Pruritus

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Educational aims

The object of this paper is:

- To provide a comprehensive overview of the protean nature of pruritus as a manifestation of normal physiology, of diseased internal organs, and of mental aberrations as well as of very many skin diseases proper.
- To provide an overview of the pathophysiology, clinical features, and conventional therapies of the commoner conditions.

Keywords: pruritus, causes of pruritus, treatment of pruritus

Everybody is familiar with pruritus. It is defined formally as an unpleasant sensation which provokes scratching. Indeed, the biological advantage of pruritus seems to lie in the elimination of cutaneous parasites by the scratching which it provokes. Pruritus is the predominant symptom in dermatology.[1]

Clinical pruritus varies in quality, mode of onset, intensity, duration, anatomical location, provoking factors, as well as in its relationships to time, emotion, bathing, physical exertion, sleep and so many other everyday activities. Itch is part of the normal human condition, but the pruritus of skin disease can be very annoying and it impairs the quality of life.[2] Severe, intractable pruritus of metabolic origin may drive the patient to distraction. Pruritus is always perceived as arising from the skin itself, as indeed it most often does, for example following insect bites. Pruritus may, however, arise from sources outside the skin like organic lesions of the central nervous system (e.g. brain tumour), from the action of circulating chemical mediators on receptors in the central nervous system (e.g. opioid peptides of cholestasis), or for purely psychogenic reasons (e.g. parasitophobia). The evaluation of pruritus may therefore constitute a difficult diagnostic task. Pruritus of primary skin disease is usually associated with a characteristic eruption which can be diagnosed by a careful history and physical examination (e.g. urticaria; atopic dermatitis) or with the help of simple bedside procedures like Wood's light illumination (e.g . cat ringworm) or microscopy (e.g. scabies). Biopsy or mycology may additionally be required to clinch the diagnosis. However, pruritus may be due to underlying systemic problems like thyroid disease, lymphoma, or cholestasis. Excoriations may be abundant in these cases, but there is no specific rash visible to help with the diagnosis. Haematology, blood chemistry, and imaging are important in the diagnosis of such patients and their further management mandates liaison with internists. In all cases of pruritus the treatment is primarily of the underlying condition, but general measures to relieve symptoms are also applicable when, as is often the case, no specific remedy is available.

Pathophysiology

Pain and pruritus are distinct modalities but they share broadly similar molecular mechanisms and neural pathways. The signals generated in the free nerve endings near the dermo-epidermal junction by external physical and chemical agents or by the pruritogenic mediators of skin disease (such as histamine, neuropeptides, opioid peptides, proteases, prostaglandins, cytokines and others) are carried along unmyelinated nerve fibres via the dorsal root ganglion of the spinal cord to the spinothalamic tract of the contralateral side whence they ascend to the thalamus. The signals are then relayed to the cerebral cortex where the sensation of pruritus is perceived in consciousness. In the spinal cord, the electric activity of these ascending fibres is modulated by descending myelinated fibres carrying sensory signals of touch and pressure also originating in the skin (e.g. by the act of scratching). Thus the flow of itch and pain traffic is regulated by means of a metaphorical 'gate' on its journey to the cerebral cortex.[3] Increased gating may explain why scratching provides temporary relief from pruritus. It may also explain the beneficial effect of acupuncture and trans-cutaneous nerve stimulation in the relief of both pain and pruritus, and why pruritus cannot be induced in areas of skin previously exposed to pain. On the other hand, reduced gating may explain why the itch threshold is reduced in patients with dry skin. It may also explain why mild mechanical stimuli

which do not provoke pruritus in normal skin can produce severe pruritus in the skin of atopic individuals (an empirical observation called 'allokenesis'). The important role of central factors in modulating pruritus has been further emphasized by more recent discoveries which highlight the role played by opioid peptides (mentioned above already in their peripheral role) and serotonin in modulating the traffic of pain and itch in the central nervous system. Thus, morphine administered spinally or epidurally frequently causes intense facial pruritus which can be blocked by the intravenous injection of its antagonist naxolone. Contrariwise, serotonin antagonists reduce itching, including that induced by morphine. These central mechanisms offer opportunities for therapeutic manipulation by pharmacological substances, but the anatomy and physiology of the central perception and regulation of pruritus is still fragmentary and awaits further investigation.[4]

