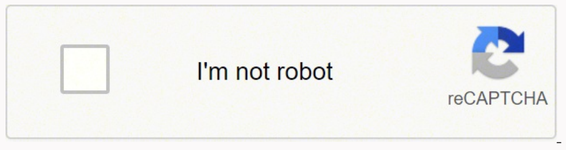

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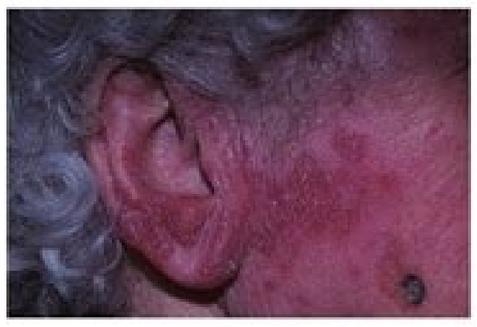

Fig. 14.8 A plaque with the brownish tinge characteristic of lupus vulgaris. Diascopy was positive.

plaque slowly enlarges, and can damage deeper tissues such as cartilage, leading to ugly mutilation. Scarring and contractures may follow.

Diascopy (p. 32) shows up the characteristic brownish 'apple jelly' nodules and the clinical diagnosis should be confirmed by a biopsy. A warty variant exists.

Scrofuloderma

The skin overlying a tuberculous lymph node or joint may become involved in the process. The subsequent mixture of lesions (irregular puckered scars, fistulae and abscesses) is most commonly seen in the neck.

Tuberculides

A number of skin eruptions have, in the past, been attributed to a reaction to internal foci of tuberculosis. Of these, the best authenticated—by finding mycobacterial DNA by polymerase chain reaction (PCR)—are the 'papulonecrotic tuberculides'—recurring crops of firm dusky papules, which may ulcerate, favouring the points of the knees and elbows.

Erythema induratum (Bazin's disease)

In erythema induratum, deep purplish ulcerating nodules occur on the backs of the lower legs, usually in women with a poor 'chilblain' type of circulation. Sometimes this is associated with a tuberculous focus elsewhere. Erythema nodosum (p. 101) may also be the result of tuberculosis elsewhere.

Investigations

Biopsy for:
- microscopy (tuberculoid granulomas);
- bacteriological culture; and
- detection of mycobacterial DNA by PCR.

Mantoux test.

Treatment

The treatment of all types of cutaneous tuberculosis should be with a full course of a standard multidrug antituberculosis regimen. There is no longer any excuse for the use of one drug alone.

Prevention

Outbreaks of pulmonary tuberculosis are reminders that this disease has not yet been conquered and that vigilance is important. Bacillus Calmette–Guérin (BCG) vaccination of schoolchildren, immunization of cattle and pasteurization of milk remain the most effective protective measures.

Leprosy

Cause

Mycobacterium leprae was discovered by Hansen in 1874, but has still not been cultured *in vitro*, although it can be made to grow in some animals (armadillos, mouse foot-pads, etc.). In humans the main route of infection is through nasal droplets from cases of lepromatous leprosy although, interestingly, some cases have occurred in Louisiana from eating infected armadillos.

Epidemiology

Some 15 million people suffer from leprosy. Most live in the tropics and subtropics, but the ease of modern travel means that some cases are seen in northern Europe and the USA.

Presentation

The range of clinical manifestations and complications depends upon the immune response of the patient (Fig. 14.9). Those with a high resistance develop a paucibacillary tuberculoid type (Fig. 14.10) and those

5 Psoriasis

One to three per cent of most populations have psoriasis, which is most prevalent in European and North American white people, uncommon in American black people and almost non-existent in American Indians. It is a chronic non-infectious inflammatory skin disorder, characterized by well-defined erythematous plaques bearing large adherent silvery scales. It can start at any age but is rare under 10 years, and appears most often between 15 and 40 years. Its course is unpredictable but is usually chronic with exacerbations and remissions.

Cause and pathogenesis

The precise cause of psoriasis is still unknown. However, there is often a genetic predisposition, and sometimes an obvious environmental trigger.

