MIDDLE AGE (35 - 65 YEARS)

Persons of middle age probably represent the bulk of patients who present with dermatological problems.

Steven J Glassman
BSc, MB BCh, FCDerm (SA)
Consultant Dermatologist
Division of Dermatology
Department of Medicine
University of the Witwatersrand and
Johannesburg Hospital

Steven Glassman obtained a BSc and MB BCh from Wits, and served his internship at Hillbrow Hospital. He obtained the FCDerm in 1996. A year was spent as a resident at New York University Medical Center Dermatology Department in 1997. Since then he has been a full-time consultant at Johannesburg Hospital with a limited private practice. He enjoys teaching medical students and registrars the exciting field of clinical dermatology in the new millennium. Steven enjoys hiking, classical music and philately.

At this busy time, when family and work commitments reach a peak, skin problems can cause significant distress or disability, with potentially serious consequences. The concept of a 'skin examination' as a proactive measure has recently emerged, particularly for the early detection of skin cancer. Such promotive efforts by patients are to be encouraged.

ALLERGIC CONTACT DERMATITIS

Eczematous skin changes (itch, redness, swelling, blisters, crusts) of sudden onset, and at localised sites such as the face, eyelids, hands and feet, are suggestive of allergic contact dermatitis. This is a true type IV allergy (delayed hypersensitivity). Sensitisation can take months or years. Once sensitised, subsequent exposure is rapidly followed by clinical symptoms and signs of dermatitis at the site of contact, with distant spread via the hands. Common causes include nickel, cement, leather, rubber additives (not latex), glues, fragrance, medicaments, plants and nail polish (Fig.1). Diagnosis requires an elaborate history, and is confirmed with patch tests. In the case of cosmetics, a 'use test' is very helpful: the suspected product is applied twice daily to the inner forearm for 7 days. Allergic contact dermatitis is an important industrial dermatosis, and compensation or medical boarding may be necessary. Treatment is aimed at finding and avoiding the allergy; symptomatic treatment with corticosteroid tablets, creams or ointments is dependent on the severity of the reaction. The condition can be severe with frank blistering, pain and secondary infection. Soaks with tepid tap water or dilute potassium permanganate solution are helpful, together with a short course of oral steroids (e.g. prednisone (Meticorten, Panafcort, Pulmison) 60 mg daily for 5 days), a potent steroid cream and an oral antibiotic like cloxacillin, co-amoxyclav or cephalosporin. Note that antihistamines are not helpful.

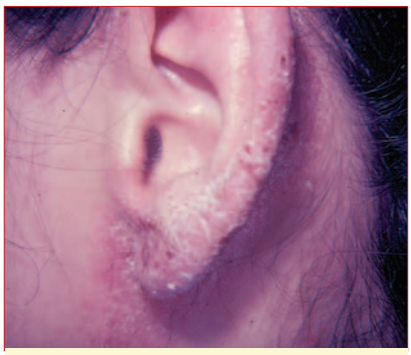

Fig. 1. Allergic contact dermatitis — earring.

ANDROGENETIC ALOPECIA

Androgenetic alopecia is thought to be an autosomal dominant condition. New hair growth becomes thinner and shorter as a result of the gradual transformation of normal-sized terminal hair follicles on the scalp to small, hypopigmented, miniaturised hair follicles, which are not lost completely. The condition occurs in men and women, starting in the teens, twenties or thirties. It is fully established in men in the forties, but can take much longer in women. Men notice hair thinning in the temporal and frontal areas, with the parietal areas and vertex affected later (Fig. 2). Women notice thinning over the vertex, with more of the scalp becoming visible. Progression to bitemporal recession and parietal loss can occur, but the frontal margin is usually retained. The condition causes considerable body-image problems and should not be brushed aside. Minoxidil solution

2% (Regaine) applied bd to the affected scalp can be used in both sexes, and can prevent further loss, or even stimulate new growth. In men the addition of 1 mg finasteride (Propecia) daily improves the situation. Baseline PSA should be obtained prior to treatment. Surgical implants are successful, but expensive. Causes of recurrent telogen effluvium (thyroid disease, drugs, iron deficiency, connective tissue diseases) should be excluded in atypical suspected androgenetic alopecia.

Fig. 2. Androgenetic alopecia — early.