Pruritus as a manifestation of systemic disease

Pruritus is common among patients with chronic uraemia, especially if they are on dialysis. The itch bears no relation to levels of serum urea or creatinine. Many plausible mechanisms have been implicated in the pathogenesis, but the real cause has yet to be identified. Ultraviolet therapy (UVB) offers valuable symptomatic relief for pruritus of renal origin.[5]

Pruritus also generally accompanies a decrease or arrest in the flow of bile, while hepatitis B and C may cause pruritus in the absence of overt liver disease. Bile salts, histamine and opioids may play a part in the pathogenesis of cholestatic pruritus. Exchange resin drugs and the anti-opioid naxolone relieve cholestatic pruritus.

Hyper- and hypo-thyroidism can both induce pruritus, as can iron deficiency and mastocytosis. Up to 10% of patients with recalcitrant generalized pruritus may have an underlying malignancy, usually of the lympho-reticular system, and occasionally it may be the presenting complaint.[6] Patients with HIV disease frequently complain of pruritus. It can be severe and may occur early in the course of the disease. Many healthy people experience a distressing amount of pruritus following exposure to water, irrespective of its temperature. A small number of these may have underlying polycythaemia rubra vera or myelodysplasia.[7]

Generalized pruritus of sudden onset in otherwise healthy individuals and in the absence of simple causes mandate a complete history, physical examination as well as routine laboratory studies. Extensive imaging and endoscopic studies should be reserved for patients with intractable pruritus. Generalized pruritus may persist for several years without issue, but follow-up examination of patients with chronic pruritus is important in monitoring the emergence of systemic disease.

Psychogenic pruritus

Due to its visibility and accessibility for manipulation, the skin may serve as a vehicle of a person's psyche. Emotional disturbances can be readily transformed into obsessive-compulsive dermatological manifestations like pruritus and hair pulling.[8] A diagnosis of psychogenic pruritus may be entertained only after skin pathology or an underlying medical disease is very carefully excluded.

Emotionally disturbed individuals may suffer from a reduced threshold to pruritus leading to an itch-scratch vicious cycle which results in a defensive thickening of the epidermis called lichenification. The ano-genital area is an archetypal site of involvement in both sexes, producing well-defined clinical presentations labelled pruritus vulvae, pruritus scroti, and pruritus ani. These may be mis-diagnosed as ringworm simply because they occur in the vicinity of the groin. Physical signs in the genital areas are often mis-interpreted as evidence of a sexually-transmitted condition by neurotic individuals as well as by those who may have good reason to fear that they may have acquired one. Psychosomatic pruritus may prove recalcitrant to the best of therapies. In addition, misuse of topical antibiotics, anaesthetic, and steroid creams may be complicated by allergic contact dermatitis (see below).

Delusion of parasitosis is an unusual and rather bizarre mono-symptomatic phobia in which the patient is incontrovertibly convinced of being infested by parasites and often brings along minute specimens (in reality consisting of fluff or skin scales) to prove it. The psychotropic drug pimozide is helpful if the patient can be cajoled into taking it, but psychiatric referral is usually resented and opposed due to lack of insight.

Pruritus in pregnancy

Pregnancy may influence the course of a pre-existing pruritic disease like eczema, possibly for the better. Nevertheless, there are some pruritic conditions that are peculiar to pregnancy itself.[9] Pruritus, in fact, occurs quite commonly in pregnancy.

The usual variety of pruritus, called prurigo gravidarum, affects mainly the lower abdomen. There are excoriations but no specific rash. It may develop at any stage of pregnancy, but occurs particularly in the last three months. Liver function is normal. The foetus is not at risk, but the condition may re-occur in subsequent pregnancies.

