"PREVALENCE OF METABOLIC SYNDROME AMONG PSORIATIC PATIENTS ATTENDING KLE'S DR. PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTER,BELAGAVI"

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Endorsement

This is to certify that the dissertation entitled **"PREVALENCE OF METABOLIC SYNDROME AMONG PSORIATIC PATIENTS ATTENDING KLE'S DR. PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTER, BELAGAVI"** is a bonafide research work done by **(REG NO. BT0116002)** .

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LIST OF ABBREVIATIONS USED

HLA	-	Human leucocyte antigen
IL	-	Interleukin
MHC	-	Major histocompatibility complex
GM-CSF	-	Granulocyte- monocyte- colony stimulating factor
IFN-γ	-	Interferon gamma
CD	-	Cluster differentiation
TCR	-	T cell receptor
APC	-	Antigen presenting c
ICAM	-	Intercellular adhesion molecule
AMP	-	Anti-microbial peptide
CCL	-	Chemokine ligand
TGF-α	-	Transforming growth factor
ACE	-	Angiotensin converting enzyme
UV	-	Ultraviolet
FDA	-	Food and drug administration
CVD	-	Cardiovascular disease
T2DM	-	Type 2 Diabetes mellitus
ROS	-	Reactive oxygen species
RAAS	-	Renin-angiotensin system
CRP	-	C-reactive protein
VLDL	-	Very low density lipoprotein
LDL	-	Low density lipoprotein
HDL	-	High density lipoprotein
Lp (a)	-	Lipoprotein (a)

NAFLD	-	Non-alcoholic fatty liver disease
CPP	-	Chronic plaque psoriasis
PA	-	Psoriatic arthritis
PPK	-	Palmoplantar keratoderma
WC	-	Waist circumference
TG	-	Triglyceride
SBP	-	Systolic blood pressure
DBP	-	Diastolic blood pressure
FBS	-	Fasting blood sugar
mg/dl	-	Milligram per decilitre
CI	-	Class interval
OR	-	Odd’s ratio

ABSTRACT

Background

Psoriasis is a multi-system inflammatory disease where the skin, nails and joints are commonly affected. Psoriasis is associated with an increased risk of cardiovascular atherosclerosis. Metabolic syndrome, a conglomerate of various clinical and biochemical parameters is a significant predictor of atherosclerotic disease and the associated risk for cardiovascular events in such patients. Dermatologists should be aware of these associations as they may be in a position to detect them early, thus, allowing early intervention that may improve the overall quality of life of the patient. There are many reports that psoriatic patients tend to have concurrent illnesses that are termed as comorbidities, though there are remarkably few studies from India. Hence, this study has been taken up to know the prevalence of metabolic syndrome in psoriasis.

Methodology

The present one year hospital based cross sectional study was done on a total of 100 psoriatic patients who attended KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi from January 2017 to December 2017. A written consent form was obtained. All the patients were screened for metabolic syndrome according to NCEP-ATP III criteria. Venous samples were taken at the enrolment visit after the subjects had fasted overnight (at least 8 h). Serum cholesterol and triglycerides were measured with enzymatic procedures. Plasma glucose was measured using a glucose oxidase method. Sample size was calculated using the Chi square test. Analysis of data was performed by SPSS software. An ethical committee clearance was obtained prior to the start of the study.

Results

In our study, 32 out of 100 psoriatic patients(32%) where positive for metabolic syndrome. 69% of the study population were males and 31% were females. PASI <9 was seen in 64% of the population. Chronic plaque psoriasis was the commonest type of psoriasis, seen in 75% of the patients. 41% of the patients had psoriasis since 1-5 years. Hypertension, diabetes mellitus, duration of the disease, PASI were statistically significant in relation to occurrence of metabolic syndrome

Conclusion

Psoriasis is a systemic disease with significant morbidity and mortality. Patients with psoriasis should be routinely screened for metabolic syndrome and treated accordingly to manage cardio-metabolic risk, while clinicians should monitor potential effects on treatment efficacy and safety in patients with co-morbid psoriasis and metabolic syndrome. Further research will be necessary to establish the directionality of this association and to explore the impact of treatment on these co-morbid diseases.

Keywords- Psoriasis, PASI, metabolic syndrome, cardiovascular risk

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INTRODUCTION

Psoriasis is a common, chronic, inflammatory, papulosquamous, proliferative condition of the skin, in which both genetic and environmental influences have a critical role. The disease is variable in duration, periodicity of flares and extent.[1]

Psoriasis affects nearly 2-3% of the world's population and presents as erythematous, indurated and scaly plaques over the skin and sometimes with involvement of the nails and joints.[2] Several studies have recently concluded that psoriasis is associated with systemic disorders such as cardiovascular disease, the metabolic syndrome (MS) cancer, chronic obstructive pulmonary disease inflammatory bowel disease, depression and osteoporosis.[3,4]

Metabolic syndrome is defined as a cluster of risk factors including central obesity, atherogenic dyslipidemia, hypertension and diabetes mellitus. It is a strong predictor of cardiovascular disease, that confers a cardiovascular risk higher than the individual components[5,6] .The prevalence of metabolic syndrome has been estimated to be 15-24% in the general population and 30‑50% among psoriasis patients in the recent studies.[7]

The data suggest that the association of psoriasis with metabolic syndrome occurs early in the course of the disease as psoriasis is associated with obesity and as well as elevated lipids even in childhood[8]. These prevalence studies clearly establish an association between psoriasis and metabolic syndrome but they cannot establish the directionality of the association.

Several studies suggest that obesity which is a primary component of metabolic syndrome, is a risk factor for future development of psoriasis with an estimated incidence 30% of new psoriasis, cases being attributable to obesity.[9,10] Alternatively, several studies have shown that psoriasis patients are prone to the future development of key components of metabolic syndrome such as diabetes, independent of traditional risk factors.[11,12]

OBJECTIVE

To study the prevalence of metabolic syndrome among psoriatic patients attending KLE'S Dr. Prabhakar Kore hospital & MRC, Belagavi.

REVIEW OF LITERATURE

HISTORY

The term 'lepra' was applied to various cutaneous disorders including psoriasis, vitiligo, eczema, boils and alopecia areata[13].The Roman sage Aurelius Cornelius Celsus was the first person who gave clinical description of psoriasis.[14] Galen was the first person to use the term psoriasis and Robert Willan (1808) specifically distinguished and described it as a recognizable entity[15].Willan described a term, Lepra vulgaris, which was a variety of psoriasis. In 1841, Hebra distinguished the clinical features of psoriasis from those of leprosy.

EPIDEMIOLOGY

Psoriasis is a genetically determined immune-mediated inflammatory disease that is mediated by T-helper 1 (Th1)/Th17 T cells. Prevalence being 0.44-2.8 per cent in India. It commonly affects individuals in third or fourth decade with males being affected two times more common than females[16].Two types of psoriasis have been distinguished considering the age of onset. Type I (onset 15– 40 years) accounts for the majority of cases (>75%) and shows a high degree of familial aggregation and has a strong association with HLA Cw6. Correspondingly, type II psoriasis begins after the age of 40 years.[17]A positive family history may be elicited in 9.8-28% of the population.

The age at onset of psoriatic arthritis varies from 35 to 50 years with no sex predilection. Nearly 70% of the patients develop psoriasis before arthritis; in another

15%,arthritis precedes the onset of psoriasis by more than 1 year, and in the remaining 15% of the cases, the two conditions occur within 12 months of each other.[18]

Psoriasis has a significant impact on the quality of life of patients and their families resulting in great physical, emotional and social burden.It is characterized by exaggerated and disordered epidermal cell proliferation and keratinization.

ETIOPATHOGENESIS

Psoriasis is a disease of multifactorial origin where certain environmental factors that act on individuals with specific genetic predisposition lead to an immune dysregulation and abnormal keratinization which results in the appearance of typical cutaneous lesions. Currently, the most accepted hypothesis is that psoriasis is an immune-mediated inflammatory skin disease that manifests in a genetically predisposed person exposed to certain environmentaltriggers.[19-21]

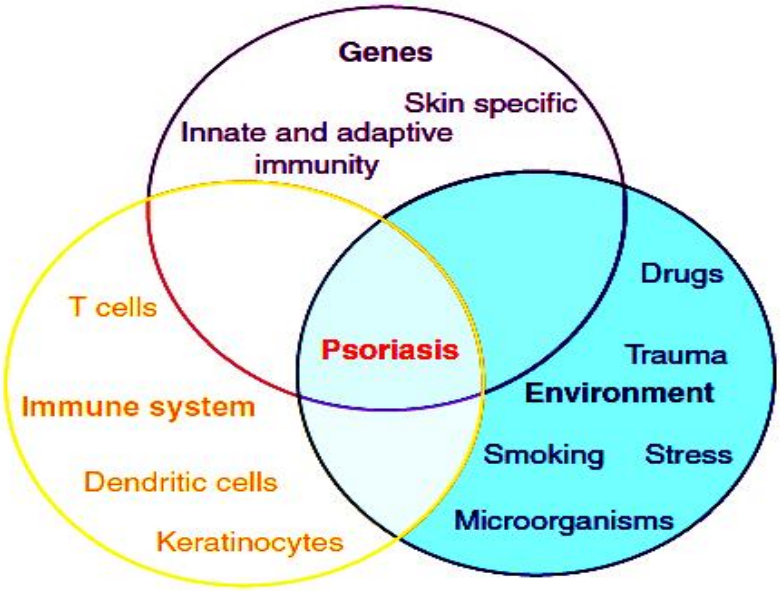


Figure 1- Etiopathogenesis of psoriasis

- ***ROLE OF GENETIC FACTORS***

There is a considerable evidence that genetic factors play an important role in the pathogenesis of psoriasis. In a study conducted in North India, 9.8% of children had a family history of psoriasis.[21] If only one parent has psoriasis, then 16% is the risk for the child developing psoriasis and it increases to a 50% chance if both parents have psoriasis.[23]Twin pair analysis has demonstrated 72% concordance among monozygotic twins compared to 22% concordance among dizygotic twins.[24]

Psoriasis is associated with many (human leukocyte antigen) HLA haplotypes. 35-50% of heritability of the disease is accounted by the presence PSORS1 which is seen in the HLA Class I region of chromosome 6p. The most likely susceptibility gene in the PSORS1 region is HLA-C-*06. Given the important role in antigenic presentation, its association reflects the role of the adaptive immune response in psoriasis.[25]This locus also harbours the corneodesmosin (CDSN) gene, which encodes a protein that is expressed in differentiated keratinocytes and is considered a genetic risk factor for psoriasis development. As PSORS1 harbours both the CDSN gene and HLA-C-*06, it is possible that both adaptive immunity and defective barrier function are involved in the pathogenesis of psoriasis.[26] Psoriatic patients have an increased frequency of HLA-B13, HLA-B17 and HLA-Bw16.[27-29]

Significant associations have been found in gene regions involving specific inflammatory pathways, such as, IL-23 signalling (IL-23A, IL-12B and IL-23R), modulation of Th2 immune responses (IL-4 and IL-13), and nuclear factor (NF) κB signaling.[30,31]

Certain other factors like, T cells, antigen-presenting cells, keratinocytes, Langerhans' cell, macrophages, natural killer cells, an array of Th1 type cytokines, certain growth factors like vascular endothelial growth factor (VEGF) keratinocyte growth factor (KGF) and others have also been suggested to play a key role in pathogenesis of psoriasis.

- ✓ ***ROLE OF IMMUNITY***

1. T cell Activation

It has been believed that abnormal regulation of T cells coupled with interaction between keratinocytes and complex cytokine network is involved in the pathogenesis of the disease.[32,33] If the primary defect resides in keratinocytes, any physical or chemical injury to the defective keratinocytes could activate synthesis and release of cytokines and hence resulting in antigen-independent activation of T lymphocytes. This further leads to release of additional cytokines followed by proliferation of keratinocytes, T lymphocytes and inflammation. Chang *et al*[34] have demonstrated that cytokines that are secreted by psoriatic epidermal cells potentiate T lymphocyte activation to a greater extent than the ones secreted from normal epidermal cells. It has also been postulated that only psoriatic keratinocytes respond to activated T cell messages with hyper-proliferation, because of their specific receptors or signal-transducing mechanisms. [33]

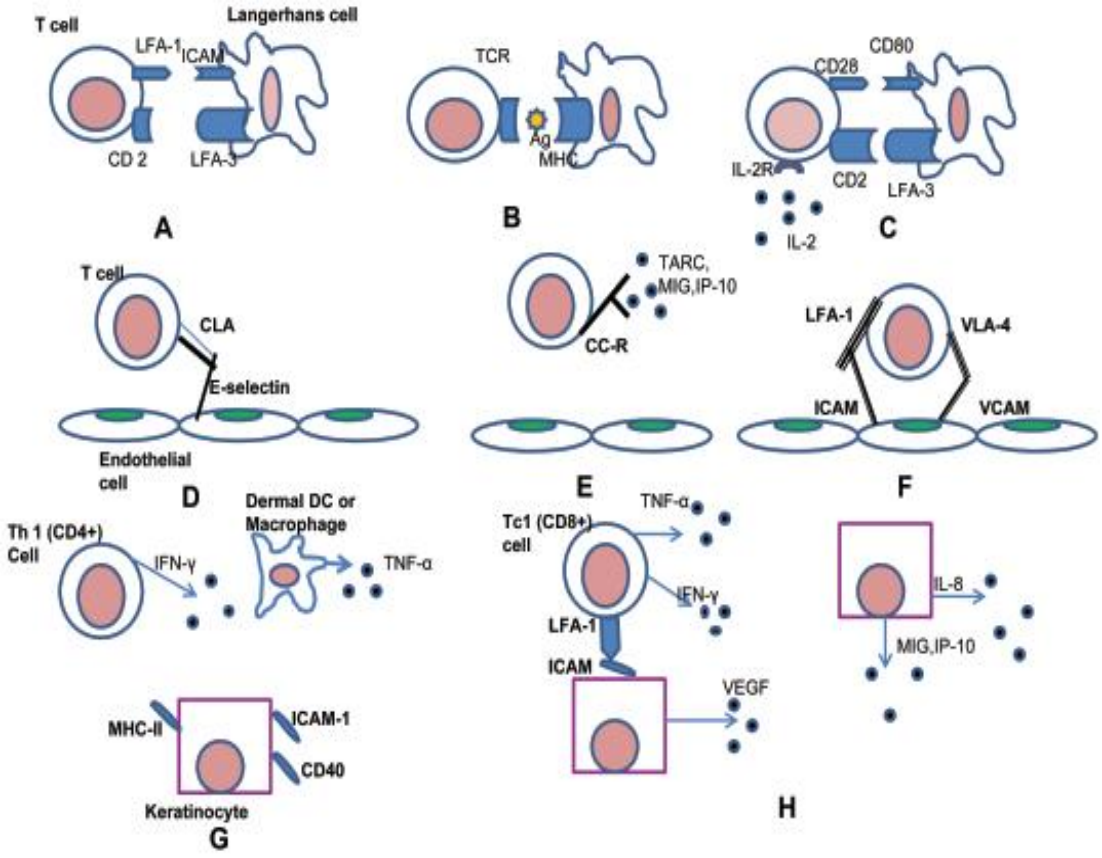


Figure 2- T cell activation

(A)T cell binds to an Antigen-Presenting Cell, (B) T cell receptor recognizes the antigen presented on MHC of the APC in an antigen specific interaction, (C) Non-antigen specific cell-interaction. The stimulation of both TCR and CD28 pathways lead to transcription of IL-2, TNF-α, GM-CSF and IFN-γ, (D) T cell is rolling on the endothelium, (E) T cell surface proteins are activated, (F) T cell binds to the endothelium and diapedesis occurs, (G) Dermal Th1 cells release IFN-γ and other cytokines, which lead to increases expression of inflammatory and adhesion proteins on keratinocytes, (H) Keratinocytes proliferate; synthesize angiogenic cytokines / chemokines that cause leukocyte trafficking and increase leukocyte adhesion to the endothelial cells.[33]

2. Hyperproliferation of Keratinocytes

A hyperproliferating psoriatic epidermis takes only 4 days to complete the epidermal cell cycle, while the maturation and shedding of a normal epidermal keratinocyte takes 26 days[35].

3. Angiogenesis

Although, the precise mechanism for angiogenesis in psoriasis is still not known, keratinocytes, however, are believed to be a major source of proangiogenic cytokines (VEGF, IL-8). In a developing psoriatic plaque, endothelial cells swell up and become activated showing prominent Golgi apparatus and Weibel-Palade bodies.[36]Activated endothelial cells migrate, sprout, and lay down a basement membrane with pericytes for structural support to form novel vessel networks.[37] Activation and swelling of these endothelial cells results in widening of the intercellular spaces, and dilation of dermal blood vessels. A venous phenotype is adopted by the lesional capillary loops, which includes bridged fenestrations and expression of E-selectin, thus making it easier for leukocytes to migrate into the skin.[38]

4. Cytokine Mediators

Pathogenesis of psoriasis involves a complex and multi-dimensional network of various cytokines[39] like TNF-α, IFN-γ, IL-1, IL-2, IL-8, IL-12, IL-17, IL-22, IL-23, GM-CSF, VEGF.