BASAL CELL CARCINOMA (BCC)

Also known as a rodent ulcer because of its tendency to slow but relentless local invasion, this tumour is very common in sun-exposed Caucasians. It presents as an asymptomatic pearly, translucent nodule with central ulceration and telangiectases on the edge (Fig. 3), commonly on the nose, face, scalp, ears, neck, arms, legs and chest. Neglected lesions can be very disfiguring; metastasis is rare. Albinos develop particularly aggressive BCCs. Pigmented BCCs can mimic melanomas. Treatment depends on site and size of the tumour, and the age of the patient. Surgical excision with a 5 mm clinical margin is a good option; in uncertain cases a small incisional or punch biopsy can be done beforehand. Small lesions (< 2 cm) respond well to curettage and electrodesiccation. Imiquimod cream (Aldara) works well for biopsy-proven superficial BCCs. Other options for troublesome or recurrent BCCs include Mohs' surgery and radiotherapy. BCCs recur frequently no matter which treatment option is chosen, usually within the first 2 years after treatment. Frequent check-ups are advisable.

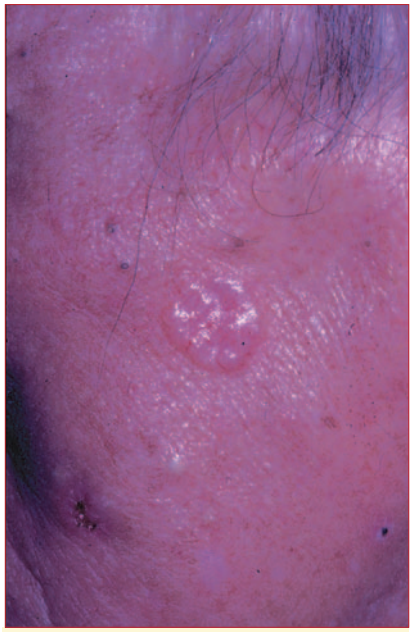

Fig. 3. Basal cell carcinoma.

BOWEN'S DISEASE

This is a squamous cell carcinoma (SCC) *in situ* in chronically sun- exposed skin, and presents as an indolent but slowly enlarging erythematous scaly plaque resembling psoriasis or eczema (Fig. 4). It may be several centimetres in diameter, and can be pigmented in dark-skinned individuals. Unlike inflammatory dermatoses, Bowen's disease is seldom itchy, and does not respond to topical steroid therapy. Multiple lesions are typical of previous arsenic exposure (cattle dip, Fowler's solution). Bowen's disease eventually progresses into invasive SCC. Definitive diagnosis requires an incisional or punch biopsy, and the treatment of choice is excision, but smaller lesions can be treated with liquid nitrogen cryotherapy, curettage and electrodesiccation, 5-fluorouracil ointment (Efudix) or imiquimod cream (Aldara).

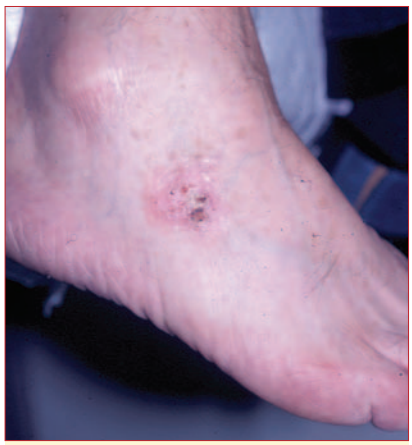

Fig. 4. Bowen's disease.

DERMATITIS ARTEFACTA

See the article on teenagers, pp. 507 - 511

DERMATOMYOSITIS

In the presence of a characteristic rash the sudden development of severe proximal muscle weakness should alert one to possible dermatomyositis: purplish-red discolouration and swelling of the eyelids, photosensitive erythema of the face and chest, erythematous papules on the knuckles and erythema and superficial erosion of the shoulders and thighs (Fig. 5). Note that the erythematous rash is similar to that seen in systemic lupus erythematosus. Nail-fold telangiectasia is also typical. The rash can precede the myopathy by months, or occur simultaneously.

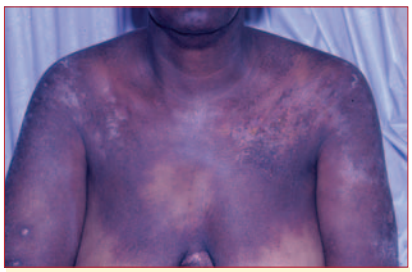

Fig. 5. Dermatomyositis — photosensitive rash.