Obstetric cholestasis is a less common but potentially more serious situation in which the function of the liver is deranged, and the total serum bile acids is raised.[10] The pruritus which it provokes is worse at night, and involvement of the palms and soles is a distinguishing feature. Obstetric cholestasis occurs most frequently during the last month of pregnancy. Possible foetal complications like unexplained stillbirth and postpartum haemorrhage may be prevented by induction at 38 weeks. Obstetric cholestasis may re-occur in subsequent pregnancies.

The polymorphic eruption of pregnancy, also known as pruritic urticarial papules and plaques of pregnancy (PUPP), occurs quite commonly. It is a very itchy urticarial rash which occurs late in the course of the first pregnancy. It starts typically in the abdominal stretch marks which look very red. The area around the umbilicus is usually not involved. The rash consists of the red papules, plaques, and weals of the title. It occurs more frequently in twin pregnancies but the foetuses are not at an increased risk. PUPP disappears shortly after delivery and it does not occur in subsequent pregnancies.

Pemphigoid gestationis (herpes gestationis)is a rare auto-immune disorder which usually starts around the umbilicus in the second trimester but which may commence in the puerperium. The rash consists of very itchy papules and polycyclic weals which eventually turn into sup-epidermal blisters and which become distributed symmetrically over a very wide area. Clinically, pemphigoid gestationis resembles bullous pemphigoid of the elderly but there are other important differences apart from the age of onset. Essentially the foetus is not at risk, but may be born with transient blisters from trans-placental

passage of antibodies. Systemic steroids are usually necessary to control pemphigoid gestationis in the mother. Remission generally occurs a month after delivery, but the condition is liable to relapse in subsequent pregnancies and there may also be exacerbations with menstruation or oral contraceptives.

Urticaria

Urticaria is characterized by itchy swellings which come up anywhere on the skin and leave without a trace after a variable length of time, only to be replaced by others while the disease remains active. The swellings are due to transient leakage of plasma from small dermal blood vessels. This is mediated by histamine and other vasoactive pruritogenic agents which are released by degranulation of dermal mast cells and basophils. Degranulation may be the result of an immunological process like the activation of IgE receptors on the surface of mast cells by autoantibodies. However, other substances can provoke degranulation by a direct effect without invoking an immunological process. These include aspirin, codeine, non-steroidal anti-inflammatory drugs, and dietary pseudoallergens including salicylates, azo dyes, and preservatives. Superficial swellings of the skin are called weals . Deeper swellings of the skin and alimentary tract are called angio-oedema. Angio-oedema often involves the lips and eyelids. It is more painful than itchy, and it tends to last longer. Weals and angio-oedema may occur on their own but usually they occur together. Urticaria is a great nuisance but generally it does not produce systemic symptoms. Patients, who may have been warned to dread the onset of laryngeal oedema, should be reassured on this point. On the other hand, urticaria occurs commonly during the course of dangerous systemic anaphylactic reactions which may, for example, follow an allergic reaction to penicillin.

There are several clinical varieties of urticaria.[11] In ordinary urticaria weals occur spontaneously, with or without angio-oedema, practically on a daily basis. Individual weals typically last from 2 to 24 hours. The course of ordinary urticaria may be short-lived, or intermittent, or long-lasting. Physical urticarias are triggered by physical stimuli. Varieties of physical urticaria include symptomatic dermographism, delayed pressure urticaria, vibratory angio-oedema, cholinergic urticaria, cold contact urticaria, aquagenic urticaria, and solar urticaria. In physical urticarias swellings are induced rather than spontaneous. The weals of physical urticaria last for one hour except in delayed pressure urticaria where they start much later and persist for longer. Contact urticaria occurs when an allergen is absorbed through the skin or mucous membrane. This is the basis of allergy to latex which may produce anaphylaxis in patients who are highly sensitive to the material. Weals last up to 2 hours. In urticarial vasculitis there is damage to small blood vessels due to impaction with circulating immune complexes. Urticarial vasculitis is suspected when individual weals persist for more than 48 hours. Urticaria may also be a presenting feature of the auto-inflammatory syndromes which are accompanied by fever, malaise and other features pertaining to each particular syndrome. Angio-oedema without weals may be caused by angiotensin converting enzyme inhibitors. It may also be a manifestation of C1 esterase inhibitor deficiency which is a rare hereditary autosomal dominant condition.

