"A ONE YEAR HOSPITAL BASED CROSS SECTIONAL STUDY OF DERMATOLOGICAL LIFE QUALITY INDEX (DLQI) IN PSORIASIS PATIENTS IN KLES DR PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE, BELGAUM"

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This is to certify that the dissertation entitled **"A ONE YEAR HOSPITAL BASED CROSS SECTIONAL STUDY OF DERMATOLOGICAL LIFE QUALITY INDEX (DLQI) IN PSORIASIS PATIENTS IN KLES DR PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE, BELGAUM"** is a bonafide research work done by **REG NO. BT0113002.**

Dr. A. M. Pandit MD
Professor and Head,
Department of Dermatology,
Venereology and Leprosy,
J. N. Medical College,
Nehru Nagar,
Belgaum – 590 010

Date:
Place: Belgaum

Dr. N. S. Mahantshetti MD
Principal,
J. N. Medical College,
Nehru Nagar,
Belgaum – 590 010

Date:
Place: Belgaum

LIST OF ABBREVIATIONS USED

Qol	=	Quality of life
HRQoL	=	Health Related Quality of Life
DLQI	=	Dermatological life quality index
PASI	=	Psoriasis area severity index
HAM-A	=	Hamilton Anxiety scale
HAM-D	=	Hamilton Depression scale
TGF	=	Transforming growth factor
IL	=	Inteleukin
TNF	=	Tumour necrosis factor
IFN	=	Interferon
ICAM	=	Inter cellular adhesion molecule
KGF	=	Keratinocyte growth factor
GM-CSF	=	Granulocyte monocyte , Colony stimulating factor
SP	=	Substance P
VIP	=	Vasoactive Intestinal Polypeptide
CSF	=	Colony Stimulating Factor
Th	=	T lymphocyte helper
CGRP	=	calcitonin gene related peptides
NY	=	neuropeptide Y
CRP	=	Corticotrophin-releasing hormone
ACTH	=	Adrenocorticotrophic hormone
NGF	=	Nerve growth factor
CAMP	=	Cyclic adenosine monophosphate
IFN-γ	=	Interferon-γ

ABSTRACT

Background and objectives

Psoriasis is a chronic disfiguring, inflammatory and proliferative condition of the skin in which both the genetic and environmental factors have a critical role. Psoriasis is linked with social stigma, pain, discomfort, physical disability and psychological stress. Thus the purpose of this study was to assess the impact of psoriasis on quality of life and also to evaluate the psychological distress; anxiety and depression in patients of Psoriasis.

Methodology

The present one year cross sectional study from January 2014 to December 2014 was done in the Department of Dermatology, Venereology and Leprosy, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum A total of 64 psoriasis patients were examined dermatologically and psychiatrically and then administered the Dermatological Life Quality Index (DLQI), Hamilton Anxiety Rating Scale (HAM-A), Hamilton Depression Rating Scale (HAM-D). Symptom severity was measured by the Psoriasis Area Severity Index (PASI).

Results

Majority of patients, 50% were in the age group of 31- 50years. Male to female ratio was 3.1:1. In 17% of patients, the duration of the disease was less than 1year. Stress was initiating or exacerbating factor in 56% of patients. Chronic plaque psoriasis(83%) was the commonest clinical type seen followed by palmoplantar psoriasis. As measured by DLQI scores, psoriasis had small effect

on quality of life in 27% of the patients, moderate effect on 20% of patients, very large effect on 33% of patients and extremely large effect on 17% of patients. The mean PASI score was 8.6. Significant correlation was noted between the PASI score and the DLQI. According to Hamilton- anxiety scale, 91% had mild severity and 9% had mild- moderate anxiety. Hamilton-depression scale revealed that, 45% were normal, 33% had mild depression, 14% had moderate, 2% had severe and 6% had very severe depression. With increase in DLQI scores, HAM-A scores were found to increase but it was not statistically significant (P value=0.862).With increase in DLQI scores, Hamilton-D scores were found to increase. The difference was statistically significant (P value- **<0.001**).

Conclusion

This study concludes that, psoriasis has a significant negative impact on physical, emotional and psychological wellbeing of the affected patients, thus causing a substantial impact on patient's Quality of life. There is also an association of Psoriasis with psychiatric co-morbidity especially, anxiety and depression.

Key words:

Psoriasis; Quality of Life; Hamilton-A; Hamilton-D; Dermatological life quality index; PASI score

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INTRODUCTION

Psoriasis is a chronic skin disease which can occur at any age and affects both sexes equally[43]. It affects the way a person sees himself and the way he is seen by others. Psoriasis is linked with social stigma, pain, discomfort, physical disability and psychological stress. Thus assessment of the extent of apparent disease in terms of the clinical severity alone may not suffice, and a more holistic approach to the quality of life is mandatory.

Recently psychological intervention has proved to be a valuable adjunct to normal dermatological treatment and followed by early improvement in majority of patients[9]. However there are not many studies supporting this observation. Hence more clinico-epidemiological studies using appropriate psychometric instrument for assessing Quality of Life are required for better understanding of disease burden of patients.

So what is psoriasis? Is it genetic disease? Immunological disease? A proliferative disease? Psychosomatic disease? Or is it all of these?

Psoriasis and psychology: a multitude of links

Psoriasis is a chronic disfiguring, inflammatory and proliferative condition of the skin in which both the genetic and environmental factors have a critical role[1]. Many environmental factors like the trauma, infection, drugs, sunlight, metabolic factors, psychogenic factors, alcohol and smoking have been linked to psoriasis and have been implicated in initiation of disease process and exacerbation of pre-existing disease[2,3,4]. Psoriasis is made worse by psychological distress in approximately 30-

40% of patients[1]. The chronic course with intermittent exacerbation and disfigurement can cause further increase in the sufferings of the patients. The embarrassment, stigma and social anxiety caused by the illness often leads to emotional distancing, isolation, anxiety and depression affecting the quality of Life[5,6,7,8]. Thus the purpose of this study was to assess the impact of psoriasis on quality of life and psychology of patients, so as to provide early psychological intervention as an adjunct to dermatological treatment for good outcomes.

AIMS AND OBJECTIVE

We must be daring and search after truth; even if we don't succeed in finding it , we shall at least have come close to it.

- By Galen.

Aims and Objectives:

1. To evaluate the quality of life in patients suffering from all types of psoriasis.
2. To evaluate the psychological distress; anxiety and depression in patients with psoriasis

REVIEW OF LITERATURE

Historical background:

Psoriasis is the disease that has existed from centuries since the history of man. Many of the "unclean" lepers of biblical times were probably psoriatics[10]. The biblical term "lepra" was applied to various cutaneous disorders including psoriasis, vitiligo, eczema, boils and alopecia areata[11].

The earliest description of what appears to represent psoriasis are given at the beginning of medicine in the Corpus Hippocratum. Hippocrates (400-377BC)[12] grouped scaly lesions under the term lopoi(from "lepo" meaning "to scale"). But the first recognizable description of psoriasis is attributed to Celsus (25BC-45BC) in his "Des Medical" nearly 2000 years ago. The disease was described under the heading of impetigo (latin word "impetigo" meaning "to attack")[13].

Galen (133-200AD) is credited as the first person to use the word psoriasis and though he was probably describing seborrhoeic eczema, he used the word correctly as it comes from greek word "psora" meaning " itch".

In 1809, Robert Willan first accurately described psoriasis and separated two diseases as psoriasiform entities, a discoid lepra graecorum and a polycyclic confluent "psora leprosa" but he did not separate it with certainty from leprosy[10]. Ferdinand Von Hebra, in 1841 unequivocally showed that Williams "Lepra Graecorum" and "Psora Leprosa" were one disease. He separated psoriasis from leprosy[12,13].

Review of literature on psychological and psychosocial aspects of psoriasis:

Psoriasis is a disease with profound impact on the psychological and social aspect of the patient, particularly because of its lesions with characteristic body involvement. Individuals with psoriasis may also feel self-conscious about their appearance and have a poor self-image that stems from fear of public rejection and psychosexual concerns.

- In a survey by the National Psoriasis Foundation almost 75% of patients had moderate-large negative impact on their quality of life, with alterations in their daily activities[14].

- Gupta MA and Gupta AK in their study of 127 psoriasis patients found that 9.7% of patients reported their wish to be dead and 5.5% reported active suicidal ideation at the time of the study[8].

- In a cohort study by Kurd SK and Gelfand JM they estimated that in the UK, in excess of 10,400 diagnoses of depression, 7,100 diagnoses of anxiety, and 350 diagnoses of suicidality are attributable to psoriasis annually[15].

- Study by Mazzotti E,Picardi A,Sampogna F,Sera F,Pasquini P,Abeni D contributes to building evidence of validity for the DLQI, because the instrument demonstrated the ability to detect small but meaningful changes in clinical status over time in a large sample of patients with psoriasis[25].

- In a study on Quality of life in patients with Psoriasis in northern Taiwan. The mean score on the DLQI was 9.16 ± 6.3 and 67% of all patients reported a moderate to extremely large impact on their quality of life (DLQI > 6). A

higher psoriasis area and severity index (PASI), younger age and initial lesions on the nails significantly negatively impacted patients' quality of life[16].

- The Hamilton Anxiety Scale (HAM-A) was tested for reliability and validity in two different samples, one sample (*n*=97) defined by anxiety disorders, the other sample (*n*=101) defined by depressive disorders. The reliability and the concurrent validity of the HAM-A and its subscales proved to be sufficient[17].

An article[18] published examined the literature concerning psychological aspects of psoriasis published since 1995. The literature is concerned with :

1. The consequences of psoriasis in terms of quality of life, disability, anxiety, depression and stigmatization and factors that may predict such outcomes.
2. Potential mechanism of interaction between psychological factors, stress and the pathophysiology of psoriasis.
3. Examinations of the clinical utility of psychological interventions on extent of psoriasis and psychological distress[18].

This investigation confirms that DLQI is a reliable and valid instrument to assess the patients perceived impact of skin disease. Also, it supports the uni-dimensionality of DLQI and hence corroborates the common practice of using the total score[21].

This study confirms the enormous burden caused by skin disease in the form of impairment and stigmatization, depending on somatic severity, even when the current affection is rather mild. In connection with psychosocial consequences, future study should also focus on the disease burden of psoriasis[22].

The impact of psoriasis on HRQL is similar to that of other major medical diseases. The different aspects of psoriasis are related to the different variables of HRQL supporting the need for multidimensional treatment models[23]. Patients with more extensive skin involvement have greater reduction in quality of life. Female patients and young patients are affected to greater extent[24].

Epidemiology

Incidence and prevalence:

Psoriasis is universal in occurrence. In United States psoriasis affects about 2% of the population with approximately 150,000 newly diagnosed cases per year. The worldwide incidence varies considerably[27].

Its prevalence varies from 0.3% or less in Mongoloids and more than 2% in Scandinavia. In both North America and Europe the prevalence has been estimated to be between 1% to 2%. In a recent community based study from U.K, a prevalence of 1.48% was found. The incidence of psoriasis ranges from 0.97% in South America to 1.3% in Germany, 1.6% in Great Britain, 1.7% in Denmark and 2.3% in Sweden[28].

In India the reported incidence of psoriasis in different studies are 5.6%, 0.84%, 1.25%, 1.5%, 1.4% and 2.8%[29,30,31,32,33,34].

Racial and ethnic variation:

The incidence of the disease is particularly low in the Japanese, in Native American blacks especially those of West African origin[35]. The incidence is low in Eskimos, Mongolians and nearly absent in North American indians[27]. Psoriasis appears to be more common in Kenyans and Ugandans[36].

In India, Jews, Parsis have been found to be more susceptible than Hindus and Muslims.

Age of onset:

Onset of psoriasis is most common in 2nd to 4th decade of life though it can occur just after birth or in old age[37]. It has been reported to be present at birth and has been described recently as having its onset at age as late as 108years[38.]

In German study, evaluation of age of onset revealed 2 peaks: an early one at 16- 22years and a later one at 57-60years[39]. A North Indian study found that the mean age of onset was higher for males than females (37 Vs 29years)[33].

It is also seen that earlier the onset, severe is the disease[40], so much so that one of the patients with congenital psoriasis reported by Lerner and Lerner had developed crippling psoriatic arthritis before the age of 13years[41]. Also earlier the age of onset, greater is the possibility of a positive family history of psoriasis[42].

Sex ratio:

The earlier age of onset in females suggest a greater incidence in young females than in young males[28]. However the incidence of psoriasis in adult men and women is usually reported to be about equal[43]. But a higher prevalence in males have been noted in most Indian studies[30]. In a study in India, ratio of incidence in males to females was found to be 2.4:1[34].

Hereditary:

The evidence that psoriasis may be inherited is beyond doubt. Evidence has been presented for single gene Autosomal dominant inheritance with reduced

penetrance, for the presence of multifactorial genetic components as well as for no genetic component[28]. The major support for concluding that there is genetic predisposition to psoriasis comes from studies showing:

1. An increased incidence of psoriasis among relatives of affected probands.
2. A proportionally increased incidence of psoriasis in offsprings of matings in which one or both parents are affected.
3. High rates of concordance for psoriasis among monozygotic twins when one is affected.
4. Susceptibility loci located on various chromosomes including disequilibrium of certain MHC antigens[44,45].

Etiopathogenesis and pathology

Genetic factors:

Genetic factors play an important role in the Etiopathogenesis of psoriasis. Based on population studies, the risk of psoriasis in an offspring has been estimated to be 41% if both the parents are affected, 14% if one of the parents is affected, and 6% if one sibling is affected, compared to 2% when no parent or sibling is affected. Concordance for psoriasis in monozygotic twins ranged from 35% to 73% in various studies. Despite multiple genome wide linkage studies, only 1 locus termed psoriasis susceptibility 1(PSORS1) has been consistently confirmed. PSORS1 is located in the major histocompatibility complex (MHC, chromosome 6p21.3), home of HLA gene. Multiple HLA alleles have been associated with psoriasis, particularly HLA-B13, HLA-B37, HLA-B46, HLA-B57, HLA-Cw1, HLACw6, HLA-DR7 and HLA-DQ9, HLA-Cw6[46.]

HLA typing in Indian psoriatic identified HLA-BW17 to be significantly associated with the disease. Patients with HLA-B17 have an earlier onset of disease than those with HLA-BW16. HLA-BW38 shows a strong association with distal psoriatic arthritis and HLA-B27 with generalized pustular psoriasis and psoriatic sacroiliitis. It has been suggested that HLA-B13 and BW-17 reduce the threshold for clinical expression in subjects predisposed to psoriasis[37].

The main pathogenetic features of psoriatic lesions are abnormal keratinocyte differentiation and hyperproliferation, infiltration of inflammatory cells and vascular changes. Accordingly, keratinocytes, fibroblasts and cells involved in the immune response (antigen-presenting and T cells) or vascular system (endothelial cells) are all suggested as primary defects in the disease process. Interactions between the different cell types via cytokines play an important role in the disease process, but the pathogenetic mechanisms are still largely unsolved[47.]

1. Keratinocytes

In psoriatic skin, keratinocyte differentiation is abnormal, resulting in parakeratosis and loss of the granular layer. The keratinocytes are also hyperproliferative. These changes are believed to be secondary to altered growth and maturation kinetics of keratinocytes. In psoriatic plaques, the cell cycle of proliferating keratinocytes is eight times shorter and the proliferating cell population is two times greater than in control skin[48]. Normally, only a small portion of the keratinocyte stem cells in the basal layer is active in cell cycling. In psoriatic skin, the percentage of stem cells participating in cell division seems to be increased[49]. Alternatively, the number of cell cycles in a transiently amplifying cell population might be increased. The factors leading to these changes in keratinocytes remain

obscure. There is also a disturbance in keratinocyte adhesion in psoriatic skin lesions. Integrins are important in cell-cell interactions and in adhesive properties of keratinocytes. They also play a role in the initiation of terminal differentiation of keratinocytes[50]. The polarized topography of integrin expression and the integrin-cytoskeleton association are altered in non-lesional and lesional psoriatic skin compared with normal skin, but whether the changes are primary or secondary to some other stimulus is not known[51].

2. Inflammatory cells

Strong evidence indicates that inflammatory cells, especially T lymphocytes, have an important role in the pathogenesis of psoriasis. T lymphocytes accumulate early in the developing plaque and immunosuppressive drugs targeted against T lymphocytes (e.g. cyclosporin A) are effective in treating active psoriasis[52]. Psoriasis can be exacerbated by HIV infection, which is known to infect the CD4+ T cells[53]. Experiments with an immunodeficient SCID mouse model have also supported the important role of immuno-mechanisms in pathogenesis. In one study, non-lesional skin from psoriasis patients and skin from healthy controls were grafted onto SCID mice. After injection of autologous immunocytes, psoriatic plaques developed in uninvolved skin from psoriasis patients but not in control skin.

3. Cytokines

The expression of several cytokines is altered in psoriatic skin. Because of the complex interaction of the cytokines, it is unlikely that over- or under expression of a single cytokine could be the sole pathogenetic mechanism. IL-6 is a major mediator of the host response to injury and infection. It also enhances B and T cell proliferation

and activation of B cells, T cells and macrophages. It is present in increased amounts in psoriatic skin. IL-8 is a potent T cell and neutrophil chemoattractant and is also overexpressed in the psoriatic skin (Gillitzer et al. 1991). Both IL-6 and IL-8 are produced in part by keratinocytes and have been shown to stimulate keratinocyte proliferation in vitro[54]. TGF-alfa is produced by keratinocytes and has mitogenic and angiogenic properties. Both TGF-alfa mRNA and protein levels are overexpressed in psoriatic lesions. TGF-alfa, by contrast, inhibits epithelial growth and its mRNA levels in psoriatic skin are not significantly different from those in normal skin[55]. IFN-alfa is produced by activated lymphocytes and has the ability to induce the expression of the adhesion molecule ICAM-1. TNF- alfa can also induce ICAM- 1 but to a lesser extent. Both IFN- alfa and TNF- alfa are believed to be important in the trafficking of T lymphocytes to the psoriatic epidermis[56]. The amount of TNF- alfa in psoriatic lesions is elevated compared with that in control skin[57]. IL-1 stimulates production of other cytokines by keratinocytes and induces vascular endothelial cell adhesion molecules for leukocytes (ELAM-1, VCAM-1, ICAM-1), which could account for the infiltration of leukocytes into psoriatic lesions. The expression of ELAM-1 and ICAM-1 has been shown to be higher in psoriatic skin than in normal skin[58]. Keratinocytes themselves also produce IL-1, which stimulates the expression of KGF and GM-CSF in fibroblasts. These fibroblast derived factors in turn stimulate keratinocyte proliferation and differentiation via a paracrine regulation mechanism.