- TNF-α causes stimulation of keratinocytes which produce IL-8, ICAM-1, TGF-α, β-defensins, GM-CSF and PAI2; and stimulation of endothelial cell to

secrete VEGF; increases keratinocyte proliferation and enhances pro-inflammatory cytokine secreting capacity of macrophage[35,39,40,41]

- IFN-γ has anti-proliferative effect on normal keratinocyte *in-vitro*; causes induction of ICAM-1 expression on keratinocytes and endothelial cells, influencing the trafficking of T lymphocytes into lesional epidermis and stimulates APC activity and TNF-α release by phagocytes and up regulation of TNF-α receptors[39,42,43]
- IL-2 regulates the production of TNF-α and IFN-γ[44]
- IL-6 Produced by keratinocytes production increased under the influence of TNF-α Mediates T-cell activation, proliferation of keratinocyte, acute phase inflammation[44]
- IL-8 Increased expression under the influence of TNF-α[44]
- IL-17 Stimulates keratinocytes to produce β-defensins, AMPs, IL-8, CCL20, CCL2[44]
- VEGF- Produced by Keratinocytes, Macrophages, mast cells, promotes dermal angiogenesis[44]

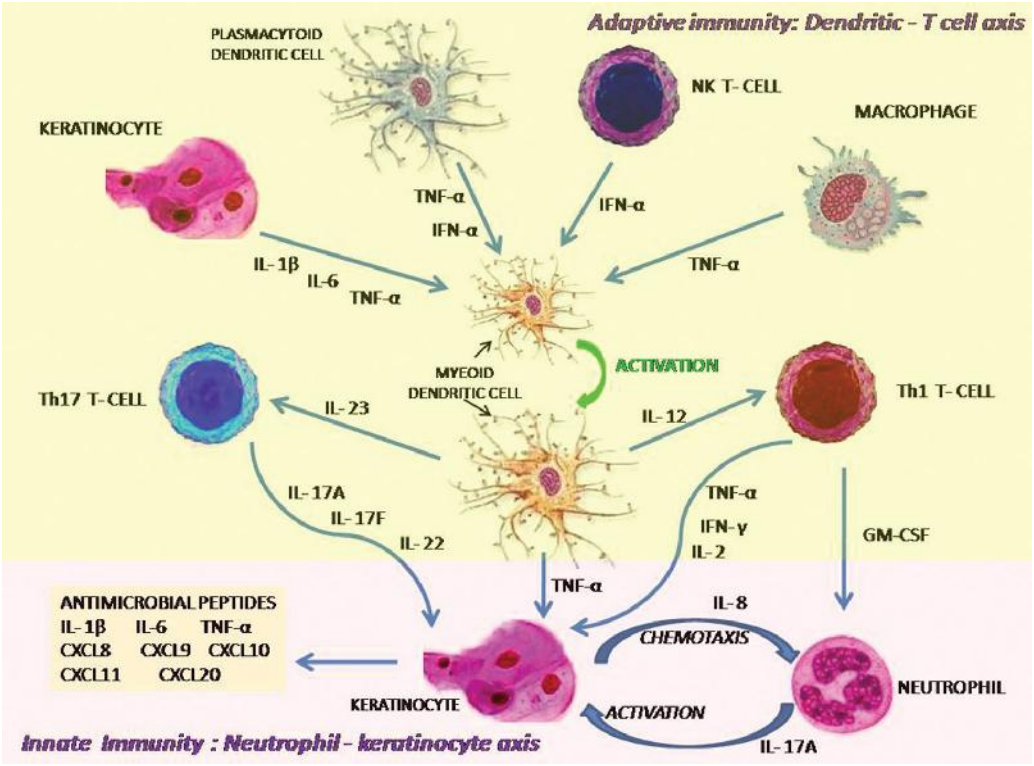


Figure 3- Cytokine mediators

TRIGERRING FACTORS

Environmental factors are involved in the expression of the disease. In a genetically predisposed individual, several factors, such as physical trauma, psychological stress, drugs[45] and infections, may trigger the disease.

1. Local Factors

Psoriatic lesions usually develop at sites of injury to the skin. The Koebner phenomenon, which is also known as the isomorphic response, refers to the induction of lesions by cutaneous trauma. The trauma may be of any kind- physical, chemical, mechanical, allergic or of any other nature. This phenomenon can be elicited at sites of sunburn, operation wounds, vaccination and other skin lesions[46]

2. Seasonal Variation

Most of the patients (89% in one study)[48] experience worsening of their skin lesions during winter[49,50]whereas, high humidity is usually beneficial. Sunlight may worsen psoriasis in some but improves it in many.[51]

3. Emotional Stress

Psoriasis is more 'stress sensitive' than other skin diseases. Some patients with psoriasis have an abnormal hypothalamic–adrenal axis response to acute stress. Increased beta-endorphin in psoriatic skin might affect both substance P mediated neurogenic inflammation and transmission of sensory stimuli by its local antinociceptive effects.[52]Stress may induce alterations in the lesion by increasing the neuropeptide content with a concomitant decrease in activity of neuropeptide degrading enzymes, especially mast cell chymase.[53]

4. Infections

Upper respiratory tract infections and tonsillitis, when they are especially caused by streptococci, may cause a flare-up of existing psoriasis or may precipitate an attack of acute guttate psoriasis.[54-56] This is common in children and is usually associated with elevated levels of antistreptolysin 'O' titer. Other bacterial and viral infections may also exacerbate psoriasis.

5. Drugs

Many drugs can precipitate or exacerbate psoriasis, particularly beta-blockers, lithium, antimalarials, imiquimod, interferons α and γ, and ACE inhibitors.[57-65]

6. Alcohol and Smoking

Psoriasis patients have high rates of excess intake of alcohol, alcoholism, and death due to alcohol related diseases. Heavy drinking exacerbates pre-existing psoriasis. Smoking (past or present) more than 20 cigarettes daily is associated with a two-fold increased risk of severe psoriasis and may play a role in its onset as well.

7. Obesity

The prevalence of obesity is twice that in the normal population or those with other types of skin disease. Obese patients are more likely to present with severe psoriasis.

CLINICAL FEATURES

Classically, psoriasis presents as a well-demarcated, raised, red plaque with a white scaly surface size varying from pinpoint papules to plaques which can cover large surface areas of the body. Under the scales, the skin appears glossy with homogeneous erythema, and bleeding points appear when the scale is removed due to traumatizing the dilated capillaries below (the Auspitz sign)[66.]

Psoriasis tends have a symmetric eruption, and symmetry is a helpful feature in establishing a diagnosis.

Koebner phenomenon (also known as the *isomorphic response*) is the traumatic induction of psoriasis on non-lesional skin which occurs more frequently during flares of disease and is an all-or-none phenomenon (i.e., if psoriasis occurs at one of the sites of injury, it will occur at all the sites of injury). The Koebner reaction usually occurs 7–14 days after injury[67]

CLASSIFICATION[68]

Psoriasis can be clinically classified as follows:

1. Guttate psoriasis.
2. Chronic plaque psoriasis.
3. Erythrodermic psoriasis.
4. Pustular psoriasis.
5. Psoriasis unguis.
6. Mucous membrane psoriasis.
7. Arthropathic psoriasis.
8. Regional variations in psoriasis: Scalp, face, eyes, body flexures, scrotum, napkin area, palms and soles.

1. GUTTATE (ERUPTIVE) PSORIASIS

Guttate psoriasis (from the Latin gutta, meaning "a drop") is characterized by eruption of small (0.5–1.5 cm in diameter) papules which is mainly over the upper trunk and proximal extremities. It typically manifests at an early age and is found frequently in young adults. This form of psoriasis has the strongest association to HLACw6,[69] and is associated with streptococcal throat infection preceding or is concomitant with the onset or flare of guttate psoriasis.[70]

2. CHRONIC PLAQUE PSORIASIS

Psoriasis vulgaris is the most common form of psoriasis, approximately seen in 90% of patients. Red, scaly, symmetrically distributed plaques are characteristically present over the extensor aspects of the extremities, particularly the elbows and knees, scalp, lower lumbosacral, buttocks, and genital involvement.

Single small lesions may confluent to form plaques in which the borders resemble a land map (psoriasis geographica). Because of the confluence of several plaques, lesions may extend laterally and become circinate (psoriasis gyrata). Occasionally, there is partial central clearing which results in ring-like lesions (annular psoriasis). *Rupioid psoriasis* refers to lesions in the shape of a cone or limpet. *Ostraceous psoriasis* refers to a ring-like, hyperkeratotic concave lesion, resembling an oyster shell. Finally, elephantine psoriasis is an uncommon form characterized by thickly scaling, large plaques usually over the lower extremities. A hypopigmented ring (Woronoff ring) surrounding individual psoriatic lesions may occasionally be seen and is usually associated with treatment, most commonly UV radiation or topical corticosteroids.[71]

3. ERYTHRODERMIC PSORIASIS

Erythrodermic psoriasis, is uncommon variant occurring in 1–2% of patients[72,73] involves most or all of the body surface. Psoriasis has been found to be the underlying cause in about 25% of cases of erythroderma.[74] Erythroderma in psoriasis may be chronic or can be due to the gradual extension of plaque psoriasis, or acute , a part of the spectrum of ‘ unstable’ psoriasis. The acute form is precipitated by environmental or therapeutic triggers including systemic illness, alcoholism,

antimalarials, irritating topical treatments, ultraviolet radiation or by withdrawal of systemic corticosteroids, cyclosporin or methotrexate.

The patient is febrile and systemically ill. Dependent oedema is usually present. Itching is severe. The entire skin may be affected and the clinical characteristics of psoriasis are often lost. Complications are those of skin failure, including sepsis[75], hypothermia or hyperthermia, hypoalbuminaemia, anaemia, dehydration and high output cardiac failure.

4. PUSTULAR PSORIASIS

When the surface of a plaque is studded with tiny, superficial, sterile pustules, it is called pustular psoriasis. Pustular psoriasis is precipitated by overtreatment with topical tar, anthralin or potent steroids or by systemic therapy with progesterone or corticosteroids.[76,77] Foci of infection, pregnancy and hypocalcemia[76] can also precipitate it. Pustular psoriasis is broadly classified into a localized form and a generalized form.

Generalized pustular psoriasis has been further classified into:[68]

i. Von Zumbusch type
ii. Annular pustular psoriasis
iii. Impetigo herpetiformis
iv. Exanthematic Type
v. Localised type

Localized pustular psoriasis has been further classified into:[68]

i. Pustulosis palmaris et plantaris
ii. Acrodermatitis continua of Hallopeau.

In children, pustular psoriasis is complicated by manifesting as sterile, lytic bone lesions [78,79] and can be a manifestation of the SAPHO syndrome (synovitis, acne, pustulosis, hyperostosis, osteitis).[80]

5. PSORIASIS UNGUIS

Nail changes are common in psoriasis patients , up to 40% of patients having them [81] and are rare in the absence of skin lesion . Nail involvement increases with age, with duration and extent of disease, and with the presence of psoriatic arthritis. The fingernails are more commonly involved than the toenails. The incidence of nail involvement is higher if there is associated arthropathy.[68] Several distinct changes have been described and can be grouped according to the portion of the nail that is affected.[82]

Nail Segment Involved	***Clinical Sign***
Proximal matrix	Pitting, onychorrhexis, Beau lines
Intermediate matrix	Leukonychia
Distal matrix	Focal onycholysis, thinned nail plate, erythema of the lunula
Nail bed	“Oil drop” sign or “salmon patch,”subungual hyperkeratosis, onycholysis, splinter hemorrhages
Hyponychium	Subungual hyperkeratosis, onycholysis
Nail plate	Crumbling and destruction plus other changes secondary to the specific site
Proximal and lateral nail folds	Cutaneous psoriasis

6. MUCOUS MEMBRANE PSORIASIS

It has been postulated that involvement of the mucosal surfaces(oral and vaginal) is uncommon occurrence in psoriasis because the epithelial surface is as rapidly proliferating as psoriatic skin.[83,84] Mucosal lesions are usually confined to the pustular and exfoliative forms of the disease and mostly occur on the buccal mucosa and dorsum of the tongue. They main layre discrete and confluent sharply circumscribed gyrated denuded areas with white elevated margins. Similar lesion on the tongue may evolve and spread, changing shape on a daily basis, and can assume distinct annular patterns and resemble a map('geographic tongue').[68] Geographic tongue which is also known as benign migratory glossitis or glossitis areata migrans, is an idiopathic inflammatory disorder resulting in the local loss of filiform papillae[71]

7. PSORIATIC ARTHRITIS

Psoriatic arthritis is an inflammatory arthritis and is associated with psoriasis with a negative test for rheumatoid factor.[85]Arthritis occurs in 5% to 10% of patients with psoriasis. Onset is concurrent with the skin disease in 10% of cases or may very rarely precede it. Similar to cutaneous psoriasis, psoriatic arthritis also is a genetically determined disorder. HLA studies show that the B27, DR3, A26 and B38 haplotypes are significantly associated with psoriatic arthritis. Arthritis may be precipitated by environmental factors such as trauma. It occurs commonly between the ages of 30 and 55. [68]

Five clinical patterns of arthritis occur:[86]

i. Asymmetrical distal interphalangeal joint involvement with nail damage (16%)
ii. Arthritis mutilans with osteolysis of phalanges and metacarpals (5%)
iii. Symmetrical polyarthritis-like rheumatoid arthritis, with claw hands (15%)
iv. Oligoarthritis along with swelling and tenosynovitis of one or a few hand joints (70%)
v. Ankylosing spondylitis alone or with peripheral arthritis (5%)

8. SCALP PSORIASIS

The well-defined nature of the plaques of psoriasis is seen on the scalp in most cases. However, if there is seborrheic dermatitis present , diffuse involvement may occur. Most often a band or corona of psoriasis, 2–5 cm wide, projects beyond the hairline on the forehead (‘corona psoriatica’)[68]

9. PALMOPLANTAR PSORIASIS

Palmoplantar lesions of psoriasis can occur alone or along with involvement of other body areas. In most cases, the lesions is well defined, less scaly and the surface usually shows fissures. Three forms of lesions can appear at these sites: diffuse hyperkeratotic plaques, erythematous patches or plaques studded with minute superficial pustules, and discrete scaly plaques or patches.[68]

HISTOPATHOLOGY OF PSORIASIS

There is parakeratosis associated with focal orthokeratosis and the accumulation of neutrophils in the stratum corneum (Munro microabscesses), near absence of the granular layer, spongiform pustules in the Malpighian layer, hyperplasia with elongation of rete ridges and suprapapillary epidermal thinning. The rete ridges are often clubbed, branched or fused at their bases, with mononuclear leukocyte infiltrates in the lower half of the epidermis. Dilated and tortuous papillary blood vessels almost at the undersurface of the thinning suprapapillary epidermis and are surrounded by a mixed mononuclear and neutrophil infiltrate, as well as extravasated erythrocytes. Invasion of the epidermis with leukocytes takes place particularly in the suprapapillary region.[87]

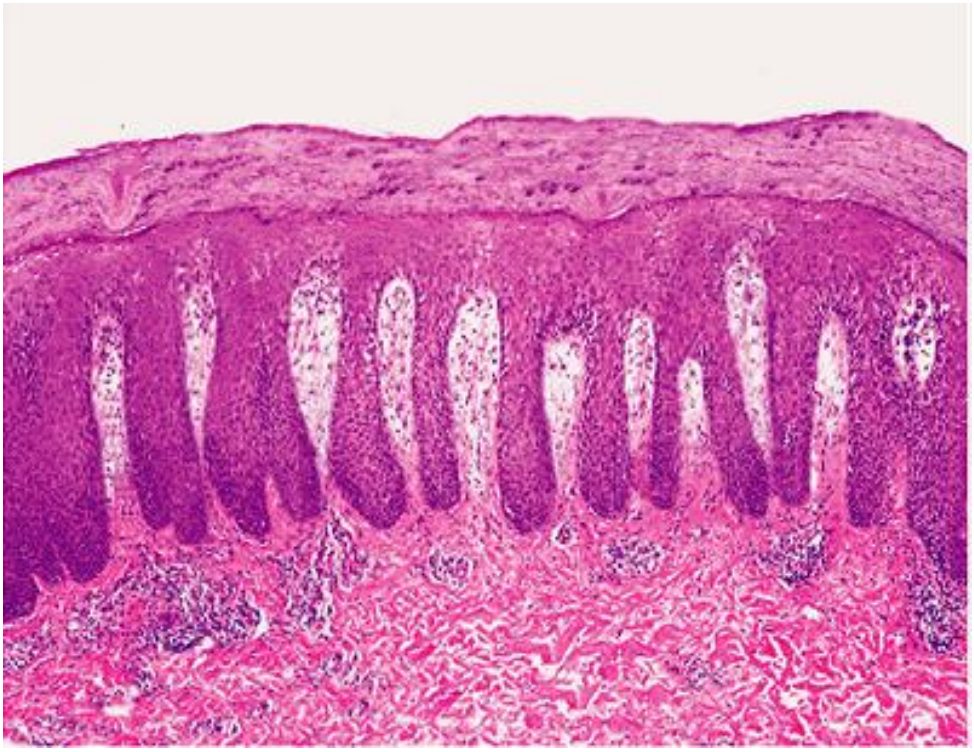


Figure 4- Histopathology of psoriasis

CLINICAL SCORES IN PSORIASIS[88]

A variety of scoring systems have been devised to assess the severity of plaque type psoriasis, which includes the clinical severity scores and quality of life scores. The clinical scores incorporate and grade the typical clinical features of psoriasis, such as, erythema, infiltration, scaling and extent of body surface area involved. Although PASI, BSA, and PGA have been approved by FDA for use in clinical trials, all have certain pitfalls. The most widely used scale is PASI as it is the most extensively validated, however, it lacks sensitivity for mild disease and does not evaluate quality of life impairment and comorbidities.

- ✓ **Assessing clinical severity**

Psoriasis area severity index (PASI)

Psoriasis log based area and severity index (PLASI)

Body surface area (BSA)

Physician global assessment (PGA)

Lattice system Physician global assessment (LSPGA)

Self assessment Psoriasis area severity index (SAPASI)

Salford psoriasis index (SPI)

- ✓ **Assessing quality of life**

Dermatology life quality index (DLQI)

Short form 36 (SF36)

World organization quality of life (WHOQOL)

Psoriasis disability index (PDI)

Salford psoriasis index (SPI)

Patients global psoriasis assessment (PGA)

PASI Score for the Evaluation of Psoriasis[68]

Four sites of affection, i.e. head (h), upper limbs (u), trunk (t), and lower limbs (l) are scored separately. Morphological scoring of psoriatic plaques is done by evaluating three parameters, viz. erythema, induration and desquamation. Each one of them is graded on a severity scale of 0 to 4 where, 0 = nil, 1 = mild, 2 = moderate, 3 = severe and 4 = very severe.

The addition of all these scores for each site is multiplied by the grading for area-wise percentage involvement of that particular site in the following manner: 1 = less than 10% area, 2 = 10%–29%, 3 = 30%–49%, 4 = 50%–69%, 5 = 70%–89% and 6 = 90% or more are involved by psoriasis. Hence if 10% of the trunk is affected, the morphologic score (E1+ l1+ D1) would be multiplied by a factor of 2. Since the four body regions (head, upper limbs, trunk, and lower limbs) represent about 10%, 20%, 30% and 40% of the body surface area respectively, they are given corresponding weightage in scoring by multiplying their scores by 0.1, 0.2, 0.3 and 0.4 respectively.

The final formula for calculating PASI score is as follows:

PASI = 0.1 (Eh + Lh+Dh) Ah + 0.2 (Eu + Lu+ Du) Au + 0.3 (El+ Ll+Dl) AL + 0.4 (El+ Ll+ Dl) Al

The score varies between 0 and 72.

COMORBIDITIES IN PSORIASIS

Comorbidities can be physical and psychosocial. Onumah et al. observed that the a serious risk for the development of these comorbidities is attributable to the severity of psoriatic skin disease. Patients with moderate to severe psoriatic skin disease may have a higher association with these comorbidities,[89]which may be related through common pathogenic mechanisms.