The diagnosis of urticaria is essentially clinical. Laboratory investigations should be guided by the history and interpreted in the clinical context. In chronic ordinary urticaria a white cell count may detect the eosinophilia of parasitic infestation or the leucopaenia of systemic lupus erythematosus. The erythrocyte sedimentation rate may be raised in urticarial vasculitis. Thyroid autoantibody and thyroid function tests may be positive in urticaria of auto-immune origin. However, many cases of chronic urticaria remain unexplained despite thorough investigation.

Many cases of ordinary, physical and vasculitic urticarias tend to resolve spontaneously with time. Meanwhile non-specific aggravating factors should be avoided. The modern generation of long-acting non-sedating H1 histamine receptor antagonists are the mainstay of treatment in chronic idiopathic urticaria. Responses and tolerance may vary between individuals, and there is now a tendency to exceed the manufacturers' recommended doses in non- responders. In patients with liver or kidney damage some antihistamines may need to be avoided or their dosage reduced. Antihistamines which prolong the Q-T interval should not be taken concurrently with drugs that inhibit hepatic metabolism via cytochrome P450 and with drugs that have potential arrhythmic activity. The manufacturer's data sheet should be consulted in pregnancy and younger children. Concern about impaired concentration and performance has diminished the use of older generation sedating antihistamines. Oral steroids are effective in acute urticaria, but in chronic urticaria the long term risks outweigh the benefits. Epinephrine can be life-saving in anaphylaxis. Immunosuppressive therapy may be considered for some patients who are disabled by chronic auto-immune urticaria which has not responded to conventional therapy.

Pruritus due to ectoparasites

Biting insects are a ubiquitous source of human misery not only by dint of the irritation they provoke but also because they are the vectors of deadly epidemics. Plague, typhus, and malaria, to name a few, have altered the course of human history down the ages. This review will ignore the wider picture and focus its attention on pediculosis, scabies, and insect bites as recurring motifs in the differential diagnosis of pruritus in any dermatology clinic.

Pediculosis

Pediculosis is an infestation caused by two species of human lice – Pediculus humanus and Phthirus pubis. Pediculus humanus can affect the head (pediculosis capitis) or the body (pediculosis corporis) but in sanitized affluent societies the latter are hardly ever seen under normal circumstances except among vagrants (vagabond disease). By comparison, head lice are ubiquitous.

Head lice are particularly common in all socio-economic groups of schoolchildren among whom there has been a recrudescence in recent decades. Head lice spread from one person to another by general physical contact or by fomites (combs, brushes, headbands, hats). The female attaches the eggs (nits) to the proximal end of the hair shafts to which they remain strongly stuck. The blood-sucking activity of adult lice induces an allergic reaction. The scalp then becomes intensely itchy and urticated papules may occur around the hair margin, particularly the nape. In neglected cases, scratching may result in secondary bacterial infection with enlargement of

regional lymphnodes. Adult head lice are difficult to see because they move around so quickly, but the identification of grayish nits containing the developing nymphs is diagnostic. Wet combing may be used to harvest adults in dubious cases.

Phthirus pubis, known colloquially as the 'crab louse' due to its appearance, is usually transmitted between human adults during sex. The infestation occurs primarily in the pubic hair but may spread to contiguous areas in hairy men. The combination of genital pruritus, nits, squat lice grasping pubic hairs, and black specs of dessicated iron-containing faeces spotting the underpants are diagnostic.

Topical applications containing malathion 0.5 %, carbaryl 0.5%, or pyrethroids effectively kill adult lice and developing nymphs despite their protected location.[12] Treatment should be repeated after a week to eliminate nymphs emerging from surviving nits. Family contacts should be treated simultaneously. The smell and stinging properties of alcoholic preparations may be objectionable to some or irritant to others. Aqueous-based formulations are preferable in these cases. The brief contact conferred by shampoos may be insufficient for successful elimination of the infestation. Essential oils demonstrate variable efficacy. Nits may be physically removed with a fine-toothed comb and lots of patience. Paper white nits attached to the outer reaches of the hair shafts are empty and are not indicative of a current infestation. Outbreaks of head lice infestation should be addressed on a school-wide and community-wide basis.

