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PSORIASIS: A COMPREHENSIVE REVIEW

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ABSTRACT

Psoriasis is a disease characterized by the presence of papules and plaques over the surface of skin with variable morphology, distribution and severity. The lesions of psoriasis are distinct from these other entities and are classically very well circumscribed, circular, red papules or plaques with a grey or silvery-white, dry scale. In addition, the lesions are typically distributed symmetrically on the scalp, elbows, knees, lumbosacral area, and in the body folds. The oral manifestations of psoriasis may involve the oral mucosa or the tongue. The dorsal surface of the tongue shows characteristic red patches surrounded with a yellow white border. The relationship between eye lesions and psoriasis are the current findings in the literature. The ocular complications along with the several extracutaneous manifestations are common complications seen in psoriasis. The pathogenesis of exact relationship between these two is still controversial. Immunological studies have shown a positive relationship between T helper cells and uveitis. Various signs and symptoms of ocular psoriasis may be overlooked. Thus, a complete understanding of ophthalmic involvement is important to the comprehensive care of patients with psoriasis. Almost any part of the body can be affected in psoriasis, but the ophthalmic complications of psoriasis usually remain clinically subtle. This review highlights the various manifestations of psoriasis with their clinical sign and symptoms. Psoriasis is a chronic, multisystem inflammatory disease with predominantly skin and joint involvement. Beyond the physical dimensions of disease, psoriasis has an extensive emotional and psychosocial effect on patients, affecting social functioning and interpersonal relationships. As a disease of systemic inflammation, psoriasis is associated with multiple comorbidities, including cardiovascular disease and malignancy. The diagnosis is primarily clinical and a skin biopsy is seldom required. Depending on the severity of disease, appropriate treatment can be initiated. For mild to moderate disease, first-line treatment involves topical therapies including corticosteroids, vitamin D3 analogues, and combination products. These topical treatments are efficacious and can be safely initiated and prescribed by primary care physicians. Patients with more severe and refractory symptoms might require further evaluation by a dermatologist for systemic therapy.

KEYWORDS: Inflammatory cells; manifestations; patches; psoriasis.

INTRODUCTION

Psoriasis is a chronic inflammatory dermatitis that affects about 2% of the population. It usually appears first between the age of 15 and 30 years. The lesions are characterised by brownish-red papules and plaques which are sharply demarcated and are covered with fine, silvery white scales. Psoriatic arthritis resembling rheumatoid arthritis is produced in about 5% of cases but rheumatoid factors is absent.Psoriasis is a serious skin disorder affecting 2-3 percent of the world's population. It can be defined as inflammatory skin condition characterized by abnormal differentiation and proliferation. It is an immune disease in which environmental and genetic factors plays very important role. Psoriasis name is derived from Greek word 'psora' which means 'itch'. As discussed earlier it is inflammatory, dry, non –contagious disease which may covers entire system of person. It can be identified by scaly mariginated erythematous plaques develop on skin with symmetrical manner. Psoriasis affects on common sites such as scalp, fingers tips, plams, soles, toes, genitals, under breast, elbows, knees, shins having chances of relapse after some interval. The disease is commonly observed as white and red hues of scaly patches appearing on first layer i.e. epidermis of skin. This kind of psoriasis is known as plaque psoriasis. In some cases there are no dermatological signs and symptoms.

HISTOLOGY

Histologically the following features are observed in fully developed lesions

- Acanthosis with regular downgrowth of rete ridges to almost the same dermal level with thickening of their lower portion.
- Elongation and oedema of the dermal papillae with bordening of their tips.
- Suprapapillary thinning of stratum malpighii.
- Absence of granular cell layer.
- Prominent parakeratosis.
- Presence of Munro microabscesses in the parakeratotic horny layer is diagnostic of psoriasis.