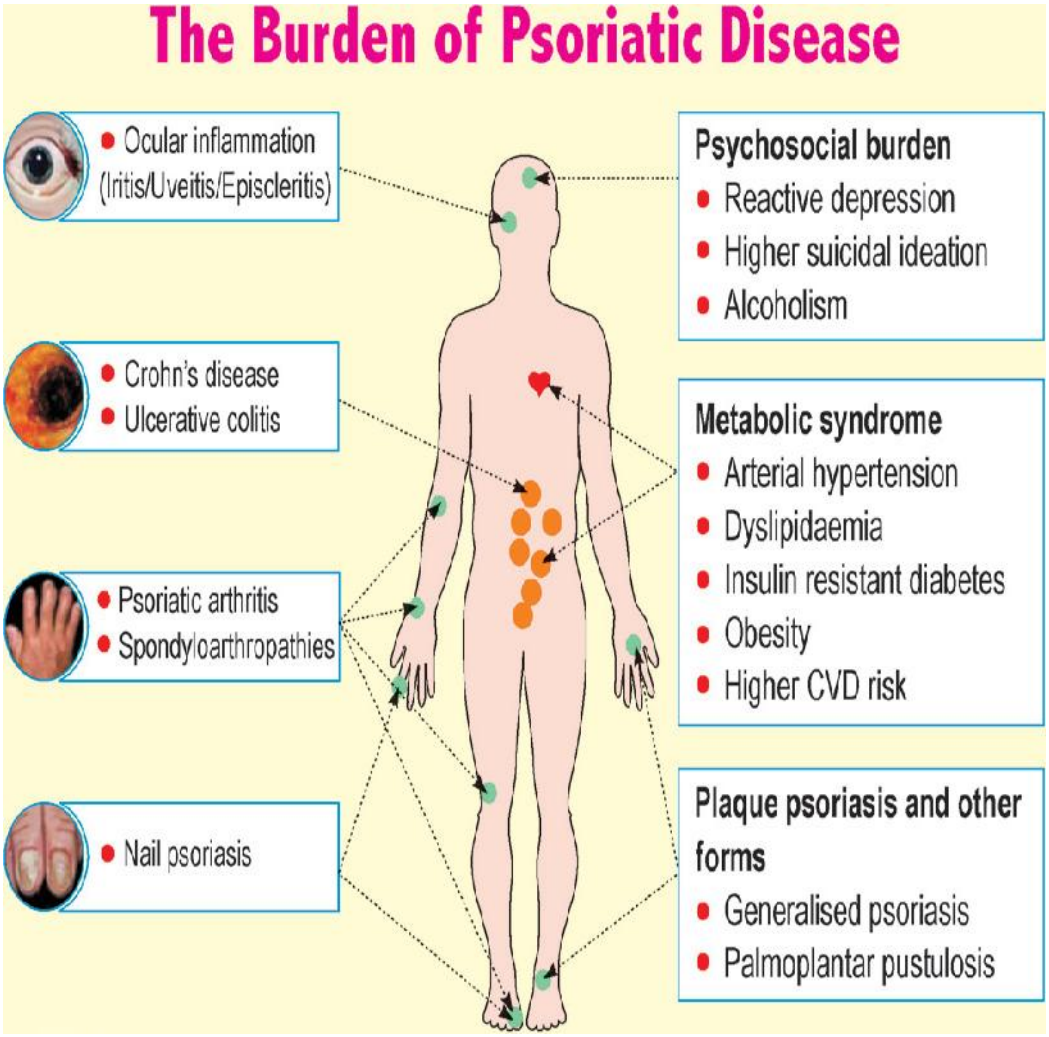


Figure 5- Comorbidities in psoriasis

METABOLIC SYNDROME

Metabolic syndrome is defined as a constellation of an interconnected physiological, biochemical, clinical, and metabolic factors which directly increase the risk of atherosclerotic cardiovascular disease (ASCVD), T2DM, and all of them cause mortality[90,91].

There have been several definitions of metabolic syndrome, but the most commonly used criteria for definition at present are from the World Health Organization (WHO)[92], the European Group for the study of Insulin Resistance (EGIR)[93], the National Cholesterol Education Programme Adult Treatment Panel III (NCEP ATP III)[94], American Association of Clinical Endocrinologists (AACE)[95], and the International Diabetes Federation (IDF).

Metabolic syndrome is diagnosed by the presence of three or more of the following five criteria of the National Cholesterol Education Programme's Adult Treatment Panel III (ATP III)[96]:

1. waist circumference > 102 cm(40 inches)in males or > 88 cm(35 inches) in females;
2. hypertriglyceridemia> 1.7 mmol/l (150mg/dl);
3. high density lipoprotein (HDL) cholesterol <1.0mmol/l (40mg/dl) in men or < 1.3mmol/dl (50mg/dl) in women;
4. blood pressure > 130/85 mmHg;
5. fasting plasma glucose of > 6.1 mmol/l (100mg/dl)

ATP III[97]identified 6 components of the metabolic syndrome that relate to CVD:

- Abdominal obesity
- Atherogenic dyslipidemia
- Raised blood pressure
- Insulin resistance _ glucose intolerance
- Proinflammatory state
- Prothrombotic state

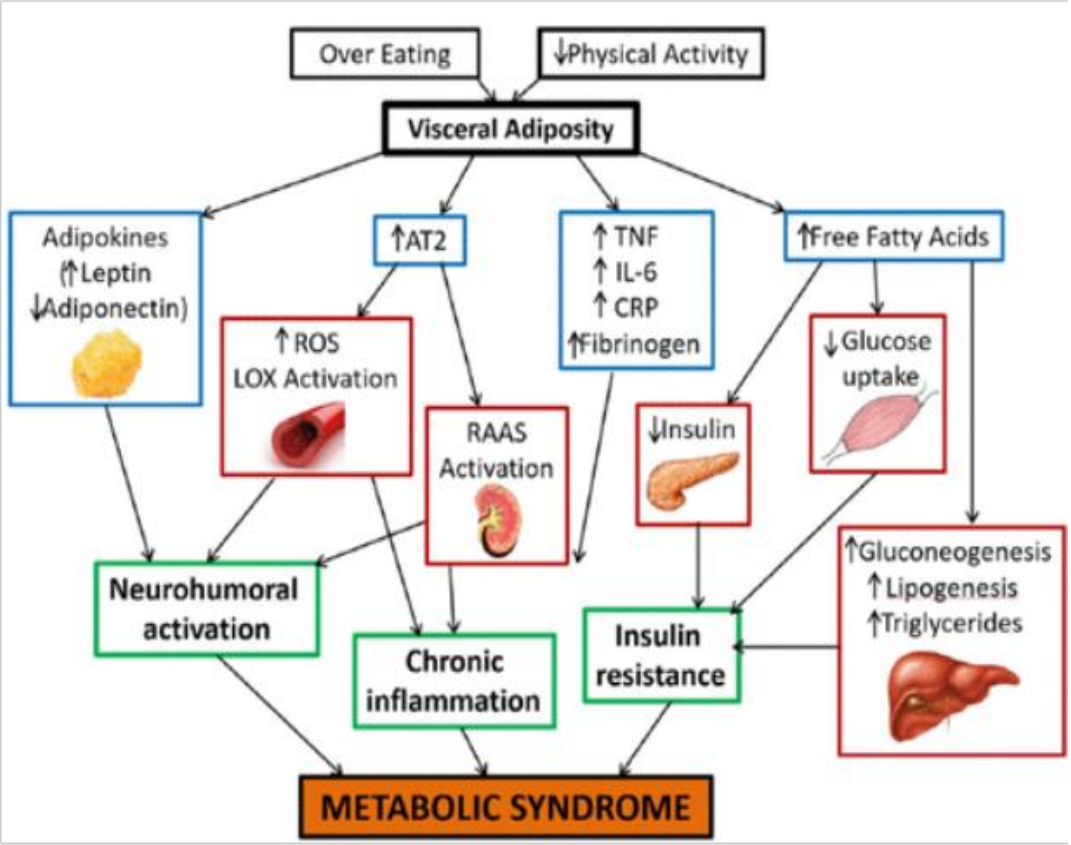


Figure 6 -Pathogenesis of metabolic syndrome

OBESITY

Obesity is a common and a preventable disease of clinical and public health importance. There is at present a global epidemic of obesity in all age groups and in both developed and developing countries. It is often a major risk factor of several non-communicable diseases, significant disability and premature death.

Obesity is defined as abnormal or excessive fat accumulation in adipose tissue, to the extent that health is impaired. [98]The amount of fat in absolute terms, and it's distribution in the body- either around the waist and trunk (abdominal, central obesity) or peripherally around the body(gyoid obesity)- have important health implications. Obesity is associated with a higher risk of disability or premature death because of type 2 diabetes mellitus (T2DM) and cardiovascular diseases (CVD) such as hypertension, stroke and coronary heart disease as well as gall bladder disease, certain cancers(endometrial, breast, prostate, colon) and non-fatal conditions including gout, respiratory diseases, gastro oesophageal reflux disease, osteoarthritis and infertility. Individuals with CVD risk factors like T2DM, hypertension and smoking are exposed to significant health risks at a lower level of obesity.

Currently, the general and central obesity anthropometric measures used for assessing adiposity-related risk include: body mass index (BMI- weight in kilograms has to be divided by square of height in meters), waist circumference (WC), hip circumference (HC), waist-to-hip ratio (WHR; ratio of WC to HC), waist-to-stature ratio (WSR; ratio of WC to height) and body adiposity index[99] (BAI; HC divided by height$^{1.5}$, and subtracting 18 from the result). BMI or WC is most commonly used to measure body fatness.[100]

The currently accepted indicators of excessive abdominal fat accumulation is an abdominal girth in excess of 108 cm (40 inches) for men and 98 cm (35 inches) for women or a WHR > 1.0 and 0.85 in men and women, respectively, which correlate with a substantially increased risk of metabolic complications[101,102]

The regulation of obesity is related to the molecular regulation of appetite that affects energy homeostasis, particularly as positive energy balance upsets lipid and glucosemetabolism.[103,104] Furthermore, obesity appears to play a key role in the dysregulation of cellular metabolism that accounts for insulin resistance in diabetes mellitus type 2. Cytokines are secreted by excess adipocytes which contribute to vascular dysfunction in hypertension and dyslipidemia, as manifested by hypercholesterolemia and triglyceridemia. These conditions eventually contribute to significant atherosclerosis, and when associated with obesity and/or diabetes and insulin resistance, they constitute the metabolic syndrome.[105,106]

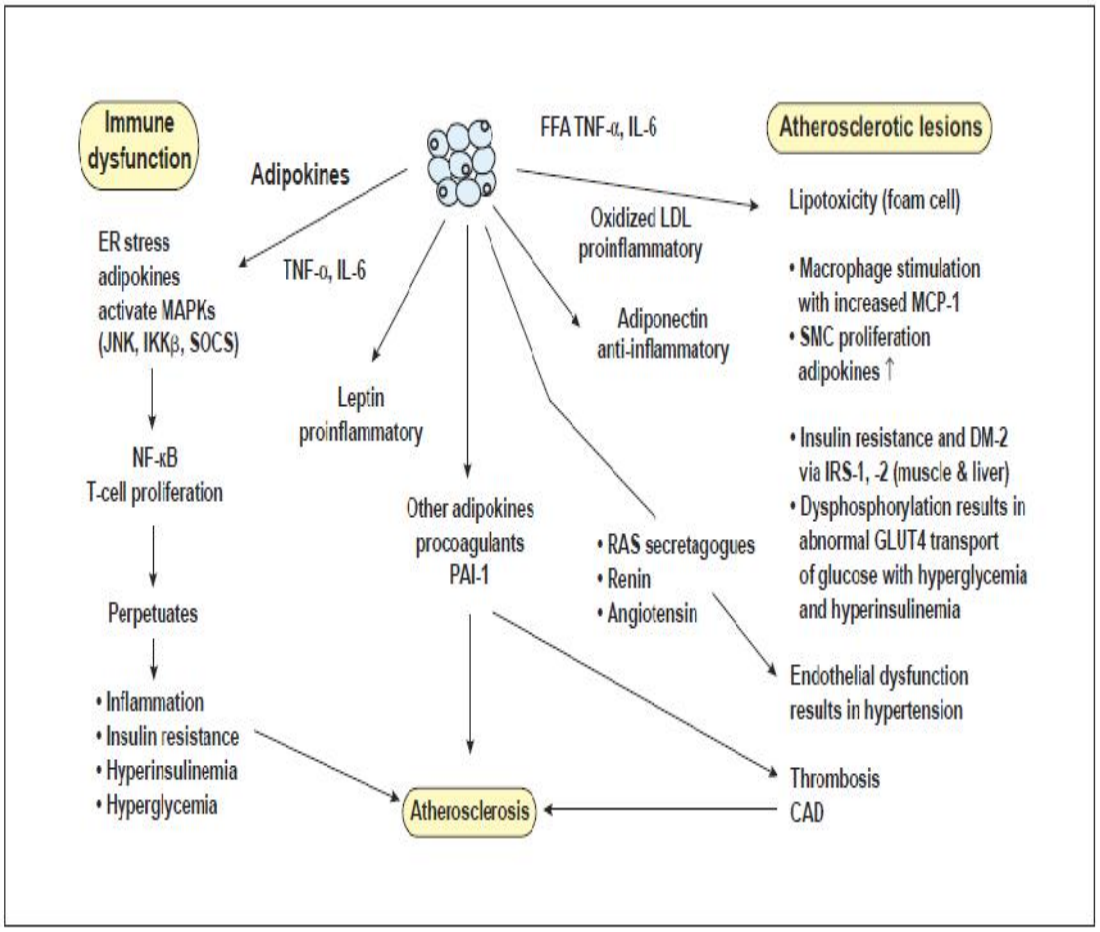


Figure 7- Role of inflammation and immune dysfunction in obesity

Role of inflammation and immune dysfunction in obesity. The immune dysfunction (left column) and inflammation (center column with arrows) are correlated with atherosclerotic lesions (right column).

CAD=coronary artery disease; DM=diabetes mellitus; ER=endoplasmic reticulum; FFA=free fatty acids; IKKβ=inhibitor of NF-KB kinase b; IL=interleukin; IRS=insulin receptor substrate; JNK=Jun N-terminal kinase; LDL=low-density lipoproteins; MAPK=mitogen-activated protein kinase; MCP-1=monocyte chemotactic protein; NF-KB=nuclear factor kappa beta; PAI-1=plasminogen activator inhibitor-1; RAS=renin angiotensin system; SMC=smooth muscle cell; SOCS=suppressor of cytokine signaling; TNF=tumor necrosis factor.

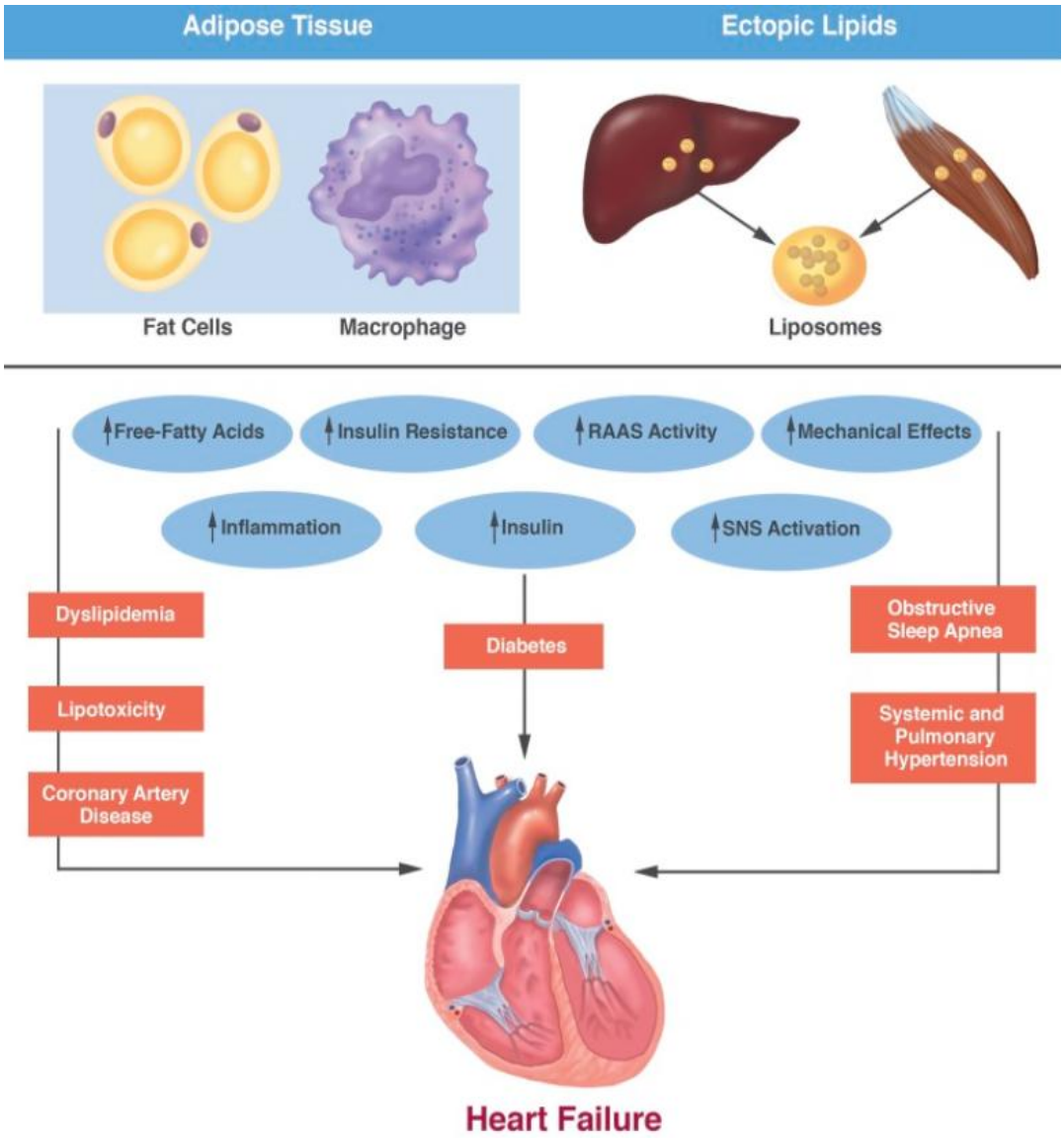


Figure 8- Role of obesity in heart failure

The Associated Inflammatory State in Obesity as a Major Contributor to the Metabolic Syndrome X

The understanding of the pathophysiology of obesity and its comorbidities reveals the central role that obesity plays as a result of the action of inflammatory adipokines in metabolic syndrome X. These comorbidities include diabetes mellitus type 2, whereby insulin resistance is worsened by TNF-α and other inflammatory adipocyte secretagogues[107]; endothelial dysfunction and hypertension, which results

from the activity of RAS-secreting adipokines[108,109]; and dyslipidemia, which is caused by hypercholesterolemia and hypertriglyceridemia. These comorbidities and the effects of fatty acid lipotoxicity[110] culminate to promote atherogenesis, including coronary artery disease. All these disorders are adversely affected by enhanced upregulation of NF-KB from visceral WAT inflammatory adipokines.[105,111,112]

HYPERTENSION

Hypertension is a major public health issue due to its high prevalence across the globe[113-116]. Around 7.5 million deaths or 12.8% of the total of all annual deaths worldwide occur due to high blood pressure[117]. It is predicted to be increased to 1.56 billion adults with hypertension in 2025[118].