Diagnosis is aided with skin and muscle biopsy and serological tests (ANA), and a search for underlying malignancy is warranted, as dermatomyositis in adults may be a paraneoplastic phenomenon (~30%). Treatment consists of high-dose oral or parenteral corticosteroids, sometimes with methotrexate, azathioprine or chloroquine. Healing with cutaneous

calcification is particularly seen in childhood dermatomyositis.

DRUG ERUPTION

Adverse cutaneous reactions to oral medications are extremely common, and among the leading causes of stopping or changing chronic medication. Reactions range from trivial and self-limiting, to severe and potentially fatal. Drug eruptions are best classified descriptively, because there are therapeutic and prognostic differences between the various reactions. Drug eruption should be suspected when an unexpected, usually itchy, generalised rash occurs soon after starting a new drug. In reality the situation is complicated by the use of several new drugs at the same time, or the concurrent use of established medicines with the introduction of a new agent. Most reactions occur within 3 weeks of starting a new drug, but this is not always the case. Viral infections like infectious mononucleosis can alter one's susceptibility to drug eruption, and HIV infection greatly increases the risk. Occasionally a family history of drug allergy is relevant. Other causes of generalised rashes, notably viral exanthemata, must be excluded if possible. The following drug eruptions will be described briefly: morbilliform, urticarial, Stevens-Johnson syndrome, toxic epidermal necrolysis, fixed eruption, vasculitis, photosensitivity, exfoliative dermatitis and lichenoid eruptions.

Morbilliform rashes are the commonest type of drug eruption, and account for about 75% of cases. Itchy, erythematous macules and barely perceptible papules start in dependent sites like the legs and lower back, and become generalised (Fig. 6). On withdrawing the drug the rash gradually fades over 1 - 2 weeks. If the causative agent is continued many reactions will also subside, but a chronic erythematous rash can progress to exfoliative dermatitis. Virtually all drugs can cause morbilliform rashes, but the most frequent agents are antibiotics. Antiretrovirals are an important emerging cause. Treatment consists of drug withdrawal, antihistamines and topical steroid creams or gels.

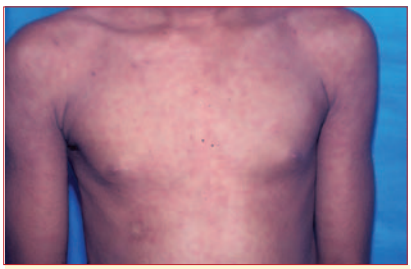

Fig. 6. Morbilliform drug eruption.

Urticarial reactions present with itchy, erythematous, oedematous weals or hives anywhere on the skin, and can represent true type I allergy caused by IgE antibodies. Angioedema and anaphylaxis can occur within minutes of exposure to the causative drug. Again, antibiotics are the major cause, especially penicillins. Other important causes include ACE inhibitors, NSAIDs, sera, blood, radiocontrast medium and latex. Urticaria is managed according to the severity of the reaction, and adrenaline may be required. In milder cases oral or parenteral antihistamines like promethazine suffice. In all cases the suspected drug should be withdrawn and never given again. A Medic-alert bracelet is mandatory.

Stevens-Johnson syndrome is a severe form of erythema multiforme with a fulminant presentation including fever, malaise, sore throat, red eyes and a painful and itchy generalised rash. See the section on erythema multiforme in the article on toddlers, pp. 496 - 498. Stevens-Johnson syndrome normally resolves in 1 - 3 weeks with postinflammatory pigmentation but no scarring. A Medic-alert bracelet is mandatory to prevent recurrence.

Toxic epidermal necrolysis (TEN) is similar to Stevens-Johnson syndrome, but has more diffuse skin involvement, and variable mucous membrane involvement. It starts with the sudden onset of painful and tender generalised erythema, which rapidly progresses to blistering and denudation in large sheets (Fig. 7). There is appreciable mortality, especially in the elderly, infirm or immunosuppressed. The most important causes of TEN are sulphonamides, ampicillin, amoxycillin, antituberculous drugs, NSAIDs, allopurinol, phenytoin, phenobarbitol and phenolphthalein laxatives. Treatment is as for severe burns, and largely supportive. There is evidence that cyclosporin, administered early, is effective. TEN in South Africa has also become much more common because of HIV infection.