The immunologic basis of psoriasis:

Whether some immunological mechanisms play a role in the etiopathogenesis of psoriasis is controversial. Most drugs like corticosteroids, methotrexate, PUVA and retinoids, beneficial in the treatment of psoriasis, have profound immunosuppressive

effects. An important mediator system, believed to be involved in the pathogenesis of psoriasis, is stratum corneum antigen-antibody interaction. In a psoriasis-prone person, vasodilatation occurs following trauma or infection, and is accompanied by an influx of neutrophils into the epidermis. Proteolytic enzymes released by neutrophils unmask the stratum corneum antigen. Stratum corneum antibodies leak into the epidermis and fix the newly exposed antigen. It has been found that stratum corneum antibodies are regularly found in the parakeratotic horny layer of fully developed psoriatic lesions. Usually IgG deposits are found, though other immunoglobulins and complement components are also often seen. The antigen-antibody reaction triggers the complement cascade and further inflammatory responses. How these immunological events cause the abnormal proliferation and differentiation of epidermal cells in psoriasis is unknown[37].

Another site of interest for immunologists is the basal layer of the epidermis. In a normal person the basal cell nuclear material is not "recognized" by the immunological system. A clone of suppressor T cells prevents such recognition. It is postulated that a genetic defect or a virus leads to malfunctioning of such a clone of suppressor cells, leading to the recognition of basal cell nuclear material as antigen. Subsequently antibodies are formed against this antigen, leading to an immunological response that results in the epidermal cell proliferation typical of psoriasis. Langerhans cells are activated in psoriasis but their role in its pathogenesis is not clear[37].

Recently, it has been demonstrated that in psoriatic skin the basal and suprabasal keratinocytes express CDW60, probably induced by intralesionally activated T cells and partly due to IL-13. These findings represent a normal

mechanism by which T cells participate in the pathogenesis of psoriasis. Levels of T cell products SIL-2R and sCD27 decrease during immunosuppressive treatment with cyclosporine[37].

Three theories have been proposed for the relationship between epidermal keratinocytes and immunocyte activation. The first theory proposes direct activation of epidermal keratinocytes by physical, chemical or ultraviolet injury, increasing the synthesis and release of cytokines, which trigger T lymphocyte activation in an antigen independent fashion[37]. The other two theories propose persistent T lymphocyte stimulation as a result of either antigen/super antigen presentation by antigen presenting cells, or as a result of auto reactivity. One or more of these mechanisms may be operating in different patients at different times, or in response to different environmental stimuli.

Arachidonic acid metabolism and psoriasis

Psoriasis tends to occur at sites of trauma. Trauma stimulates activation of the arachidonic acid cascade with subsequent release of various mediators like leukotrienes, prostaglandins and 12-hydroxyei-cosatetraenoic acid (HETE). Psoriatic epidermis contains elevated levels of 12-HETE and arachidonic acid. It appears that psoriatic skin has an endogenous inhibitor of cyclo-oxygenase resulting in diversion of arachidonic acid to the lipoxygenase pathway[95].

Nonsteroidal anti-inflammatory drugs like salicylates, indomethacin, phenylbutazone, oxyphenbutaxone, ibuprofen and meclofenamate have been reported to exacerbate psoriasis. These drugs act mainly by inhibiting prostaglandin and cAMP production and thereby precipitates the abnormal epidermal activity characteristic of

psoriasis. Another alternative explanation is that inhibition of prostaglandin synthetase results in the increased availability of the prostaglandin precursor, 5-arachidonic acid, for the lipoxygenase pathway mechanism. This leads to production of leukotriene B4 and other potent neutrophil chemotactic factors, which in turn promote leukocytic infiltration of the epidermis and contributes to the eruption. The beneficial effect of benoxaprofen, a NSAID belonging to the propionic acid group, in psoriasis further strengthens the hypothesis that psoriatic skin contains an endogenous inhibitor of cyclo-oxygenase that leads to diversion of arachidonic acid to the lipoxygenase pathway[37].

Cyclic nucleotides and psoriasis

It has been suggested that the cell cycle is controlled by the intracellular concentration of cyclic nucleotides. The epidermal cells are subject to a block in the G1 phase which is maintained by a high level of cyclic AMP in relation to the concentration of cyclic GMP. Cell proliferation is stimulated either by a fall in cAMP or by an increase in cGMP. Adrenaline and its analogue isoprenaline, which are known to increase the intracellular level of cAMP, inhibit mitosis in cultures of normal or psoriatic human keratinocytes. An actual or functional decrease in the cAMP-to-cGMP ratio could account for the clinical manifestation of psoriatic lesions.

Psoriasiform lesions can develop during therapy with beta-adrenergic receptor blocking drugs for various cardiovascular disorders. Lithium compounds inhibit adenyl cyclase and reduce hormone-induced accumulation of cAMP in vitro and thus exacerbate psoriasis[37].

Polyamines and psoriasis

Polyamines are low molecular weight organic amines such as putrescine, spermidine and spermine. They are important in the regulation of cellular proliferation and are increased in the involved and uninvolved skin of psoriatic. The activity of Ornithine decarboxylase, which is the rate-limiting enzyme in the biosynthesis of polymines, is increased during the early stages of epidermal hyperplasia. Treatment with PUVA, retinoids and topical corticosteroids reduces the level of polyamines in psoriasis lesions. Hence it is possible that these polyamines play a role in the pathogenesis of psoriasis[37].

Protease-antiprotease system and psoriasis

Certain proteases like plasminogen activator and various cathepsins and their inactivating antiproteases like alpha-1-antitrypsin have a role in epidermal proliferation and differentiation. Cathepsin-1 is chemotactic to polymorphonuclear leukocytes and a potent complement activator. Protease activity is increased in psoriatic epidermis and plasminogen activators are increased in psoriatic scales. The cell surface proteases can be activated or inactivated at a local level, which explains the local expressivity of the disease[37].

Contact inhibition theory in psoriasis

Normally, firm contact between epidermal cells especially those of the superficial layers cause a feedback inhibition of basal cell proliferation. Any defect in this intercellular contact will result in uncontrolled cellular proliferation. It has been observed that the glycoprotein-rich cell surface coat is completely absent in cells of the stratum malpighii of psoriatic skin. This results in diminished coherence between

the epidermal cells and thus there is a loss of contact inhibition of growth. This leads to accelerated epidermopoiesis. A reduced cell surface coat also decreases the activity of various cell membrane-bound enzymes. Adenyl cyclase synthesizes cAMP from adenosine triphosphate. Disturbance of intracellular regulation of cAMP and cGMP may occur due to deficiency of membrane bound enzymes[37,59].

Exacerbating Factors:

Psoriasis is marked by periods of exacerbations and remissions. Remissions may last for few weeks to many years. Exacerbating factors may be local or systemic.

1. **Local factors:**

Psoriatic lesions develop at sites of injury to skin. The koebner phenomenon,also known as the isomorphic response, refers to induction of lesions by cutaneous trauma. Epidermal trauma alone will not induce the lesions, it should involve the papillary dermis. The trauma may be of any kind: physical, chemical, mechanical, allergic or of any other nature .The koebner phenomenon is elicitated at sites of sun burn, operation wounds, vaccination and other skin lesions. It usually with in 7-14 days, but the interval may be as short as 3 days or as long as 3 weeks. Psoriasis may occur as a koebner phenomenon at sites of bites, burns, drug reactions, dermatitis, lichen planus, miliria, pressure, skin tests, vitilgo, herpes zoster[60].

2. Seasonal variations:

Most patients experience worsening of their skin lesions during winter. High humidity is usually beneficial. Sunlight may worsen psoriasis in some but improves it in many[30,31].

3. Infections:

Upper respiratory tract infections and tonsillitis, especially when caused by streptococci may cause flare up existing psoriasis or may precipitate an attack of acute guttate psoriasis[60]. This is common in children and is usually associated with an elevated antistreptolysin O titre. Infections by other bacteria and viruses may also exacerbate psoriasis. Psoriasis as a koebner phenomenon may develop at the sites of impetigo, herpes zoster, pityriasisversicolor and dermatophytosis.

4. Pregnancy:

In most cases pregnancy induces remissions, though raised levels of progesterone in the latter half of pregnancy can precipitate generalized pustular psoriasis in some[61].

5. Drugs that precipitate or exacerbate psoriasis:

Many drugs can exacerbate or precipitate psoriasis. A number of beta adrenoreceptor blocking drugs like propranolol, practolol, metaprolol and oxprenolol induce papulosquamous eruptions that resemble psoriasis. The cyclic AMP levels in psoriatic epidermis is decreased and this responsible for accelerated epidermopoiesis. Treatment with beta blockers further reduces the cAMP levels thereby exacerbating psoriasis. Psoriasiform lesions induced by such drugs are less scaly and less erythematous. The palms and the soles and the elbows are only rarely involved. The eruptions usually subsides within 2 to 6 weeks of cessation of beta blockers. Other drugs which precipitate psoriasis are, NSAIDS, indomethcin, salicylates, meclofenamate, pheny butazone and ibuprufen.

Severe psoriasis is associated with depression and may be its cause. Treatment of depression with lithium compounds in these patients may destabilize and exacerbate the psoriasis. Lithium has an inhibitory effect on adenyl cyclase and reduce hormone induced accumalation of cAMP invitro[92]. Trazodone a new antidepressant can also cause Generalised pustular psoriasis.

Chloroquine is known to exacerbate psoriasis, causing exfoliative dermatitis. The chemical structure of chloroquine is very similar to dansyl putresine, a potent transglutaminase inhibitor hence alters the the transamidation of glutamine residues in cellular protiens[37].

Rapid withdrawal of corticisteroids results in precipitation of generalised pustular psoriasis or exfoliative dermatitis as a rebound phenomenon. Sodium valproate and carbamazepine can also induce psoriasiform eruptions.

6. Emotional stress and psoriasis:

Psoriasis is more stress sensitive than any other skin disease. Many stressful events of daily life may exacerbate psoriasis. The disease itself can cause a reactive anxiety and depression in patients which could further exacerbate the psoriasis.

There is a relatively large body of literature implicating stressful life situations in precipitating and/or exacerbating psoriasis[62,63,64,65,66,67,68,69]. It is possible that a subgroup of psoriatics who are “stress reactors” experience a relatively benign clinical course, as their symptoms subside after the stress provoking situation becomes less bothersome or subsides. Identification of such patients early in the course of treatment and incorporation of specific psychosocial interventions in the overall treatment regimen may improve the course of illness.

Increased beta-endorphin in psoriatic skin might affect both substance P-mediated neurogenic inflammation and transmission of sensory stimuli by its local antinoceptive effects[73]. Stress might induce alterations in the psoriatic lesion by increasing the neuropeptide content with a concomitant decrease in the activity of neuropeptide degrading enzymes especially mast cell chymases.

Clinical features:

Psoriasis is a papulosquamous disease with variable morphology, distribution, severity, and course. Papulosquamous diseases are characterised by scaling papules (raised lesions <1 cm in diameter) and plaques (raised lesions >1 cm in diameter). The lesions of psoriasis are classically very well circumscribed, circular, red papules or plaques with a grey or silverywhite, dry scale. The lesions are typically distributed symmetrically on the scalp, elbows, knees, chest, abdomen, lumbosacral area, and in the body folds.

Psoriasis may also develop at the site of trauma or injury, known as Koebner's phenomenon. If psoriasis is progressive or uncontrolled, it can result in a generalised exfoliative erythroderma. Nail involvement may be present, particularly if psoriatic arthritis (PsA) is present.

Occasionally psoriasis may involve the oral mucosa or the tongue. When the tongue is involved, the dorsal surface may have sharply circumscribed gyrate red patches with a white-yellow border. The patches may evolve and spread, changing on a daily basis, can assume distinct annular patterns and may resemble a map, hence the term geographic tongue.

Psoriasis can be highly variable in morphology, distribution, and severity. Psoriasis may be symptomatic with patients complaining of intense pruritus or burning. The various types and presentations of psoriasis are outlined below[37].

1. Guttate Psoriais.
2. Chronic plaque psoriasis
3. Exfoliative psoriasis
4. Pustular psoriasis
5. Psoriasis unguis.
6. Mucous membrane psoriasis
7. Arthropathic psoriasis.

Regional variations in psoriasis: scalp, face, eyes, body flexures, scrotum, napkin area, palms and soles.

1. Guttate psoriasis:

Guttate psoriasis, is derived from the Greek word gutta meaning a droplet, describes the 2–10 mm diameter lesions of psoriasis. These are usually distributed in a centripetal fashion. But can also involve the head and limbs. Classically, guttate psoriasis occurs shortly after an acute group B haemolytic streptococcal infection of the tonsils or pharynx and can be the presenting episode of psoriasis in children or, occasionally, adults. The number of lesions may range from 5 or 10 to over 100. Guttate psoriasis accounts for 2% of the total cases of psoriasis. In children, an acute episode of guttate psoriasis is usually self limiting; in adults, guttate flares may complicate chronic plaque disease. Sometimes patients with guttate psoriasis can eventually develop Plaque psoriasis.

2. Chronic plaque psoriasis:

This is common type of psoriasis which manifests either as coin sized lesion called nummular psoriasis or Palm sized lesions called psoriasis geographica. The lesions are stable and remain unchanged for long period. The extensor surfaces of the body, the elbow and the knees, lumbosacral area and back are commonly involved.

3. Exfoliative psoriasis (psoriatic erythroderma):

This is a severe generalised form of the disease and is characterized by universal erythema and scaling. It is usually precipitated by sudden withdrawal of Systemic Corticosteriods, therapy with chloroquine or beta adrenergic receptor blockers or by overt treatment with tar or dithranol.

4. Pustular psoriasis:

Usually in Psoriasis vulgaris, the surface of a plaque is dry with silvery white, loose scales. When it is studded with tiny, superficial, sterile pustules, it is called pustular psoriasis. Pustular psoriasis is precipitated by over treatment with topical tar, Anthralin or potent steroids or by systemic therapy with progesterone or corticosteroids. Pregnancy, infection and hypocalcemia may also precipitate it. Pustular psoriasis is broadly classified into.

a. Localized form

b. Generalized form.

a. Localised Form:

The true localized form of pustular psoriasis affects the palms and soles and is also known as ‘palmo-plantar pustulosis’. It is more common in females. Erythematous, scaly,well defined plaques with numerous tiny pustules are distributed

bilaterally symmetrical on the thenar and hypothenar eminences of the palms, soles and sides of heels. Sometimes they may extend to the Achilles tendon area. Typical skin lesions of psoriasis may develop elsewhere on the body. It may be associated with arthropathy, especially of the distal interphalangeal type. Nail changes typical of psoriasis occur in the majority of patients. Some authors consider acrodermatitis continua of Hallopeau to be a variant of localized pustular psoriasis.

b. Generalized pustular psoriasis:

There are 4 types of generalized pustular psoriasis (GPP):

i. Von Zombusch type

This is the most severe form of GPP. The skin lesions start abruptly as multiple erythematous, tender plaques, which soon become studded with pinhead-sized, tiny sterile pustules. Features of systemic toxicity such as fever, chills, polyarthralgia and malaise may be associated. The plaques coalesce to form generalized erythema, scaling and crusting. Pustules may coalesce to form lakes of pus. Each pustule dries up in 2-3 days and is exfoliated as thin scales or as brown crusts. At the same time, fresh crops of pustules erupt on the shiny underlying skin and the cycle is thus repeated. The disease may last for weeks or even months.

Mucous membrane involvement, especially of the oral cavity, is not uncommon and consists of discrete and confluent denuded areas with white elevated margins. This type of lesion is known as ‘annulus migrans’ and occurs on the buccal mucosa and on the dorsum of the tongue. It may occur in Reiter’s syndrome and in acrodermatitis continua of Hallopeau. Systemic complications like pericholangitis and renal oligemia with resultant acute tubular necrosis may rarely occur.

ii. Exanthematic type of GPP

Usually following an upper respiratory tract infection, there is an abrupt eruption of erythema and pustules. It often begins on the palms and soles and spreads to other parts of the body. General signs and symptoms of toxicity may be present.

iii. Annular type of GPP:

The eruption consists of annular lesions with erythema, scaling and pustules at the periphery. Each lesion grows slowly and persists for weeks or months and usually not associated with systemic symptoms.

iv. Localized type of GPP

The chronic plaque type of psoriasis may be transformed into unstable psoriasis by over treatment with tar, anthralin or potent topical corticosteroids. Small pustules develop in and around the psoriatic plaques. Features of general toxicity, if present, are very mild. By gentle topical therapy, this unstable pustular form of psoriasis reverts to the chronic plaque type again.

5. Psoriasis unguis:

Nail changes are seen in about 20% to 50% of psoriatics. The common changes are pitting of the nail plate, Onycholysis or separation of the nail plate from the nail bed, subungual hyperkeratosis, and crumbling of the nail plate Grooves and ridges on the nail plate, yellowish discoloration and splinter hemorrhages, leukonychia, are also occasionally observed. Sometimes oval or round, brownish-red lesions resulting from accumulation of parakeratotic material in the nail bed can be seen through the nail plate. This is the ‘Olfleck’ phenomenon. The fingernails are

more frequently involved than the toenails. The incidence of nail involvement is higher if there is associated arthropathy.

6. Mucous membrane lesions in psoriasis:

Mucosal lesions are seen only in the pustular and exfoliative forms of the disease. They consist of discrete and confluent denuded areas with white elevated margins. Such lesions are called ‘annulus migrans’ and occur on the buccal mucosa and dorsum of the tongue. Rarely, lesions may occur on the gingival and ventral lingual mucosa. About 2% of psoriatics have a lesion on the glans penis, usually a solitary patch. The color and well defined nature of psoriasis is retained, but scaling is less. The lips may show a silvery scaling in generalized cases.

7. Psoriatic arthritis:

Psoriatic arthritis is an inflammatory arthritis associated with psoriasis usually with a negative test for rheumatoid factor. Arthritis occurs in about 5-10% of patients with psoriasis. It occurs commonly between the ages of 30 and 55. Onset of arthritis is concurrent with the skin disease in 10% of cases but rarely may precede it. Like cutaneous psoriasis, psoriatic arthritis is a genetically determined disorder. HLA studies reveal that the B27, DR3, A26 and B38 haplotypes are significantly associated with psoriatic arthritis. Environmental factors like trauma may precipitate arthritis. QOL is severly affected in psoriatic arthritis. There are five clinical patterns of psoriatic arthritis.

a. Classic psoriatic arthritis.