High blood pressure is a major risk factor for chronic heart disease, stroke, and coronary heart disease. Apart from coronary heart disease and stroke, its complications include heart failure, peripheral vascular disease, renal impairment, retinal hemorrhage, and visual impairment[117].

Hypertension (or HTN) is defined as abnormally high arterial blood pressure. High blood pressure is a classical feature of the metabolic syndrome, and it has been reported that the metabolic syndrome is present in up to one third of hypertensive patients[119,120]. Thus, high blood pressure is included in the definition for the metabolic syndrome that presented by the World Health Organization, the National Cholesterol Education Program, the International Diabetes Federation, and the American Heart Association/National Heart, Lung, and Blood Institutes[121-124].

According to National Cholesterol Education Programme's Adult Panel III (NCEP ATP III), blood pressure > 130/85 mmHg, is included under metabolic syndrome[96]. According to the Joint National Committee 7 (JNC7), normal blood pressure is a systolic BP < 120 mmHg and diastolic BP < 80 mm Hg. Hypertension has been defined as systolic BP level of ≥140 mmHg and/or diastolic BP level ≥ 90 mmHg. The grey area falling between 120–139 mmHg systolic BP and 80–

89 mmHg diastolic BP is defined as "prehypertension"[125,126].Blood pressure levels are strongly associated with visceral obesity and insulin resistance[127]

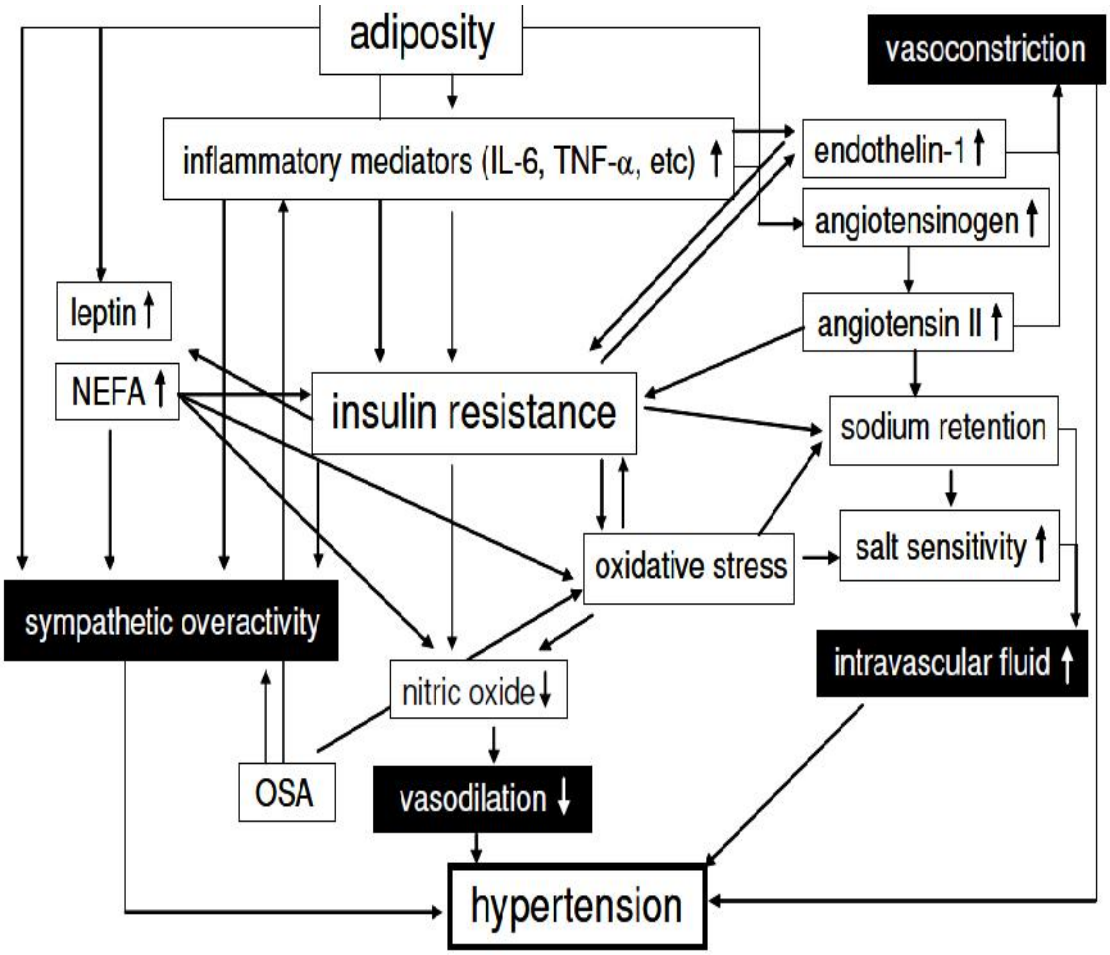


Figure 9- Pathogenesis of hypertension

DIABETES MELLITUS

Diabetes is a group of metabolic diseases which is characterized by hyperglycemia due to defects in insulin secretion, insulin action, or both. The chronic hyperglycemia of diabetes is associated with long-term damage such as dysfunction, and failure of different organs, especially the eyes, kidneys, nerves, heart, and blood vessels. Several pathogenic processes have been involved in the development of diabetes.

They range from autoimmune destruction of the β-cells of the pancreas with insulin deficiency to abnormalities which result in resistance to insulin action. The classification of diabetes as proposed by the American Diabetes Association (ADA) in 1997 as type 1, type 2, other types, and gestational diabetes mellitus (GDM) is still the most accepted classification and adopted by ADA. [128]

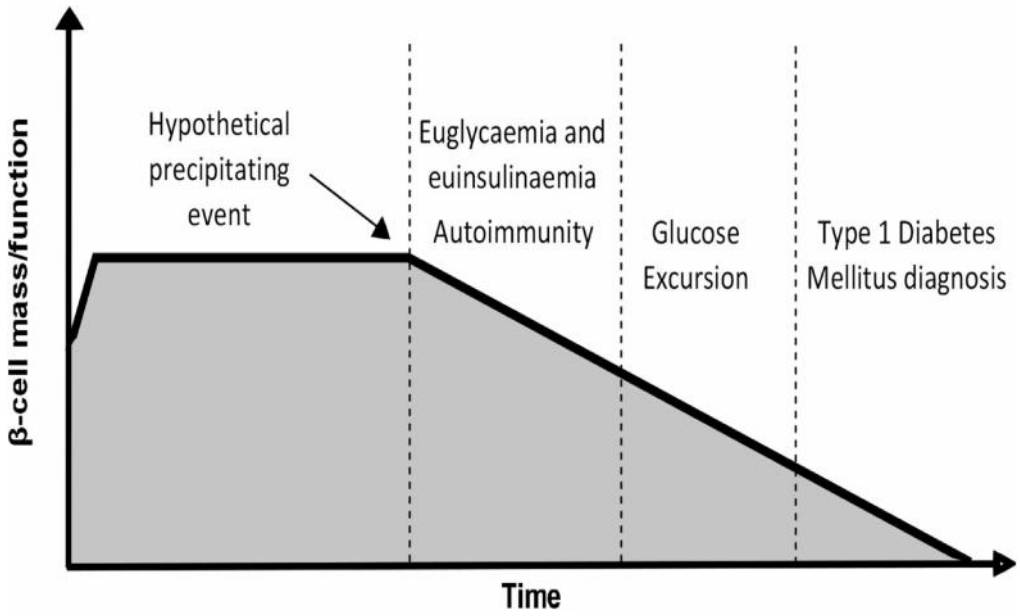


Figure 10 -Pathogenesis of type 1 Diabetes

Type 2 diabetes mellitus and obesity are major health problems worldwide, both of which are closely related[129-134]. In the majority of cases, type 2 diabetes is considered to be one component within a group of disorders called the metabolic syndrome. Certain factors that are characteristic of the metabolic syndrome, also known as dysmetabolic syndrome X are abdominal obesity, atherogenic dyslipidemia (elevated triglyceride [TG] levels, small low-density lipoprotein [LDL] particles, low high-density lipoprotein cholesterol [HDL-C] levels), elevated blood pressure, insulin resistance (with or without glucose intolerance), and prothrombotic and proinflammatory states[135-138].

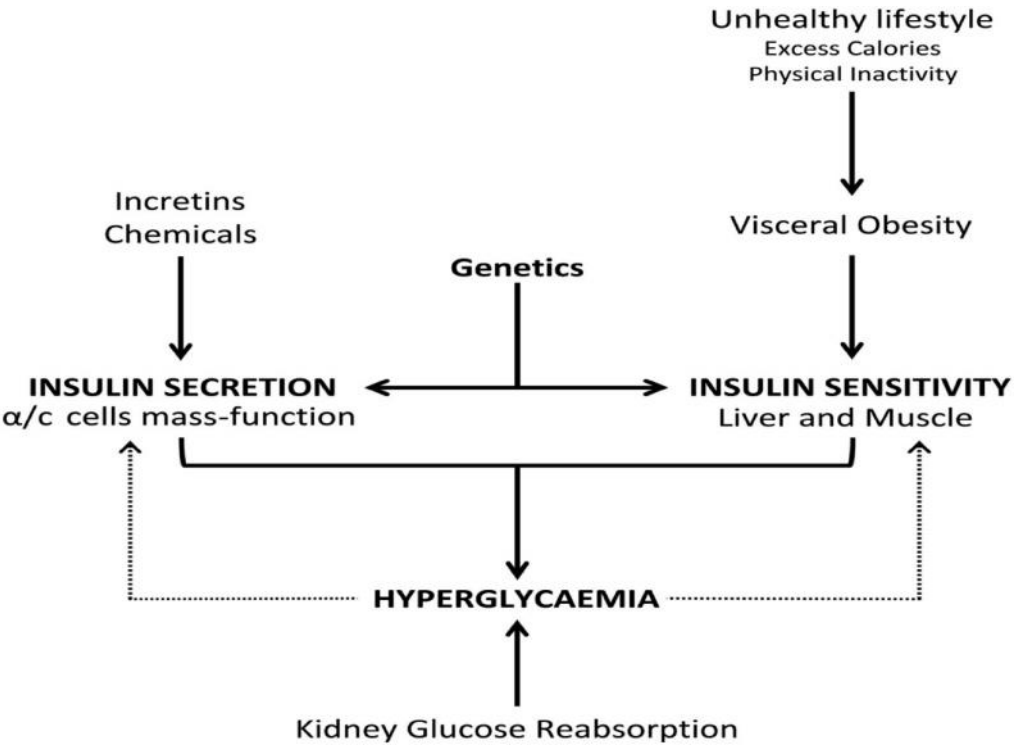


Figure 11 -Pathogenesis of type 2 Diabetes

A person is diagnosed to have diabetes when classic signs and symptoms of hyperglycemia are associated with a single random plasma glucose measurement of 200 mg per dL (11.1 mmol per L) or greater. Alternatively, the diagnosis can be made with HbA1C level of 6.5% or greater, a fasting plasma glucose level of 126 mg per dL

(7.0 mmol per L) or greater, or a two-hour plasma glucose level of 200 mg per dL or greater during an oral glucose tolerance test with 75-g glucose load[139]. According to National Cholesterol Education Programme's Adult Panel III (NCEP ATP III) fasting plasma glucose of > 6.1 mmol/l (100mg/dl)[96]

The factors like, the permanent elevation of plasma free fatty acid (FFA) and the predominant utilization of lipids by muscles inducing a diminution of glucose uptake and insulin resistance, dominate in obesity. An insulin-resistant state – as the key phase of metabolic syndrome – constitutes the major risk factor for the development of diabetes mellitus. Hyperinsulinemia is a compensatory mechanism that responds to increased levels of circulating glucose. People when diagnosed to have type 2 diabetes usually pass through the phases of excessive adipogenesis (obesity), nuclear peroxisome proliferator activated receptors (PPAR) modulation, insulin resistance, hyperinsulinemia, pancreatic beta cells stress and damage leading to progressively decrease of insulin secretion, impaired glucose postprandial and fasting levels[140-143]. Fasting glucose level usually remains normal as long as insulin hypersecretion can compensate for insulin resistance. Hyperglycemia occurs as a late phenomenon due to the fall in insulin secretion and, in fact, separates the patients with metabolic syndrome from those with or without overt diabetes.

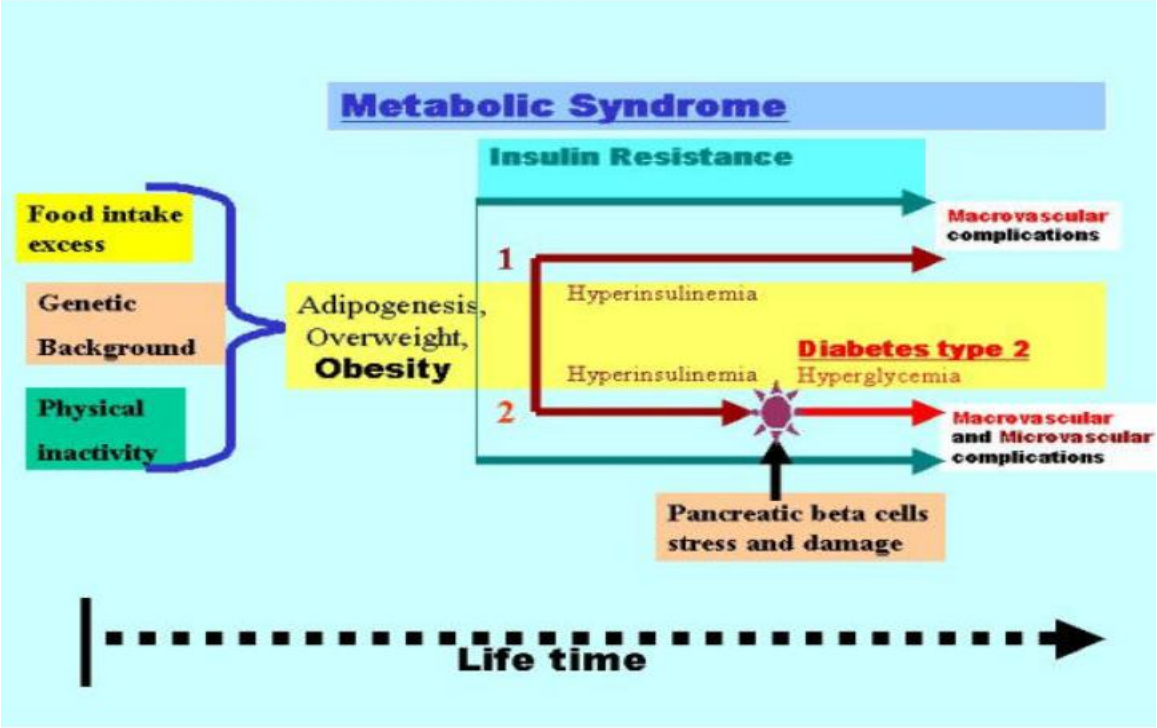


Figure 12- The relationship between metabolic syndrome, insulin resistance, hyperinsulinemia and hyperglycemia

Time-related scheme.

The relationship between metabolic syndrome, insulin resistance, hyperinsulinemia and hyperglycemia (overt type 2 diabetes). An insulin-resistant state following nuclear peroxisome proliferator activated receptors (PPAR) deactivation is the key phase of metabolic syndrome initiation. Afterwards, there are 2 principal pathways of metabolic syndrome development: 1)With preserved pancreatic beta cells function and insulin hypersecretion which can compensate for insulin resistance. This pathway leads mainly to the macrovascular complications of metabolic syndrome; 2)With massive damage of pancreatic beta cells leading to progressively decrease of insulin secretion and to hyperglycemia (e.g. overt type 2 diabetes). This pathway leads both to microvascular and macrovascular complications.

DYSLIPIDEMIA

As lipids, such as cholesterol and triglycerides, are insoluble in water these lipids must be transported in association with proteins in the circulation. Large quantities of fatty acids from meals must be transported as triglycerides to avoid toxicity. The lipoproteins play an important role in the absorption and transport of dietary lipids by the small intestine, in the transport of lipids from the liver to peripheral tissues, and also the transport of lipids from peripheral tissues to the liver and intestine (reverse cholesterol transport). A secondary function is to transport toxic foreign hydrophobic and amphipathic compounds, such as bacterial endotoxin, from areas of invasion and infection[144].