Scabies

Scabies is a contagion caused by *Sarcoptes scabiei var. hominis*. It is spread by prolonged, intimate physical contact. The gravid female mite lays its eggs in a burrow beneath areas of thick stratum corneum. The wrists in adults and the soles in infants are among the sites of predilection. After a few weeks, the mite elicits an allergic immune reaction that produces a generalized papular eruption which is intensely pruritic especially in a warm bed. Secondary infection, eczema, and persistent nodules especially in the genital area may follow. Burrows in the finger webs are pathognomonic, while extraction of the mite with a needle provides solid proof. Sulphur, gamma benzene hexachloride and benzyl benzoate have been superceded by 5% permethrin and 0.5% malathion which are both effective if applied secundum artem and left to act for the stipulated period. Ivermectin by mouth may also be considered under certain circumstances.[13] In all cases, it is imperative to treat the patient's contacts to interrupt the chain of transmission.

Papular urticaria

Papular urticaria is the generic name given to the typical cutaneous response to the bites of a variety of insects, including biting flies, mosquitoes, mites, fleas, and bed-bugs. The sources vary with geographical location and this is one branch of clinical practice where a knowledge of local insect behaviour is useful. Pruritus is a cardinal symptom, and the rash usually consists of papules and weals which are sometimes surmounted by a vesicle. The punctum produced by the proboscis may be visible with a hand lens. The lesions are the result of an allergic reaction to the proteins injected by the biting insects into the wound to keep the blood flowing in their sucking apparatus. Some people do not react to these antigens, while people who are very allergic may develop large weals and bullae. Immunological tolerance to insect bites can develop over time in indigenous populations. Mosquitoes usually bite at night and the lesions are discovered on the exposed parts the morning after. The worst cases are seen in children and foreigners on short visits who sleep with the windows open in the warm months. Other typical scenarios for insect bites are at the beach during twilight and visits to a farm.

In the domestic arena animal fleas are another common source of papular urticaria. The usual natural hosts are cats, dogs, and, in Malta, rabbits. Eggs, larvae, pupae and adults get lodged in the furnishings of the environment inhabited by the infested mammals and humans get bitten when they frequent these areas which may indeed be in someone else's abode. Fleas cannot fly but they can jump a long distance compared to their size and so the ankles are the areas usually attacked. They may otherwise creep up the legs until their progress is impeded by a belt or other article of tight clothing and then they bite in that locality. The insect often bites three or four times in the same area giving rise to the typical appearance of grouped lesions sometimes referred to as 'breakfast', 'lunch', 'tea' and 'dinner'. Owners who cherish their pets are often in denial. The lengths they go to put the blame elsewhere is often a hurdle which must be overcome with tact and diplomacy. It is important to treat the infested pet as well as the areas it frequents because fleas may survive in the environment for several weeks in starving anticipation of a prey.

Eczema

Eczema (syn. dermatitis) refers to a pattern of pruritic cutaneous inflammation which can assume several forms. Atopic eczema, seborrhoeic eczema, discoid (nummular) eczema, pompholyx (dyshidrotic eczema), juvenile plantar dermatosis, lichen simplex, lichen striatus, gravitational eczema, and contact dermatitis are all types of eczema with different aetiologies and distinctive clinical features. It is impossible to do justice to them all within the remit of this review.

Morphologically, acute eczema is distinguished by erythema and vesicles or bullae which burst on the skin surface releasing serum. In subacute eczema the exudate is less profuse but nevertheless still visible as a glisten. Skin affected by chronic eczema is dry, thickened, and fissured but there are no vesicles. Excoriations, secondary bacterial infection, and residual hyper- or hypo-pigmentation may alter the clinical picture. Post-inflammatory changes in skin pigmentation may persist for a long time, especially in individuals with dark skin. Acute, subacute and chronic features may co-exist in the same individual.