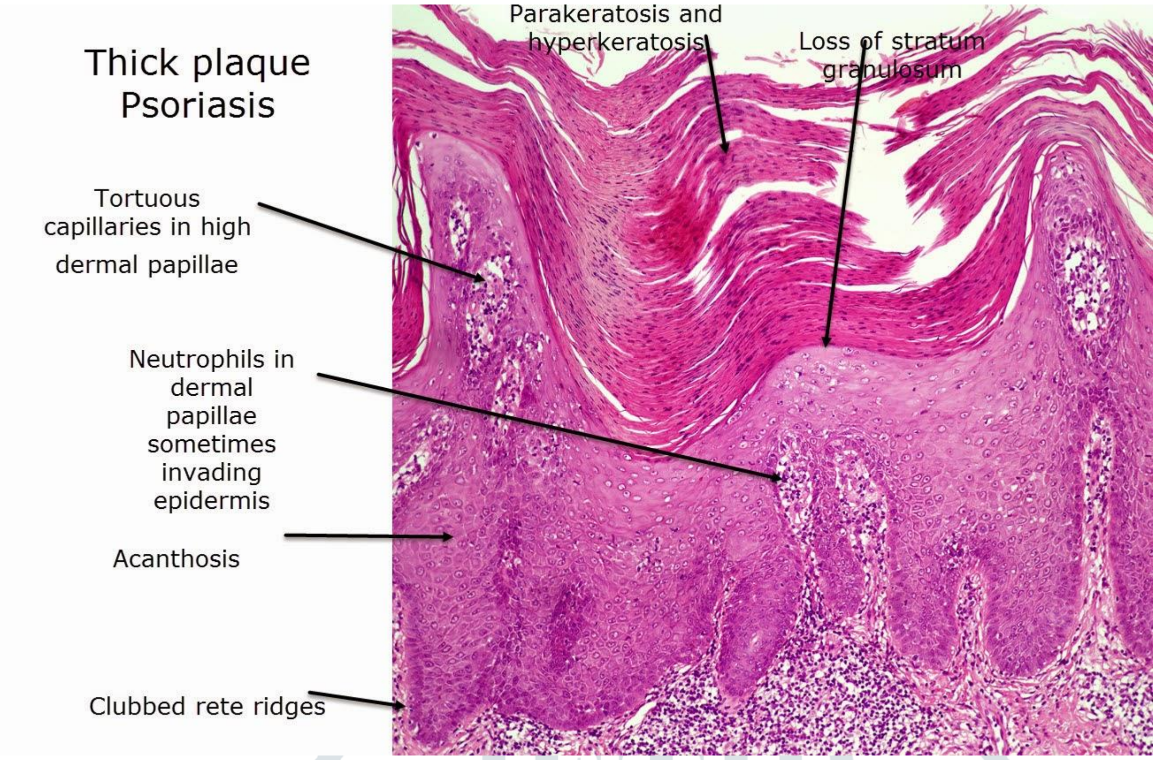

Figure 1: Psoriasiform reaction pattern

EPIDEMIOLOGY

Although psoriasis occurs worldwide, its prevalence varies considerably. In the USA, approximately 2% of the population is affected. High rates of psoriasis have been reported in people of the Faroe islands, where one study found 2.8% of the population to be affected. The prevalence of psoriasis is low in certain ethnic groups such as the Japanese, and may be absent in aboriginal Australians and Indians from South America.

Psoriasis can present at any age and has been reported at birth and in older people of advanced age. Accurate determination of the age of onset of psoriasis is problematic, as studies which do so typically rely on a patient's recall of the onset of lesions or determine the onset from the physician's diagnosis as recorded on the initial visit. Data based on patient recall can be inaccurate; determining onset based on first visit to a physician could underestimate the time of disease occurrence, as minimal disease may be present for years before a consultation is sought. A bimodal age of onset has been recognised in several large studies. The mean age of onset for the first presentation of psoriasis can range from 15 to 20 years of age, with a second peak occurring at 55–60 years.