Lipoproteins are the complex particles with a central core containing cholesterol esters and triglycerides surrounded by free cholesterol, phospholipids, and apolipoproteins, which facilitate lipoprotein formation and function. Plasma lipoproteins have been divided into seven classes based on size, lipid composition, and apolipoproteins (chylomicrons, chylomicron remnants, VLDL, IDL, LDL, HDL, and Lp (a)). Chylomicron remnants like, VLDL, IDL, LDL, and Lp (a) are all pro-atherogenic while HDL is anti-atherogenic.[145]

Structure of lipoprotein

Lipoproteins are complex particles that have a central hydrophobic core of non-polar lipids, primarily cholesterol esters and triglycerides. This hydrophobic core is surrounded by a hydrophilic membrane consisting of phospholipids, free cholesterol, and apolipoproteins[145]

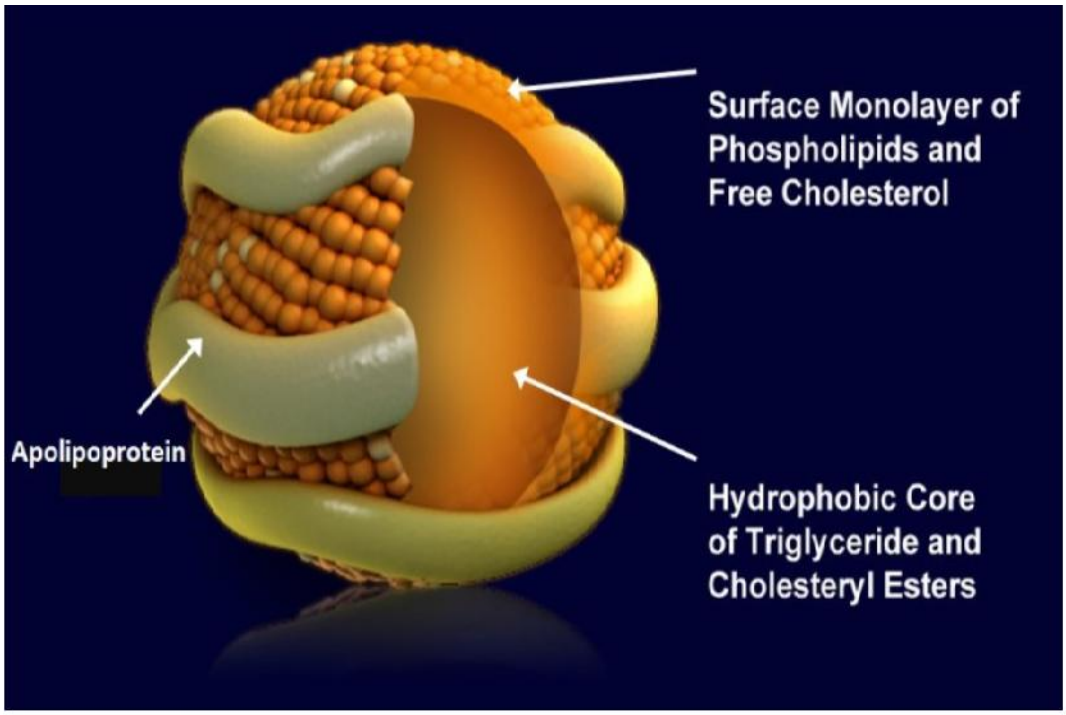


Figure 13- Lipoprotein Structure

Lipoprotein Structure (figure modified from Biochemistry 39: 9763, 2000)

High density lipoproteins (HDL)

These particles play a major role in reverse cholesterol transport from peripheral tissues to the liver, which is the reason for HDL being anti-atherogenic. In addition, HDL particles have anti-oxidant, anti-inflammatory, anti-thrombotic, and anti-apoptotic properties, which also contribute to their ability to inhibit atherosclerosis. HDL particles are enriched in cholesterol and phospholipids. The apolipoproteins A-I, A-II, A-IV, C-I, C-II, C-III, and E are associated with these particles. Apo A-I is the core structural protein and each HDL particle may contain multiple Apo A-I molecules[145]

Skin Surface and Epidermal Lipids

The stratum corneum is made of corneocytes and intracellular lipids, mainly ceramides, sterols, and free fatty acids which form the barrier for diffusion of substances into the skin[146-149]. The lipids are organised into multilamellar intercellular membranes which are derived from glycerophospholipids, glucocerebrosides, sphingomyelin of the stratum granulosum-stratum corneum interface[149,150]. Then these precursors are converted to ceramides and free fatty acids by the hydrolytic enzymes[151,152]. In psoriasis, alterations in ceramide content have been observed [153] and abnormal lipid structures reported[154]. Total lipids, phospholipids, triacylglycerols, and cholesterol were found to increase both in blood and in epidermis of psoriatic patients[155,156]. The proportion of an esterified fraction decreased mainly in the normally appearing epidermis areas, especially in severe psoriasis[157].

Lacroix demonstrated significant quantity of cholesterol in scaly plaques and in serum. He suggested that psoriasis might be the form of cholesterol elimination through the skin[158]. The regulation of cellular cholesterol metabolism is fully developed in the foetal life. The maintenance of its steady cellular levels is a key element of cellular and systemic homeostasis. It is already known that this homeostasis is disturbed in psoriasis[159]. Every day approximately 85 mg of cholesterol is secreted through the healthy skin. In psoriasis, the patients loose daily 12–23.5-times more lipids from the scales than compared to healthy subjects[160-162]

Serum Lipids

In psoriasis, there is a decrease of HDL synthesis and HDL structural changes have been observed, due to various biochemical disturbances such as abnormalities of receptor function, changes of hepatic structure and function, activity changes of

hepatocyte membranes, impaired reverse cholesterol transport (RCT), esterification, and lipases[161]

Apolipoproteins are the protein part of lipoproteins, their composition is specific for each lipoprotein. They have a varied molecular structure, amino acid composition, and anti-atherosclerotic properties. Apolipoprotein A1 is immunocytochemically detected at the psoriatic skin dermo-epidermal junction, vascular walls, and the perivascular region of papillary dermis. Apolipoprotein B100 and apolipoprotein E are observed intracellularly both in normal epidermis and psoriatic epidermis, and they are also detected in parakeratotic regions in the horny layer[163]. Elevated levels of apolipoprotein B is associated with the increased risk of atherosclerosis, because of its role in the cholesterol accumulation in the endothelium, which initiates the atheromatous process.[164]

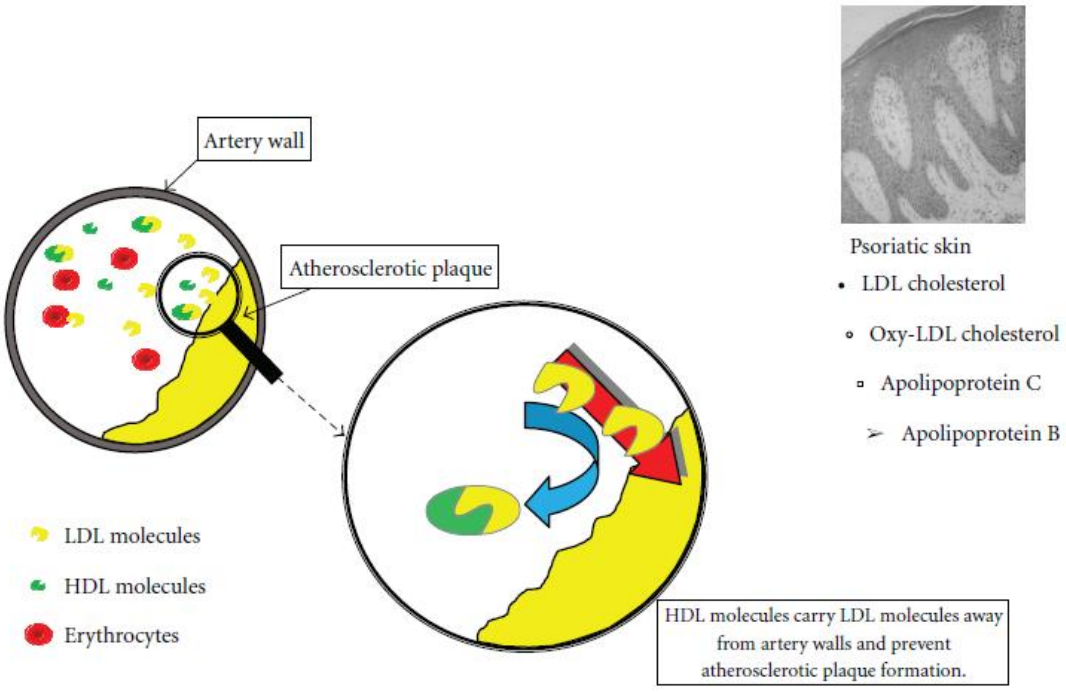


Figure 14- Lipoprotein replacement in circulation from artery walls and peripheral blood into psoriatic skin lesions

Lipoprotein replacement in circulation from artery walls and peripheral blood into psoriatic skin lesions[165]

Peroxisome Proliferator-Activated Receptors (PPARs) and Liver X Receptors (LXRs)

The epidermis is an active site of lipid metabolism, and all peroxisome proliferator-activated receptor (PPAR) and liver X receptor (LXR) isoforms are expressed in the epidermis. An increased expression of PPAR*β/δ* and a decreased expression of PPAR*α* and PPAR*γ* were observed in the lesional skin of patients with psoriasis and atopic dermatitis[166-168]. Since the prevalence of metabolic syndrome is increased in psoriasis[169], a combination of insulin resistance, obesity, or chronic inflammation may trigger the expression of PPAR*β/δ*, which in turn contributes to a nonterminated regenerative skin phenotype. This disease mechanism would be expected to be aggravated by acute inflammation, or stress via the induction of PPAR*β/δ* by TNF-*α* and stress-activated kinase[170].

The lipid disturbances are a very important part in the pathogenesis of psoriasis. The results of the majority of the studies are coherent and indicate that the increased total cholesterol, LDL cholesterol and/or triglycerides, and decreased HDL cholesterol in psoriatic patients' serum the composition of apolipoproteins, and increased production of oxygen metabolites are characteristic of the metabolic syndrome. These parameters have a great impact on some comorbidities observed in psoriatic patients especially on cardiovascular diseases.

CO-RELATION OF METABOLIC SYNDROME AND PSORIASIS

Psoriasis is a chronic skin condition which is T cell mediated inflammatory disease and occasionally affects the joints.[5] Till date, the exact underlying pathways that link metabolic syndrome to psoriasis not fully understood. Increased mortality from cardiovascular disease in patients with severe psoriasis has been documented and psoriasis may be an independent risk factor for myocardial infarction, especially in young patients.[5] Psoriasis is related to metabolic syndrome, independent of its severity.[5] Several factors may contribute to an unfavorable cardiovascular risk profile in patients with psoriasis, such as cigarette smoking, alcohol consumption, obesity, physical inactivity, homocysteinemia, psychological stress, and depression, all of which are more prevalent in patients with psoriasis.[3,5]

Numerous studies have suggested that patients with psoriasis have an increased risk of myocardial infarction, stroke, vascular inflammation and atherosclerotic conditions independent of conventional risk factors for cardiovascular disease.[171-174] Similarly, metabolic syndrome, which is a group of cardiovascular risk factors, specifically obesity, hypertension, dyslipidemia, and insulin resistance, is associated with chronic inflammation.[175,176]

Psoriasis and metabolic syndrome share multiple inflammatory and cytokine-mediated mechanisms. Both are part of an intriguing network of genetic, clinical, and pathophysiologic features. The mechanisms underlying the association between psoriasis and metabolic syndrome are multifactorial (involving both genetic and environmental factors) and often overlap with metabolic abnormalities, which frequently coexist in psoriatic patients. In particular, altered transcription in genes biologically significant for psoriasis and metabolic disorders, including renin,

cytotoxic T-lymphocyte antigen 4 (CTLA4), and Toll-like receptor 3 (TLR3), was identified.[177]

Psoriasis is a T cell–mediated inflammatory disease characterized by the proliferation and activation of Th-1, Th-17, and Th-22 cells, which lead to local overproduction of multiple proinflammatory mediators by lymphocytes and keratinocytes into the skin of psoriatic patients, including tumor necrosis factor (TNF)-α, interleukin (IL)-6, IL-1, IL-17, IL-22, IL-23, vascular endothelial growth factor, and interferon-γ.[178-180]There is evidence that these locally overproduced proinflammatory mediators can migrate into the systemic circulation, and potentially induce systemic insulin resistance, circulatory endothelial dysfunction, increased oxidative stress, increased angiogenesis, and hypercoagulation, all of which are common features of inflammatory conditions and cardiovascular damage.[181-183]

TNF-alpha, overexpressed in patients with psoriasis, is found to be elevated in people with abdominal obesity, a component of metabolic syndrome. It promotes the production of adhesion molecules by endothelial cells, promoting monocyte binding in the early phases of atherosclerosis.[184] If left uncontrolled, the immunologic mediators common to both disease processes may lead to cardiovascular impairment or death.

Abdominal adipose tissue accumulation, which is a key pathogenic factor of Metabolic syndrome, is also a major source of several proinflammatory cytokines and adipokines.[185]Activatedmacrophages and T cells infiltrate abdominal visceral adipose tissue, stimulates adipocytes to release nonesterified fatty acids (NEFA) and secrete a myriad of adipokines and proinflammatory molecules, such as TNF-α, IL-6, leptin, resistin, chemerin, vascular endothelial growth factor, and procoagulant

factors, which induce a chronic low-grade inflammatory state, thus further contributing to the development of systemic insulin resistance, dysglycemia, atherogenic dyslipidemia, vascular dysfunction, and NAFLD.[185,186]

In addition, evidence indicates that NAFLD (especially in its more severe forms, ie, steatohepatitis and advanced fibrosis) exacerbates systemic/hepatic insulin resistance, induces atherogenic dyslipidemia, and releases a series of proinflammatory, procoagulant, pro-oxidant, and profibrogenic mediators (eg, C-reactive protein, IL-6, fibrinogen, plasminogen activator inhibitor-1,transforming growth factor-β) which play important roles in the pathophysiology of psoriasis. It is conceivable that the release of these mediators from the steatotic and inflamed liver may also adversely influence the severity of psoriasis by increased keratinocyte proliferation, increased inflammation, and upregulation of various vascular adhesion molecules.[186]

In addition, the existence of pleiotropic genetic loci, e.g., PSORS2-4, CDKAL1, and ApoE4, has also been implicated in the shared genetic susceptibility to both psoriasis and metabolic syndrome.[187]

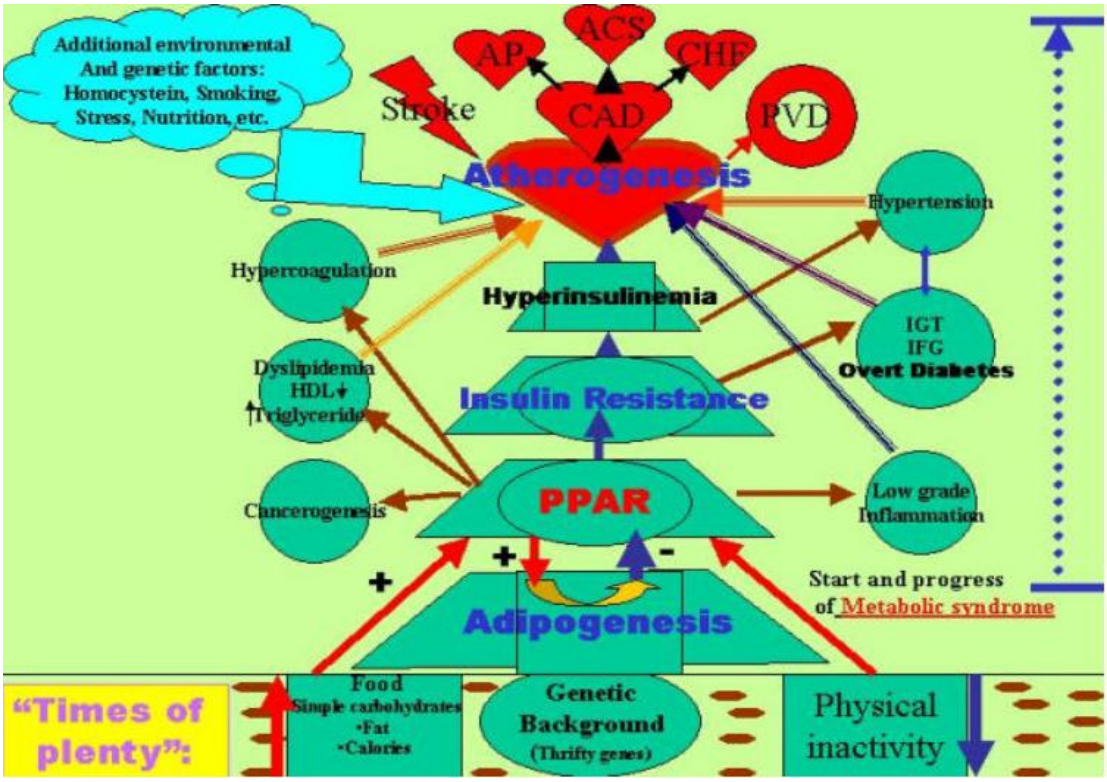


Figure 15- The atherogenesis tree, showing the complex interrelationship between hereditary and environmental factors in the pathogenesis of metabolic syndrome and atherothrombotic events

The atherogenesis tree, showing the complex interrelationship between hereditary and environmental factors in the pathogenesis of metabolic syndrome and atherothrombotic events. The central role of an insulin-resistant state following adipogenesis and nuclear peroxisome proliferator activated receptors (PPAR) deactivation is emphasized. CAD – coronary artery disease; AP – angina pectoris; ACS – acute coronary syndromes; CHF – congestive heart failure; PVD – peripheral vascular disease; HDL – high density lipoproteins cholesterol; IGT – impaired glucose tolerance; IFG – impaired fasting glucose.

METHODOLOGY

This study was conducted within a duration of twelve months (January 2017 to December 2017) in the Department of Dermatology, Venereology and Leprosy, KLE'S Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi. The study was a hospital-based cross sectional study involving 100 patients.

INCLUSION CRITERIA

Subjects of either sex of age group 15-60 years, with psoriasis, attending KLE'S Dr. Prabhakar Kore hospital & MRC, Belagavi.

EXCLUSION CRITERIA

1) Subjects who are on drugs like anti-malarials, lithium, interferons, imiquimoid, NSAIDs, glibenclamide.
2) Subjects who are known cases of hypertension, DM, hyperlipidaemia before the onset of psoriasis.
3) Subjects who have renal or cardiac abnormalities.

An informed consent was taken from all patients and patient characteristics were recorded on a standard proforma. Statistical analysis of the data was done using statistical processing software (SPSS-17) and epi-info software.

Relevant data included age, gender, waist circumference, blood pressure, smoking habit, age of onset and duration of psoriasis, type and severity of psoriasis. To determine waist circumference, the upper hip bone was located and the measuring tape was placed at the level of the upper most part of the hip bone around the

abdomen (ensuing the tape measure was horizontal). The tape measure was snug but did not cause compression on the skin. Blood pressure was recorded as the average of two measurements after subjects have been sitting for five minutes. Severity of psoriasis was assessed according to psoriasis area and severity index (PASI).[188]

Metabolic syndrome was diagnosed by the presence of three or more of the five criteria of the National Cholesterol Education Programme's Adult Panel III (ATP III):[96]

1. waist circumference > 102 cm(40 inches) in men or > 88 cm(35 inches) in women;
2. hypertriglyceridaemia> 1.7 mmol/l (150mg/dl);
3. high density lipoprotein (HDL) cholesterol < 1.0mmol/l (40mg/dl) in men or < 1.3mmol/dl (50mg/dl) in women;
4. blood pressure > 130/85 mmHg;
5. fasting plasma glucose of > 6.1 mmol/l (100mg/dl)

Venous samples were taken at the enrolment visit after the subjects had fasted overnight (at least 8 h). Serum cholesterol and triglycerides were measured with enzymatic procedures. Plasma glucose was measured using a glucose oxidase method.

The study was approved by the institutional ethical committee.