Eczema is divided into exogenous and endogenous varieties where environmental and constitutional factors play dominant roles respectively. In practice, endogenous and exogenous aetiologies often coincide.

Contact dermatitis develops in response to interaction with external substances. These may act as allergens, where T-cell mediated immune responses are involved, or as irritants, where they are not. Contact dermatitis is a common cause of loss of productivity.[14]

Strong irritants produce acute dermatitis. Repeated exposure to weaker irritants produces cumulative chronic changes. This occurs most commonly on the hands of people whose occupation requires frequent contact with water, detergents and other irritants. Housewives, hairdressers, nurses, chefs, cleaners, and mechanics are at risk. Another common pattern of dermatitis is asteatotic eczema which develops on the shins of elderly people when the tendency

for dry skin is intensified by cold weather and excessive washing.

Nickel, cobalt, rubber, perfumes, nail varnish, plants, dyes, preservatives and lanolin in cosmetics, and colophony are frequent causes of allergic dermatitis in the home. Chromates in cement, resins in the plastic industry, dyes, rubber, and glues are common causes in industrial settings. Topical medicaments, including corticosteroids, may also be allergenic *(dermatitis medicamentosa)*.[15] In photo-allergic dermatitis combined exposure to a chemical and ultraviolet light is necessary for the rash to develop. Tracing the source of an allergic dermatitis may prove difficult. The distribution of the rash may be helpful since it occurs at the point of contact. Diagnosis is confirmed by patch testing. In this procedure, batteries of allergens in appropriate vehicles are loaded into small inert aluminium pans fitted onto adhesive tape strips which are applied to the patient's back and removed only after 48 hours. Dermatitis is reproduced at the application site of allergens to which the individual is specifically sensitized. Patch tests must be interpreted by experts. Epidemiological data using these results show that the frequencies of positive reactions to some common allergens like chromate have decreased due to improved legislation but that nickel allergy continues to rise.[16]

Recovery is possible if the responsible irritants or allergens are avoided, but this may be difficult. Meantime, emollients and topical steroids provide symptomatic relief. A short course of systemic steroids may be justifiable to quell very acute reactions.

In atopic dermatitis constitutional factors play an important role. It is increasingly common, chronic, and typically begins in childhood but tends to resolve after some years following episodes of remissions and relapses. More severe cases persist into adult life. Other manifestations of atopy like asthma, hay fever, urticaria and allergic conjunctivitis are commonly associated with dermatitis in the subject or close members of the family. The pathogenesis of atopic dermatitis is complex and imperfectly understood. The two traditional hypotheses implicate a primary immunological defect leading to IgE-mediated sensitization to allergens, and epidermal-barrier dysfunction. T-Helper type 2 lymphocytes play an important role in driving the immune-histochemical processes which broker the immune response. Environmental and emotional factors play additional roles. Atopic dermatitis exhibits a typical age-related distribution of skin lesions. It often affects the cheeks in early childhood. Later it settles in the flexures of the wrists, elbows, knees and feet. The skin is dry and constant scratching and rubbing produces lichenification of the affected areas.