There are two key abnormalities in a psoriatic plaque: hyperproliferation of keratinocytes; and an inflammatory cell infiltrate in which neutrophils and TH-1 type T lymphocytes predominate. Each of these abnormalities can induce the other, leading to a vicious cycle of keratinocyte proliferation and inflammatory reaction; but it is still not clear which is the primary defect. Perhaps the genetic abnormality leads first to keratinocyte hyperproliferation that, in turn, produces a defective skin barrier (p. 11) allowing the penetration by, or unmasking of, hidden antigens to which an immune response is mounted. Alternatively, the psoriatic plaque might reflect a genetically determined reaction to different types of trauma (e.g. physical wounds, environmental irritants and drugs) in which the healing response is exaggerated and uncontrolled.

To prove the primary role of an immune reaction, putative antigens (e.g. bacteria, viruses or autoantigens) that initiate the immune response will have to be identified. This theory postulates that the increase in keratinocyte proliferation is caused by inflammatory cell mediators or signalling. Theories about the pathogenesis of psoriasis tend to tag along behind fashions in cell biology, and this idea is currently in vogue.

Genetics

A child with one affected parent has a 16% chance of developing the disease, and this rises to 50% if both parents are affected. Genomic imprinting (p. 301) may explain why psoriatic fathers are more likely to pass on the disease to their children than are psoriatic mothers. If non-psoriatic parents have a child with psoriasis, the risk for subsequent children is about 10%. In one study, the disorder was concordant in 70% of monozygotic twins but in only 20% of dizygotic ones. These figures are useful for counselling but psoriasis does not usually follow a simple Mendelian pattern of inheritance. The mode of inheritance has therefore to be categorized as genetically complex, implying a polygenic inheritance.

Psoriasis is also genetically heterogeneous. Early onset psoriasis shows an obvious hereditary element and linkage analysis (p. 300) revealed the first psoriasis susceptibility locus (*PSI*), on 6p—close to the major histocompatibility complex Class I (MHC-I) region, but probably not HLA-C itself. The risk of those with the HLA-CW6 genotype developing psoriasis is 20 times that of those without it; 10% of CW6+ individuals will develop psoriasis. Other MHC-I associated diseases include Behçet's disease, ulcerative colitis and anterior uveitis. Interestingly, T-cell mediation is also seen in these diseases. The hereditary element and the HLA associations are much weaker in late-onset psoriasis.

Untreated lesions usually clear in 6–9 months, often after a brief local inflammation. Large solitary lesions may take longer. Some leave depressed scars.

Complications

Eczematous patches often appear around mollusca. Traumatized or overtreated lesions may become secondarily infected.

Differential diagnosis

Inflamed lesions can simulate a boil. Large solitary lesions in adults can be confused with a keratoacanthoma (p. 262), an intradermal naevus (p. 259), or even a cystic basal cell carcinoma (p. 266). Confusion with warts should not arise as these have a rough surface and no central pore.

Investigations

None are usually needed, but the diagnosis can be confirmed by looking under the microscope for large swollen epidermal cells, easily seen in unstained preparations of debris expressed from a lesion.

Treatment

Many simple destructive measures cause inflammation and then resolution. They include squeezing out the lesions with forceps, piercing them with an orange stick (preferably without phenol), and curettage. Liquid nitrogen may also be helpful.

These measures are fine for adults, but young children dislike them and it is reasonable to play for time using imiquimod or chlortetracycline cream, or instructing the mother carefully how to apply a wart paint once a week to lesions well away from the eyes.

LEARNING POINTS

1 If you cannot tell mollusca from warts, buy a lens.
2 Do not hurt young children with mollusca. You will not be able to get near them next time something more serious goes wrong.

Sometimes a local anaesthetic cream (EMLA; see Formulary 1, p. 339), under polythene occlusion for an hour, will help children to tolerate more attacking treatment. Sparse eyelid lesions can be left alone but patients with numerous lesions may need to be referred to an ophthalmologist for curettage. Common sense measures help to limit spread within the family.