RESULTS

Table 1: Distribution by socio demographic and other factors

Factors	Number of cases	Percentage
Gender		
Male	69	69.00
Female	31	31.00
Age groups		
<=20yrs	7	7.00
21-30yrs	15	15.00
31-40yrs	31	31.00
41-50yrs	22	22.00
51-60yrs	25	25.00
Duration		
<1yr	14	14.00
1-5yrs	41	41.00
6-10yrs	21	21.00
>=11yrs	24	24.00
Hypertension		
No	93	93.00
Yes	7	7.00
Diabetic mellitus		
No	90	90.00
Yes	10	10.00
PASI		
<=9	64	64.00
>=9.1	36	36.00
Types		
CPP	75	75.00
CPP,PA	3	3.00
Erythroderma	5	5.00
Guttate	2	2.00
PPK	8	8.00
Pustular	2	2.00
Pustular, PA	2	2.00
Scalp	3	3.00
Total	100	100.00

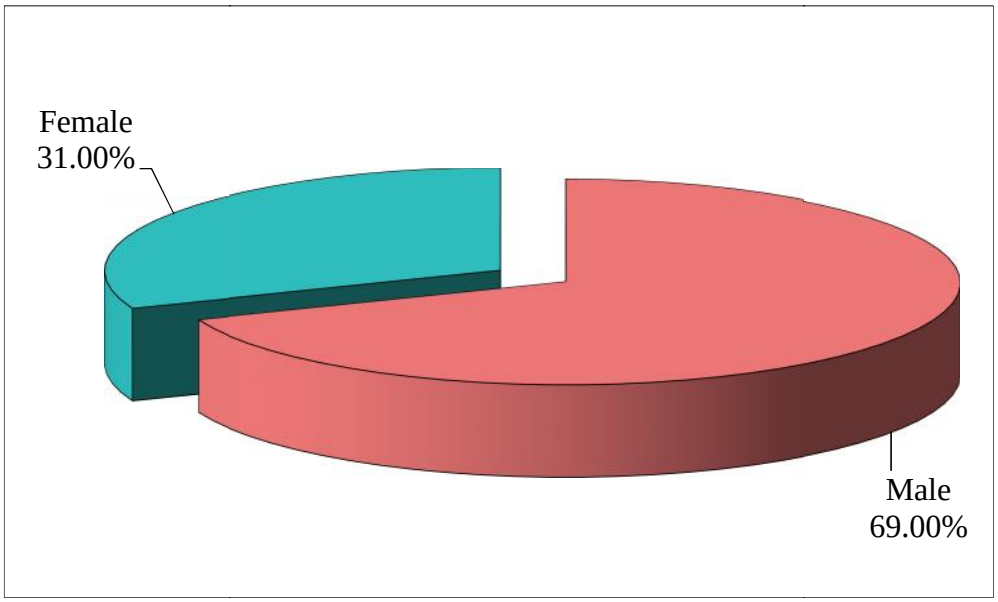
The number of males in the study population were n=69, 69% and females were n=31, 31%, and 68% of study population belonged to age group between 21 to 50 years.

Only few were diabetic[n=10,10%] and hypertensive[n=7,7%].

PASI <9 was seen in n=64,64% compared to PASI >9 in n=36,36%

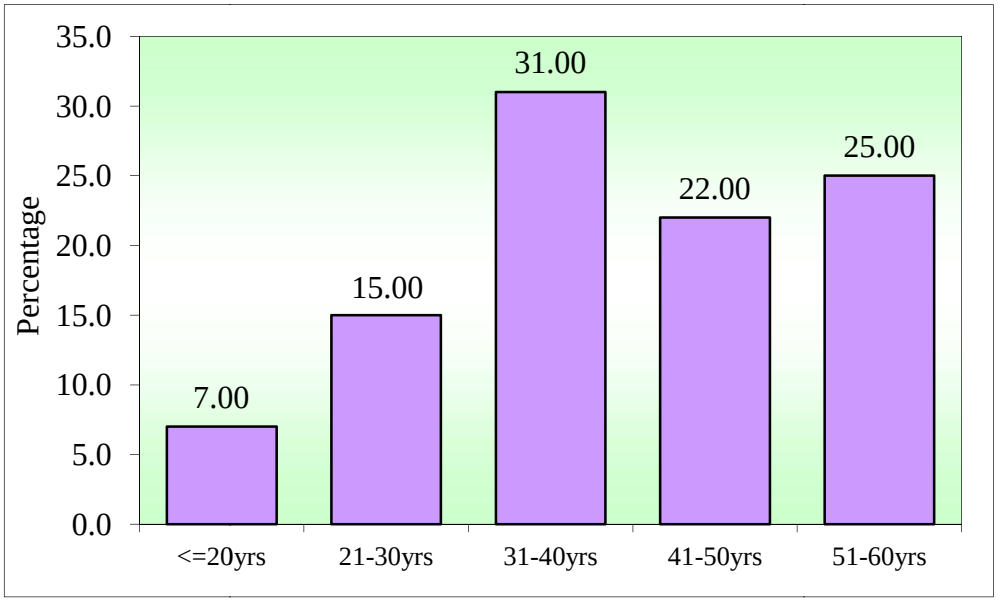
Regarding the type of psoriasis, CPP was the most commonest variety [n=75,75%] followed by PPK=n=8,8%], CPP,PA[n=5,5%] and other variety

Graph 1: GENDER DISTRIBUTION



69%[(n=69] of the study population were males whereas,31%were females[n=31]

Graph 2: AGE WISE DISTRIBUTION

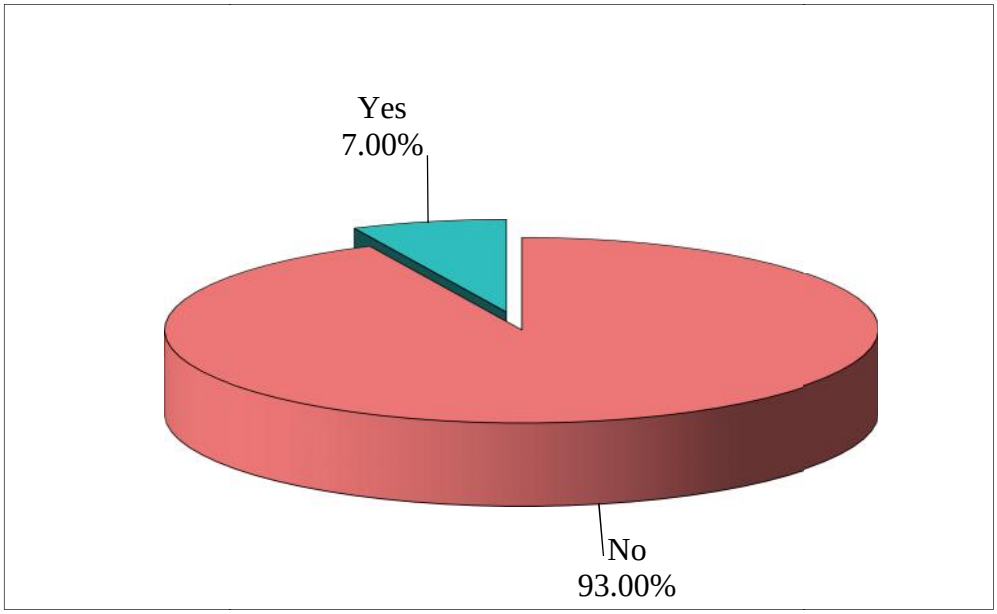


Majority of the population belonged in the age group of 31-40 years [n=31,31%] followed by 51-60 years[n=25,25%], 41-50 years, [n=22,22%], 21-30 years [n=15,15 %] and the least being less than 20yrs[n=7,7%]

Graph 3: DURATION OF THE DISEASE WISE DISTRIBUTION

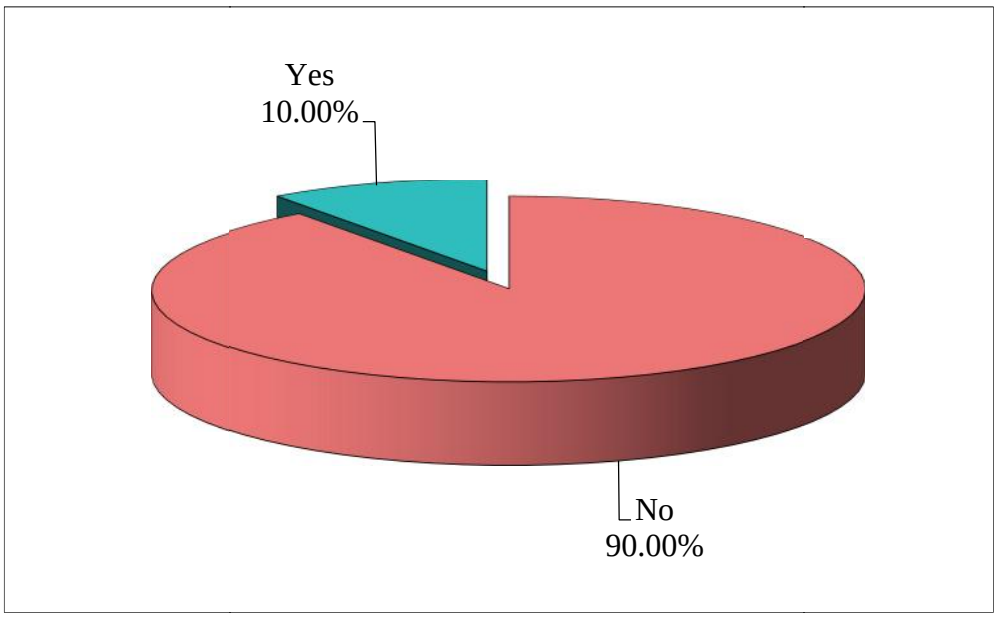
Majority of the population belonged in the group of 1-5years [n=41,41%], followed by more than 11years [n=24,24%], 6-10years [n=21,21%] and less than 1year[n=14,14%]

Graph 4: HYPERTENSION WISE DISTRIBUTION



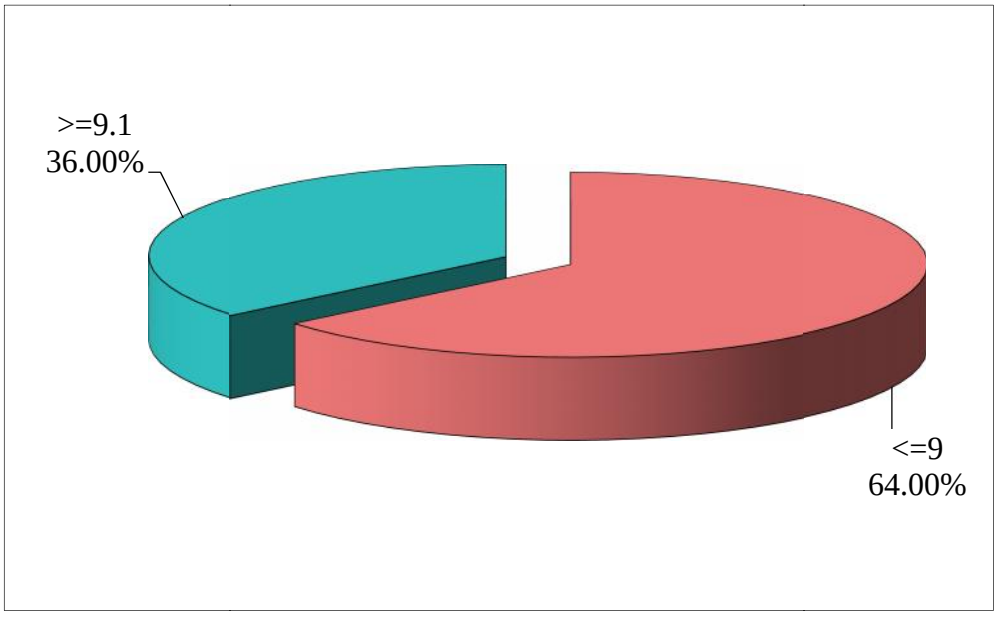
Overall number of hypertensives was less[n=7,7%] compared to normotensives [n=93,93%]

Graph 5: DIABETES MELLITUS WISE DISTRIBUTION



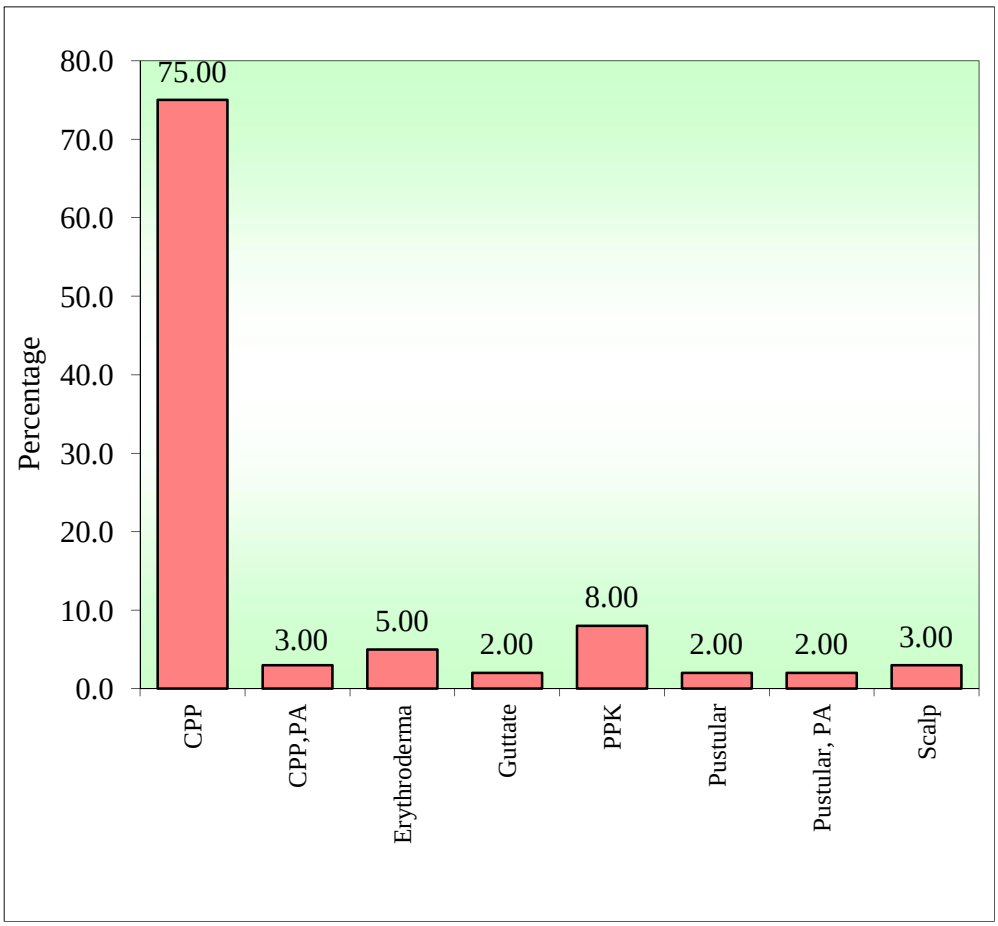
Overall number of diabetics was less[n=10,10%] compared to non-diabetics [n=90,90%]

Graph 6: PASI WISE DISTRIBUTION



PASI <9 was higher [n=64,64%] compared to PASI >9[n=36,36%]

Graph 7: TYPES OF PSORIASIS WISE DISTRIBUTION



CPP was the most commonest type of psoriasis [n=75,75%] followed by PPK[n=8,8%], erythroderma[n=5,5%], CPP with PA[n=3,3%],scalp psoriasis[n=3,3%], pustular[n=2,2%], pustular with PA[n=2,2%] and guttate[n=2,2%].

Table 2: Association between gender and prevalence of metabolic syndrome

Factors	Metabolic syndrome				Total	Chi-square	p-value
	Normal	%	Abnormal	%			
Gender							
Male	43	62.32	26	37.68	69	3.3010	0.0690
Female	25	80.65	6	19.35	31		

There was no statistically significant relationship between gender [p>0.05] , in relation to occurrence of metabolic syndrome.

However, metabolic syndrome was higher in males(n=26, 37.68%) compared to females (n=6, 19.35%).

Table 3: Association between age and prevalence of metabolic syndrome

Factors	Metabolic syndrome				Total	Chi-square	p-value
	Normal	%	Abnormal	%			
Age groups							
<=20yrs	6	85.71	1	14.29	7	7.4430	0.1140
21-30yrs	13	86.67	2	13.33	15		
31-40yrs	23	74.19	8	25.81	31		
41-50yrs	12	54.55	10	45.45	22		
51-60yrs	14	56.00	11	44.00	25		

There was no statistically significant relationship between age categorization [p>0.05] in relation to occurrence of metabolic syndrome.

However, metabolic syndrome was highest in the age group 51-60yrs (n=11,44%), followed by 41-50yrs(n=10, 45.5%) and the least in the age group, less than 20yrs (n=1, 14.29%)

Table 4: Association between duration of psoriasis and prevalence of metabolic syndrome

Factors	Metabolic syndrome				Total	Chi-square	p-value
	Normal	%	Abnormal	%			
Duration							
<1yr	9	64.29	5	35.71	14	8.5390	0.0360*
1-5yrs	28	68.29	13	31.71	41		
6-10yrs	19	90.48	2	9.52	21		
>=11yrs	12	50.00	12	50.00	24		

*p<0.05

Duration of disease was significant in relation to occurrence of metabolic syndrome[p<0.05].

Higher the duration of psoriasis, more is the prevalence of metabolic syndrome.

Metabolic syndrome was present 50%(n=12) of the patients who had psoriasis for more than 11yrs and the least being 9.52%(n=2) in the group 6-10yrs

Table 5: Association between hypertension and prevalence of metabolic syndrome

Factors	Metabolic syndrome				Total	Chi-square	p-value
	Normal	%	Abnormal	%			
Hypertension							
No	67	72.04	26	27.96	93	9.9800	0.0020*
Yes	1	14.29	6	85.71	7		

*p<0.05

Presence of hypertension was significant in occurrence to metabolic syndrome. (p<0.05)

Table 6: Association between Diabetis mellitus and prevalence of metabolic syndrome

Factors	Metabolic syndrome				Total	Chi-square	p-value
	Normal	%	Abnormal	%			
Diabetis mellitus							
No	64	71.11	26	28.89	90	4.0030	0.0450*
Yes	4	40.00	6	60.00	10		

*p<0.05

Presence of Diabetes mellitus was significant in occurrence to metabolic syndrome. (p<0.05)

Table 7: Association between PASI and prevalence of metabolic syndrome

Factors	Metabolic syndrome				Total	Chi-square	p-value
	Normal	%	Abnormal	%			
PASI							
<=9	49	76.56	15	23.44	64	5.9900	0.0140*
>=9.1	19	52.78	17	47.22	36		

*p<0.05

PASI score was significant in occurrence to metabolic syndrome. (p<0.05) Metabolic syndrome is associated with severe forms of psoriasis (PASI score>=9.1) 47.22%(n=17) of the patients who had PASI >/= 9.1 were positive for metabolic syndrome.

Table 8: Association between types of psoriasis and prevalence of metabolic syndrome

Factors	Metabolic syndrome				Total
	Normal	%	Abnormal	%	
Types					
CPP	52	69.33	23	30.67	75
CPP,PA	1	33.33	2	66.67	3
Erythroderma	3	60.00	2	40.00	5
Guttate	1	50.00	1	50.00	2
PPK	7	87.50	1	12.50	8
Pustular	0	0.00	2	100.00	2
Pustular, PA	2	100.00	0	0.00	2
Scalp	2	66.67	1	33.33	3
Total	68	68.00	32	32.00	100
CPP	52	69.33	23	30.67	75

Prevalence of metabolic syndrome was higher in pustular psoriasis(n=2,100%), followed by chronic plaque psoriasis with psoriatic arthritis(n=2,66.67%). However, there was no correlation between pustular psoriasis with psoriatic arthritis and metabolic syndrome.