Henseler and Christophers examined a series of 2147 patients and reported two clinical presentations of psoriasis, type I and II, distinguished by a bimodal age at onset. Type 1 begins on or before age 40 years; Type II begins after the age of 40 years. Type I disease accounts for more than 75% of cases. Patients with early onset, or type I psoriasis, tended to have more relatives affected and more severe disease than patients who have a later onset of disease or type II psoriasis. In addition, strong associations have been reported with human leucocyte antigen (HLA)-Cw6 in patients with early onset, compared with later onset of psoriasis. The course and progress of psoriasis is unpredictable. In one study, 39% of patients reported complete remission of disease for between one and 54 years. Higher figures have been reported in Japan.

The molecular genetic basis of psoriasis is complex with evidence that multiple genes are involved. Seven major psoriasis susceptibility loci have been reported. Many investigators have established that a major susceptibility locus for psoriasis is at 6p21, referred to as PSORS1 and is overrepresented in all populations tested. As noted, an association between psoriasis and other loci has also been reported on chromosomes 1p (PSORS7), 1q (PSORS4), 3q (PSORS5), 4q (PSORS3), 17q (PSORS2),and 19p (PSORS6). The strength of associations between such genes and susceptibility to psoriasis, apart from PSORS1, is variable as replication of these findings has been incomplete. The difficulty of confirming psoriasis susceptibility loci may relate, in part, to heterogeneity among different populations. Whereas the existence of a genetic component in psoriasis is certain, the exact locations of the genes involved remains to be definitely determined.

PATHOGENESIS

Any changes in the immune response in the innate and adaptive cutaneous tissue are responsible for the development of inflammation associated with psoriasis.In some patients; Cytokines and activation of innate immune system by endogenous danger signal; coexist with an auto inflammatory perpetuationsand T cell-driven autoimmune reactions in others.

Hence it can be said that Psoriasis shows the traits of an autoimmune disease with an inflammatory background with both mechanism of overlapping and even reciprocating one another. Clinical finding of psoriasis are morphological changes in the outermost layer of skin, which is made up of keratinocytes. The development of plaque psoriasis is not confined with inflammation of epidermal layer but extended by the interaction of keratinocytes with many different cells types like innate and adaptive immune cells, vasculature. The pathophysiology of psoriasis can be conceptualized into an initiation phase possibly initiated by trauma, infection drugs and a maintenance phase characterized by a chronic clinical advancement.