The impaired barrier function of the skin makes patients with atopic dermatitis prone to the effect of irritants, precluding occupations where they would come into daily contact with them. Chafing garments should be avoided next to skin. The benefits of dietary manipulation, avoidance of furry pets, and elimination of house dust mites are contentious. However, they are subject to continuing evaluation along with other promising complementary therapies like silver-coated textiles to reduce staphylococcal colonization, and probiotic treatment during pregnancy and nursing to delay the onset of the disease.[17] Soap substitutes and emollients are essential in the management and there are many commercial formulations to choose from.[18] Topical steroids are still the mainstay of pharmacological treatment of inflammatory lesions, but the potential for thinning of the skin and for percutaneous absorption are of special concern in children. The aim should be to use the weakest preparation equal to the task. Combination with a topical antibiotic may be considered as staphylococcal exotoxins aggravate atopic dermatitis.[19] Rebound of dermatitis may occur on withdrawal of topical steroids. The new topical immune-modulating calcineurin inhibitors are potent anti-inflammatory substances without steroid side-effects. They inhibit T-helper lymphocyte type 1 and 2 cytokine production as well as mediator release from mast cells and basophils.[20] Pimecrolimus cream and tacrolimus ointment are topical immune-modulating therapeutic options for mild-to-moderate and moderate-to-severe atopic dermatitis respectively in situations where the benefits outweigh the risks of optimized topical corticosteroid treatment, for example in the treatment of the face. Possible short-term side-effects include local irritation and increased susceptibility to skin infections. The long term safety and place in therapy of these medications is under continuing evaluation, and the most recent recommendations on the subject by the National Institute for Health and Clinical Excellence (NICE) should be observed.[21] Bandages impregnated with zinc oxide or ichthammol are effective but inconvenient remedies. Sometimes sedative antihistamines are administered at night to reduce scratching but concerns about their effect on concentration and learning abilities have been mentioned already. Ultraviolet therapy and systemic immunosuppressive drugs in a hospital setting are reserved for intractable cases.

Lichen planus

Lichen planus is a mysterious and rather variable disease in which there is a T-lymphocyte mediated attack on the epidermis similar to that seen in a graft-versus-host reaction. The commonest pattern consists of itchy, small, shiny, polygonal, flat-topped papules with a violaceous hue sometimes following scratch marks (Koebner phenomenon). The wrists and ankles are favourite sites of involvement, but the rash may become generalized and the lesions may coalesce into sheets. The buccal mucosa often reveals a typical white lacy network on the surface. The rash usually resolves spontaneously after a few months with a variable amount of residual hyperpigmentation due to melanin granules which drop into the dermis. Clinical variants of lichen planus include thick, hypertrophic plaques below the knees; a destructive inflammation of the hair follicles which ends in unsightly and permanent hair loss in the scalp (lichenplanopilaris); permanent loss or damage to the nails; and persistent painful atrophic lesions inside the mouth which may be related to infection with hepatitis-C.[22] Anti-malarials, beta-blockers, and gold are among the drugs which are known to cause lichen planus -like eruptions. There is no specific remedy for idiopathic lichen planus, but steroids may suppress the symptoms until spontaneous remission occurs. More severe cases, especially those with scalp, nail and mucous membrane involvement may need more intensive therapy.[23]

Conclusion

Pruritus is the predominant symptom in dermatology and it is one of the important features which accounts for the impairment in the quality of life which skin diseases produce. The treatment of pruritus is usually symptomatic because the aetiology of the majority of underlying skin diseases is not known. The benefits attributed to many remedies in common use remain largely based on anecdotal evidence. Placebo effects

probably account for the perceived effects in many others. Meanwhile, the cost of treatment continues to spiral upwards while economic resources dwindle. It behoves clinicians to increase their efficiency by improving their diagnostic abilities and basing their treatment on scientific evidence. More reliable and reproducible methods for measuring treatment outcome continue to be developed and the evidence base for current remedies is being collated and scrutinised.[24] The resulting guidelines help to provide a consistent level of medical care and reassure health care politicians that doctors carry out their jobs according to the highest degree of professionalism. However, it is not possible to have for every situation an evidence-based answer and an evidence-based treatment. Many patients still need an individualized approach which is patient based with the help of an experienced practitioner.[25]

Practice points

- Pruritus is the predominant symptom in dermatology and it impairs productivity and the quality of life.
- The evaluation of pruritus may prove difficult because it can be provoked by very many unrelated skin conditions as well as internal medical and psychological disorders.
- Important systemic causes of pruritus include chronic uraemia, cholestasis, thyroid disease, HIV infection, and malignancy of the lymphoreticular system.
- Common dermatological causes of pruritus include urticaria, ectoparasites, eczema (dermatitis), and lichen planus.
- Epicutaneous patch tests may be required to confirm the source of an allergic contact eczema, but most dermatological causes of pruritus can be diagnosed and managed successfully without special investigations.
- Most remedies in dermatology are symptomatic but evidence-based guidelines promote a consistent level of care.

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