Table 9: Association between prevalence of WC (inc) with other characteristics

Factors	WC (inc)				Total	Chi-square	p-value
	Normal	%	Abnormal	%			
Gender							
Male	45	65.22	24	34.78	69	3.5570	0.0590
Female	14	45.16	17	54.84	31		
Age groups							
<=20yrs	7	100.00	0	0.00	7	8.3420	0.0800
21-30yrs	6	40.00	9	60.00	15		
31-40yrs	17	54.84	14	45.16	31		
41-50yrs	12	54.55	10	45.45	22		
51-60yrs	17	68.00	8	32.00	25		
Duration							
<1yr	7	50.00	7	50.00	14	0.6860	0.8760
1-5yrs	25	60.98	16	39.02	41		
6-10yrs	12	57.14	9	42.86	21		
>=11yrs	15	62.50	9	37.50	24		
Hypertension							
No	54	58.06	39	41.94	93	0.4810	0.4880
Yes	5	71.43	2	28.57	7		
Diabetic mellitus							
No	52	57.78	38	42.22	90	0.5560	0.4560
Yes	7	70.00	3	30.00	10		
PASI							
<=9	37	57.81	27	42.19	64	0.1040	0.7480
>=9.1	22	61.11	14	38.89	36		
Types							
CPP	44	58.67	31	41.33	75		
CPP,PA	2	66.67	1	33.33	3		
Erythroderma	3	60.00	2	40.00	5		
Guttate	1	50.00	1	50.00	2		
PPK	7	87.50	1	12.50	8		
Pustular	0	0.00	2	100.00	2		
Pustular, PA	0	0.00	2	100.00	2		
Scalp	2	66.67	1	33.33	3		
Total	59	59.00	41	41.00	100		

There was no statistically significant relationship between gender, age categorization, hypertension, Diabetes mellitus, PASI or types of psoriasis in relation to waist circumference.[p>0.05]

Table 10: Association between abnormal of SBP (mm of Hg) with other characteristics

Factors	SBP (mm of Hg))				Total	Chi-square	p-value
	Normal	%	Abnormal	%			
Gender							
Male	25	36.23	44	63.77	69	4.1600	0.0410*
Female	18	58.06	13	41.94	31		
Age groups							
<=20yrs	6	85.71	1	14.29	7	10.9110	0.0280*
21-30yrs	9	60.00	6	40.00	15		
31-40yrs	12	38.71	19	61.29	31		
41-50yrs	5	22.73	17	77.27	22		
51-60yrs	11	44.00	14	56.00	25		
Duration							
<1yr	6	42.86	8	57.14	14	1.4740	0.6880
1-5yrs	20	48.78	21	51.22	41		
6-10yrs	9	42.86	12	57.14	21		
>=11yrs	8	33.33	16	66.67	24		
Hypertension							
No	42	45.16	51	54.84	93	2.5320	0.1120
Yes	1	14.29	6	85.71	7		
Diabetic mellitus							
No	40	44.44	50	55.56	90	0.7660	0.3810
Yes	3	30.00	7	70.00	10		
PASI							
<=9	31	48.44	33	51.56	64	2.1450	0.1430
>=9.1	12	33.33	24	66.67	36		
Types							
CPP	34	45.33	41	54.67	75		
CPP,PA	0	0.00	3	100.00	3		
Erythroderma	2	40.00	3	60.00	5		
Guttate	1	50.00	1	50.00	2		
PPK	5	62.50	3	37.50	8		
Pustular	0	0.00	2	100.00	2		
Pustular, PA	0	0.00	2	100.00	2		
Scalp	1	33.33	2	66.67	3		
Total	43	43.00	57	57.00	100		

*p<0.05

There was statistically significant relationship between gender and age categorization in relation to SBP[$P<0.05$]

There was no statistically significant relationship between hypertension, Diabetes mellitus, PASI or types of psoriasis in relation to SBP [$p>0.05$]

Table 11: Association between abnormal of DBP (mm of Hg) with other characteristics

Factors	DBP (mm of Hg))				Total	Chi-square	p-value
	Normal	%	Abnormal	%			
Gender							
Male	46	66.67	23	33.33	69	4.5300	0.0330*
Female	27	87.10	4	12.90	31		
Age groups							
<=20yrs	7	100.00	0	0.00	7	6.2200	0.1830
21-30yrs	13	86.67	2	13.33	15		
31-40yrs	22	70.97	9	29.03	31		
41-50yrs	16	72.73	6	27.27	22		
51-60yrs	15	60.00	10	40.00	25		
Duration							
<1yr	10	71.43	4	28.57	14	5.8920	0.1170
1-5yrs	30	73.17	11	26.83	41		
6-10yrs	19	90.48	2	9.52	21		
>=11yrs	14	58.33	10	41.67	24		
Hypertension							
No	72	77.42	21	22.58	93	13.1650	0.0001*
Yes	1	14.29	6	85.71	7		
Diabetic mellitus							
No	68	75.56	22	24.44	90	2.9820	0.0840
Yes	5	50.00	5	50.00	10		
PASI							
<=9	51	79.69	13	20.31	64	4.0340	0.0450*
>=9.1	22	61.11	14	38.89	36		
Types							
CPP	55	73.33	20	26.67	75		
CPP,PA	1	33.33	2	66.67	3		
Erythroderma	4	80.00	1	20.00	5		
Guttate	2	100.00	0	0.00	2		
PPK	6	75.00	2	25.00	8		
Pustular	1	50.00	1	50.00	2		
Pustular, PA	2	100.00	0	0.00	2		
Scalp	2	66.67	1	33.33	3		
Total	73	73.00	27	27.00	100		

*p<0.05

There was statistically significant relationship between gender, hypertension and PASI in relation to DBP[$p<0.05$]

There was no statistically significant relationship between age categorization, Diabetes mellitus or types of psoriasis in relation to DBP.[$p>0.05$]

Table 12: Association between abnormal of FBS (mg/dl) with other characteristics

Factors	FBS (mg/dl)				Total	Chi-square	p-value
	Normal	%	Abnormal	%			
Gender							
Male	44	63.77	25	36.23	69	1.8320	0.1760
Female	24	77.42	7	22.58	31		
Age groups							
<=20yrs	6	85.71	1	14.29	7	15.5830	0.0040*
21-30yrs	14	93.33	1	6.67	15		
31-40yrs	25	80.65	6	19.35	31		
41-50yrs	11	50.00	11	50.00	22		
51-60yrs	12	48.00	13	52.00	25		
Duration							
<1yr	10	71.43	4	28.57	14	8.6640	0.0340*
1-5yrs	29	70.73	12	29.27	41		
6-10yrs	18	85.71	3	14.29	21		
>=11yrs	11	45.83	13	54.17	24		
Hypertension							
No	66	70.97	27	29.03	93	5.3770	0.0200*
Yes	2	28.57	5	71.43	7		
Diabetic mellitus							
No	66	73.33	24	26.67	90	11.7650	0.0010*
Yes	2	20.00	8	80.00	10		
PASI							
<=9	51	79.69	13	20.31	64	11.1600	0.0010*
>=9.1	17	47.22	19	52.78	36		
Types							
CPP	51	68.00	24	32.00	75		
CPP,PA	2	66.67	1	33.33	3		
Erythroderma	3	60.00	2	40.00	5		
Guttate	1	50.00	1	50.00	2		
PPK	8	100.00	0	0.00	8		
Pustular	1	50.00	1	50.00	2		
Pustular, PA	1	50.00	1	50.00	2		
Scalp	1	33.33	2	66.67	3		
Total	68	68.00	32	32.00	100		

*p<0.05

There was statistically significant relationship between age categorization, duration of disease, Diabetes mellitus, hypertension and PASI in relation to FBS[$p<0.05$]

There was no statistically significant relationship between gender in relation to FBS[$p>0.05$]

Table 13: Association between abnormal of TG (mg/dl) with other characteristics

Factors	TG (mg/dl)				Total	Chi-square	p-value
	Normal	%	Abnormal	%			
Gender							
Male	52	75.36	17	24.64	69	9.2020	0.0020*
Female	31	100.00	0	0.00	31		
Age groups							
<=20yrs	6	85.71	1	14.29	7	1.3280	0.8570
21-30yrs	13	86.67	2	13.33	15		
31-40yrs	24	77.42	7	22.58	31		
41-50yrs	18	81.82	4	18.18	22		
51-60yrs	22	88.00	3	12.00	25		
Duration							
<1yr	10	71.43	4	28.57	14	4.8090	0.1860
1-5yrs	35	85.37	6	14.63	41		
6-10yrs	20	95.24	1	4.76	21		
>=11yrs	18	75.00	6	25.00	24		
Hypertension							
No	77	82.80	16	17.20	93	0.0390	0.8430
Yes	6	85.71	1	14.29	7		
Diabetic mellitus							
No	75	83.33	15	16.67	90	0.0710	0.7900
Yes	8	80.00	2	20.00	10		
PASI							
<=9	55	85.94	9	14.06	64	1.0870	0.2970
>=9.1	28	77.78	8	22.22	36		
Types							
CPP	62	82.67	13	17.33	75		
CPP,PA	3	100.00	0	0.00	3		
Erythroderma	5	100.00	0	0.00	5		
Guttate	1	50.00	1	50.00	2		
PPK	5	62.50	3	37.50	8		
Pustular	2	100.00	0	0.00	2		
Pustular, PA	2	100.00	0	0.00	2		
Scalp	3	100.00	0	0.00	3		
Total	83	83.00	17	17.00	100		

*p<0.05

There was statistically significant relationship between gender in relation to Triglyceride levels[$p<0.05$]There was statistically significant relationship between age categorization, duration of disease, Diabetes mellitus, hypertension and PASI in relation to Triglyceride levels[$p>0.05$]

Table 14 : Association between abnormal of HDL with other characteristics

Factors	HDL				Total	Chi-square	p-value
	Normal	%	Abnormal	%			
Gender							
Male	19	27.54	50	72.46	69	7.9010	0.0050*
Female	1	3.23	30	96.77	31		
Age groups							
<=20yrs	1	14.29	6	85.71	7	6.3740	0.1730
21-30yrs	1	6.67	14	93.33	15		
31-40yrs	6	19.35	25	80.65	31		
41-50yrs	3	13.64	19	86.36	22		
51-60yrs	9	36.00	16	64.00	25		
Duration							
<1yr	3	21.43	11	78.57	14	0.3810	0.9440
1-5yrs	8	19.51	33	80.49	41		
6-10yrs	5	23.81	16	76.19	21		
>=11yrs	4	16.67	20	83.33	24		
Hypertension							
No	20	21.51	73	78.49	93	1.8820	0.1700
Yes	0	0.00	7	100.00	7		
Diabetic mellitus							
No	20	22.22	70	77.78	90	2.7780	0.0960
Yes	0	0.00	10	100.00	10		
PASI							
<=9	15	23.44	49	76.56	64	1.3130	0.2520
>=9.1	5	13.89	31	86.11	36		
Types							
CPP	15	20.00	60	80.00	75		
CPP,PA	0	0.00	3	100.00	3		
Erythroderma	0	0.00	5	100.00	5		
Guttate	1	50.00	1	50.00	2		
PPK	3	37.50	5	62.50	8		
Pustular	0	0.00	2	100.00	2		
Pustular, PA	0	0.00	2	100.00	2		
Scalp	1	33.33	2	66.67	3		
Total	20	20.00	80	80.00	100		

*p<0.05

There was statistically significant relationship between gender in relation to HDL levels[$p<0.05$]

There was statistically significant relationship between age categorization, duration of disease, Diabetes mellitus, hypertension and PASI in relation to HDL levels[$p>0.05$]

Table 15:Multiple logistic regression analysis of metabolic syndrome

Factors	OR	95%CI for OR		P-value
		Lower	Upper	
Gender				
Male				
Female	0.61	0.19	1.91	0.3930
Age groups				
<=20yrs				
21-30yrs				
31-40yrs	0.36	0.04	3.76	0.3960
41-50yrs	0.26	0.05	1.35	0.1090
51-60yrs	0.60	0.20	1.84	0.3750
Duration				
<1yr				
1-5yrs	0.92	0.26	3.20	0.8950
6-10yrs	0.48	0.16	1.45	0.1940
>=11yrs	0.09	0.01	0.51	0.0070*
Hypertension				
No				
Yes	0.69	0.21	2.23	0.5330
Diabetic mellitus				
No				
Yes	10.99	0.99	124.34	0.0500*
PASI				
<=9				
>=9.1	1.36	0.25	7.44	0.7230

*p<0.05

Longer duration of disease [p<0.05] and presence of diabetes mellitus [p<0.05] were the only factors that were significantly associated with the metabolic syndrome on doing multiple logistic regression in different factors.

DISCUSSION

The present study is a hospital- based cross sectional study conducted over a period of 12 months from January 2017 to December 2017 in the Department of Dermatology, Venereology and Leprosy, KLE'S Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi. 100 psoriasis patients who met the inclusion criteria were selected for the study.

Since there is a bimodal age on onset, the first peak at 15-20years of age and a second one at 55-60years, patients between age of 15-60years are included in this study.

In our study, 32 out of 100 psoriatic patients(32%) where positive for metabolic syndrome. Our study was similar to a study conducted by Lakshmi S et al.[7], in Puducherry, India, over a period of two months between June and July 2012 ,which was a hospital based comparative study, which was conducted involving 40 adult patients with psoriasis and 40 age- and sex-matched controls, metabolic syndrome was present in 13 out of 40 (32.5%) patients with psoriasis.

Gender incidence

In a study, 'Prevalence of metabolic syndrome and cardiovascular changes in patients with chronic plaque psoriasis and their correlation with disease severity: A hospital-based cross-sectional study', performed on 140 patients with chronic plaque psoriasis and 140 controls carried out by Kothiwala SK et al.[189], the incidence of psoriasis in males was 102(72%) and in females was 38(27.1%).

In a study, 'Metabolic syndrome in patients with psoriasis: A comparative study' performed by Lakshmi et al.[7], of the 40 cases and controls, 31 each were male and 9 female.

In a study by Morgaonkar M et al.[190,] 'Prevalence of metabolic syndrome in psoriasis vulgaris: a cross sectional study from a tertiary care hospital of South-East Rajasthan', it was found that metabolic syndrome was more prevalent in female psoriasis patients than male patients (P =0.039). But in contrast to study by Gisondi et al.[5] in which prevalence of metabolic syndrome was independent of gender.

In the present study, majority of the study population were males[n=69, 69%] compared to females[n=31, 31%]. However, there was no statistically significant relationship between gender [p>0.05] , in relation to occurrence of metabolic syndrome.

Age incidence

In a study by Lakshmi et al.[7,] the mean age of cases was 49.95 years (±12.17), with age ranging from 26 to 76 years. The mean age of male and female psoriasis patients were 50.26 years and 48.89 years, respectively. The mean age of the controls was 49.35 years (±12.06). There was no statistically significant difference in age between the cases and controls.

In a study carried out by Kothiwala SK et al.[189], the mean age of cases and controls was 37.9 years(±13.26) and 36.1years (±11.63) respectively.

In the present study, majority of the patients were of the age group of 31-40 years [n=31 %] and the least were of age less than 20yrs[n=7%]. There was no association found between age and prevalence of metabolic syndrome.

Duration of the disease

In a study by Morgaonkar M et al.[190] , the mean duration of the disease in patients with and without metabolic syndrome was 7.6 years(±7.9) and 5.7 years(±4.8) respectively.

In a study by Lakshmi et al.[7] ,the mean duration of psoriasis was 4.5 years (±6.52). Mean duration in males was 3.94 years and in females was 6.44 years.

Madanagobalane S et al.[191]did not observe any difference between the presence of metabolic syndrome and the duration of the disease. Mallbris et al.[192], in their study, have shown that patients with new onset psoriasis had increased total cholesterol and HDL than controls, proving the presence of lipid abnormalities even in those with shorter duration of disease. On the contrary, an Indian study by Nisa et al.[188] has shown a positive association between longer duration of psoriasis and metabolic syndrome.

In our study, majority of the population belonged in the group of 1-5years duration [n=41,41%] and the least were less than 1year duration [n=14,14%]. Duration of disease was significant in relation to occurrence of metabolic syndrome[$p<0.05$]. Our study has shown a positive association between longer duration of psoriasis and metabolic syndrome.

Psoriasis and hypertension

In one of the studies, mean systolic blood pressure (mm Hg) was 129.4(±14.42) in psoriasis cases and was 121.5(±11.90) in controls.[189]

In a study by Morgaonkar M et al.[190], 42% of the cases and 21% of the controls were hypertensives.

Whereas, in the present study, 32% of the patients were hypertensives. Amongst them, (n=44)63.77% of the males and (n=13)41.94% of the females had raised SBP. (n=23)33.33% of the males and (n=4) 12.90% of the females had raised DBP. Presence of hypertension was significant in occurrence to metabolic syndrome. ($p<0.05$)

Psoriasis and Diabetes mellitus

In a study by Madanagobalane S et al.[191] 61% of the cases and 47.5% of the controlshad FBS>100mg/dl.

In a study[7],on analysing the individual components of metabolic syndrome among psoriasis patients, they found that fasting blood sugar level was significantly higher among those with metabolic syndrome ($P < 0.001$).

The prevalence of raised FBS among psoriatic patients was found to be 29.7% in a study performed by Das S et al.[193], which was an institution-based case-control study where one hundred and eleven patients of psoriasis and 162 healthy volunteers were screened during the study period of 36 months.

In our present study, 32% of the patients were diabetics. Amongst them, 36.23% were males and 22.58% were females. Presence of Diabetes mellitus was significant in occurrence to metabolic syndrome. ($p<0.05$)

Psoriasis and waist circumference

In a study by Kothiwala SK et al.[189] , central obesity was abnormal in 26.4% of the psoriatic cases versus 11.4% of the controls.

In the present study, we observed a higher abnormal waist circumference in females, 54.84%(n=17) compared to males, 34.78%(n=24). However, the difference was not significant.

Psoriasis and dyslipidemia

Several studies have demonstrated higher lipid levels in psoriasis. Dreiher et al.[194] found a significant increase in lipid levels among psoriasis patients than in controls ($P < 0.001$). Shapiro et al.[195] found that psoriasis was associated hyperlipidemia, but was not associated with an increase in LDL level. Cohen et al.[196] have found that psoriasis is associated with dyslipidemia ($P < 0.015$). In contrast, LDL and total cholesterol were significantly higher among controls with MS than among psoriasis patients with MS ($P = 0.0170$ and 0.0164, respectively) in our study.