This form of arthritis involves the distal interphalangeal joints of the fingers and toes. In the acute stage the involved joint is swollen and tender; the swelling often includes juxtaarticular tissues leading to the so called ‘sausage’ appearance of the affected fingers and toes. The nails of the affected fingers are usually involved. If there are more than 30 pits on the fingernail of a patient with chronic inflammatory arthritis, the diagnosis of psoriatic arthritis is confirmed. About 16% of patients with psoriatic arthritis have the classic form of arthropathy.

b. Rheumatoid type of psoriatic arthritis

This is a symmetric polyarthritis similar to rheumatoid arthritis. It accounts for about 15% of all forms of psoriatic arthritis. Unlike the classic type, the proximal interphalangeal joints of the fingers and toes are affected. They are swollen and tender, and result in a ‘swan neck’deformity as in rheumatoid factor are usually negative. The arthropathy is less extensive and more benign in its course. Early morning stiffness, fusiform swelling of the proximal interphalangeal joints of the fingers, symmetrical involvement and the late deformity of ulnar deviation-all pathognomonic of rheumatoid arthritis-are usually not seen in psoriatic arthritis.

c. Arthritis mutilans

This type accounts for about 5% of all forms of psoriatic arthritis. Osteolysis and destruction of bones, especially of the hands and feet, result in telescoping of the soft tissue and deformities.

d. Oligoarticular arthritis

This is the most common type (70%) of psoriatic arthritis. Usually a single joint or sometimes a few interphalangeal or metacarpophalangeal joints are affected. This form is characteristically asymmetrical in distribution.

e. Psoriatic spondylitis

The concept of psoriatic spondylitis has been strengthened by epidemiologic studies showing an increased incidence of ankylosing spondylitis and sacroiliitis and patients with psoriasis. Radiological evidence of spondylitis is seen in 30% of patients with psoriasis while clinical disease occurs in few.

Rare types:

1. Follicular psoriasis

Psoriasis may present with erythematous scaly follicular papules on the trunk and extremities. Plaque type psoriasis of the scalp may be associated.

2. Psoriasis verrucosa

Psoriasis verrucosa appears as verrucous papules along with typical lesions of psoriasis on other areas. Two types of verrucous papules can be recognised: dome shaped papules with a keratotic plug and crater-shaped papules with a central depression[37].

3. Psoriasis in HIV-infected persons

The prevalence of psoriasis is not increased in HIV infected patients. But psoriatic arthritis, dactylitis and enthesitis are more common in them indicating that HIV may alter the course and severity of psoriasis, but not its frequency[70].

Regional variations:

Palms and soles

Palmoplantar lesions of psoriasis may occur alone or along with involvement of other areas. In most cases the lesions are well defined, but they are less scaly and the surface often shows fissures.

Three forms of lesions can appear at these sites:

a. Diffuse hyperkeratotic plaques,

b. Erythematous patches or plaques studded with minute superficial pustules,

c. Discrete scaly plaques or patches .

Scalp

Diffuse involvement occurs if there is associated seborrheic dermatitis. Usually well defined plaques of psoriasis are retained on the scalp in most cases, but often a band or corona of psoriasis, 2-5 cm wide, projects beyond the hairline on the forehead (‘corona psoriatica’). Sometimes, the scales on the scalp are firmly adherent and asbestos-like known as ‘tinea amiantacea’ or ‘pityriasis amiantacea’. On the retroauricular area the plaques are often fissured and painful.

Face

The face is rarely involved in psoriasis. Sometimes facial involvement can be seen in children or when the disease becomes erythrodermic. Facial lesions are less thick and scaly, but more erythematous than those elsewhere.

Eyes

Conjunctivities, blepharitis, xerophthalmia and symblepharon may rarely develop.

Lumbosacral area

This is commonly affected site in psoriasis. Here it usually forms a large, thick, dry, keratotic plaque with deep fissures. It is also known as ‘elephantine’ or ‘inveterate’ psoriasis.

Body flexures

Typical psoriasis characteristically affects the extensor surfaces of the body, flexural psoriasis is often called ‘psoriasis inversus’. The axillae, groins and the skin under a pendulous breast may be involved. The well-defined nature of a psoriatic lesion is retained but they are less scaly and appear smooth and glistening with a few deep, painful fissures. These plaques are often confined to the areas of skin-to-skin contact. In patients with seborrheic dermatitis, psoriatic lesions may develop as a Koebner phenomenon at the site of seborrheic dermatitis on the body flexures. It is then called ‘sebopsoriasis’.

Scrotum

The psoriatic plaques at this site are less scaly but more erythematous.

Napkin area:

In infants and children, psoriatic lesions are sometimes localized to the napkin area. Whether it develops as a Koebner phenomenon to associated diaper dermatitis or

candidial skin infection is not known. About 17% of such children develop typical psoriasis in later life.

Psychoneuroimmunology:

Relationship between skin disease and psychological or neurotic influences has been established for many years[72]. The skin and mind relationship has vastly been described by the burgoeing science of Psychoneuroimmunology. Psychoneuroimmunology is the study of integrated system of behavioural-neuroendocrine- immune system interactions.

Structures in the brain, nerves and skin, are all embryologically derived from the ectoderm. They show their origins from the neural plate where both the central nervous system (CNS) and peripheral nerves develop from the neural crest, as do the cutaneous structures, melanocytes and Merkel cells[73]. Communication between the immune system and the brain is bi-directional and probably largely mediated by chemical messengers called neuropeptides, released within nerves and also locally at tissue sites, in this case, the skin. This neuroimmuno- cutaneous-endocrine system (NICE)[74,75] seems to form part of an integrated system relating behaviour to neuroendocrine and immune function. The epidermal cells, keratinocytes, dendritic cells, such as Langerhans cells and melanocytes, function with dermal structures, such as macrophages, mast cells, leucocytes and dermal dendritic cells to provide this immune reaction. The mechanisms involved include the cell-signalling interactions provided by the glycoproteins, termed cytokines, which are produced by different cell types in all organs and tissues. In basic terms they are classified as interleukins (ILs), colony stimulating factors (CSFs), interferons (IFNs) and tumour necrosis factors (TNFs). When binding to specific receptor sites, cytokines may upregulate or

downregulate the inflammatory, proliferative and immune reactions in target cells. Cytokines may also inhibit or stimulate the production of further cytokines in a cascade of activity. Furthermore, cytokines can fundamentally influence the direction of T lymphocyte helper (Th) subset differentiation into either Th1, cell-mediated immune reactions or Th2, humoral immune responses[108].

Stress induces the production of neuropeptides not only in cutaneous nerves but also in most skin cells including keratinocytes, fibroblasts and langerhans cells. The role of vasoactive peptides such as calcitonin gene related peptides (CGRP), nocioceptive neurotransmitter SP, VIP, neuropeptide Y and the newer pituitary adenylcyclate activating peptides are increasingly evident in cutaneous inflammatory disease. SP receptors found on mast cells, neutrophills and macrophages suggest a link between neurogenic stimuli and infiltration of the skin by inflammatory cells. CGRP has a crucial immunomodulating effects on disease expression, perhaps by inhibition of antigen presentation by langerhans cells. Lymphocytes have receptors for corticotrophin-releasing hormone(CRH), adrenocorticotrophic hormone(ACTH) and endogeneous opioids and both endorphins and enkephalins are known to directly control antigen specific and non specific in vivo and invitro responses[108].

MIND-BODY Afferent Immune Interactions:

Exposure to either naturally occurring or experimentally induced stress affect both the Humoral and cell mediated immune responses. Bereavement is often associated with depression and many changes in the immune function have been shown in depressed patients. Depression is associated with a decreased number of T and B lymphocytes, natural killer cells, and helper and suppressor cytotoxic T cells and an increased number of circulating neutrophils,. There are also alterations in B

cell function, manifest by increased antibody titres to herpes simplex and cytomegalovirus. Changes in both humeral and cell mediated immunity have been found following marital separation and divorce and alteration in immune functions occurs during exams. Slowing of human wound healing has been associated with stress, possibly mediated by depression of local production of the cytokine, interleukin1B[108].

BODY MIND Efferent Immune Interactions:

The bi-directional communication between the central nervous system and the immune system provides the basis not only for behaviorally induced alterations in immune functions, but also for immunologically based changes in behavior. Those patients who present with disease causing intense afferent stimuli such as scratching, rubbing or traction are probably in a NICE loop where the inflammatory reactions stimulate an immune response which in turn releases cytokines to activates CNS neurotransmitters. Endorphins, for example, modify not only the perception of pain, but also other sensory perceptions such as beauty. The compulsive and irresistible nature of scratching, excoriating and picking may relate to pleasurable release of endorphins. These pathways may explain how emotional stress influences a wide range of disorder[108].

Responses:

Stressful influences initiate autonomic, neuroendocrine or immune responses.

Common stressful events appear to induce immune changes.

Stress producing stimuli:

1. Psychogenic: Fear, rage, anger, frustration and helplessness
2. Environmental: Heat or cold, noise, pollutants, change in diurnal rhythms
3. Behavioural: Isolation, overcrowding, physical restraint, enforced starvation, change in diet and hierarchical challenge

Long-term stress may produce chronic immune dysfunction. Whatever the recognized stressor, the response is to trigger the neuroendocrine pathway via the hypothalamic–pituitary–adrenal (HPA) axis[76]. The hypothalamic paraventricular nuclei stimulate the secretion of corticotrophin releasing hormone (CRH) and in addition, vasopressin. These travel down the hypophyseal-pituitary portal system to the anterior pituitary and release adrenocorticotropic hormone (ACTH). The adrenal cortex responds by secreting cortisone into the circulation and this classic feedback mechanism then suppresses ACTH, CRH and vasopressin. During the neuroendocrine response, the autoimmune nervous system is also activated by the brainstem nuclei, predominantly the nucleus ceruleus, which provokes the production of noradrenaline and neuropeptides from spinal ganglia and the adrenal medulla. In addition, these neuropeptides released by autonomic stress systems, such as propiomelanocortin exert a modulating control on both CRH secreting neurons and pain control secretions in the hindbrain[77]. The neuromediators and neurohormones involved are predominantly neuropeptides, existing as small, simple compounds of 40 or less amino acids. The major group of neuropeptides include substance P (SP), calcitonin gene-related peptide (CGRP) and vasoactive intestinal peptide (VIP). Other significant neuropeptides are neuropeptide Y, somatostatin, neurokins A and B and the

opiomelanocortins, which include the endorphins. A brief summary of actions of important neuropeptides and their target cells is illustrated below.

Table 1 . Neuropeptides and their effect on skin cell type

Target cells	SP	CGRP	VIP
1.Endothelial cells	Proliferation, Permeability	Proliferation, IL-8 secretion.	proliferation
2.Keratinocytes	Stimulates IL-1 and IL-8 Comitogen, LTB4, CGRP	Proliferation in conjuction with SP	Mitogen proliferation
3.Lymphocytes	Proliferation,IL-2 synthesis, B cell differentiation	Chemotactic to T cells, Increases proliferation	Nk cell activity
4.Macrophage	IL- 1, IL- 6 synthesis	Impairs antigen presentation	
5.Mast cells	Histamine andTNF alfa Release.	Histamine andTNF alfa Release	Histamine release
6.Monocytes	Chemotaxis, Phagocytosis		
7.Neutrophills	Chemotaxis, Phagocytosis	Enhances chemotaxis	Enhanced migration

Cytokines in depression and anxiety

There is mounting evidence that both depression and depressive symptoms can induce immune dysregulation by the production of proinflammatory cytokines including IL-6[78]. Similar responses in chronic anxiety with the production of IL-6 and reduced IL-2 receptor production (which is an essential cytokine to counter infection) was deemed a factor in increased URTI episodes[79]. Persistent elevation of pro-

inflammatory cytokines may lead to chronicity in disease, poor healing and increased disability[80].

It seems, therefore, that negative emotions can directly affect the immune system to up or down regulate the response via inflammatory cytokines. This affects not only immediate reaction to the challenge of infection, but also the mechanisms of inflammatory disease.

Psychoneuroimmunology with respect to Psoriasis

Psoriasis, like atopic eczema, is an inflammatory skin disease with a multifactorial aetiology. It is a chronic, immune-mediated disorder[81] associated with significant physical and psychological morbidity. Stress plays an important role in the initiation and exacerbation of psoriasis (Fortune et al., 2002).

Raychaudhuri and farber[82] have proposed a psychoneuroimmunological basis for psoriasis. Thus psoriatic skin has been shown to have significant increase in SP containing nerves. Psoriatic skin is rich in SP and VIP. Proliferation of keratinocytes, a central feature of psoriasis could be triggered by release of both SP and VIP, while SP could also induce localised lymphocytic proliferation. Moreover the initiation and maintainence of lymphocytic infilterate characteristics of psoriasis could be in part related to SP, CGRP and VIP; indeed, SP can induce expression of E-selectin on the endothelium.

In psoriasis there is an over expression of INF gamma and TNF alpha and a relative under expression of the Th2 cytokines, IL-4 and IL-10. It appears that the T-cells involved are Th1 lymphocytes and that the disease may be influenced by a cell-mediated autoimmune process. There is an early influx of T-cells into psoriatic

lesions, increased antigen presentation in psoriatic cells and ablative effect with anti-T cell therapy, and the common antigens considered in the pathogenesis of psoriasis are bacterial proteins and super antigens.

However, clinical and histological features in psoriasis show the Koebner phenomenon on trauma, clearance of psoriatic plaque after desensitizing injury, symmetry of clinical lesions and increased nerve density in psoriatic plaques, which have led to the proposal of a neurogenic hypothesis for the disease[82]. Nerve growth factor (NGF) is a peptide whose functions have been shown to stimulate and direct nerve growth. At a cellular level in skin it is mitogenic for keratinocytes and promotes the migration and degranulation of mast cells leading to dilated vessels and oedema. In addition to this promotion of the release of inflammatory mediators via mast cells, NGF also induces keratinocytes to produce the cytokine RANTES, a key participant in the activation of memory T-cells in psoriasis[82]. NGF, together with neuropeptides, have been shown to produce neutrophil response in psoriasis via CGRP and IL-8. They are mitogenic for keratinocytes and inhibitors of apoptosis (cell death) via SP, CGRP and VIP, and thus probably contribute to the influences producing the epidermal proliferation in psoriasis[83].

Stress and psoriasis:

There is a large literature implicating stressful life situations in precipitating and/or aggravating psoriasis. Though, the nature of this union remains uncertain. In a survey of more than 4500 dermatological patients, 2% of the patients had psoriasis, and emotional factors were reported to "trigger the onset of symptoms" in 62% of the psoriatics[84]. Ingram[85] observed that psychosocial stressors exerted "the most potent influence" on psoriasis, whereas Baughman et al[86] reported that the effect of "stress"

was "modest but significant." These conclusions are based on uncontrolled observations. Studies using controls show that psychosocial causes were significant in the onset and/or exacerbating of symptoms in 39%[87] to 80%[88] of psoriatic patients v/s 10% to 50% of controls which consisted of patients with an assortment of non dermatological disorders respectively. A Danish study involving 245 children with psoriasis reported "stress" was observed to be a stimulating factor in 90% of patients[89].

In the studies where a significant relationship was noted between the commencement or exacerbation of psoriasis and life proceedings, the subjects were asked whether they had experienced "specific stress" one month before the onset of psoriasis[87], or whether they had a "stressful" life event six months before the onset of psoriasis in interview[88]. It is seen that the important factor is the psychologic stress or "stress" dealt by the patient rather than the life proceedings per se. This is supported by a earlier observation that the onset of psoriasis was evidently related with a stressful life event only when the incident was of "an acute catastrophic nature," for eg, sudden demise of a relative. In such a situation it is rational to assume that most people would experience considerable psychologic distress.

Furthermore, a recent study by Ametz et al[90] established that psoriatics experienced "significantly higher stress levels" in contrast to healthy controls, when both cases and controls were exposed to the same stress aggravating situation. This was calculated by scores on standard questionnaires and increased urinary adrenalin levels[90].

Psoriatics who reported "specific stress" a month prior to the onset of psoriasis were also seen to have better prognosis three years later on[87]. It is likely that a

subgroup of psoriatics who are "stress reactors" experience a comparatively benign clinical course, as their symptoms settle after the stress-aggravating situation becomes less worrisome or subsides. Recognition of such patients in the early hours in the course of treatment and inclusion of specific psychosocial interventions in the treatment schedule may improve the course of illness. Facilitation of grieving and supportive psychotherapy may prove to be important clinical interventions in some patients.

Biochemical and Physiological Correlates of Psoriasis that Interface with Psychiatry:

Lithium and Psoriasis:

Lithium may precipitate and often exacerbate psoriasis[91]. This result is believed to be mediated by the effect of lithium on the 2 intracellular "second messenger" systems, cyclic adenosine monophosphate (cAMP) and the phosphoinositides. Lithium has an inhibitory effect on adenylate cyclase, leading to decreased levels of cAMP[92]. Psoriasis has been associated with decreased responsiveness of the P-adrenergic receptors in the epidermal cells[93]. These receptors are correlated with the adenylate cyclase cAMP system. Further inhibition of adenylate cyclase by lithium therefore can worsen psoriasis. Lithium also affects the phosphoinositide pathway by inhibiting the enzyme inositol monophosphatase, thus slowing the rate of synthesis of phosphatidylinositol[94]. In psoriasis abnormalities have been found in the arachidonic acid transformation cascade[95]. This can be further exacerbated by the effect of lithium on the phosphoinositide pathway. Major depressive disorder has been associated with reduction in lymphocytes P-adrenergic

responsiveness, as measured by agonist-induced cAMP production[96]. The lymphocytes have been implicated as peripheral models of central p-adrenergic receptor function. This possible defect in P-adrenergic receptor function in both depressive illness and psoriasis, along with some reports of increased prevalence of depressive symptoms[88] and alcoholism among psoriatics, suggests that the association between psoriasis and affective disorders requires further investigation.

Neuropeptides and Psoriasis:

Farber et al[97] proposed that substance P, a neuropeptide involved in itch and pain perception and the modulation of inflammation, may be involved in psoriasis, particularly in cases where the lesions follow a symmetric dermatomal distribution. Substance P has been demonstrated in intraepidermal nerve endings and there have been reports of resolution of psoriasis with cutaneous nerve resection. Since the epidermis and the nervous system are developmentally both derived from the embryologic ectoderm, neural factors may affect epidermal cells .

Cutaneous Blood Flow:

Psoriasis has been associated with increased cutaneous blood flow[97], and improvement in psoriasis has been associated with decrease in the cutaneous blood flow[98,99,100]. This may be because of the change in morphology of the capillaries of psoriatic skin[98]. More than three decades ago Graham[103] had observed that cutaneous blood flow, as measured by skin temperature and the reactive hyperemia threshold, increased significantly in the patient with psoriasis when the topic concerning distressing life situation was discussed. Several studies have reported that temperature

biofeedback training was associated with a noteworthy decrease in the severity of psoriasis.

Depression in Psoriasis patients:

Studies have reported depression to the range of 10% to 58%[104] in patients with psoriasis. Severity of major depressive disorder was determined with HAM-D score. 53.8% had mild depression, 30.7% moderate depression and 15.5% severe depression[19]. Patients with psoriasis have been reported to be more depressed than controls on the Beck Depression Inventory[105] and to have usually higher rates of depression or psychopathology than patients with leprosy[106], lichen planus[104], and vitiligo[107]. Studies[8] suggest that inpatients with psoriasis express more suicidal ideation than outpatients with rates in psoriasis higher than that seen in patients with acne, alopecia areata, atopic dermatitis, or general medical conditions[8].