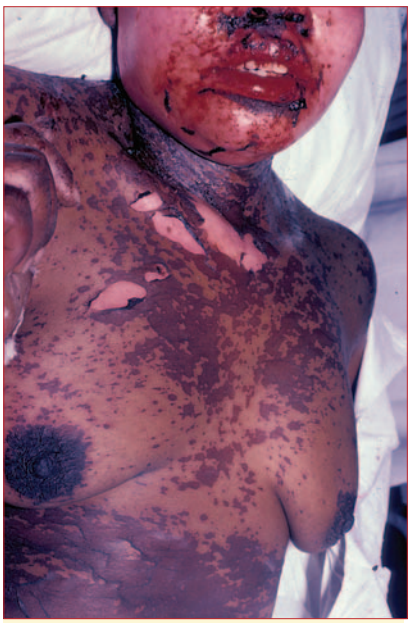

Fig. 7. Toxic epidermal necrolysis.

Fixed drug eruption is a distinctive eruption of oval, erythematous, oedematous plaques, sometimes with central blisters, in the acute phase (Fig. 8), resulting in slate-coloured pigmentation, and on re-exposure the acute reaction occurs at the same site (hence 'fixed'), with further pigmentation afterwards. Common sites include the face, lips, hands, feet and genitalia. Severe cases can mimic Stevens-Johnson syndrome, but the mucosal involvement is much less, and the patient is not as ill. Common causes include ampicillin,

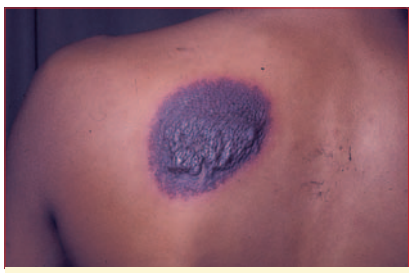

Fig. 8. Fixed drug eruption.

aspirin, paracetamol, barbiturates, muscle relaxants, metronidazole, NSAIDs, oral contraceptives, phenolphthalein laxatives, sulphonamides and tetracyclines. Treatment is aimed at drug avoidance; the pigmentation fades very slowly. In the acute phase potent topical steroids are useful.

Vasculitis can be a drug reaction, and usually represents leukocytoclastic vasculitis, presenting with palpable purpura on dependent sites, and can progress to blistering and ulceration. Symptoms and signs of systemic vasculitis can occur. Agents implicated include allopurinol, antibiotics, furosemide, gold, NSAIDs, phenytoin and thiazides. Skin biopsy is important to establish the diagnosis of this sometimes severe reaction, which can also be caused by non-drug factors. Treatment is with non-adherent dressings, antihistamines and oral steroids in severe cases.

Photosensitivity is often triggered by drugs and can be either phototoxic or photoallergic. Phototoxicity is exaggerated sunburn (Fig. 9). Common causes include doxycycline (NB: malaria prophylaxis + holiday in the sun), NSAIDs, benzodiazepines, quinolones, amiodarone and phenothiazines including promethazine and hydroxyzine. Ketoprofen gel and other topical agents can also cause phototoxicity. It is self-limiting but can be severe and painful, but does not recur with subsequent sun exposure unless the same agent is taken. Photoallergic reactions are insidious and chronic, and present as intensely itchy eczematous patches mainly, but not only, on sun-exposed sites. The condition can easily be mistaken for a chronic eczema like atopic or seborrhoeic eczema. Several commonly used drugs can cause photoallergy, including sulphonamides, thiazides, sulphonylureas, furosemide, NSAIDs and phenothiazines. Topical medicaments and plants can also cause photoallergy. The condition can be persistent even after drug withdrawal. Treatment is difficult in these cases, and involves oral and topical steroids, possibly including immunosuppressants. Mild cases are treated with UVB sunscreens, sun avoidance, physical cover-up and potent topical steroids.

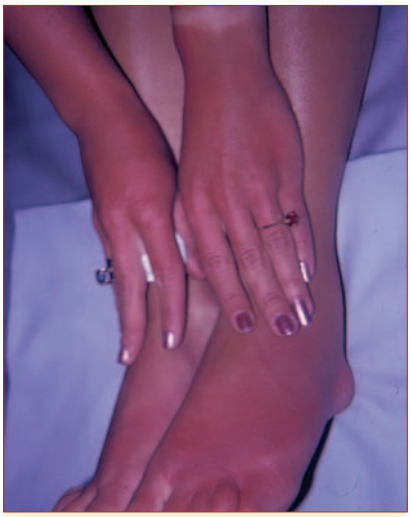

Fig. 9. Phototoxicity.