It is notable that dendritic cells assume a significant job in the underlying phases of ailment. Dendritic cells are proficient antigen-introducing cells. Be that as it may, their enactment in psoriasis isn’t altogether clear. One of the proposed systems includes the acknowledgment of antimicrobial peptides (AMPs), which are discharged by keratinocytes because of injury and are typically over communicated in psoriatic skin. Among the most considered psoriasis-related AMPs are LL37, defensins, and S100 proteins. LL37 or cathelicidin has been credited a pathogenic job in psoriasis. It is discharged by harmed keratinocytes, and along these lines structures buildings with self-hereditary material from other harmed cells. LL37 bound to DNA animates cost like receptor (TLR) 9 in Plasmacytoid dendritic cells (pDCs).The actuation of pDC is key in beginning the improvement of the psoriatic plaque, and is portrayed by the creation of type I (IFN-_ and IFN-). Type I IFN flagging advances myeloid dendritic cells (mDC) phenotypic development, and has been ensnared in Th1 and Th17 separation and capacity, including IFN-and interleukin (IL)- 17 creation, separately .While LL37–DNA edifices animate pDCs through TLR9, LL37 bound to RNA invigorates pDCs through TLR7. Furthermore, LL37–RNA edifices follow up on mDCs by means of TLR8. Enacted mDCs relocate into depleting lymph hubs and discharge tumor rot factor (TNF)-, IL-23, and IL-12, with the last two tweaking the separation and expansion of Th17 and Th1 cell subsets, individually. Moreover, slan+ monocytes, which are significant master fiery cells found in psoriasis skin sores, react to LL37–RNA initiation by discharging high measures of TNF-, IL-12, and IL-23. The enactment of the versatile safe reaction by means of the unmistakable T cell subsets drives the upkeep period of psoriatic aggravation. Th17 cytokines, in particular IL-17, IL-21, and IL-22 actuate keratinocyte expansion in the epidermis. The provocative milieu actuates keratinocyte expansion by means of TNF-, IL-17, and IFN-.Keratinocytes are likewise enacted by LL37 and DNA, and enormously increment the creation of type I IFNs. Moreover, they take part effectively in the incendiary course through cytokine (IL-1, IL-6, and TNF-), chemokine, and AMP emission. A broadly utilized psoriasis-like aggravation mouse model depends on the impact of the TLR7/8 agonist imiquimod, and is subsequently on the side of the TLR7/8 infection inception model. Moreover, the reaction to imiquimod was obstructed in mice lacking of IL-23 or IL-17R, which features the association of the IL-23/IL-17 hub in skin irritation and psoriasis-like pathology. The TNF_–IL-23–Th17 fiery pathway portrays plaque-type psoriasis.The IL-17 cytokine family is made out of six individuals: IL-17A–F. They are created by various cell types, and are significant controllers of incendiary reactions. Up until now, the clinically pertinent motioning in psoriasis is intervened for the most part by IL-17A and IL-17F; both act through a similar receptor, yet have various potencies. IL-17A applies a more grounded impact than IL-17F, and the IL-17A/IL-17F heterodimer has a middle of the road impact. IL-17A ties to its trimeric receptor complex made out of two IL-17RA subunits and one IL-17RC subunit, bringing about the enlistment of the ACT1 connector protein. The connection among ACT1 and the IL-17 receptor complex prompts the initiation of a progression of intracellular kinases including: extracellular sign directed kinase (ERK), p38 MAPK, TGF-beta-enacted Kinase 1 (TAK1), I-kappa B kinase (IKK), and glycogen synthase kinase 3 beta (GSK-3 beta).These kinases empower NF_B, AP-1, and C/EBP interpretation of star provocative cytokines, chemokines, and antimicrobial peptides. Th1 and Th2 cytokines act through Janus kinase (JAK)- STAT flagging pathways, though Th17 reactions are interceded by ACT1 and NF_B. On the other hand, T cells can create IL-17A autonomously of the IL-23 boost. Medications focusing on TNF, IL-23, and IL 17 and flagging pathways, for example, JAK/STAT are viable in the clinical administration of plaque psoriasis. Be that as it may, interchange incendiary pathways might be substantial for particular psoriatic variations.