Predictors of depression: - There is general agreement that the clinical severity of psoriasis is not the principal predictor of depression in psoriasis. Although many studies have shown a correlation between depression and psoriasis severity[105,8] or visibility of plaques[109], one study suggested this might be more prevalent in women than in men[110]. Other studies have reported the absence of an association that suggests that any effect of disease severity on depression is mediated by other factors[111]. Symptoms of psoriasis, especially pruritus, are related to depression in patients with psoriasis[112] with the implication that the depressed state decreases the threshold for itch perception via an increase in central nervous system opiate levels. Similarly, there is evidence[113] that patient who report high levels of stress experience pruritus more frequently than patients with lower stress levels. The effects of other symptoms are less clear. In a survey of 225 hospital outpatients with psoriasis, Fortune and

colleagues[111] reported that depression was best predicted by gender, reporting more symptoms, a stronger belief in the severity of the consequences of psoriasis, and less use of coping strategies that help the patient to reappraise the impact of the condition on his or her life. Regardless of the clinical severity of patient's psoriasis, depression also has been associated with feelings of stigma that arise from deprivation of social touch[114]. Attempts to cope through venting emotions and the use of alcohol and non-prescription drugs also have been associated with depression in psoriasis outpatients. Although the non effectiveness of treatment has been shown to be associated with higher levels of distress[115]. Schmid-Ott and colleagues showed that patients whose psoriasis was rated as worse after 1 year were significantly more likely to engage in coping strategies such as the use of religion and rumination[20].

Anxiety in Psoriasis patients:

Studies of anxiety in psoriasis also have tended to employ different methods of assessment and to report on different aspects of the experience. The proportion of patients meeting criteria for an anxiety disorder is notably higher than that for depression in patients with psoriasis. Anxiety symptoms were assessed using the HAM-A by kumar S et al. They found that 52% psoriasis patient showed mild anxiety, 36% showed mild to moderate anxiety, and 12% showed moderate to severe anxiety[26]. In another study by Shrikanth B. Deshmukh et al using HAM-A in psoriasis patients they found that 40% had mild anxiety, 40% had moderate anxiety and 20% had severe anxiety[19]. Some studies have not found that patients with psoriasis have elevated levels of anxiety. Devrimci Ozguven and collegues[105] reported no significant differences between patients with psoriasis and controls on levels of anxiety as assessed by the Speilberg State-Trait Anxiety Scale. The sample size was small

(n=50), and the authors emphasize that the concentration of low psoriasis severity scores may have influenced results. Fear of negative evaluation[117] gives a particular social component to the anxiety experienced by patients with psoriasis, and social anxiety/ avoidance has been reported to be elevated in patients with psoriasis than in patients with atopic or contact dermatitis, acne, and vitiligo.

Predictors of Anxiety:

Anxiety is an important feature associated with psoriasis. Inevitably the cognitive processes that accompany anxiety, in particular, fears relating to interpersonal process such as perceived stigma experiences and worrying, also seem to be important in terms of its impact and in terms of informing patients understanding of the condition.

Anatomic location of psoriasis has not been assessed adequately in terms of its relation to distress in patients. Some studies have suggested a relationship between anxiety and presence of psoriasis on the face and hands[118]. Williamson and collegues[119] reported results from patients with nail involvement and found that greater nail involvement was associated with anxiety. Patients with more severe nail involvement by psoriasis also tended to have more severe arthritis and psoriatic arthropathy, however, which makes inferences about the specificity of the findings solely to anatomic location of psoriasis problematic. Greater use of two coping strategies, avoidance of anxiety-provoking situations and emotion-focused coping, tend to be associated significantly with greater levels of anxiety[111]. When patient are explained that the exacerbation is due to the stress , wide array of psychologic well being was noted[139].

One research group has begun to examine the effects of excessive worrying in psoriasis[120,121]. Some initial work has suggested that 40% of patients attending a tertiary referral clinic for psoriasis meet criteria for pathologic levels of worry[125]; 25% of patients have worry scores similar to scores found in patients with a diagnosis of generalized anxiety disorder[120]. Further work has illustrated this finding to be consistent[121]. Pathologic worry in patients with psoriasis seems to be associated with social evaluative concerns and beliefs about the cause of psoriasis being due to the patient's state of mind, rather than more disease-oriented variables[120,111]. A role for pathologic worrying in slowing the response of psoriasis to photo chemotherapy has been reported[121].

Quality of life assessment In Dermatology Patients:

What is quality of life?

Quality of life is a broad term without precise definition. It depends on number of factors: support from the friends and relatives, ability to work and interest in ones occupations, accommodation appropriate to expectations and of course, health and disabilities whether congenital or recently acquired disorder[129].

Quality Of Life and Psoriasis:

Psoriasis is a serious state strongly disturbing the way a person looks at himself and the way he is looked upon by others. It has great financial and economic ramifications. Psoriasis has a markedly negative impact on patients health related QoL. A survey by the national psoriasis foundation found that 75% of patients assumed that psoriasis had moderate to large negative impact on their QoL, with change in their every day activities[14]. Another study showed that at least 20% of psoriasis patients had tried committing suicide[18]. Furthermore, emotional and physical

effects of psoriasis were reported to have a markedly negative impact at patients' place of work as calculated by the validated scales like SF-8, Work Productivity Assessment Index (WPAI) and Hamilton Anxiety and Depression Scale (HAM-A and HAM-D).

Psoriasis patients frequently experience problems like maladaptive coping responses, self-esteem, problems in body image, self concept, have feelings of stigma, embarrassment and shame regarding their appearance[18]. This is commonly accompanied by a insight of being judged by others based on their disfigurement. People with psoriasis usually engage in coping strategies to keep away from unpleasant and unwanted social consequences. Though, many of these strategies do not succeed to improve patient's quality of life. Discussing their skin condition, avoiding contact with people and covering their lesions are significantly related with negative impact on life[8]. Studies have shown that talking to others in relation to the non-contagious nature of psoriasis reduces the negative impact on the Quality of Life and thereby decreases social distress.

Studies that have shown the association between psoriasis and depression hint towards a mutual connection between them. Psoriasis patients were more prone to be depressed than the general population with patients' age, disease severity and education being chief factors of psychological distress in the patient cohort[139]. In a study by Gupta et al of 127 psoriasis patients found that 9.7% of patients wanted to die, and 5.5% had suicidal tendency at the time of the study[8]. These studies have focussed the need for psychosocial planning in managing patients with psoriasis and helping them to progress in their overall Quality of Life.

Why try to measure quality of life?

QOL in dermatology is measured for clinical, research audit and for financial and political purposes.

All practitioners use an instinctive view of how much the skin disease is affecting their patients when taking treatment decisions, but patients may measure Quality Of Life in a different way from their doctors[129]. When there are data's to infer the scores, more precise measurement of Quality Of Life might be helpful in taking treatment decisions, for example where costly or perilous therapy is being initiated. The use of straightforward Quality Of Life, measures is equally welcomed by patients who wish to communicate their concerns.

Methods of measurement of quality of life

The techniques used to measure QOL are questionnaire based. The concepts involved in the development of QOL measures have been explained in detail. These measures either cover all ways in which patients lives can be affected by any disease, or are more specific to diseases of systems or individual diseases. The detail asked about and the question base-vary widely. In dermatology several general health quetionaires have been used and disease specific and speciality specific questionnaires have been created.

Generic QOL measures:

1. **Short Form 36 (SF-36)**

The widely used health survey is SF-36 ,contains 36-item, 8 domains of health status are checked by self reporting health status questionnaire(1) physical activities; (2) usual physical role activities; (3) social activities; (4) bodily pain; (5)

usual emotional role activities; (6) general mental health; (7) vitality ;(8) general health perceptions. A score ranges from 0 to 100, with higher values indicating superior HRQL[122]. The SF-36 may be the best considered measure for comparing Quality of Life differences across diverse diseases. The SF-36 was used to demonstrate that the impact of psoriasis is as enormous as that of other major medical diseases[123].

2. **Subjective Well Being Scale (SWLS)**

This is a short tool consisting of 5-varibles intended to measure global life satisfaction. It is validated and correlated with other method of subjective well-being (SWB). This was made to gauge contentment with the respondent's life completely, without assessing contentment with definite life event[122].

3. **EuroQoL 5D (EQ-5D)**

EQ-5D is the standardized generic tool developed for describing and valuing health states. This tool is developed for the use in population health surveys or in combination with a condition-targeted instrument for evaluation of outcomes related to definite health conditions or their treatment. It particularly refers to health status at the time of inquiring. The initial two parts measures a patient's health state along 5 variables (mobility, usual activities, self-care, anxiety/depression and pain/discomfort). Each variable has three levels referring to no problem, some problem, and extreme problem. Patients are asked to point to one of the 3 levels along each of the 5 variables. This classifies patients into 1 out of 243 distinct health states. The second part of the EQ-5D is the EQ-VAS which records the respondent's self-rated health on a vertical, visual analogue scale where the endpoints are labelled 'The

best health you can imagine' and 'The worst health you can imagine'. Patients are asked to demonstrate how they rate their own health state by drawing a line to that best represent to routine activities, their own health state on that day[122].

4. **Sickness Impact Profile assessment:**

The sickness Impact Profile (SIP) is a broadly based assessment of performance of daily activities. One hundred thirty- six individuals statements relating to daily activities can be agreed or disagreed by patients. These are grouped into 7 main categories and 5 subcategories which addresses to physical, psychosocial, sleep and rest, eating, work, home management, recreation and pastimes[14].

5. **Nottingham Health Profile:**

The Nottingham health profile consists of 38 statements combined to form six scales reflecting health problems, such as physical mobility and pain, and severe other statements about areas of daily life affected most often by health. A weighting formula is applied to the tick box answers[14].

6. **General health questionnaire:**

GHQ is a self administered screening questionnaire. There are 60,30,28,12 item versions. The 12 and 28 item versions are used for skin conditions. The 28 item version has 4 subscales addressing somatic symptoms, anxiety and insomnia, social dysfunction and severe depression[14].

Mixed QoL measures

1. **Salford Psoriasis Index (SPI)**

This index is derived from combining a score of present severity of psoriasis based on the PASI, score based on past information, and a score demonstrating psychosocial disability. The resultant 3- figure SPI (signs, psychosocial disability, interventions) is an analogous concept to the TNM (tumor, nodes, metastasis) classification used for cancer staging[123].

2. **Koo-Menter Psoriasis Instrument (KMPI)**

This is a diagnostic algorithm and a prescribed measure, to assist in identifying patients with significant impact on Quality of Life warranting systemic therapy. In addition, this instrument can also be used to file and validate treatment decisions for patients. While the choice to undertake a systemic treatment and the selection of treatment plan must be made by the patient and the physician mutual conclusion, these tools are intended to provide information that will be essential in making knowledgeable decisions regarding treatments[124]

Skin-specific measures:

1. **Questionnaire on Experience with Skin Complaints (QES)**

The short form of the QES with 23 variables is a valid tool for examination of psychic and social burdens of psoriasis. The recording of stigmatization feeling and of QoL determines diverse additional aspects of the illness-related stress of patients with chronic skin diseases[125].

2. Dermatology Life Quality Index (DLQI)

The DLQI is a condensed self-reported questionnaire to measure HRQoL over the former weeks in dermatologic patients. It consists of 10 parameters covering symptoms and feelings (items 1 and 2), daily activities (items 3 and 4), leisure (items 5 and 6), work and school (item 7), personal relationships (items 8 and 9) and treatment (item 10). Each parameter is scored on a 4 point scale, with greater scores demonstrating greater impairment in HRQoL[129].

Psoriasis-specific measures:

1. Psoriasis Index of Quality of Life (PSORIQoL)

This index is based on a "needs-based" approach. This tool is based on the theory that "life gains its quality from the ability and capacity of individuals to satisfy their needs". This 25 dichotomous variable tool was developed through interviews conducted in 3 countries in Europe. An American report has also been recently developed. It has the advantage of being based on theory and measuring the impact of the disease on Quality of Life rather than assessing disability or impairment. Furthermore, it is likely to work in a uniform manner across patient samples, irrespective of gender and age [126].

2. **Psoriasis Life Stress Inventory (PLSI)**

The PLSI is a 15-variable questionnaire that measures the daily hassles of psychosocial stress linked with having to cope up with daily events in living with psoriasis. Scores on this scale range from 0 to 45. The PLSI also allows patients to categorize into two groups according to scores: those patients who react extensively to the stress related with having psoriasis (score of > 10); and those patients who are not

noticeably affected with having psoriasis-related stress (score of<10). The PLSI is scored by having the respondent rate the total incidence with which each item has been experienced in the last 4 weeks from (scoring 0) to a great deal (scoring 3)[127].

3. Psoriasis Disability Index (PDI)

The PDI is a 15-variable scale that particularly addresses self-reported disability in areas of routine activities, leisure, employment, personal relationships and treatment effects[127].

4. Psoriasis Area and Severity Index (PASI) and Self Administered PASI (SAPASI)

Four major areas were assessed to calculate the PASI scores: head, trunk, upper extremities, and the lower extremities, corresponding to 10%, 20%, 30%, and 40% of the total body area, respectively. The highest score for PASI is 72. The SAPASI is a self-assessed, using the similar criteria as the PASI, but presented in amateur terminology. Scores on the SAPASI range from 0 - 72. Though in essence the PASI and the SAPASI are measures for severity of psoriasis, they provide an sufficient picture of the impact of the disease on patients' Quality of Life. Studies have shown an inverse relationship between Quality of Life and severity of psoriasis. Moreover, PASI is the most widely accepted measure of severity in the research as well as the clinical situation. This makes it a key tool in measuring the impact of the disease on Quality of Life. Since PASI or SAPASI do not gauge the impact of psoriasis on patients' Quality of Life directly, use of other QoL scales is suggested.

Dermatology Life Quality Index (DLQI):

The Dermatology Life Quality Index is a condensed self-reported questionnaire to gauge HRQoL over the previous week in patients with skin diseases. It consists of 10 variables covering symptoms and feelings (item 1 and 2), daily activities (items 3 and 4), leisure (items 5 and 6), work and school (item 7), personal relationships (items 8 and 9) and treatment (item 10). Each item is scored on a 4 point scale, with higher scores representing greater impairment in HRQoL[129].

INSTRUCTIONS FOR USAGE:

DERMATOLOGY LIFE QUALITY INDEX (DLQI)

The DLQI questionnaire is designed for use in adults, i.e. > 16 years. It is self descriptive and can be simply given to the patient who is requested to fill it without the need for detailed explanation. It take one to two minutes to complete the questionnaire.

Scoring

The scoring of each question is as follows:

1) Very much- scored 3
2) A lot- scored 2
3) A little- scored 1
4) Not at all- scored 0
5) Not relevant- scored 0
6) Question unanswered- scored 0
7) Question 7(prevented work or study) scored 3

The DLQI is calculated by adding the score of each question, score ranges between 0-30 . The higher the score, the more is the impairment of quality of life. The DLQI can also be expressed as a percentage of the highest probable score of 30.

Meaning of DLQI score:

0-1 = no effect at all on patients life

2-5 = small effect on patients life

6-10 = moderate effect on patients life

11-20 = Very large effect on patients life

21-30 = Extremely large effect on patients life

The DLQI can be analyzed under following 6 headings:

	Questions	**Maximum score**
Symptoms/feelings	1 and 2	6
Daily activities	3 and 4	6
Leisure	5 and 6	6
Work/School	7	3
Personal relationships	8 and 9	6
Treatment	10	3

Interpretation of incorrectly completed questionnaires

1. There is a very high success rate of precise conclusion of the DLQI. Nevertheless, occasionally subjects do make mistakes.
2. If one question is left unanswered this is scored 0 and the scores are added and expressed as usual out of a maximum of 30.
3. If two or more questions are left unanswered the questionnaire is not scored.
4. If question 7 is answered 'yes' the score is given as 3. If question 7 is answered 'no' or 'not relevant' but then either 'a lot' or 'a little' is marked this then score is given as 2 or 1.
5. If two or more answer options are ticked, the response option with the maximum score should be recorded.
6. If there is a response between two tick boxes, the lesser of the two score options should be recorded.

The DLQI can be measured by calculating the score for each of its 6 sub-scales (see above). If the answer to one question in a sub-scale is missing, that sub-scale should not be scored.

The DLQI scale is attached with the Proforma.(Annexure IV)

Hamilton Anxiety Rating Scale:

It is a psychological questionnaire used by clinicians to rate the severity of a patient's anxiety. It contains 14 symptom oriented questions. Each item is scored on a scale of 0 (not present) to 4 (severe), with a total score range of 0–56,

where:-

<17 indicates mild severity

18–24 mild to moderate severity

25–30 moderate to severe.

>30 indicates very severe.

The HAM-A scale is attached with the proforma.(Annexure IV)

Hamilton Depression Rating Scale:

It is a multiple item questionnaire used to provide an indication of depression, and as a guide to evaluate recovery. In all it has 17 questions of which Eight items are scored on a 5-point scale, ranging from 0 = not present to 4 = severe. Nine are scored from 0-2. With a total score range of 0-50.

where:-

0-7 = Normal

8-13 = Mild Depression

14-18 = Moderate Depression

19-22 = Severe Depression

≥ 23 = Very Severe Depression

The HAM-D scale is attached with the proforma.(Annexure IV).

Since significant number of patients with psoriasis have Anxiety and depression. The treatment of the same is given below.

Treatment of Depression in Psoriasis Patients:

Selective serotonin reuptake inhibitors (SSRIs) are the first line treatment for depression. Doxepin is probably the only tricyclic antidepressants (TCA) worth considering as first line treatment in dermatology, because of its combined antipruritic, antidepressant and antihistaminic effects. There are two more antidepressants with altered MOA other than SSRIs that are worth considering when treating psychodermatologic patients who have depression: Bupropion (Wellbutrin) and Venlafaxine(Effexor). Nefazodone (Serzone) was removed from market recently because of hepatic toxicity[14].

Treatment of Anxiety in Psoriasis Patients:

Psychodermatologic cases like Psoriasis, involving anxiety can be grouped into two groups: acute and chronic anxiety. For acute anxiety in dermatological patients treatment can be started with Alprazolam, 0.25mg tablet 6 hourly, on as needed basis. Buspirone is choice for the treatment of chronic anxiety because it is an anti-anxiety medication that does not cause dependency and is nonsedating[14].

Psychotherapy in psoriasis patients:

Increasingly then, within the dermatological literature, attention was given to the therapeutic benefits that might derive from psychological interventions beyond those of standard medical care. Given the close and clear associations between psychological factors and cutaneous conditions, it is not surprising that the effects of such treatment have been investigated. The literature has documented psychological

interventions for a number of cutaneous conditions, such as vitiligo, psoriasis, acne and atopic dermatitis, which have been suggested to be as effective for each of these types of disorders as classical medical procedures[132,133].

