
iii) Lentigo simplex
iv) Senile lentigines

Cellular naevi

Common moles (cellular or naevocytic) appear in the first years of life; increase over 20 – 30 years, and then decline. Females have more than males; whites more than blacks. More common in relatively sun-exposed areas.

Histological position of melanocytes determines the type of mole:

i) Epidermo-dermal junction (junction naevus)
ii) Cell nests in the dermis (intradermal naevus)
iii) Both positions (compound naevus)

Evolution takes place from junction to intradermal naevi.

Many associated abnormalities, ie enlarged hair.

(Malignant degeneration discussed under "Malignant melanoma").

Benignancy suggested by:

i) diameter 3 mm or less
ii) uniform pigmentation
iii) uniform border
iv) soft epidermis
v) unchanging size or colour
vi) histology: melanocytes in nests, not singly

Evaluation of naevi.

i) Clinical examination
ii) Dermoscopy (10-fold magnification; oil placed on lesion)
iii) Computerized image analysis systems (Mole Max)
iv) Excision biopsy

Dermal melanocytic lesions

i) Mongolian Spot
ii) Naevus of Ota
iii) Naevus of Ito
iv) Blue naevus

VASCULAR NAEVI

Telangiectatic (capillary) naevi (capillary malformation)

Naevus flammeus (port-wine stain). Commonest site: unilateral distribution on face. Present at birth; blanches on diascopic pressure.
Naevus flammeus nuchae ("stork bite"): congenital lesion on midline of posterior neck.
Midline naevus flammeus ("salmon patch"): on glabella, or one upper eyelid; fades during childhood.
Sturge-Weber-Syndrome: port-wine stain in ophthalmic division of trigeminal nerve, associated with a cerebrovascular defect (encephalo-trigeminal angiomatosis)

Treatment of naevus flammeus:

Pulsed dye laser
Cosmetic concealment

Infantile haemangioma (Strawberry or "capillary" haemangioma)

Present at birth in one third of cases; in the rest it appears between two weeks to two months. Commonest on head and neck, but may appear anywhere. Dome-shaped, red tumour, which grows for a year, remains for a period, then involutes spontaneously (30% by third year; 50% by fifth; 70% by seventh).

Treatment:

Spontaneous involution mostly yields better result than intervention.
Intervention allowed in special circumstances, ie haemorrhage; thrombocytopaenia; cardiovascular compromise; interference with vital functions (feeding, respiration, excretion, limb movement, vision); or disfigurement.

Treatment:

i) Oral prednisone or beta blockers
ii) Intralesional steroids
iii) IFN–α 2a or 2b: danger of spastic diplegia

iv) Topical imiquimod
v) Ultrasound
vi) Laser

Kasabach Merritt Syndrome

Haemangioma (with lymphatic component) and thrombocytopaenia.
Average age: seven weeks
Bleeding into haemangioma itself, or chest or abdominal cavities.
Often precipitated by surgery.

Treatment:

Systemic steroids, IFN–α 2a, cytostatic agents (vincristine, vinblastine, cyclophosphamide, etc); excision; compression.

OTHER BENIGN TUMOURS

Naevus sebaceus

Congenital, commonest on scalp.
Yellow colour, warty surface; manifests as bald, yellow patch. Adnexal tumours often associated with it; occasionally basal cell carcinoma.

Epidermal naevus

Congenital, linear lesions; brown in colour, with verrucous surface; esp on limbs. Epidermal hypertrophy.

Neurofibromatosis

Autosomal dominant condition, presenting with pigmented café-au-lait macules, axillary freckles, neurofibromas (from peripheral nerve Schwann cells), and Lisch nodules in the irides. 85% of patients belong to type 1 (typical); type 2 is characterized by bilateral acoustic neuromas, without skin lesions; type 3 (mixed) and type 4 (variant) have both acoustic neuromas and skin lesions.
Associated abnormalities: mental retardation, epilepsy, acoustic neuroma, cerebral glioma, spinal cord abnormalities, endocrine disorders (ie phaeochromocytoma).

Tuberous sclerosis (epiloia).

Autosomal dominant condition with variable penetrance.
Triad: adenoma sebaceum, mental retardation, epilepsy.
Adenoma sebaceum: angiofibromas, esp nasolabial fold.
Other cutaneous signs: nail-fold fibromas, connective tissue naevi (shagreen patches), ash leave macules.

Diverse skin tumours

i) Kerato acanthoma: looks like squamous cell carcinoma, but regresses within months.
ii) Squamous cell papilloma: small, warty lesion.
iii) Campbell de Morgan spots: capillary proliferation.
iv) Seborrhoeic keratosis (basal cell papilloma): common after age 40.
v) Acrochordons (fibro-epithelial polyps): in areas of friction, ie neck, axillae.
vi) Lipoma: soft, poorly circumscribed, often large.
vii) Pyogenic granuloma: develops rapidly in area of trauma; soft red nodule, bleeds easily.
viii) Epidermoid cyst: fluctuating, tense cyst.
ix) Milium: small subepidermal keratin cyst.
x) Dermatosis papulosa nigra: small, dark facial papules, common in Africans.
xi) Chondrodermatitis nodularis chronica helicis: painful, degenerative papule on ear.