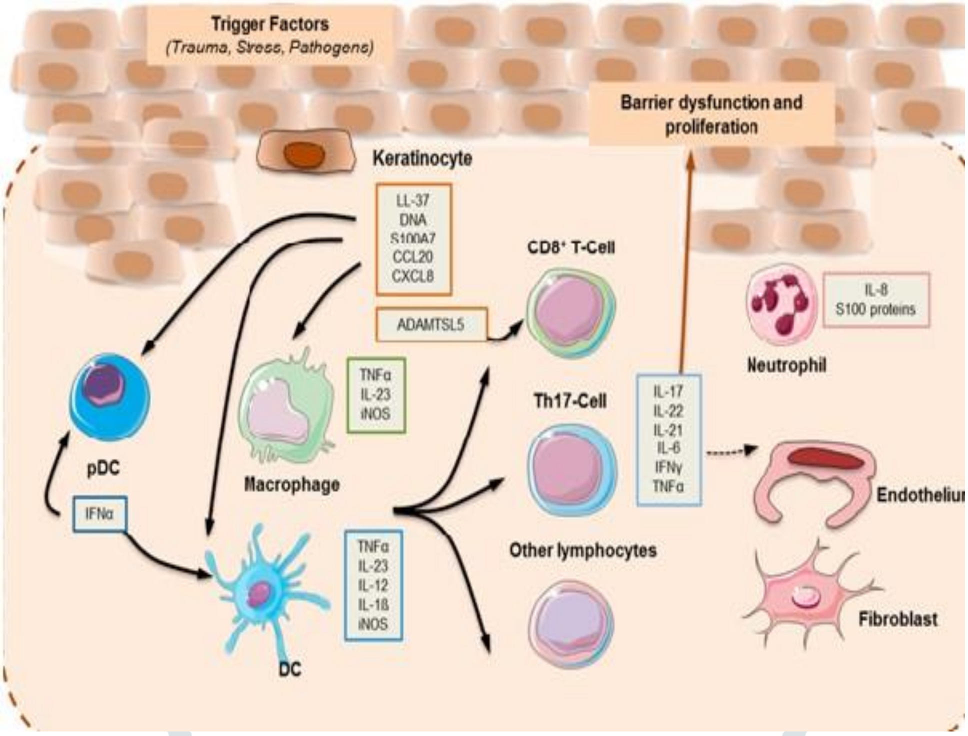

Figure 2

CLASSIFICATION OF PSORIASIS

The term psoriasis (from the Greek psora, to itch) encompasses a number of distinct clinical phenotypes, sometimes representing a dynamic, anatomical, or qualitative spectrum of the same disease (e.g., large and small plaque psoriasis), whereas, in other cases, most likely corresponds to a quite different entity (e.g., generalized pustular psoriasis [GPP]).

Historically, disease classification has been based on clinical appearance, mainly differentiating according to localization and morphology. Here, we follow the recent classification proposed by the International Psoriasis Council, which identifies four main forms of psoriasis: plaque-type, guttate, GPP, and erythroderma, and several further subphenotypes according to distribution (localized vs. widespread), anatomical localization (flexural, scalp, palms/soles/nail), size (large vs. small) and thickness (thick vs. thin) of plaques, onset (early vs. late), and disease activity (active vs. stable).

a. Plaque-Type Psoriasis :

Plaque-type psoriasis, occurring in 85%–90% of affected patients, is the most common form of psoriasis and is characterized by oval or irregularly shaped, red, sharply demarcated, raised plaques covered by silvery scales. Plaques occur mainly on the extensor surface of elbows and knees, on the scalp, and in the lower back, but can affect every area of the body, often with a symmetrical distribution. Size of the lesions can vary, from pinpoint to larger individual lesions or confluent areas leading to two clinical subphenotypes. The term large (>3 cm) plaque psoriasis describes thick (>0.75 cm), well-demarcated, red plaques with silvery scales. Small (<3 cm) plaque psoriasis presents with numerous lesions; the plaques are thinner (<0.75 cm), pinkish in color with a fine scale, and can be well-defined or merge with surrounding skin. A further classification takes into account the age of onset. Type I psoriasis has early onset (<40 yr), is often associated with familiar disease history and shows high association with the human leukocyte antigen (HLA)-Cw0602 allele, whereas type II psoriasis develops after the age of 40.

Figure 3

b . Guttate Psoriasis :

Guttate psoriasis, from the Latin “gutta” for tear drop, is characterized by multiple small scaly plaques usually occurring around the trunk and upper arms and thighs. The rash has often sudden onset, usually within 2–4 wk after a bacterial infection of the upper ways, notably streptococcal pharyngitis in children and young adults, and is therefore associated with type I psoriasis. Guttate psoriasis can either completely clear spontaneously or following topical treatment, become chronic, or worsen into the plaque type.