Psychological approaches, such as psychoanalysis and hypnosis[135] as well as behavioural[136] and cognitive–behavioural therapy[133] have been used to treat people affected by skin disorders (see Table2.) Indeed, such interventions have been shown to produce clinically significant improvements in cutaneous conditions, such as atopic dermatitis (eczema), psoriasis, vitiligo and virus-mediated diseases[132] and have helped patients to improve their psychological well-being and quality of life[137,133]. Outlined below are the main therapeutic techniques used in dermatology settings.

Table 2. Approaches to treatment of dermatological conditions

	Behavior therapy	Cognitive behavior therapy	Group therapy	Psychodynamic psychotherapy
Time frame	Here and now	Here and now	Here and now	Understanding the past, focuses on current relationship
Cost	Cost effective	Cost effective	Cost effective	Expensive
Technique	Systematic desensitization, relaxation,habit reversal training, imagery	Problem solving, Cognitive restructuring, Guided imagery, modelling	Psycho education, social and assertiveness skills training, role play	Analysis of transference and countertransference,hypnosis
Time	Short term	Short term	Short term	Long term
Efficacy	Psoriasis,Eczema, Vitilgo, Acne	Psoriasis,Eczema, Vitilgo, Acne	Psoriasis,Eczema,Vitilgo	Eczema

MATERIAL AND METHODS

Source Of Data:

Patients having Psoriasis and visiting Dermatology outpatient department of KLE's Dr Prabhakar Kore Hospital and Medical Research Centre, Belgaum over a period of 1 year.

Methods Of Collection Of Data:

1. Informed consent: Patients who fulfilled the selection criteria were briefed about the nature of study and a written informed consent was obtained (Annexure I).
2. 64 patients of Psoriasis were evaluated under the study. Relevant history, clinical examination along with PASI were done for all the patients. (Annexure II).
3. Copy right permission, for the use of DLQI scale in our study was taken by Dr. A. Y. Finlay, Proffessor and HOD Department Of Dermatology, University of medicine Cardiff, Wales, U.K. (Annexure III).
4. Quality of life of all psoriasis patients was evaluated by using DLQI structured questionnaire. (Annexure IV).
5. Severity of Psoriasis was calculated using PASI score. (Annexure IV).
6. Screening for anxiety and depression in all the above patients was done using Hamilton anxiety and depression scales (HAM-A and HAM- D). (Annexure IV).

Sample Size:

Mean from last 3 years data record of psoriasis patients visiting dermatology, venereology and leprosy opd of kle hospital is 80.

80% of mean i.e 64 was our sample size.

Inclusion Criteria:

1. Patients with all types of Psoriasis above age of 18 years.
2. Psoriasis patients of both sexes were evaluated
3. All newly diagnosed cases, as well as old cases of psoriasis with exacerbations were evaluated under study.

Exclusion Criteria:

1. All patients of psoriasis below 18 years were excluded from the study.
2. Patients having any other chronic skin disease than psoriasis.
3. Patients not willing to be a part of the study.

Ethical clearance:

Prior to the commencement, the ethical clearance was obtained from Institutional Ethics Committee dated 7/2/13, Jawaharlal Nehru Medical College, Belgaum.

Statistical analysis

The data obtained was coded into Microsoft excel spreadsheet (Annexure III). The continuous data was expressed as mean ± standard deviation (SD). Categorical data was expressed in terms of rates, ratios and percentages and comparison was done using chi-square test. A probability value ('p' value) of less than or equal to 0.050 was considered as statistically significant.

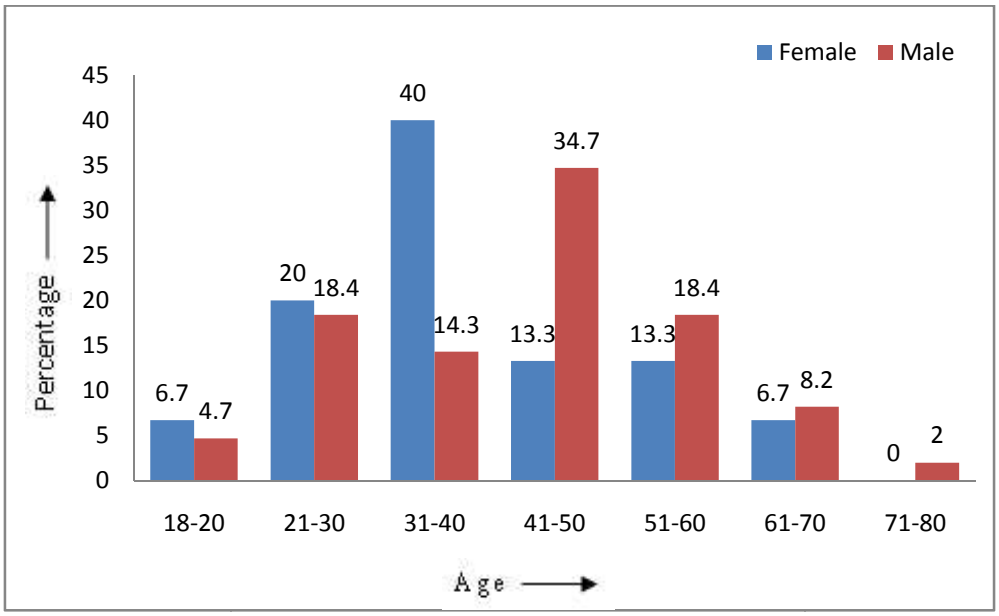
RESULTS

Table 3: **Distribution according to Age and Sex in the study group** n=64

Age(years)	Female	Male	Total
18-20	1 (6.7%)	2 (4.7%)	3 (4.6%)
21-30	3 (20%)	9 (18.4%)	12 (18.8%)
31-40	**6 (40%)**	7 (14.3%)	13 (20.3%)
41-50	2 (13.3%)	**17 (34.7%)**	**19 (29.7%)**
51-60	2 (13.3%)	9 (18.4%)	11 (17.2%)
61-70	1 (6.7%)	4 (8.2%)	5 (7.8%)
71-80	0	1 (2%)	1 (1.6%)
TOTAL	15	49	64

Fisher exact test P-value= 0.348

Graph 1: **Showing the distribution according to Age and Sex in the study**



In this study, out of 64 patients of psoriasis 49 were males and 15 were females.

In the present study the commonest age group was 41 to 50 years comprised of 30% of the patients.

Among males, the commonest age group was 41-50 years(34.7%) while among females 31-40 years(40%) was the the commonest.

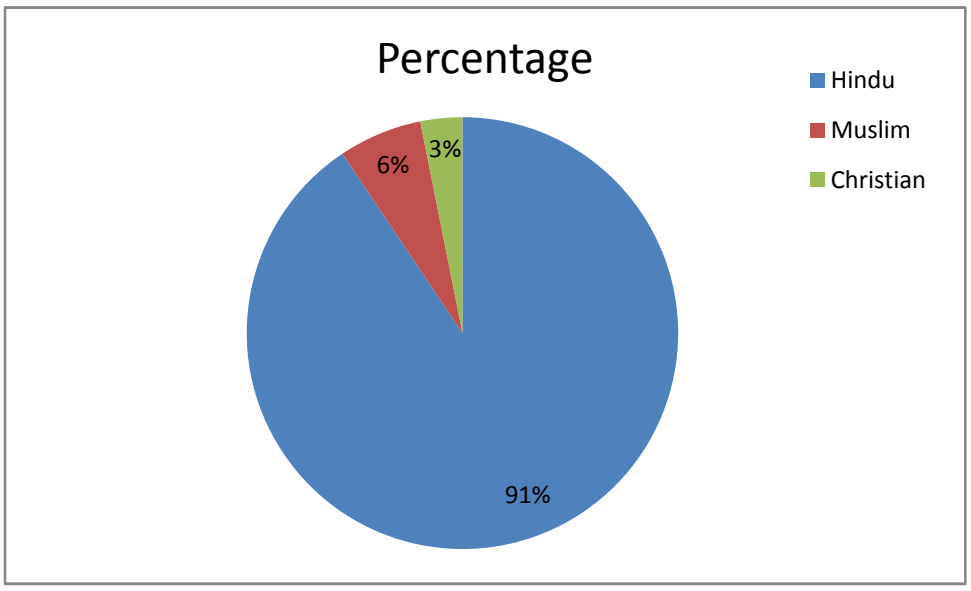
The difference was not statistically significant (P-value= 0.348).

The distribution of other age groups is as shown in Table 1 and Graph 1.

Table 4: **Distribution according to Religion in the study group** n=64

Religion	Number of Patient's	Percentage
Hindu	58	90.6
Muslim	4	6.3
Christian	2	3.1
TOTAL	64	100

Graph 2: **Showing the distribution according to Religion and Sex in the study group**



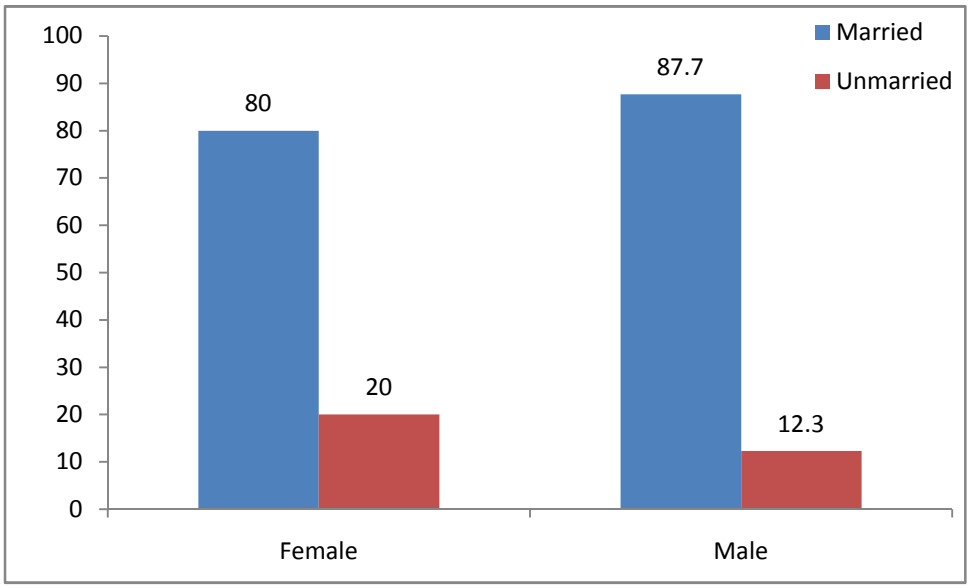
In this study, out of 64 patients 58 (91%) were hindus, 4 (6%) were muslims and 2 (3%)were christians.

Table 5: **Distribution according to Marital Status in the study group** n=64

Marital Status	Female	Male	Total
Married	12 (80%)	43 (87.7%)	55 (85.9%)
Unmarried	3 (20%)	6 (12.3%)	9 (14.1%)
TOTAL	15	49	64

$\chi 2$ with yates correction= 0.111; P-value= 0.746

Graph 3: **Showing the distribution according to Marital Status and Sex in the study**



In this study, out of 64 patients 86% of them were married and 14% unmarried.

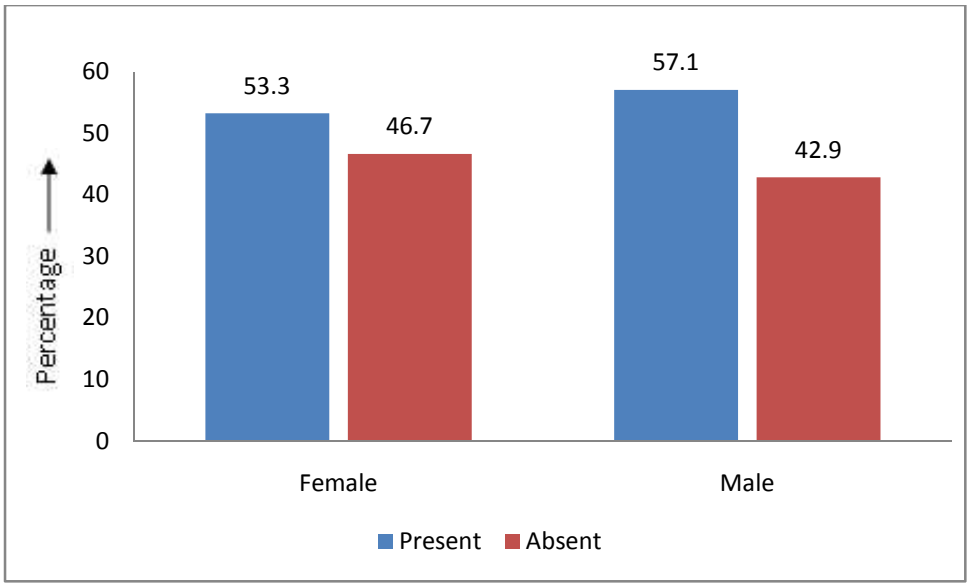
Number of married females were 80% whereas number of married males were 88%.

Table 6: **Distribution according to Prior Stress and Sex** n=64

Stress	Female	Male	Total
Present	8 **(53.3%)**	28 **(57.1%)**	36 **(56.3%)**
Absent	7 (46.7%)	21 (42.9%)	28 (43.7%)
TOTAL	15	49	64

$\chi_1^2 = 0.068$; P-value= 0.795

Graph 4: **Showing the distribution according to Prior Stress and Sex in the study group**



In this study, out of 64 patients with psoriasis, 56% of the patients reported stress as one of the exacerbating factor.

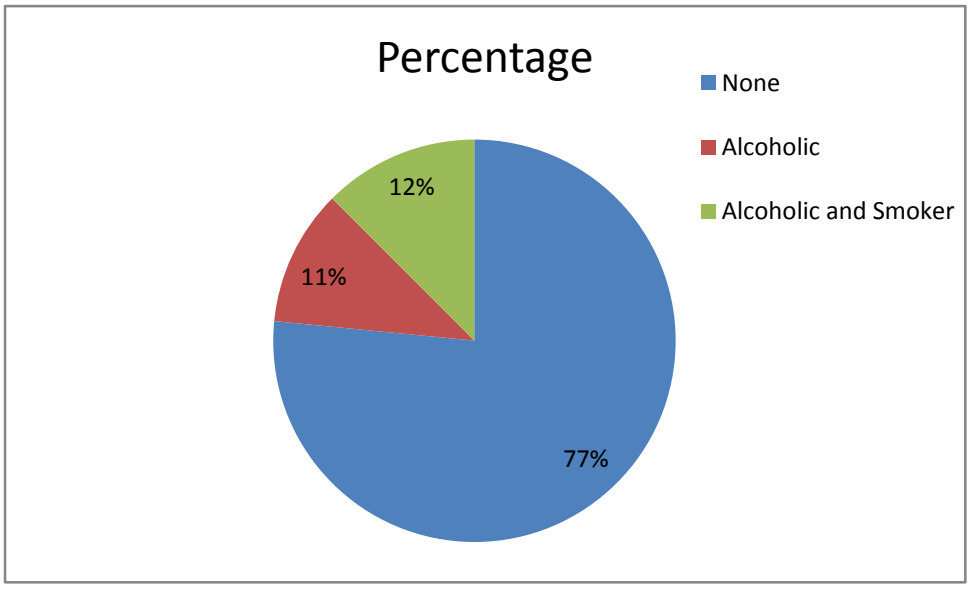
Prior stress reported among males was 57% and among females was 53%.

There was no significant difference in the incidence of stress among males and females (P-value= 0.795).

Table 7: **Distribution according to Habits in the study group** n=64

Habits	Number of Patient's	Percentage
None	49	76.6%
Alcoholic	7	10.9%
Alcoholic and Smoker	8	12.5%

Graph 5: **Distribution according to Habits in the study group**

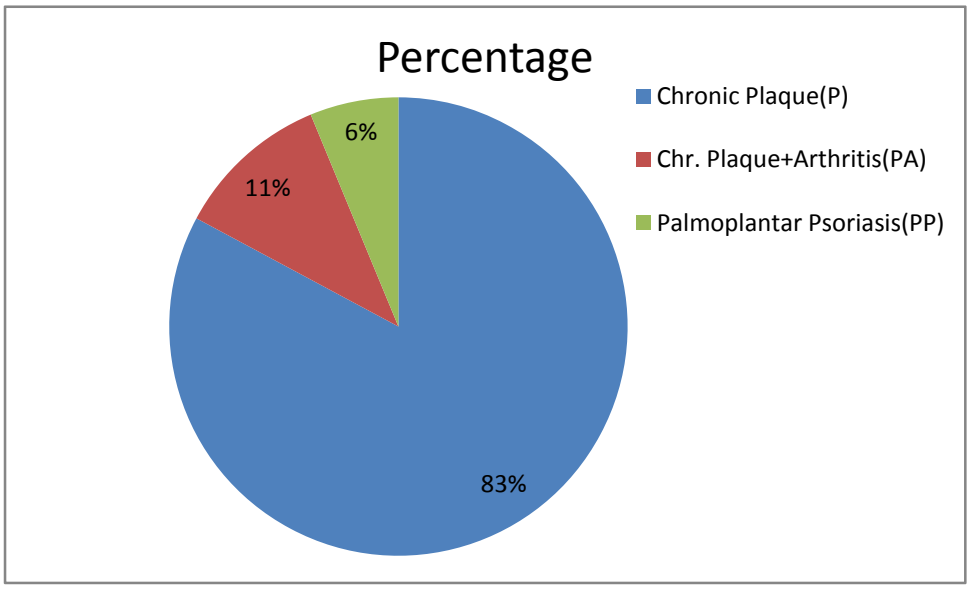


In this study, out of 64 patients with psoriasis, 11% of the patients were alcoholic and 12% of the patients were alcoholic and smoker.

