*ABC of Dermatology*P K BUXTON
D KEMMETT

BLACK SPOTS IN THE SKIN

Compound naevi.

Any dark lesion of the skin may be a cause for concern, sometimes regarded by the patient with the same dread as Long John Silver's "black spot" in *Treasure Island*—a sign of imminent demise.

Most of us acquire "moles" or pigmented naevi in childhood and early adult life. Most of these fade gradually in old age. These naevi are composed of cells derived from melanocytes of the epidermal basal layer, with a variable amount of connective tissue as well.

The clinical appearance varies from a flat pigmented macule (melanocytic naevus) to a protruberant nodule bearing hair (compound naevus) with variable proportions of pigmented naevus cells and connective tissue. Malignant change is very rare. The exception is found in patients with the uncommon dysplastic naevi syndrome, in which multiple naevi develop during adolescence, many of which show the features of malignant change outlined below.

Benign naevi

Congenital hairy naevus.

Blue naevus.

Halo naevus.

Spitz naevus.

Congenital pigmented naevi present at, or soon after, birth as dark hairy lesions, which become more protruberant in time. There is a tendency to malignant change. The very large lesions covering much of the trunk or buttocks (bathing trunk naevi) present considerable problems in their removal.

Other benign pigmented naevi include the following.

The blue naevus, presenting as a stable, deep blue, dermal lesion in children and young adults.

The Spitz naevus is a fleshy pink papule in children—it is composed of benign monomorphic spindle cells. The old name of juvenile melanoma should be abandoned.

A *halo naevus* consists of a central melanocytic naevi with a depigmented halo, caused by an autoimmune reaction. The central naevus gradually involutes. Vitiligo may be present elsewhere.

Becker's naevus consist of a faintly pigmented area, often on the upper trunk of shoulder with an increase in hair density. It is benign.

Freckles or *ephelides* are small (less than 0.5 cm) pigmented macules that occur on exposure to sunlight in fair skinned people and fade during the winter.

Becker's naevus.

Malignant melanomas

Melanoma is an invasive malignant tumour of melanocytes. Most cases occur in adults over the age of 30 with fair complexions. The condition is also more common in women, in whom melanomas often occur on the lower leg. The table on the next page shows the criteria for suspecting malignant changes in pigmented lesions. Any change in a skin lesion is a reason to assess the lesion carefully, and if more than two of the features in the table are present it is generally accepted that the patient should be referred for a specialist opinion.

Criteria for suspecting malignant changes in pigmented lesions

- (1) *Itching*—Normally a mole does not itch but a melanoma may. Irritated seborrhoeic warts also itch.
- (2) *Inflammation* is, of course, a feature of irritated or infected benign lesions, but it occurs in melanoma spontaneously, particularly in the early stages.
- (3) *Size*—Apart from congenital pigmented naevi, most benign moles are less than 1 cm in diameter. Any lesion over 0.5 cm should be carefully checked.
- (4) *Growth*—Benign pigmented naevi continue to appear in adolescents and young adults. Any mole increasing in size in an adult over the age of 30 may be a melanoma.
- (5) *Shape*—Moles usually have a symmetrical, even outline, any indentations being quite regular, but melanomas usually have an irregular edge with one part advancing more than the others.
- (6) *Colour*—Variation in colour of benign moles is even but a melanoma may be intensely black or show irregular coloration varying from white to slate blue, with all shades of black and brown. Inflammation may give a red colour as well. The "amelanotic" melanoma shows little or no pigmentation.
- (7) *Surface breakdown* with bleeding and crusting occurs in an actively growing melanoma.

If more than two of these features are present refer the patient for a specialist opinion

A simple summary:

- A—Asymmetry of the lesion
- B—irregularity of the Border
- C—variations in Colour
- D—Diameter, larger than 0.5 cm

The commonest clinical type is a spreading lesion with irregular surface and pigmentation.

The nodular variety is less common but grows more rapidly and is more likely to be confused with benign moles.

The even flat brown lentigo on the face of elderly patients grows slowly over the years. However, a nodular raised portion indicates the development of more rapidly growing invasive melanoma and the need for urgent treatment.

Talon noir.

Acral melanoma occurs on the soles and palms and under the nails (subungual melanoma). Benign pigmented naevi may also occur in these sites, but the seven points in the table will help to differentiate them. Talon noir, due to haemorrhage from trauma—for example, playing squash—may cause much concern but the distinct blood filled papillae seen on paring the lesion make the diagnosis plain.

The prognosis of malignant melanoma is related to the depth of invasion. Superficial lesions with a tumour thickness of less than 1 mm have a good prognosis if removed early (80-90% survival at five years). Histologically, the thickness of the tumour is measured from the granular layer (Breslow thickness). Deeper invasion carries a poor prognosis (only 40-50% survival for a tumour deeper than 3 mm).

It is therefore very important to remove any malignant melanoma while it is in the superficial stage.

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New Drugs

Thrombolytic treatment and new calcium antagonists

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Injury to a blood vessel results in the formation of a haemostatic plug of platelets, and fibrin is laid down to reinforce it to form a thrombus. Once the repair process is established the fibrinolytic system is activated to remove fibrin. Tissue plasminogen activators produced at the site of the injury convert plasminogen to active plasmin, which lyses fibrin. The plasmin produced is active only within the thrombus, and any leakage is destroyed by circulating antiplasmins in the plasma. Thus the fibrinolytic system plays an important part in maintaining the patency of the vascular tree.

Thrombolytic treatment

Two thrombolytic agents—namely, streptokinase and urokinase—are available for clinical use.¹ Streptokinase, the metabolic product of β haemolytic streptococci, is, unlike urokinase (isolated

from human urine), antigenic in man. Streptokinase combines with plasminogen to form an activator complex, which converts plasminogen to plasmin, while urokinase activates plasminogen directly. The generalised lytic state caused by both of these drugs results in the dissolution of thrombi, but it also produces a haemostatic defect throughout the entire vascular system. For this reason these drugs should not be given to patients who are actively bleeding or who have had recent surgery, stroke, or cardiopulmonary resuscitation. Uncontrolled hypertension and active peptic ulceration are also contraindications to treatment.

In recent years other drugs have been developed that act locally at the site of thrombus formation and have a lesser systemic effect. Recombinant tissue plasminogen activator (t-PA) produced by recombinant deoxyribonucleic acid technology from human tissue cultures specifically activates plasminogen already bound to fibrin, and its action on circulating plasminogen is limited. A second drug, anisoylated plasminogen streptokinase activator complex (APSAC), is inactive in plasma. When it attaches to fibrin deacylation occurs, plasminogen activator complex is formed, and plasmin production is localised to the thrombus.

Myocardial infarction

Coronary angiography performed in patients within hours of the onset of pain has confirmed that coronary artery occlusion is the cause of myocardial infarction in most patients.² This has supported the view of pathologists that the rupture of an atheromatous plaque

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