Drugs are an important cause of **exfoliative dermatitis** or erythroderma, where nearly the entire skin surface is itchy, red and scaly. Other causes like eczema, psoriasis and lymphoma should be excluded, and skin biopsy is helpful. Sulphonamides, isoniazid, ampicillin, anticonvulsants, ACE inhibitors, cimetidine, furosemide, gold, lithium, NSAIDs, penicillamine and thiazides are predominant causes. In addition to drug withdrawal, treatment includes the use of topical and systemic corticosteroids, emollients and antihistamines.

Lichenoid drug eruptions consist of erythematous to violaceous papules and plaques, often in a photosensitive distribution, resembling idiopathic lichen planus (Fig. 10). Skin biopsy is usually necessary for diagnosis, and common causes include chloroquine, chlorpropamide, furosemide, gold, methyldopa, phenothiazines, thiazides, captopril and beta blockers. Drug withdrawal and treatment with potent topical steroids are usually sufficient.

Drug hypersensitivity syndrome (also termed DRESS: **D**rug **R**eaction with **E**osinophilia and **S**ystemic **S**yptoms) refers to a drug eruption together with systemic reactions like fever, lymphadenopathy and involvement of various internal organs. Any of the preceding rashes can represent the skin manifestation of the syndrome. Hepatitis, glomerulonephritis and pneumonitis can occur. Major causes include aromatic anticonvulsants (phenytoin, phenobarbital, carbamazepine), sulphonamides, dapsone, salazopyrine, minocycline and allopurinol. Treatment with oral steroids for a protracted period is usually recommended.

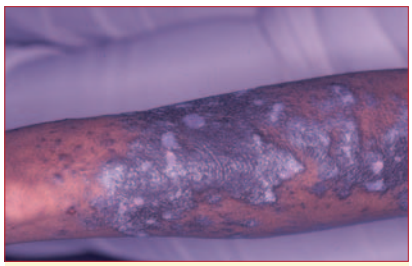

Fig. 10. Lichenoid drug eruption.

EPIDERMAL CYST

These very common cystic swellings are derived from hair follicles and can occur anywhere on the body. They grow to a certain size and then remain quiescent for an indefinite period, during which time they cause no symptoms. At this stage the cyst is a firm dome-shaped, non-tender swelling with a central dilated hair follicle orifice, the so-called punctum, through which sebum can be expressed. They can be surgically removed or expressed through an incision. The cyst can become inflamed or secondarily infected. At this stage incision and drainage under local anaesthesia is used for pain relief, and a formal excision is done later. Cysts on the scalp are often pilar cysts rather than epidermal cysts, and have a thick-walled capsule that does not rupture easily and can be easily expressed through a small incision.

ERYSIPELAS

Erysipelas is a superficial form of cellulitis caused by *Streptococcus pyogenes* and most frequently affects the lower leg or face. The portal of entry is often an insignificant fissure between the toes from tinea pedis.

Fever and malaise precede the development of a painful, red, hot, well-demarcated area of swelling, often with overlying haemorrhagic bullae (Fig. 11). The differential diagnosis includes deeper cellulitis and in the case of the leg, deep vein thrombosis. Treatment includes leg elevation, cool compresses and preferably intravenous penicillin G. Alternatives include intramuscular benzathine penicillin or oral antibiotics like penicillin, amoxycillin, cephalosporin or erythromycin. If oral therapy is chosen at least 3 weeks is necessary. If cellulitis is suspected, an anti-staphylococcal agent like cloxacillin should be added. Erysipelas is often recurrent, and with each attack lymphatics can be destroyed, causing lymphoedema which can predispose to further attacks.

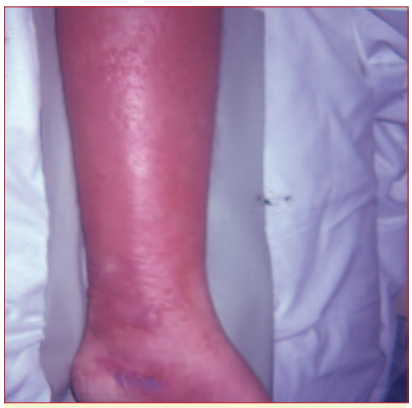

Fig. 11. Erysipelas — leg.