Figure 4

c . Generalized Pustular Psoriasis :

GPP, also known as von Zumbush psoriasis, is a rare but potentially life-threatening disease characterized by episodic, widespread skin and systemic inflammation. Typical histological feature of GPP is the presence of prominent aggregates of neutrophils infiltrating the stratum spinosum (spongiform pustules of Kogoj) and giving rise to sterile cutaneous pustules. The skin manifestations are associated with marked systemic features: high fever, fatigue, and neutrophils leukocytosis. Acute attacks often occur during pregnancy and may be triggered by infection, exposure to or withdrawal from drugs. GPP can be frequently associated with plaque-type psoriasis and/or palmoplantar pustular psoriasis. Although still classified as a variant of psoriasis, the striking clinical and histological features of GPP have long suggested that it is a disease of distinct etiology. Recent genetic data lend further support to this hypothesis with the identification of some cases of familial GPP in which the disease is inherited as an autosomal recessive trait with mutations in the IL36RN gene encoding the anti-inflammatory IL-36-receptor antagonist, IL-36Ra. IL-36Ra blocks the proinflammatory cytokines IL-36α/β/γ; when IL36RN is mutated, IL-36 signaling is uncontrolled with enhanced production of further proinflammatory cytokines. However, IL36RN mutations only occur in a minority of patients, thus, more genes are likely involved. Interestingly, a de novo mutation in the epidermal NF-κB activator CARD14 has been described to underlay a sporadic case of severe GPP, suggesting that KCs dysfunction is likely to play a predominant role in this disease phenotype.

Figure 5

d . Erythrodermic Psoriasis :

Erythrodermic psoriasis, one of the rarest forms of psoriasis (1%–2.25% of patients with psoriasis), represents the most severe phenotype; it carries substantial morbidity and can be potentially life threatening. It is characterized by diffuse erythema, with or without scaling, involving >75% of the skin surface. If present, scales are only superficial and differ from the adherent scales of plaque psoriasis. Systemic manifestations such as hypothermia and limb edema might occur because of the generalized vasodilation underlying the erythema, as well as myalgia, fatigue, and fever. GPP may revert to erythrodermic psoriasis when pustule formation stops. Both administration and abrupt withdrawals of systemic corticosteroids or methotrexate, sunburn, and emotional stress have been suggested as possible triggering factors.

Figure 6

e . Nail Psoriasis :

Up to half of those with psoriasis have nail changes. Nail psoriasis is even more common in people who have psoriatic arthritis, which affects your joints.Symptoms are Pitting of your nails, Tender, painful nails, Separation of the nail from the bed, Color changes (yellow-brown), Chalk-like material under your nails.

Figure 7

f . Inverse Psoriasis :

This type usually found in Armpits, Groin,
Under the breasts, Skin folds around the genitals and buttocks. Symptoms include Patches of skin that are bright red, smooth, and shiny, but don't have scales &Getting worse with sweating and rubbing.
Common triggers are Friction , Sweating,
Fungal infections.

Figure 8

g . Psoriatic Arthritis :

Psoriatic arthritis is a condition where you have both psoriasis and arthritis (joint inflammation). In 70% of cases, people have psoriasis for about 10 years before getting psoriatic arthritis. About 90% of people with it also have nail changes.

Figure 9

ASSESSMENT OF PATIENTS WITH PSORIASIS

Psoriasis is assessed by the extent of skin involvement (body surface area (BSA)) and the severity of erythema, induration and scaling. In secondary care, validated scores such as Psoriasis Area Severity Index (PASI) and Physician Global Assessment Scale are routinely used along with patient reported outcome measures such as Dermatology Life Quality Index (DLQI).Attention to its psychological impact is essential as this may contribute to disengagement and non-adherence to therapy. Every patient encounter is also an opportunity to screen for multimorbidities. In addition to improving overall health, recognition of multimorbidities may influence psoriasis treatment choice. For instance, chronic liver disease may contraindicate methotrexate use. A multidisciplinary approach is, therefore, crucial and often involves rheumatologists, hepatologists and clinical psychologists.

TREATMENTS FOR PSORIASIS

This will depend on the type of psoriasis that you have, and on its severity.