Orf

Cause

Contagious pustular dermatitis is common in lambs. Its cause is a parapox virus that can be transmitted to those handling infected animals. The condition is therefore most commonly seen on the hands of shepherds, of their wives who bottle-feed lambs, and of butchers, vets and meat porters.

Presentation and course

The incubation period is 5–6 days. Lesions, which may be single or multiple, start as small firm papules that change into flat-topped apparently pustular nodules with a violaceous and erythematous surround (Fig. 14.31). The condition clears up spontaneously in about a month.

Complications

- Lymphadenitis and malaise are common.
- Erythema multiforme.
- 'Giant' lesions can appear in the immunosuppressed.

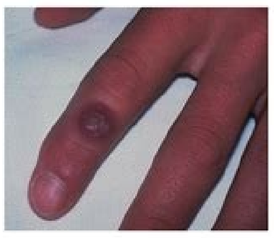

Fig. 14.31 The pseudopustular nodule of orf.

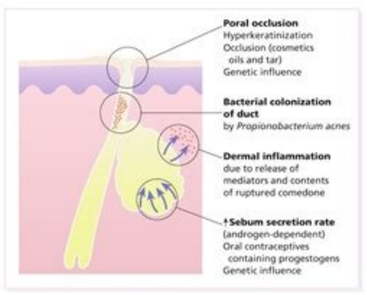

Fig. 12.1 Factors causing acne.

and free fatty acids. Rupture of these follicles is associated with intense inflammation and tissue damage, mediated by oxygen free radicals and enzymes such as elastase, released by white cells.

- *Bacterial. Propionibacterium acnes*, a normal skin commensal, plays a pathogenic part. It colonizes the pilosebaceous ducts, breaks down triglycerides releasing free fatty acids, produces substances chemotactic for inflammatory cells and induces the ductal epithelium to secrete pro-inflammatory cytokines. The inflammatory reaction is kept going by a type IV immune reaction (p. 26) to one or more antigens in the follicle.
- *Genetic*. The condition is familial in about half of those with acne. There is a high concordance of the sebum excretion rate and acne in monozygotic, but not dizygotic, twins. Further studies are required to determine the precise mode of inheritance.

Variants of acne

- *Infantile acne* may follow transplacental stimulation of a child's sebaceous glands by maternal androgens.
- *Mechanical*. Excessive scrubbing, picking, or the rubbing of chin straps or a fiddle (see Fig. 12.2) can rupture occluded follicles.
- *Acne associated with virilization*, including clitoromegaly, may be caused by an androgen-secreting

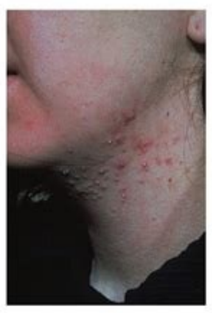

Fig. 12.2 Papulopustular lesions in an odd distribution. The patient played the violin ('fiddler's neck').

tumour of the adrenals, ovaries or testes or, rarely, to congenital adrenal hyperplasia caused by mild 21-hydroxylase deficiency. The gene frequency for this autosomal recessive disorder is high in Ashkenazi

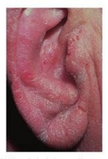

Fig. 7.20 Dry scaly seborrhoeic eczema of the ear.

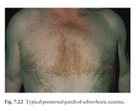

Fig. 7.22 Typical presternal patch of seborrhoeic eczema.

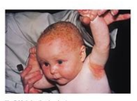

Fig. 7.23 Infantile seborrhoeic eczema.

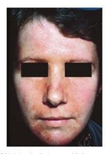

Fig. 7.21 Active seborrhoeic eczema of the face.

3 Intertriginous lesions of the armpits, umbilicus or groins, or under spectacles or hearing aids.

Cause

This condition is not obviously related to seborrhoea. It may run in some families, often affecting those with a tendency to dandruff. The success of treatments directed against yeasts has suggested that overgrowth of the pityrosporum yeast skin commensals plays an important part in the development of seborrhoeic eczema. This fits well with the fact that seborrhoeic eczema is often an early sign of AIDS, and that it responds to antiyeast agents such as topical ketoconazole shampoo or cream.