ERYTHEMA INDURATUM

Chronic, tender, inflamed nodular swellings on the lower legs, particularly the calves, point to the possibility of erythema induratum. This form of panniculitis, or inflammation of the fat, is much commoner in women and is thought to represent an immunological reaction to a focus of active tuberculosis in the lungs or elsewhere. The nodules can ulcerate and cause scars (Fig. 12). Often there are no overt signs of tuberculosis. A skin biopsy of a nodule can be helpful to exclude other causes of panniculitis. The tuberculin test should be strongly positive, and sometimes empirical anti-TB treatment is advisable.

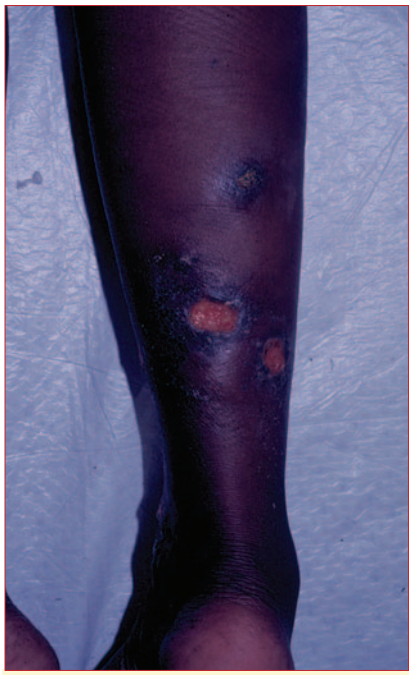

Fig. 12. Erythema induratum.

ERYTHEMA NODOSUM

This form of panniculitis is generally milder, and of shorter duration than erythema induratum, affecting mainly women. Fever, malaise and arthralgia of the ankles herald the onset of the disease, which also causes tender, red, hot nodular swellings on the lower legs, especially the shins (Fig. 13). The condition is usually self-limiting, lasting several weeks, the nodules healing with bruising. The commonest cause is streptococcal infection but other causes include *Yersinia* and other intestinal infections, urinary tract infections, tuberculosis, systemic fungal infections like histoplasmosis, sulphonamides, oral contraceptives and sarcoidosis. Treatment entails finding the cause if possible, together with bed rest and NSAIDs. Often no obvious cause is apparent, and it might be prudent to give a broad-spectrum antibiotic like a cephalosporin. On occasion erythema nodosum is recurrent or persistent.

GROVER'S DISEASE

This is a common cause of intense, episodic itching on the upper trunk in fair-skinned men with chronic sun exposure. Small, erythematous papules, vesicles and crusts are seen on affected areas like the chest and upper back (Fig.14). The itching is exacerbated by heat and sweating. The condition is easily confused with other itchy dermatoses and ideally a skin biopsy should be performed. Treatment is difficult, and entails potent topical steroids like clobetasol propionate cream (Dermovate, Dovate). In most cases Grover's disease resolves spontaneously in a few months, but recurrences are common.

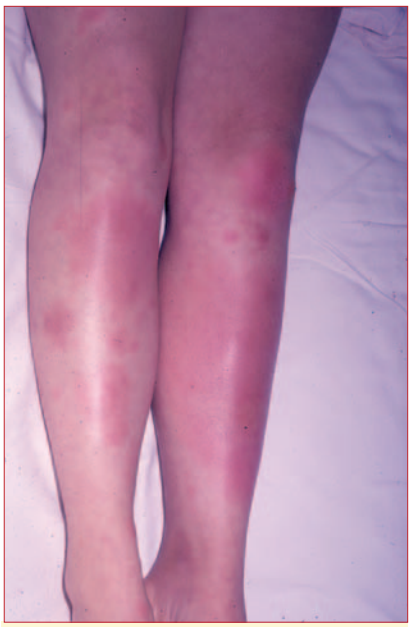

Fig. 13. Erythema nodosum.

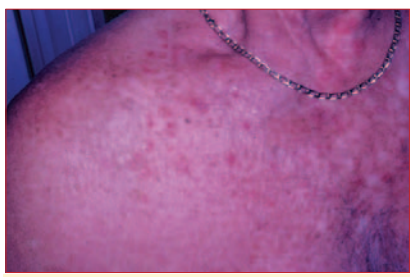

Fig. 14. Grover's disease.

HIRSUTISM

Hirsutism is the development of male-type terminal hairs in women in androgen-dependent sites like the upper lip, chin, beard area, lower abdomen and thighs (Fig.15). In most cases this is a normal familial trait, but can be most prominent at the time of puberty, pregnancy or menopause. In these cases circulating ovarian and adrenal androgens are normal. Pathological causes of hirsutism, with