Table 8: **Distribution according to different types of Psoriasis** n=64

Type of Psoriasis	Number of Patient's	Percentage
Chronic Plaque(P)	53	**82.8%**
Chr. Plaque+Arthritis(PA)	7	**10.9%**
Palmoplantar Psoriasis(PP)	4	**6.3%**

Graph 6: **Distribution according to different types of Psoriasis**

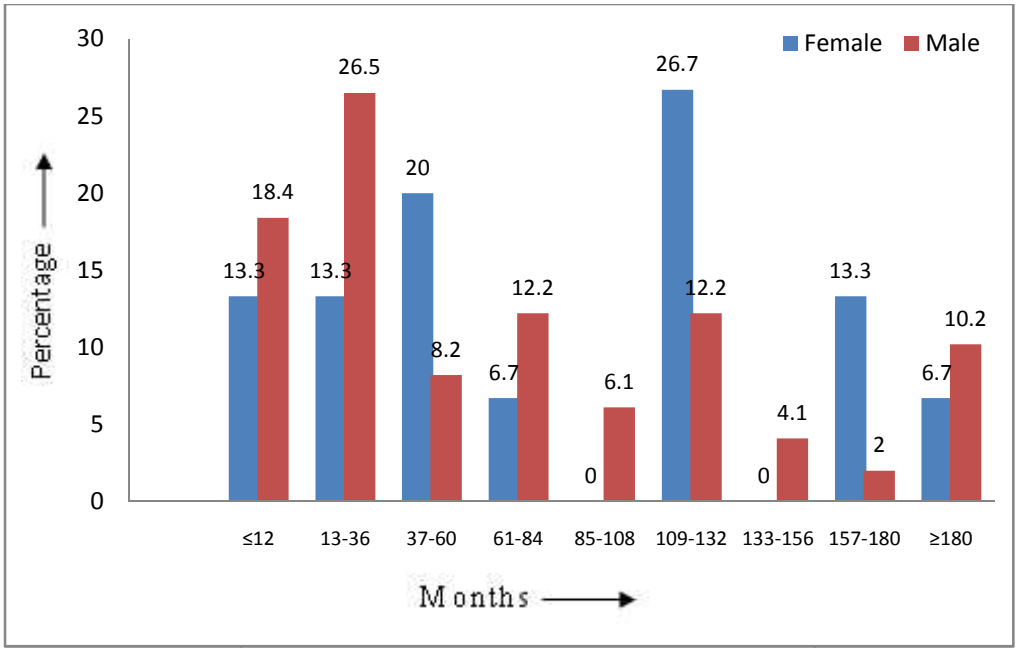


In this study, out of 64 patients with psoriasis, 83% of the patients had chronic plaque type of Psoriasis, 11% had chronic plaque associated with arthritis and 6% had palmoplantar type of Psoriasis.

Table 9: **Distribution according to Duration of disease and Sex** n=64

Duration of Disease (months)	Female	Male	Total
≤12	2 (13.3%)	9 (18.4%)	11 (17.2%)
13-36	2 (13.3%)	13 **(26.5%)**	15 **(23.4%)**
37-60	3 (20%)	4 (8.2%)	7 (10.9%)
61-84	1 (6.7%)	6 (12.2%)	7 (10.9%)
85-108	0	3 (6.1%)	3 (4.7%)
109-132	4 **(26.7%)**	6 (12.2%)	10 (15.6%)
133-156	0	2 (4.1%)	2 (3.1%)
157-180	2 (13.3%)	1 (2%)	3 (4.7%)
≥180	1 (6.7%)	5 (10.2%)	6 (9.3%)
TOTAL	15	49	64

Graph 7: **Distribution according to Duration of disease and Sex**



According to this study 17% of the patients had psoriasis for less than 1 year.

Most number of cases were between 1-3 years that is 23.4%.

There was no significant difference in the duration of disease between males and females.

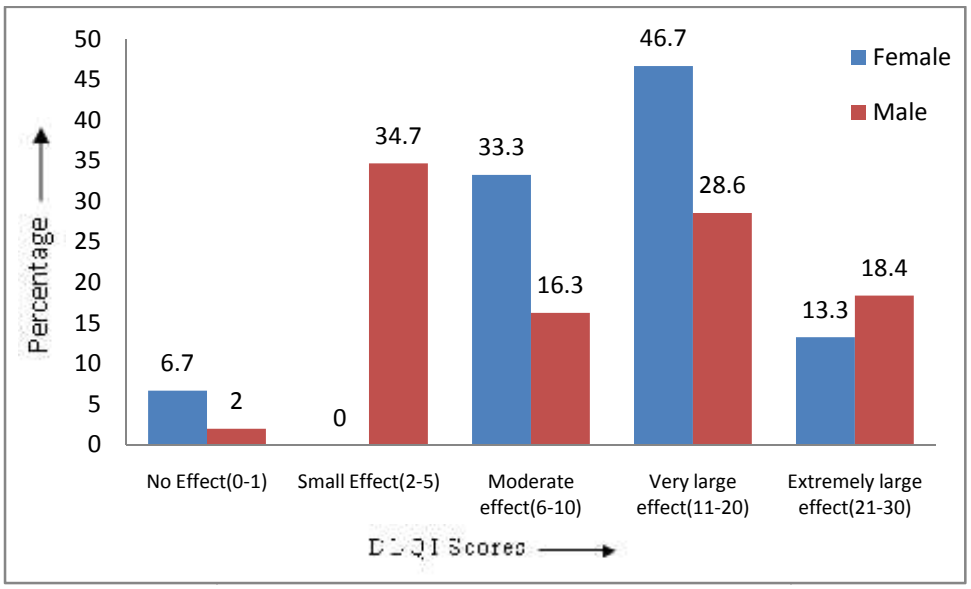
Table 10: **Distribution according to DLQI and Sex** n=64

DLQI	Female	Male	Total
No Effect(0-1)	1 (6.7%)	1 (2%)	2 (3.1%)
Small Effect(2-5)	0	17 **(34.7%)**	17 (26.6%)
Moderate effect(6-10)	5 (33.3%)	8 (16.3%)	13 (20.3%)
Very large effect(11-20)	7 **(46.7%)**	14 (28.6%)	21 **(32.8%)**
Extremely large effect(21-30)	2 (13.3%)	9 (18.4%)	11 (17.2%)
TOTAL	15	49	64

Fisher Exact Test P value= **0.020**

*DLQI= Dermatological Life Quality Index

Graph 8: **Distribution according to DLQI and Sex**



Out of 64 patients, psoriasis had no effect on quality of life in 3% of the patients, small effect in 27% of the patients, moderate effect in 20% of patients, very large effect in 33% of the patients and extremely large effect in 17% of patients.

In this study, females were found to have profound effect on their quality of life compared to men.

The difference was statistically significant (P value= **0.020)**.

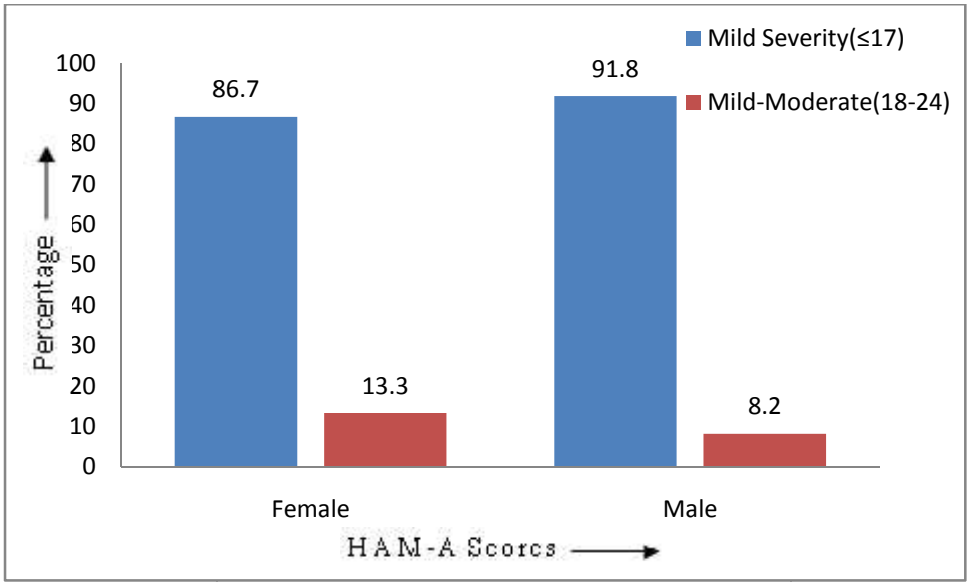
Table 11: **Distribution according to HAM-A and Sex** n=64

HAM-A	Female	Male	Total
Mild Severity(≤17)	13 **(86.7%)**	45 **(91.8%)**	58 **(90.6%)**
Mild-Moderate(18-24)	2 (13.3%)	4 (8.2%)	6 (9.4%)
TOTAL	15	49	64

$\chi 2$ with yates correction= 0.009; P-value= 0.924

*HAM-A= Hamilton-Anxiety score

Graph 9: **Distribution according to** HAM-A **and Sex**



Out of 64 patients who were screened for anxiety using Hamilton- anxiety scale, 91% had mild severity and 9% had mild- moderate anxiety.

None of the patients suffered from moderate-severe anxiety, values of which lie within the score range of 25-30 or very severe anxiety, values of which are above 30

There was not much difference seen between anxiety levels of males and females (P-value= 0.924).

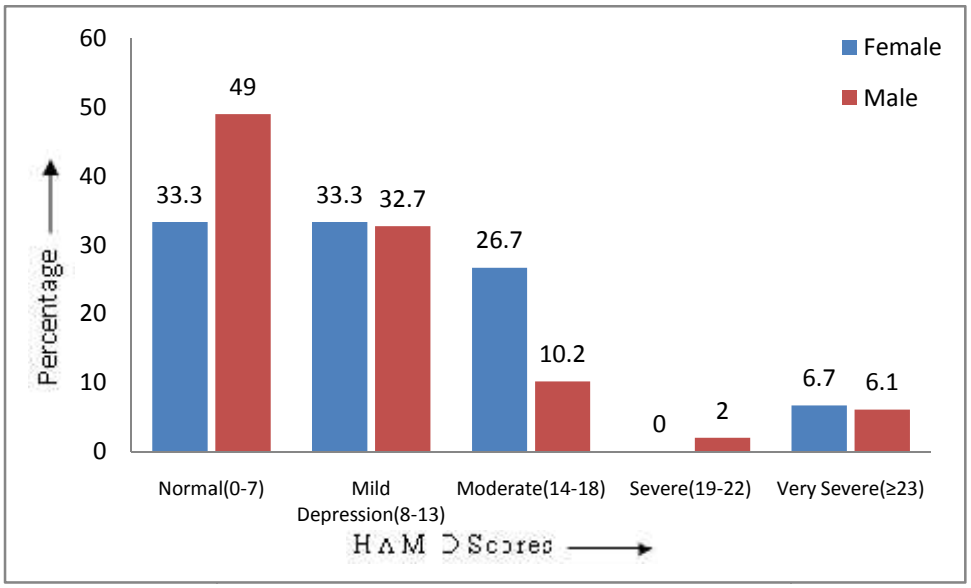
Table 12: **Distribution according to HAM-D and Sex** n=64

HAM-D	Female	Male	Total
Normal(0-7)	5(33.3%)	24(49%)	29(45.3%)
Mild Depression(8-13)	5**(33.3%)**	16**(32.7%)**	21**(32.8%)**
Moderate(14-18)	4(26.7%)	5(10.2%)	9(14%)
Severe(19-22)	0	1(2%)	1(1.6%)
Very Severe(≥23)	1(6.7%)	3(6.1%)	4(6.3%)
TOTAL	15	49	64

Fisher Exact test P value= 0.521

*HAM-D= Hamilton-Depression scores

Graph 10: **Distribution according to HAM-D and Sex**



Out of 64 patients who were screened for depression using Hamilton-depression scale, 45% were normal, 33% had mild depression, 14% had moderate, 2% had severe and 6% had very severe depression.

Slightly more depression was noted among females.

But the difference was not statistically significant (P value= 0.521).

Table 13: **Distribution according to PASI Score and Sex** n=64

	Mean	**Standard deviation**
Female	**5.1**	3.67
Male	**9.7**	6.58
Total	**8.6**	6.31

t_{62}= 2.586; P value=**0.012**

*PASI= Psoriasis Area Severity Index

Out of 64 patients mean PASI was 8.6 with standard deviation of 6.31.

Mean PASI score among females was 5.1 with a standard deviation of 3.67 whereas among males mean was 9.7 with standard deviation of 6.58. Thus males were more severely affected.

The difference between different sexes was statistically significant(P value=**0.012**).

CORRELATION ANALYSIS:

Table 14: **Comparison of Anxiety scores in patients with different DLQI scores.**

DLQI	Hamilton-A Scores		Total
	Mild Severity(≤17)	Mild-Moderate(18-24)	
No Effect(0-1)	2 (100%)	0	2
Small Effect(2-5)	16 (94.1%)	1 (5.9%)	17
Moderate effect(6-10)	12 (92.3%)	1 (7.7%)	13
Very large effect(11-20)	19 (90.5%)	2 (9.5%)	21
Extremely large effect(21-30)	9 (81.8%)	2 (18.2%)	11
TOTAL	58	6	64

Fisher Exact Test P value=0.862

In the sample size with varied DLQI score ranges, majority of the patients showed mild severity(≤17) of anxiety and few showed mild-moderate type of anxiety.

With increase in DLQI scores, anxiety was found to increase but it was not statistically significant (P value=0.862).

Table 15: **Comparison of Depression scores in patients with different DLQI levels.**

DLQI	**Hamilton-D Scores**					**Total**
No Effect(0-1)	0-7	8-13	14-18	19-22	≥23	
	2 (100%)	0	0	0	0	2
Small Effect(2-5)	13 (76.5%)	4 (23.5%)	0	0		17
Moderate effect(6-10)	9 (69.2%)	2 (15.4%)	2 (15.4%)	0		13
Very large effect(11-20)	4 (19%)	11 (52.4%)	5 (23.8%)	0	1 (4.8%)	21
Extremely large effect(21-30)	1 (9.1%)	4 (36.4%)	2 (18.2%)	1 (9.1%)	3 (27.3%)	11
TOTAL	29	21	9	1	4	64

Fisher Exact test P value- **<0.001**

In the sample size with varied DLQI score ranges, majority of the patients were found to be normal.

With increase in DLQI scores, Hamilton-D scores were found to increase.

The difference was statistically significant (P value- **<0.001**).

Table 16: **Comparison of PASI scores with DLQI scores**

S.No	DLQI	Mean PASI	Standard deviation
1.	No Effect(0-1)	4.1	0.14
2.	Small Effect(2-5)	5.1	2.96
3.	Moderate effect(6-10)	5.7	4.01
4.	Very large effect(11-20)	9.9	5.36
5.	Extremely large effect(21-30)	15.9	7.83

F=9.540; P value- **< 0.001**

With increase in DLQI scores, PASI scores were found to increase significantly.

On comparing data of S.No 2 with 5, P value- <0.001 **(Statistically significant)**

On comparing data of S.No 3 with 5, P value- <0.001 **(Statistically significant)**

On comparing data of S.No 4 with 5, P value- 0.043 **(Statistically significant)**

Table 17: **Comparison of PASI scores with HAM-A values**

n=64

S.No	HAM-A	Mean PASI	Standard deviation
1.	Mild Severity(≤17)	8.2	6.23
2.	Mild-Moderate(18-24)	12.6	6.12

t_{62}= 1.645; P value= 0.105

Out of the study sample it was seen that with increase in HAM-A Score, Mean PASI increases but the value is not statistically significant.

No patients fell under the category of moderate-severe and very severe anxiety.

Table 18: **Comparison of PASI scores with HAM-D values**

S.No	HAM-D	Mean PASI	Standard deviation
1.	Normal(0-7)	7.3	4.49
2.	Mild Depression(8-13)	9.6	5.97
3.	Moderate(14-18)	5.9	5.08
4.	Severe(19-22)	8.2	7.98
5.	Very Severe(≥23)	16.7	6.29

F= 2.808; P value= **0.033**

- Out of the study sample it was seen that with increase in HAM-D Score range Mean PASI increases and the value is statistically significant.
- On comparing data of S.No 3 with 5, P value- 0.045 **(Statistically significant)**

Table 19: **Correlation Matrix (Spearman)**

	DLQI	**HAM-A**	**HAM-D**
PASI	0.528(<0.001)	0.379(0.002)	0.352(0.004)
DLQI		0.618(<0.001)	0.692(<0.001
HAM-A			0.915(<0.001)

According to Spearman's Correlation, P-value on comparing:

a) PASI with DLQI is **<0.001** **(Statistically significant)**

b) PASI with HAM-A is **0.002** **(Statistically significant)**

c) PASI with HAM-D is **0.004** **(Statistically significant)**

d) DLQI with HAM-A is **<0.001** **(Statistically significant)**

e) DLQI with HAM-D is **<0.001** **(Statistically significant)**

f) HAM-A with HAM-D is **<0.001** **(Statistically significant)**

PHOTOGRAPHS

Photograph 1: Palmar psoriasis

Photograph 2: Psoriasis Vulgaris

Photograph 3: Psoriasis Vulgaris

Photograph 4: Psoriasis Vulgaris

Photograph 5: Psoriasis Vulgaris

Photograph 6: Psoriasis Vulgaris

Photograph7: Psoriasis vulgaris

Photograph 8: Psoriasis Vulgaris

Photograph 9: Scalp Psoriasis

Photograph 10: Scalp Psoriasis

Photograph 11: Nail Psoriasis

Photograph 12: Plantar Psoriasis

DISCUSSION

Psoriasis is a chronic disfiguring disease, often associated with physical disability, social discomfort and psychological disorders. It is important to consider this association in overall management of Psoriasis. Psoriasis patients react to stress, function sub-optimally, in terms of mental health and experience more disabilities in everyday life thus affecting the Quality of Life of the patient significantly. In order to consider the psychiatric co-morbidity, an appreciation for the effects of gender, age, marital status, duration and severity of disease and distribution of lesions is important, as well as the bi-directional relationship between skin disease and psychological distress.

Anxiety and depression remain the most common psychiatric disorders in patients with dermatological disorders. Gupta et al. have confirmed the association of anxiety and depression with psoriasis. Various questionnaires have been used, from time to time to determine the psychological effects of psoriasis. In the current study, PASI score was used to assess the severity of the disease. DLQI scale was used for assessing the quality of life of psoriasis patients and Hamilton Anxiety and Depression Scale was used to assess the anxiety and depression in psoriasis patients. The degree of psychiatric co-morbidity in dermatological patients varies in accordance with the setting of a study; type of patient, disease itself and the scale used[13].

This one year hospital based cross sectional study of 64 patients to identify Dermatological Life Quality Index (DLQI) in Psoriasis patients was conducted in the Department of Dermatology, Venereology and Leprosy, KLES Dr. Prabhakar Kore

Hospital and Medical Research Centre, Belgaum from January 2014 to December 2014.

1. Age distribution:

Maximum number of patients (30%) in the present study were between the age group of 41-50years .There was no significant difference in the age of onset in males and female.

Mehta et al[32], in their study of 300 patients revealed maximum number of patients between the age of 11years and 50 years and number of patients below 10 years and above 50years were minimum. Gunawardena et al[116] in his study reported that mean age of females was 25years and males 34.7 years.

Thus our values correlate with the data's of above- mentioned studies.

2. Sex distributions:

In the present study 76% of the psoriasis patients were male and 24% were females. Hence the sex ratio being 3.1:1

Inderjeet kaur[33] in their study of 782 patients revealed a male to female ratio of 2.3:1. Mehta et al[32], in their study reported a male to female ratio of 4:1. Bedi et al[34], in his study of 530 patients of psoriasis from north India revealed a male to female ratio of 2.4:1.