Seborrhoeic eczema may affect infants (Fig. 7.23) but is most common in adult males. In infants it clears quickly but in adults its course is unpredictable and may be chronic or recurrent. Some particularly severe

Sign guidelines hyperlipidemia.

All patients currently in this regime can continue if they have been stable for at least a year, statins should not be prescribed with geifibrizil patients who use drugs that influence the metabolism of cytochrome P450 should avoid concomitant use of atorvastatin or simvastatin. In such cases, pravastatin or rosuvastatin are acceptable to alternative therapies that lassants of lipids should not be offered to: patients with active liver disease or persistent liver tests patients who report intolerance to the statin can be rewarded, if arranged, initially With the same dose of the same statin, unless they have a significant increase in creatine chinase if the intolerance to the statin reported persists, patients should be offered an alternative statin that lowers lipids for special groups with family hypercolemiaiamia should be offered the statin therapy regardless of their calculated cardiovascular risk and can be considered for the therapy combined with Ezetimibe in which the lid-lid-logical is inadequate on the therapy of the maximum tolerated statin or for monotherapy in which the statins are contraindicated with hypercolterol heterozygous individuals heterozygous family members the Emia and the High LDL cholesterol despite the statin monotherapy or the therapy of combination of statins/ezetimibe should be considered for patients with PCSK9 inhibitors with CKD in Stadium 3 and superior, or with micro or macroalbuminuria, which are not in dialysis should be offered in therapy Ezetimibe and bile statins, kidnapping therapy should be considered only for primary prevention in patients at risk of high CVDs in which statins therapy is contraindicated and in patients with family hyperchosterolaemia ezetimibe and seizure therapy of biliary acid should be considered for the of the second based on the basis It is considered inadequately controlled fibers are not usually recommended for the primary or secondary prevention of the treatment of cardiovascular diseases with a fibrato should be considered in individuals with: â ¢ cvd or which are at high cardiovascular risk and marked hypertriglyceridemia and nicotinica at the level of Low cholesterol The acid is not recommended for the reduction of cardiovascular risk in any group of group PCSK9 inhibitors should be considered in patients at high risk of vascular events with cholesterol levels that remain above target levels despite others tolerated Therapy with lipids lowering all individuals with persistent blood pressure Â â ¥ 140 /90 mm Hg or a family history of hypertension should receive advice on lifestyle to help reduce blood pressure and the risk of CVD. Lifestyle consultancy should continue even when drug therapy is started blood pressure thresholds for individuals with symptomatic cardiovascular disease individuals with clinical evidence of cardiovascular diseases and supported clinical systolic pressure> 140 mm systolic and/or diastolic Hg> 90 MM should be offered drug therapy that I shoot the blood pressure that have had a hemorrhagic or ischemic stroke, or Tia should be offered drugs that drop for blood pressure, even when their basal blood pressure is at a level that would be considered conventionally normotesi, to reduce the risk of recurrence blood pressure thresholds for individuals without symptomatic cardiovascular diseases high -cardiovascular risk individuals, but without consolidated CVD and supported clinical systolic blood pressure> 140 mm Hg Sistolica and/or diastolic blood pressure> 90 mm Hg with hypertension on the c ui risk of ten years A first CVD event is below the threshold for the consideration of drug therapy should continue with life strategies and have blood pressure and total total The risk has re -evaluated every or five years, depending on the clinical circumstances with clinical blood pressure greater than 160 mm systolic HG or 100 mm diastolic HG should be offered anti -expertise treatment and specific lifestyles advice to lower blood pressure and risk Cardiovascular blood pressure thresholds for specific high -cardiovascular risk groups, individuals with diabetes should be offered a treatment that is prepared for the bloodocyst pressure treatment that lowers blood pressure, even if the systolic clinic is

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