A . Topical therapies:

Treatments that are applied directly to the skin are known as topical therapies. They include creams, ointments, pastes and lotions. If your psoriasis is mild, topical therapies will be the mainstay of your treatment. Topical therapies are discussed in more depth in another of our leaflets (Topical psoriasis remedies), which include the following:

1. Emollients - Reduce scaling and can be used as often as needed.
2. Salicylic acid - help heavily scaled plaques.
3. Topical steroids - Weaker steroids also do not perform very well on dense areas of psoriasis, but on the face or in the folds of skin could do well. The stronger ones have possible side effects, one of which is to make your skin thinner. Your doctor will monitor their use closely. Psoriasis sometimes comes back quickly when topical steroid treatment stops.
4. Tar preparations - Taking a medicated tar bath may help to remove loose scales. Tar creams or ointments help most patients but may be messy and can stain clothing.
5. Dithranol - This can be used at home for minor or moderate psoriasis. Patients may be also treated in specialized units in hospitals. Dithranol may be effective on patients with thick plaque psoriasis; however, it is rarely used nowadays, since it may irritate the skin and also it stains not only the skin and clothing, but baths and showers.
6. Vitamin D analogues - There are several vitamin D preparations used to treat psoriasis; calcipotriol, calcitriol and tacalcitol. They are safe, clean to use and do not stain the skin. Treatment is applied either once (tacalcitol) or twice (calcipotriol and calcitriol) daily and can go on for as long as required. Irritation may occur, especially on the face, buttocks and genitals, and these treatments should be applied to those areas only on the specific instructions of your doctor.
7. Vitamin A analogues - Tazarotene is a vitamin A gel which is added to psoriasis patches once a day. Irritation can occur when added to the folds of the face or of the head. It is important to tell your doctor if you are pregnant or breast-feeding. You will stop becoming pregnant while you are being cared.
8. Topical immunosuppressant agents - also called calcineurin inhibitors, Tacrolimus and Pimecrolimus, are creams or ointments that are used mainly for eczema but can be effective and safe on treating psoriasis on special areas like face, folds or genitals.

Topical treatments for special sites: Skin folds and the face, scalp, nails.

B . Phototherapy :

This term refers to treatment with various forms of ultraviolet light, sometimes assisted by taking particular tablets. It is helpful if the psoriasis is extensive, or fails to clear with topical treatment, or comes back quickly after seeming to clear. Topical therapy will usually continue during the phototherapy.Two types of ultraviolet (UV) light may be given, using special machines: UVA and UVB. These are different parts of normal sunlight. Treatment with UVA is helped by taking a medication known as a psoralen – a combination known as PUVA therapy. Treatment with UVB does not need tablets. Both UVB and PUVA treatments have to be given with great care, and you will have to come up to the skin department 2 or 3 times a week for a number of weeks. Full details are given in other leaflets issued by the British Association of Dermatologists (Treatments for moderate and severe psoriasis and Phototherapy).

C . Internal Treatments :

In cases, where the disease is very extensive or severe, patients may need oral treatment; however all of the different tablets have potential risks. In addition, you will usually have to continue with some topical therapy even though you are taking the tablets. Oral medications involve treatment by acitretin (vitamin A-related), ciclosporin (weakens the immune system), methotrexate (tends to slow the pace at which skin cells split into psoriasis), and hydroxycarbamide (formerly called hydroxyurea-also slows the rate at which the skin cells divide). Several injectable forms of therapy for severe form of psoriasis are also available. Biological drugs, which target more complex defense system components, include adalimumab, ustekinumab, etanercept, and infliximab.Clear description of these therapies can be given in the patient information leaflet Therapies for moderate to serious psoriasis.

CONCLUSION

Psoriasis is a multisystem inflammatory disease that is underdiagnosed and undertreated despite its prevalence and considerable effect on quality of life. Beyond skin and joint involvement, psoriasis is also associated with an array of important medical and psychiatric comorbidities that require timely therapy to improve long-term outcomes. Primary caregivers are well positioned to provide diagnosis and treatment of patients who seek initial evaluation at the primary care level. Patients with psoriasis for whom topical therapy fails can be referred to a dermatologist for further evaluation.

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