Thus our values correlate with the data's of above- mentioned studies.

3. Duration Of disease:

In the present study 17% of patients reported duration of illness below 1yr, 83% of patients had psoriasis for more than 1yr.

Sharma and sepaha[30], in their study reported 83.3% of patients having duration below 1 year and 46.6% of patients had duration of 1-6 years. Ambady et al[29], in their study reported 31.1% patients having duration of psoriasis below 1yr and 69.9% above 1yr.

The prolonged duration of the disease reveals the chronicity of the disease with frequent remissions and exacerbations.

4. Exacerbating Factors:

In the present study 56% of patients attributed the initiation or worsening of the disease due to prior stress.

In a survey of over 4500 dermatological patients, 2% of the patients had psoriasis, and emotional factors were reported to "trigger the onset of symptoms" in 62% of the psoriatics[84].

Studies using controls show that psychosocial causes were significant in the onset and/or exacerbating of symptoms in 39%[87] to 80%[88] of psoriatic patients v/s 10% to 50% of controls which consisted of patients with an assortment of non dermatological disorders respectively.

Thus the findings in our study correlates with that of above mentioned study.

5. QOL in Psoriasis Patients:

Out of 64 patients, psoriasis had no effect on quality of life in 3% of the patients, small effect in 27% of the patients, moderate effect in 20% of patients, very large effect in 33% of the patients and extremely large effect in 17% of patients. So Psoriasis affected the Qol in 97% of the patients.

Gladys Aires Martins et al in their study reported that psoriasis affected the quality of life in 99% of patients with age varying from 20 and 70years[140].

In a survey by the national psoriasis foundation almost 75% of patients believed that psoriasis had moderate to large negative impact on their quality of life, with alterations in their daily activities[138].

Thus the findings in our study correlates with that of above mentioned study.

6. Correlation between PASI scores and DLQI scores:

In our study significant correlation was found between the PASI score and the DLQI. Increase in the PASI score was associated with increased affection on quality of life.

Koo et al[141] in their study showed that there was significant correlation between PASI and DLQI.

Sampogna et al[142] in their study reported that quality of life instruments like DLQI and Skindex correlated with the PASI and SAPASI scores.

Thus our study outcomes are similar to above mentioned studies.

7. Anxiety in Psoriasis patients:

Out of 64 patients who were screened for anxiety using Hamilton- anxiety scale, 91% had mild severity and 9% had mild- moderate anxiety.

In a study by kumar S et al, they found using HAM-A that 52% psoriasis patient showed mild anxiety, 36% showed mild to moderate anxiety and 12% showed moderate to severe anxiety[26].

In another study by Shrikanth B. Deshmukh et al using HAM-A in psoriasis patients they found that 40% had mild anxiety, 40% had moderate anxiety and 20% had severe anxiety[19].

In our study there is higher incidence in mild anxiety levels and is not correlating with the above mentioned studies. This may be due to small sample size.

8. Depression in Psoriasis patients:

Out of 64 patients who were screened for depression using Hamilton-depression scale, 45% were normal, 33% had mild depression, 14% had moderate, 2% had severe and 6% had very severe depression.

In a study by Shrikant et al using HAM-D scores it was found that 53.8% had mild depression, 30.7% moderate depression and 15.5% severe depression[19].

In our study there is slightly lesser percentage of depression found among patients as compared to the above study mentioned.

CONCLUSION

This study concludes that, psoriasis has a significant negative impact on physical, emotional and psychological wellbeing of the affected patients, thus causing a substantial impact on patient's Quality of life. There is a significant correlation between disease severity and the QOL. There is also an association of Psoriasis vulgaris with psychiatric co-morbidity especially, anxiety and depression. The magnitude of this anxiety and depression can be influenced by variables like age, gender, marital status and duration of disease.

In view of this, it is very important to evaluate quality of life of psoriasis patients and also screen the patients for associated psychiatric co-morbid conditions like anxiety and depression, before making therapeutic decision.

Many more studies are needed in large group of patients with psoriasis to evolve comprehensive treatment guidelines involving the treatment of psychiatric morbidity associated with psoriasis in the overall successful management of psoriasis.

SUMMARY

A total of 64 patients of psoriasis who satisfied the inclusion criteria were included in the study. The aim was to evaluate the quality of life along with anxiety and depression in these patients.

- Majority of patients 50% were in the age group of 31- 50years.
- Majority of the patients were Hindu (91%), muslims (6%), and christians(3%).
- 86% of the patients were married.
- Male to female ratio was 3.1:1.
- In 17% of patients, the duration of the disease was less than 1year and 83% patients >1yr.
- Stress was initiating or exacerbating factor in 56% of patients.
- Chronic plaque psoriasis 83% was the commonest clinical type seen followed by palmoplantar psoriasis.
- Out of 64 patients, psoriasis had small effect on quality of life in 27% of the patients, moderate effect on 20% of patients, very large effect on 33% of patients and extremely large effect on 17% of patients. So Psoriasis affected the Quality of life in 97% of the patients.
- The mean PASI score was 8.6 with a standard deviation of 6.31.
- Significant correlation was noted between the PASI score and the DLQI.
- Out of 64 patients who were screened for anxiety using Hamilton-Anxiety scale, 91% had mild severity and 9% had mild-moderate anxiety.
- Out of 64 patients who were screened for depression using Hamilton-depression scale, 45% were normal, 33% had mild depression, 14% had

moderate, 2% had severe and 6% had very severe depression.

- With increase in DLQI scores, anxiety was found to increase but it was not statistically significant (P value=0.862).
- With increase in DLQI scores, Hamilton-D scores were found to increase. The difference was statistically significant (P value- **<0.001**).

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ANNEXURE -I

INFORMED CONSENT FORM

I.D.NO. _____

A One Year Cross Sectional study of Dermatological Life Quality Index (DLQI) in all the patients of Psoriasis

The study is conducted by _____ Post graduate student in M.D Dermatology, Venereology and Leprosy under guidance of J N Medical College, Belgaum.

Respected Sir/Madam, we invite you to participate in our study as, you are eligible for the same. During the study you will be asked some questions in detail regarding your present complaints.

Purpose of the study:

Individuals with psoriasis may feel self-conscious about their appearance and have a poor self-image that stems from fear of public rejection and psychosexual concerns. The purpose of this study is to evaluate the Quality of Life in patients suffering from all types of Psoriasis and to evaluate the psychological distress; anxiety & depression in patients of psoriasis**.** You are being asked to participate in this research because you have been diagnosed to have psoriasis. All patients attending the outpatient department, who are diagnosed to have this disease, will be requested to participate in this study during the period of one year.

Procedure and treatment:

Should you choose to participate, you will be asked to give a detailed history of your disease, undergo a dermatological physical examination, and answer questions which will be handed to you in the form of preformed questionnaire.

Risks and benefits:

The result of you taking part in this research would help health care providers towards a better understanding of this disease, and thus we will be able to provide improved patient care. There is no risk associated with the study.

Alternatives:

If you decide not to participate in this study, you will still be receiving the usual standard care for your disease.

Privacy and confidentiality:

Your privacy will be respected and all information collected about you during the course of this study will be kept confidential. Your identity will remain undisclosed.

Relations with the Institutional/Sponser's policy:

The J N Medical College will provide, within the limitations of the laws of the State of Karnataka, facilities and medical attention to patients who suffer injuries as a result of participating in this project. However, the treatment will not be free and no reimbursement will be provided.

Financial incentives:

You shall not be receiving any payment or any financial incentives for participating in this study.

Authorization to publish results:

The results of this study may be published for scientific purpose or presented to a scientific group. Your identity, however, will be maintained confidential at all times.

Voluntary participation:

Your participation in this study is voluntary. Your decision whether or not to participate will neither affect the care of your current disease, nor your future relations with the doctor or the hospital.

Questions:

In case you need further information regarding your rights as a study participant, you may please contact _____ or Dr. Ganga Pilli, Chairman of the Ethical Committee On Human subjects, J N Medical College, Belgaum on telephone No. 08312473350.

Statement of Consent:

I.D.NO:_____

I Mr/Ms/Mrs

_____

volunteer and consent to participate in this study. I have read the consent document or it has been read to me in my vernacular language. I accept to participate in the study. All the information regarding this study is provided to me and I have understood the same. I have been given the opportunity to ask questions and obtain appropriate answers.

Participant's name:

Signature or left thumb print of participant:

Witness name:

Signature of witness & Date :

Signature of the investigator & Date :

Signature of the guide :

ANNEXURE-II

PROFORMA

Case No:

Name : **IP No:**

Age: **OP No:**

Gender: **date :**

Occupation:

Address with Phone Number :

Present Complaint and Duration :

History of Present Illness:

1. a. Onset:

   i. Sudden ii. Gradual

   b. i. Progressive ii. Stationary

   H/o of erythroderma :

   1. Present 2. Absent

H/o of sore throat :

1. Present 2. Absent

H/o of stress n strain :

1. Present 2. Absent

H/o joint involvement :

1. Present 2. Absent

Triggering and modifying factor :

A. Local factors :

1. Trauma
2. Operation wound
3. Vaccination
4. Insect or animal bite

B. Seasonal Variation (exacerbation) :

1. Winter 2. Summer

C. Pregnancy

D. Drugs

E. Sunlight

F. Alcohol and smoking

G. Obesity

Initial Lesion:

1. Erythema
2. Pus filled lesion

   Any associated factors :

   i. Itching

   ii. Pain

   iii. Joint Pain

   iv. Asymptomatic

History of remissions and exacerbations :

1. Present 2. Absent

History of any other medical disorder

1. Present 2. Absent

Family History :

1. Present 2. Absent

Treatment History :

1. Topical
2. Phototherapy
3. Systemic
4. Others

Personal History :

Diet

1. Vegetarian 2. Mixed

Appetite

1. Normal 2. Stress

Bowel/Bladder:

1. Normal 2. Altered

Sleep

1. Normal 2. Altered

Alcohol intake :

1. Present 2. Absent.

Smoking

1. Present 2. Absent

Stress

1. Present 2. Absent

General Physical Examination :

1. Poor
2. Moderate
3. Good

Vitals

1. Pulse bpm
2. BP (mm/Hg)
3. Temperature (F)
4. Height (cm)
5. Weight (Kg)

Pallor

Icterus

Cynosis

Clubbing

Lymphadenopathy

Edema

Mucocutaneous Examination :

Types of lesions:

Papules

1. Present 2. Absent

Plaque

1. Present 2. Absent

Pustule

1. Present 2. Absent

Erythema

1. Present 2. Absent

Distribution :

1. Symmetrical
2. Asymmetrial
3. Localized
4. Generalized

Site of Lesions :

Scalp

1. Present 2. Absent

Face

1. Present 2. Absent

Neck

1. Present 2. Absent

Back

1. Present 2. Absent

Trunk

1. Present 2. Absent

Elbows

1. Present 2. Absent

Extensor aspect of extremities

1. Present 2. Absent

Flexures of upper limbs and lower Limbs :

1. Present 2. Absent

Palms

1. Present 2. Absent

Knees

1. Present 2. Absent

Axillae

1. Present 2. Absent

External genitalia

1. Present 2. Absent

Gluteal region

1. Present 2. Absent

Soles

1. Present 2. Absent

Size of lesion :

1. Small (0.5 to 1 cm in diameter)
2. Large (2 to 5 cm diameter)
3. Large (more than 10 cm in diameter)

Types of Scaling :

1. Firmly adherent scales
2. Loosely adherent scales
3. Mica like scales
4. Cone or limpet like scales
5. Oyster shell like scales

Clinical Pattern of Presentation :

1. Plaques
2. Annular
3. Guttate or eruptive
4. Pustules
5. Erythrodermic

Nail lesion :

1. Pitting
2. Subumgal hyperkerataxis
3. Onycholysis
4. Yellow discoloration
5. Beaus lines
6. Splinter hemorrhages
7. Oil drop

Joint involvement :

1. Present
2. Absent

If present which joints :

i. Distal interphalangeal

ii. Proximal interphalangeal

iii. Sacroiliac

iv. Metacorpophalangeal

v. Knee joint, elbow, joint wrist joint

Mucosal Examination :

Genital lesion :

1. Present
2. Absent

Oral lesion :

1. Present
. Absent.

Auspitz Sign :

1. Positve
2. Negative

Koebner's Phenomenon :

1. Present
2. Absent

Systemic Examination :

Cardiovascular System : heart sounds

1. Normal
2. Abnormal, if abnormal specify the findings,

Respiratory system : Breath sounds

1. Normal
2. Abnormal ; if abnormal specify the findings

Central nervous system :

1. Normal
2. Abnormal ; if abnormal specify the findings :

ANNEXURE III

*** Copy right permission letter from Dr.Andrew Y Finlay.***

Dear Dr Mohan

Thank you for your e-mail dated 29th September 2013. Thank you for your interest in the DLQI. We are happy to give you formal permission to use the DLQI. It is a requirement that the copyright statement must be reproduced at the end of every copy of the DLQI. There is no charge. There is considerable information concerning the DLQI on our website

www.dermatology.org.uk

(Click on Quality of Life)

I wish you every success in your research.

Yours sincerely,

Andrew Finlay

Professor Andrew Y Finlay CBE, FRCP

Research Affiliation:

Department of Dermatology and Wound Healing

Cardiff University School of Medicine, Cardiff, UK

ANNEXURE-IV

(English)

DERMATOLOGY LIFE QUALITY INDEX (DLQI)

Hospital No: **Date:**

Name: **Score:**

Address: **Diagnosis:**

The aim of this questionnaire is to measure how much your skin problem has affected your life OVER THE LAST WEEK. Please tick (✓) one box for each question.

1.	Over the last week, how **itchy**, **sore**, **painful** or **stinging** has your skin been?	Very much A lot A little Not at all	❒ ❒ ❒ ❒		
2.	Over the last week, how **embarrassed** or **self conscious** have you been because of your skin?	Very much A lot A little Not at all	❒ ❒ ❒ ❒		
3.	Over the last week, how much has your skin interfered with you going **shopping** or looking after your **home** or **garden**?	Very much A lot A little Not at all	❒ ❒ ❒ ❒	Not relevant	❒
4.	Over the last week, how much has your skin influenced the **clothes** you wear?	Very much A lot A little Not at all	❒ ❒ ❒ ❒	Not relevant	❒
5.	Over the last week, how much has your skin affected any **social** or **leisure** activities?	Very much A lot A little Not at all	❒ ❒ ❒ ❒	Not relevant	❒
6.	Over the last week, how much has your skin made it difficult for you to do any **sport**?	Very much A lot A little Not at all	❒ ❒ ❒ ❒	Not relevant	❒
7.	Over the last week, has your skin prevented you from **working** or **studying**?	Yes No	❒ ❒	Not relevant	❒
	If "No", over the last week how much has your skin been a problem at **work** or **studying**?	A lot A little Not at all	❒ ❒ ❒		
8.	Over the last week, how much has your skin created problems with your **partner** or any of your **close friends** or **relatives**?	Very much A lot A little Not at all	❒ ❒ ❒ ❒	Not relevant	❒
9.	Over the last week, how much has your skin caused any **sexual difficulties**?	Very much A lot A little Not at all	❒ ❒ ❒ ❒	Not relevant	❒
10.	Over the last week, how much of a problem has the **treatment** for your skin been, for example by making your home messy, or by taking up time?	Very much A lot A little Not at all	❒ ❒ ❒ ❒	Not relevant	❒

Please check you have answered EVERY question. Thank you.

(DLQI-Kannada)

ಡರ್ಮಟಾಲಜಿ ಲೈಫ್ ಕ್ವಾಲಿಟಿ ಇಂಡೆಕ್ಸ್

ಆಸ್ಪತ್ರೆ ಸಂಖ್ಯೆ: ದಿನಾಂಕ: ಸ್ಕೋರ್:

ಹೆಸರು: ದಯಗ್ನೊಸಿಸ್:

ವಿಳಾಸ:

ಈ ಪ್ರಶ್ನಾವಳಿಯ ಉದ್ದೇಶ ಕಳೆದ ಒಂದು ವಾರದಲ್ಲಿ ನಿಮ್ಮ ಜೀವನದ ಮೇಲೆ ನಿಮ್ಮ ಚರ್ಮದ ಸಮಸ್ಯೆ ಎಷ್ಟರ ಮಟ್ಟಿಗೆ ಪ್ರಭಾವ ಬೀರಿದೆ ಎಂದು ಅಳತೆ ಮಾಡುವುದು. ದಯವಿಟ್ಟು ಪ್ರತಿ ಪ್ರಶ್ನೆಗೂ ಒಂದು ದಾಖ್ಲೆಅನ್ನು ಟಿಕ್ ಮಾಡಿ.

1.	ಕಳೆದ ವಾರದಲ್ಲಿ, ನಿಮ್ಮ ಚರ್ಮದಲ್ಲಿ ಎಷ್ಟರ ಮಟ್ಟಿಗೆ **ನವೆ, ಹುಣ್ಣು, ನೋವು** ಅಥವಾ **ಕಡಿತ** ಉಂಟಾಗಿದೆ?	ಬಹಳ ಹೆಚ್ಚು ಬಹಳ ಸ್ವಲ್ಪ ಇಲ್ಲವೇ ಇಲ್ಲ	☐ ☐ ☐ ☐	
2.	ಕಳೆದ ವಾರದಲ್ಲಿ, ನಿಮ್ಮ ಚರ್ಮದ ಕಾರಣ ನೀವು ಎಷ್ಟರ ಮಟ್ಟಿಗೆ **ಜೀವಚೆಗೆ** ಅಥವಾ **ಸ್ವಪ್ರಜ್ಞೆಗೆ** ಒಳಗಾಗಿದ್ದೀರಿ?	ಬಹಳ ಹೆಚ್ಚು ಬಹಳ ಸ್ವಲ್ಪ ಇಲ್ಲವೇ ಇಲ್ಲ	☐ ☐ ☐ ☐	
3.	ಕಳೆದ ವಾರದಲ್ಲಿ, ನೀವು **ಅಂಗಡಿಗೆ** ಹೋಗುವುದು ಅಥವಾ **ಮನೆ** ಸ್ವಚ್ಛವೆ **ಉದ್ಯಾನವನ್ನು** ನೋಡಿಕೊಳ್ಳುವುದರಲ್ಲಿ ನಿಮ್ಮ ಚರ್ಮ ಎಷ್ಟರ ಮಟ್ಟಿಗೆ ಅಡ್ಡಿಯಾಗಿದೆ?	ಬಹಳ ಹೆಚ್ಚು ಬಹಳ ಸ್ವಲ್ಪ ಇಲ್ಲವೇ ಇಲ್ಲ	☐ ☐ ☐ ☐	ಪ್ರಕ್ನತವಲ್ಲ ☐
4.	ಕಳೆದ ವಾರದಲ್ಲಿ, ನೀವು ಧರಿಸುವ **ಬಟ್ಟೆಗಳ** ಮೇಲೆ ನಿಮ್ಮ ಚರ್ಮ ಎಷ್ಟರ ಮಟ್ಟಿಗೆ ಪ್ರಭಾವವೀರಿದೆ?	ಬಹಳ ಹೆಚ್ಚು ಬಹಳ ಸ್ವಲ್ಪ ಇಲ್ಲವೇ ಇಲ್ಲ	☐ ☐ ☐ ☐	ಪ್ರಕ್ನತವಲ್ಲ ☐
5.	ಕಳೆದ ವಾರದಲ್ಲಿ, ಯಾವುದೇ **ಸಾಮಾಜಿಕ** ಅಥವಾ **ಬಿಡುವಿನ** ಚಟುವಟಿಕೆಗಳ ಮೇಲೆ ನಿಮ್ಮ ಚರ್ಮ ಎಷ್ಟರ ಮಟ್ಟಿಗೆ ಪ್ರಭಾವವೀರಿದೆ?	ಬಹಳ ಹೆಚ್ಚು ಬಹಳ ಸ್ವಲ್ಪ ಇಲ್ಲವೇ ಇಲ್ಲ	☐ ☐ ☐ ☐	ಪ್ರಕ್ನತವಲ್ಲ ☐
6.	ಕಳೆದ ವಾರದಲ್ಲಿ, ಯಾವುದೇ **ಆಟ** ಆಡುವುದರಲ್ಲಿ ನಿಮ್ಮ ಚರ್ಮ ಎಷ್ಟರ ಮಟ್ಟಿಗೆ ತೊಂದರೆ ಕೊಟ್ಟಿದೆ?	ಬಹಳ ಹೆಚ್ಚು ಬಹಳ ಸ್ವಲ್ಪ ಇಲ್ಲವೇ ಇಲ್ಲ	☐ ☐ ☐ ☐	ಪ್ರಕ್ನತವಲ್ಲ ☐
7.	ಕಳೆದ ವಾರದಲ್ಲಿ, ನೀವು **ಕೆಲಸ** ಅಥವಾ **ಅಧ್ಯಯನ** ಮಾಡದಂತೆ ನಿಮ್ಮ ಚರ್ಮ ನಿಮ್ಮನ್ನು ತಡೆದಿದೆಯೆ?	ಹೌದು ಇಲ್ಲ	☐ ☐	ಪ್ರಕ್ನತವಲ್ಲ ☐
	'ಇಲ್ಲ' ಎಂದರೆ, ಕಳೆದ ವಾರದಲ್ಲಿ, **ಕೆಲಸ** ಅಥವಾ **ಅಧ್ಯಯನದಲ್ಲಿ** ನಿಮ್ಮ ಚರ್ಮ ಎಷ್ಟರ ಮಟ್ಟಿಗೆ ಸಮಸ್ಯೆಯಾಗಿತ್ತು?	ಬಹಳ ಸ್ವಲ್ಪ ಇಲ್ಲವೇ ಇಲ್ಲ	☐ ☐ ☐	
8.	ಕಳೆದ ವಾರದಲ್ಲಿ, ನಿಮ್ಮ **ಸಂಗಾತಿ** ಅಥವಾ ನಿಮ್ಮ **ಹತ್ತಿರದ ಸ್ನೇಹಿತರು** ಅಥವಾ **ನೆಂಟರ** ಜತೆ ನಿಮ್ಮ ಚರ್ಮ ಎಷ್ಟರ ಮಟ್ಟಿಗೆ ಸಮಸ್ಯೆ ತಂದಿತು?	ಬಹಳ ಹೆಚ್ಚು ಬಹಳ ಸ್ವಲ್ಪ ಇಲ್ಲವೇ ಇಲ್ಲ	☐ ☐ ☐ ☐	ಪ್ರಕ್ನತವಲ್ಲ ☐
9.	ಕಳೆದ ವಾರದಲ್ಲಿ, ನಿಮ್ಮ ಚರ್ಮ ಎಷ್ಟರ ಮಟ್ಟಿಗೆ ಯಾವುದೇ **ಲೈಂಗಿಕ ತೊಂದರೆಗೆ** ಕಾರಣವಾಗಿದೆ?	ಬಹಳ ಹೆಚ್ಚು ಬಹಳ ಸ್ವಲ್ಪ ಇಲ್ಲವೇ ಇಲ್ಲ	☐ ☐ ☐ ☐	ಪ್ರಕ್ನತವಲ್ಲ ☐
10.	ಕಳೆದ ವಾರದಲ್ಲಿ, ನಿಮ್ಮ ಚರ್ಮದ **ಚಿಕಿತ್ಸೆ** ಎಷ್ಟರ ಮಟ್ಟಿಗೆ ಸಮಸ್ಯೆಯಾಗಿತ್ತು, ಉದಾಹರಣೆಗೆ ಮನೆಯನ್ನು ಅವ್ಯವಸ್ಥೆ ಮಾಡುವುದು, ಅಥವಾ ಸಮಯ ತೆಗೆದುಕೊಳ್ಳುವುದು?	ಬಹಳ ಹೆಚ್ಚು ಬಹಳ ಸ್ವಲ್ಪ ಇಲ್ಲವೇ ಇಲ್ಲ	☐ ☐ ☐ ☐	ಪ್ರಕ್ನತವಲ್ಲ ☐

ದಯವಿಟ್ಟು ನೀವು ಪ್ರತಿಯೊಂದು ಪ್ರಶ್ನೆಗೂ ಉತ್ತರಿಸಿದ್ದೀರೆ ಎಂದು ಖಚಿತ ಪಡಿಸಿಕೊಳ್ಳಿ. ಧನ್ಯವಾದಗಳು.

(DLQI -Marathi)

त्वचाविज्ञान जीवन स्तर सूची

डीएलक्यूआय

रुग्णालय क्र:

तारीख:

गुण:

नाव:

रोग परिक्षण :

पत्ता:

ह्या प्रश्नावलीचा मुख्यउद्देश हा आपल्या त्वचेच्या त्रासामुळे आपल्या जीवनावर मागील आठवड्यात झालेला परिणाम मोजणे. कृपया प्रत्येक प्रश्नासाठी एका चौकोनात बरोबरची खूण करा.

1.	मागच्या आठवड्यात, तुमची त्वचा किती **खाज येणारी,सोअर(ल्सची), दुखणारी** किंवा **दंश करणारी** होती?	खूपच बराच थोडासा काहीही नाही	☐ ☐ ☐ ☐	
2.	मागच्या आठवड्यात, तुम्ही त्वचेमुळे किती **शरमिंदे** किंवा **स्वतःची जाणीव** ठेवून वागत होतात?	खूपच बराच थोडासा काहीही नाही	☐ ☐ ☐ ☐	
3.	मागच्या आठवड्यात, तुमच्या त्वचेने तुमच्या **खरेदीला जाण्यात** किंवा,आपल्या **घराची** किंवा **बागेची** देखभाल करण्यात किती अडचण आणली?	खूपच बराच थोडासा काहीही नाही	☐ ☐ ☐ ☐	संबंध नाही ☐
4.	मागच्या आठवड्यात, तुमच्या त्वचेने तुमच्या कोणते **कपडे** घालावेत ह्या निर्णयावर प्रभाव टाकला?	खूपच बराच थोडासा काहीही नाही	☐ ☐ ☐ ☐	संबंध नाही ☐
5.	मागच्या आठवड्यात, तुमच्या त्वचेमुळे तुमच्या **सामाजीक** किंवा फुरसतीच्या कामांवरती किती परीणाम झाला?	खूपच बराच थोडासा काहीही नाही	☐ ☐ ☐ ☐	संबंध नाही ☐
6.	मागच्या आठवड्यात, तुमच्या त्वचेने तुम्हाला कोणताही **खेळ खेळायला** किती कठीण केले?	खूपच बराच थोडासा काहीही नाही	☐ ☐ ☐ ☐	संबंध नाही ☐
7.	मागच्या आठवड्यात, तुमच्या त्वचेने तुम्हाला **काम करण्यात** किंवा **अभ्यासात** अडथळा आणला का?	होय नाही	☐ ☐	संबंध नाही ☐
	जर "नाही" तर मागच्या आठवड्यात तुमच्या त्वचेने **कामात** अथवा **अभ्यासात** किती त्रास दिला?	बराच थोडासा काहीही नाही	☐ ☐ ☐	
8.	मागच्या आठवड्यात, तुमच्या त्वचेमुळे तुमच्या **साथीदारसोबत** किंवा **जवळच्या मित्रांसोबत** किंवा **नातेवाईकांसोबत** काही त्रास निर्माण झाला का?	खूपच बराच थोडासा काहीही नाही	☐ ☐ ☐ ☐	संबंध नाही ☐
9.	मागच्या आठवड्यात, तुमच्या त्वचेमुळे तुम्हाला काही **लैंगिक अडचणी** आल्या का?	खूपच बराच थोडासा काहीही नाही	☐ ☐ ☐ ☐	संबंध नाही ☐
10.	मागच्या आठवड्यात, तुमच्या त्वचेच्या **उपचारांमुळे** तुम्हाला काय त्रास झाला, उदाहरणार्थ आपले घर अस्ताव्यस्त झाले किंवा आपला वेळ घेतला?	खूपच बराच थोडासा काहीही नाही	☐ ☐ ☐ ☐	संबंध नाही ☐

कृपया आपण प्रत्येक प्रश्नाचे उत्तर दिले आहे ह्याची खात्री करा, धन्यवाद

PSORIASIS AREA AND SEVERITY INDEX (PASI) WORKSHEET

HOSPITAL NO.:

PATIENT NAME:

DATE OF VISIT:

The Psoriasis Area and Severity Index (PASI) is a quantitative rating score for measuring the severity of psoriatic lesions based on area coverage and plaque appearance.

Plaque characteristic	Lesion score	Head	Upper Limbs	Trunk	Lower Limbs
Erythema	0 = None 1 = Slight 2 = Moderate 3 = Severe 4 = Very severe				
Induration/Thickness					
Scaling					
Add together each of the 3 scores for each body region to give 4 separate sums (A).					
Lesion Score Sum (A)					

Percentage area affected	Area score	Head	Upper Limbs	Trunk	Lower Limbs
Area Score (B) *Degree of involvement as a percentage for each body region affected (score each region with score between 0-6)*	0 = 0% 1 = 1% - 9% 2 = 10% - 29% 3 = 30% - 49% 4 = 50% - 69% 5 = 70% - 89% 6 = 90% - 100%				
Multiply Lesion Score Sum (A) by Area Score (B), for each body region, to give 4 individual subtotals (C).					
Subtotals (C)					
Multiply each of the Subtotals (C) by amount of body surface area represented by that region, i.e. x 0.1 for head, x 0.2 for upper body, x 0.3 for trunk, and x 0.4 for lower limbs.					
Body Surface Area		**x 0.1**	**x 0.2**	**x 0.3**	**x 0.4**
Totals (D)					
Add together each of the scores for each body region to give the final PASI Score.					

PASI Score = ☐

Hamilton Anxiety Rating Scale (HAM-A)

Below is a list of phrases that describe certain feeling that people have. Rate the patients by finding the answer which best describes the extent to which he/she has these conditions. Select one of the five responses for each of the fourteen questions.

0 = Not present, 1 = Mild, 2 = Moderate, 3 = Severe, 4 = Very severe

1 Anxious mood [0] [1] [2] [3] [4]

Worries, anticipation of the worst, fearful anticipation, irritability.

2 Tension [0] [1] [2] [3] [4]

Feelings of tension, fatigability, startle response, moved to tears easily, trembling, feelings of restlessness, inability to relax.

3 Fears [0] [1] [2] [3] [4]

Of dark, of strangers, of being left alone, of animals, of traffic, of crowds.

4 Insomnia [0] [1] [2] [3] [4]

Difficulty in falling asleep, broken sleep, unsatisfying sleep and fatigue on waking, dreams, nightmares, night terrors.

5 Intellectual [0] [1] [2] [3] [4]

Difficulty in concentration, poor memory.

6 Depressed mood [0] [1] [2] [3] [4]

Loss of interest, lack of pleasure in hobbies, depression, early waking, diurnal swing.

7 Somatic (muscular) [0] [1] [2] [3] [4]

Pains and aches, twitching, stiffness, myoclonic jerks, grinding of teeth, unsteady voice, increased muscular tone.

8 Somatic (sensory) [0] [1] [2] [3] [4]

Tinnitus, blurring of vision, hot and cold flushes, feelings of weakness, pricking sensation.

9 Cardiovascular symptoms [0] [1] [2] [3] [4]

Tachycardia, palpitations, pain in chest, throbbing of vessels, fainting feelings, missing beat.

10 Respiratory symptoms [0] [1] [2] [3] [4]

Pressure or constriction in chest, choking feelings, sighing, dyspnea.

11 Gastrointestinal symptoms [0] [1] [2] [3] [4]

Difficulty in swallowing, wind abdominal pain, burning sensations, abdominal fullness, nausea, vomiting, borborygmi, looseness of bowels, loss of weight, constipation.

12 Genitourinary symptoms [0] [1] [2] [3] [4]

Frequency of micturition, urgency of micturition, amenorrhea, menorrhagia, development of frigidity, premature ejaculation, loss of libido, impotence.

13 Autonomic symptoms [0] [1] [2] [3] [4]

Dry mouth, flushing, pallor, tendency to sweat, giddiness, tension headache, raising of hair.

14 Behavior at interview [0] [1] [2] [3] [4]

Fidgeting, restlessness or pacing, tremor of hands, furrowed brow, strained face, sighing or rapid respiration, facial pallor, swallowing, etc.

Patient Name:_____ Date:_____

Hamilton Rating Scale for Depression (17-items)

Instructions: For each item select the "cue" which best characterizes the patient during the past week.

1. **Depressed Mood**
(sadness, hopeless, helpless, worthless)
0 Absent
1 These feeling states indicated only on questioning
2 These feeling states spontaneously reported verbally
3 Communicates feeling states nonverbally, i.e., through facial expression, posture, voice and tendency to weep
4 Patient reports VIRTUALLY ONLY these feeling states in his spontaneous verbal and nonverbal communication

2. **Feelings of Guilt**
0 Absent
1 Self-reproach, feels he has let people down
2 Ideas of guilt or rumination over past errors or sinful deeds
3 Present illness is a punishment. Delusions of guilt
4 Hears accusatory or denunciatory voices and/or experiences threatening visual hallucinations

3. **Suicide**
0 Absent
1 Feels life is not worth living
2 Wishes he were dead or any thoughts of possible death to self
3 Suicide ideas or gesture
4 Attempts at suicide (any serious attempt rates 4)

4. **Insomnia - Early**
0 No difficulty falling asleep
1 Complains of occasional difficulty falling asleep i.e., more than ½ hour
2 Complains of nightly difficulty falling asleep

5. **Insomnia - Middle**
0 No difficulty
1 Patient complains of being restless and disturbed during the night
2 Waking during the night – any getting out of bed rates 2 (except for purposes of voiding)

6. **Insomnia - Late**
0 No difficulty
1 Waking in early hours of the morning but goes back to sleep
2 Unable to fall asleep again if gets out of bed

7. **Work and Activities**
0 No difficulty
1 Thoughts and feelings of incapacity, fatigue or weakness related to activities, work or hobbies
2 Loss of interest in activity, hobbies or work – either directly reported by patient, or indirect in listlessness, indecision and vacillation (feels he has to push self to work or activities)
3 Decrease in actual time spent in activities or decrease in productivity. In hospital, rate 3 if patient does not spend at least three hours a day in activities (hospital job or hobbies) exclusive of ward chores.
4 Stopped working because of present illness. In hospital, rate 4 if patient engages in no activities except ward chores, or if patient fails to perform ward chores unassisted.

8. **Retardation**
(slowness of thought and speech; impaired ability to concentrate; decreased motor activity)
0 Normal speech and thought
1 Slight retardation at interview
2 Obvious retardation at interview
3 Interview difficult
4 Complete stupor

9. **Agitation**
0 None
1 "Playing with" hand, hair, etc.
2 Hand-wringing, nail-biting, biting of lips

10. **Anxiety - Psychic**
0 No difficulty
1 Subjective tension and irritability
2 Worrying about minor matters
3 Apprehensive attitude apparent in face or speech
4 Fears expressed without questioning

11. **Anxiety - Somatic**
0 Absent
1 Mild
2 Moderate
3 Severe
4 Incapacitating
Physiological concomitants of anxiety such as:
Gastrointestinal - dry mouth, wind, indigestion, diarrhea, cramps, belching
Cardiovascular – palpitations, headaches
Respiratory - hyperventilation, sighing
Urinary frequency
Sweating

12. **Somatic Symptoms - Gastrointestinal**
0 None
1 Loss of appetite but eating without staff encouragement. Heavy feelings in abdomen.
2 Difficulty eating without staff urging. Requests or requires laxatives or medications for bowels or medication for G.I. symptoms.

13. **Somatic Symptoms - General**
0 None
1 Heaviness in limbs, back or head, backaches, headache, muscle aches, loss of energy and fatigability
2 Any clear-cut symptom rates 2

14. **Genital Symptoms**
0 Absent 0 Not ascertained
1 Mild Symptoms such as: loss of libido, menstrual disturbances
2 Severe

15. **Hypochondriasis**
0 Not present
1 Self-absorption (bodily)
2 Preoccupation with health
3 Frequent complaints, requests for help, etc.
4 Hypochondriacal delusions

16. **Loss of Weight**
A. When Rating by History:
0 No weight loss
1 Probable weight loss associated with present illness
2 Definite (according to patient) weight loss

B. On Weekly Ratings by Ward Psychiatrist, When Actual Changes are Measured:
0 Less than 1 lb. weight loss in week
1 Greater than 1 lb. weight loss in week
2 Greater than 2 lb. weight loss in week

17. **Insight**
0 Acknowledges being depressed and ill
1 Acknowledges illness but attributes cause to bad food, climate, overwork, virus, need for rest, etc.
2 Denies being ill at all

Total Score_____

KEY TO MASTER CHART

Religion :H=Hindu, M=Muslim C=Christians

Mode of onset :G=Gradual S=Sudden

Marital Status : M=Married S=Single

Stress :P=Present, A=Absent

Types of psoriasis : P= Plaque type psoriasis

PP= Palmoplantar psoriasis

A= Arthritis

Habits : S=Smoker, A=Alcoholic N=None

DLQI scores: 0-1 = no effect at all on patients life

2-5 = small effect on patients life

6-10 = moderate effect on patients life

11-20 = Very large effect on patients life

21-30 = Extremely large effect on patients life

HAM-A scores: <17 indicates mild severity

18–24 mild to moderate severity

25–30 moderate to severe.

>30 indicates very severe.

HAM-D scores: 0-7 = Normal

8-13 = Mild Depression

14-18 = Moderate Depression

19-22 = Severe Depression

≥ 23 = Very Severe Depression