In the present study, raised triglyceride levels were found in (n=17) 24.64% of males and was also statistically significant. Lower HDL levels were found in (n=50)72.46% of the males and (n=30)96.77% of the females. There was statistically significant relationship between gender and HDL levels.

Metabolic syndrome and PASI score

Studies performed by Gisondi et al.[5]and Nisa and Qazi[188] found no difference in the prevalence of metabolic syndrome based on PASI score and BSA involvement.

Zindancı et al.[197] and Mebazaa et al.[198] also found that the prevalence of MS was independent of severity of psoriasis (PASI score).

Kim et al.[199]however, found that metabolic syndrome was associated with severe forms of psoriasis ($P = 0.048$). Our study also documented a strong association (P =0.0140) between PASI score and metabolic syndrome.

CONCLUSION

- Chronic plaque psoriasis is an immune-mediated inflammatory skin disease that is strongly associated with the clinical features of the metabolic syndrome (MetS), including abdominal obesity, hypertension, atherogenic dyslipidemia, type 2 diabetes, insulin resistance, and nonalcoholic fatty liver disease.
- The strength of these associations has been repeatedly confirmed by several observational studies.

Based on the finding in this study, the following conclusions were made:

- Prevalence of metabolic syndrome in psoriasis was seen in 32% of the study population.
- There was statistically significant relationship between hypertension, diabetes mellitus, duration of the disease, PASI in relation to occurrence of metabolic syndrome.
- Psoriasis and metabolic syndrome share multiple metabolic risk factors, genetic background, and pathogenic pathways.
- The association between psoriasis and metabolic syndrome has important clinical implications.
- Systemic conventional treatments should be used with caution in psoriatic patients with metabolic syndrome, because they could adversely affect the coexisting metabolic disorders, especially in the case of their chronic use.
- As a conclusion, psoriasis should not be taken just as a skin disease but rather should be regarded as a whole result of complex interplay between

inflammatory mediators and risk factors shared by both immune and metabolic diseases.

- Dermatologists should be aware of comorbid illnesses, assess the patients for accompanying metabolic disturbances and if suspect from metabolic syndrome, refer the patient to appropriate specialists.

SUMMARY

This study "Prevalence of metabolic syndrome among psoriatic patients attending KLE's Dr. Prabhakar kore hospital and medical research centre, Belagavi" was conducted between January 2017- December 2017 by the department of Skin & VD, KLE's Dr. Prabhakar kore hospital and medical research centre, Belagavi.

A total of 100 psoriasis patients attending KLE's Dr. Prabhakar kore hospital and medical research centre were enrolled for the study.

In the present study,

- 69 (69%) were male and 31(31%) were female.
- 68% of study population belonged to age group between 21 to 50 years.
- Only few were diabetic[n=10,10%] and hypertensive[n=7,7%].
- PASI <9 was seen in n=64,64% compared to PASI >9 in n=36,36%. Metabolic syndrome was associated with severe forms of psoriasis (PASI score >=9.1). 47.22%(n=17) of the patients who had PASI >/= 9.1 were positive for metabolic syndrome.
- 41% (41) of the patients had psoriasis since 1-5 years.Metabolic syndrome was present 50%(n=12) of the patients who had psoriasis for more than 11yrs and the least being 9.52%(n=2) in the group 6-10yrs
- Chronic plaque psoriasis was the commonest type (n=75, 75%)
- An abnormal waist circumference was higher in females, 54.84%(n=17) compared to males, 34.78%(n=24). However, the difference was not significant.

- Raised triglyceride levels were found in (n=17) 24.64% of males and was also statistically significant. Lower HDL levels were found in (n=50)72.46% of the males and (n=30)96.77% of the females. There was statistically significant relationship between gender and HDL levels.

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ANNEXURE I – CONSENT FORM

I.D.NO.

Title of the study: Prevalence of metabolic syndrome among psoriatic patients attending KLE's Dr Prabhakar Kore Hospital & Medical Research Center, Belagavi.

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:

Metabolic syndrome may be associated with psoriasis. Hence this study intends to find whether metabolic syndrome is associated with psoriasis, which is measured by recording waist circumference, body mass index and blood pressure and fasting blood sugar and fasting lipid profile. You are being requested to participate in this research because you have been diagnosed to have psoriasis.

Procedure and treatment:

Should you choose to participate, you will be asked to give a detailed history of your disease, undergo a physical examination, recording waist circumference, weight and blood pressure and consent for investigations like estimation of fasting blood sugar levels and fasting lipid profile levels, which requires drawing of 2 ml blood.

Risks and benefits:

While drawing of 2 ml blood for investigations like estimation of fasting blood sugar and fasting lipid levels, you may experience slight pain due to needle prick. However all necessary steps and precautions will be taken to ensure your safety. 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.

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 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.

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:

In case you need further information regarding your rights as a study participant, you may please contact Dr. Ganga.S.Pilli, Chairman of the ethical committee, J N Medical College, Belagavi.

STATEMENT OF CONSENT

I.D.NO: | | | |

I Mr/Ms/Mrs ---------- volunteer and consent to participate in this study. I have read the consent document orit 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:

Signature of the investigator:

Date:

STATEMENT OF ASSENT

I.D.NO: | | | |

I Mr/Ms/Mrs ----------

parent/guardian/ward/ of---------- consent to enrol my daughter to participate in this study. I have read the consent document or it has been read to me in my vernacular language. I give my acceptance on behalf of my daughter for her participation 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:

Participant's parent/guardian name:

Signature or left thumb print of parent/guardian of participant:

Witness name:

Signature of witness:

Signature of the investigator:

Date:

ANNEXURE-II

PROFORMA

Case no- OP No-

Date- IP No-

Name-

Age- **DIAGNOSIS:**

Gender-

Occupation-

Address with phone number-

PRESENTINGCOMPLAINTS:

HISTORY OF PRESENT ILLNESS:

1. Onset Sudden Gradual
2. Progression Progressive Stationary
3. H/o erythema Present Absent
4. H/o sore throat Present Absent
5. H/o stress and strain Present Absent
6. H/o joint involvement Present Absent
7. H/o remissions & exacerbations Present Absent
8. H/o drug intake prior to development of lesions Present Absent
9. H/o fever/ pus filled lesions Present Absent
10. H/o sexual exposure Present Absent
11. H/o diarrhoea Present Absent
12. H/o micturation difficulties Present Absent

13. H/o chest pain/palpitations/breathlessness/ weight gain Present Absent

14. H/o loss of weight /appetite Present Absent

Triggering & modifying factors:

A. Local factors:

I. Trauma Present Absent

II. Operational wound Present Absent

III. Vaccination Present Absent

IV. Insect or animal bite Present Absent

B. Seasonal variation(Exacerabation)

1)Winter:- Present Absent 2)Summer:- Present Absent

C. Pregnancy Present Absent

D. Drugs Present Absent

E. Sunlight Present Absent

F. Alcohol & smoking Present Absent

G. Obesity Present Absent

Initial lesion: Erythema Red lesions Pus filled

Associated factors: Itching Pain Asymptomatic

PAST HISTORY:

K/c/o DM Present Absent

K/c/o HTN Present Absent

FAMILY HISTORY:

H/o similar complaints in the family (First blood relatives) Present Absent

TREATMENT HISTORY:

1)Topical ☐Present ☐ Absent

2)Phototherapy ☐ Present ☐ Absent

3)Systemic ☐ Present ☐ Absent

4)Others ☐Present ☐ Absent

PERSONAL HISTORY:

I. Diet: ☐ Vegetarian ☐Mixed
II. Appetite: ☐ Normal ☐ Disturbed
III. Sleep: ☐ Adequate ☐Stress
IV. Bowel/Bladder: ☐Normal ☐ Altered
V. Alcohol: ☐ Present ☐ Absent
VI. Smoking: ☐ Present ☐ Absent
VII. Stress: ☐Present ☐ Absent

GENERAL PHYSICAL EXAMINATION:

Built: ☐ Poor ☐ Moderate ☐ Good

Vitals: 1)Pulse- bpm2)BP- / mm of Hg 3)Temperature- F

Height- cm Weight- kg BMI- kg/sq m Waist circumference- inches

Pallor Icterus Clubbing Cyanosis Lymphadenopathy Oedema

MUCOCUTANEOUS EXAMINATION:

A)**Types of lesions**-

1)Papules ☐ Present ☐ Absent

2)Plaque☐ Present☐ Absent

3)Pustules ☐ Present ☐ Absent

4)Erythema ☐ Present☐ Absent

B)**Distribution**-

☐ Symmetrical ☐ Asymmetrical ☐ Localised ☐ Genaralised

C)**Sites of lesions**:

Scalp ☐ Present ☐ Absent

Face ☐ Present ☐ Absent

Neck ☐ Present ☐ Absent

Back ☐ Present ☐ Absent

Trunk ☐ Present ☐ Absent

Elbows ☐ Present ☐ Absent

Extensor aspect of extremities ☐ Present ☐ Absent

Flexors of upper limbs & lower limbs ☐ Present ☐ Absent

Palms ☐ Present ☐ Absent

Knees ☐ Present ☐ Absent

Axillae ☐ Present ☐ Absent

External genitalia ☐ Present ☐ Absent

Gluteal region ☐ Present ☐ Absent

Soles ☐ Present ☐ Absent

D)**Size of lesion**: ☐ Small(0.5-1cm) ☐ Large(2-5cm) ☐ Larger(more than 10cm)

E)**Types of scaling**:

☐ Firmly adherent ☐ Loosely adherent ☐ Mica like ☐ Limpet like ☐ Oyster shell like

F)**Auspitz sign** ☐ Present ☐ Absent

I)**Koebner's phenomenon** ☐ Present ☐ Absent

J)**Nail lesions:**

1)Pitting ☐ Present ☐ Absent

2)Subungual hyperkeratosis ☐Present ☐ Absent

3)Onycholysis ☐ Present ☐Absent

4)Splinter haemorrhages ☐ Present ☐ Absent

5)Oil drop ☐ Present ☐ Absent

6)Beau's lines ☐ Present ☐ Absent

7)Yellow discolouration ☐Present ☐ Absent

K)**Joint involvement**: ☐Present ☐ Absent

If present, involved joint(s)-

☐ Distal interphalangeal ☐ Proximal interphalangeal ☐ Sacroiliac
☐ Metacarpophalangeal joint

☐ Knee joint ☐ elbow joint ☐ wrist joint

L**)Mucosal Examination:**

a)Oral lesion ☐ Present ☐ Absent

b)Genital lesion ☐ Present ☐ Absent

SYSTEMIC EXAMINATION

Respiratory system- 1)Normal

2)Abnormal; specify is so

Cardiovascular system- 1)Normal

2)Abnormal; specify is so

Per abdomen- 1)Normal

2)Abnormal; specify is so

Central nervous system- 1)Normal

2)Abnormal; specify is so

INVESTIGATIONS

FBS- mg/dl

Lipid Profile- TC- mg/dl

TG- mg/dl

HDL- mg/dl

LDL- mg/dl

VLDL- mg/dl

PASI SCORE-

Scaling

Induration

Desquamation

Signature

Guide's signature

ANNEXURE III – PHOTOGRAPHS

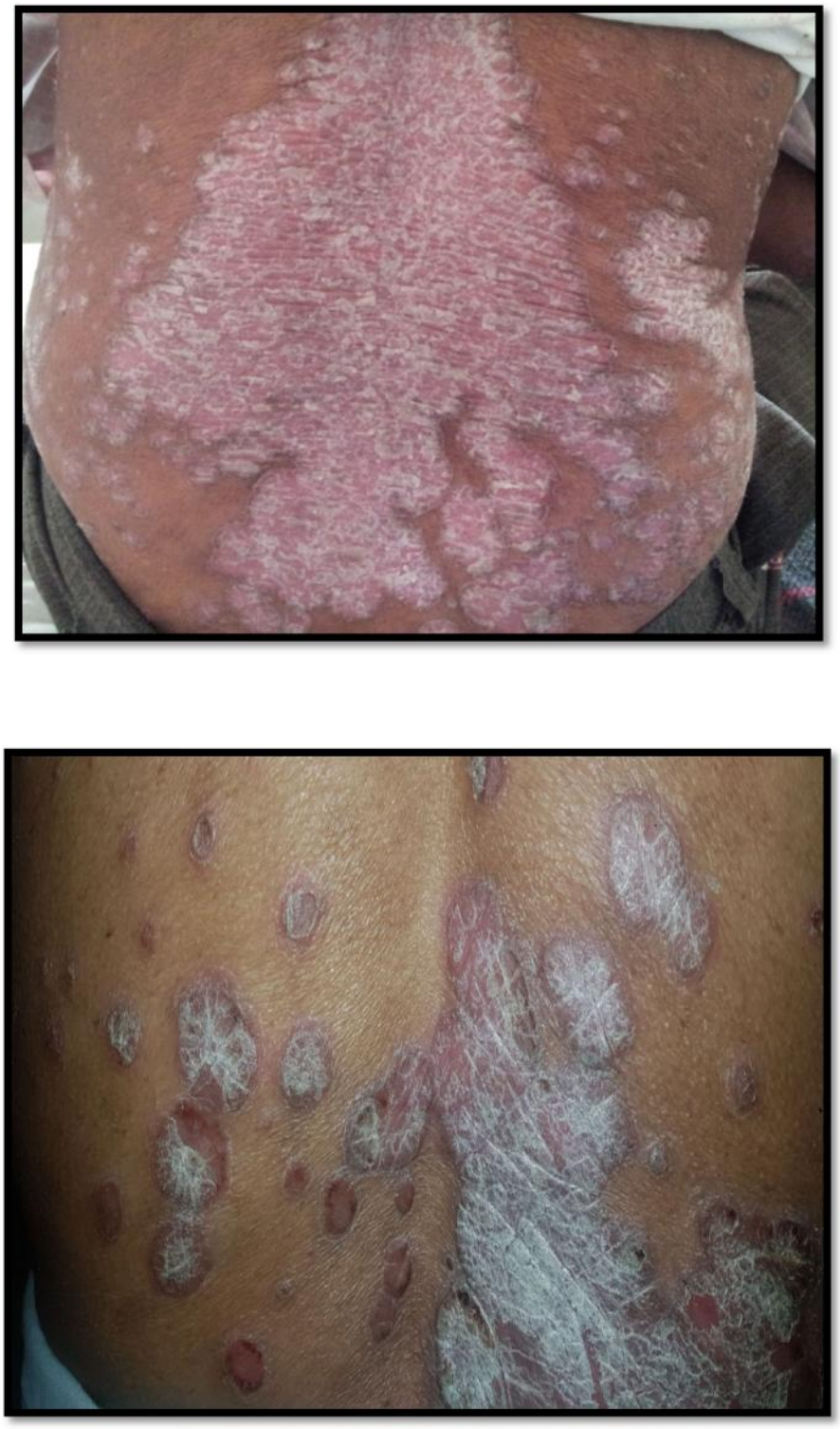

Figure 16-Chronic plaque psoriasis

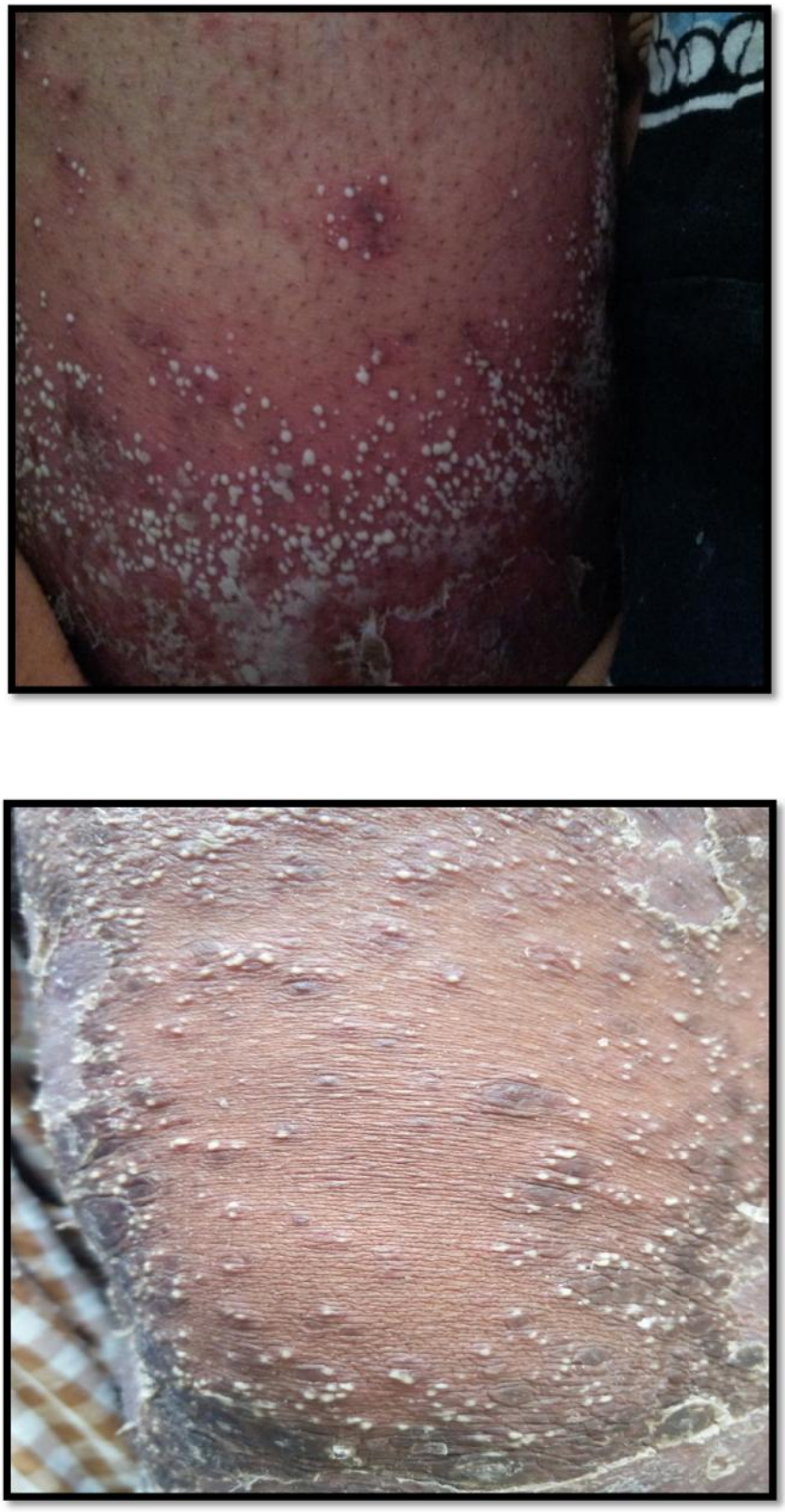

Figure 17-Pustular psoriasis

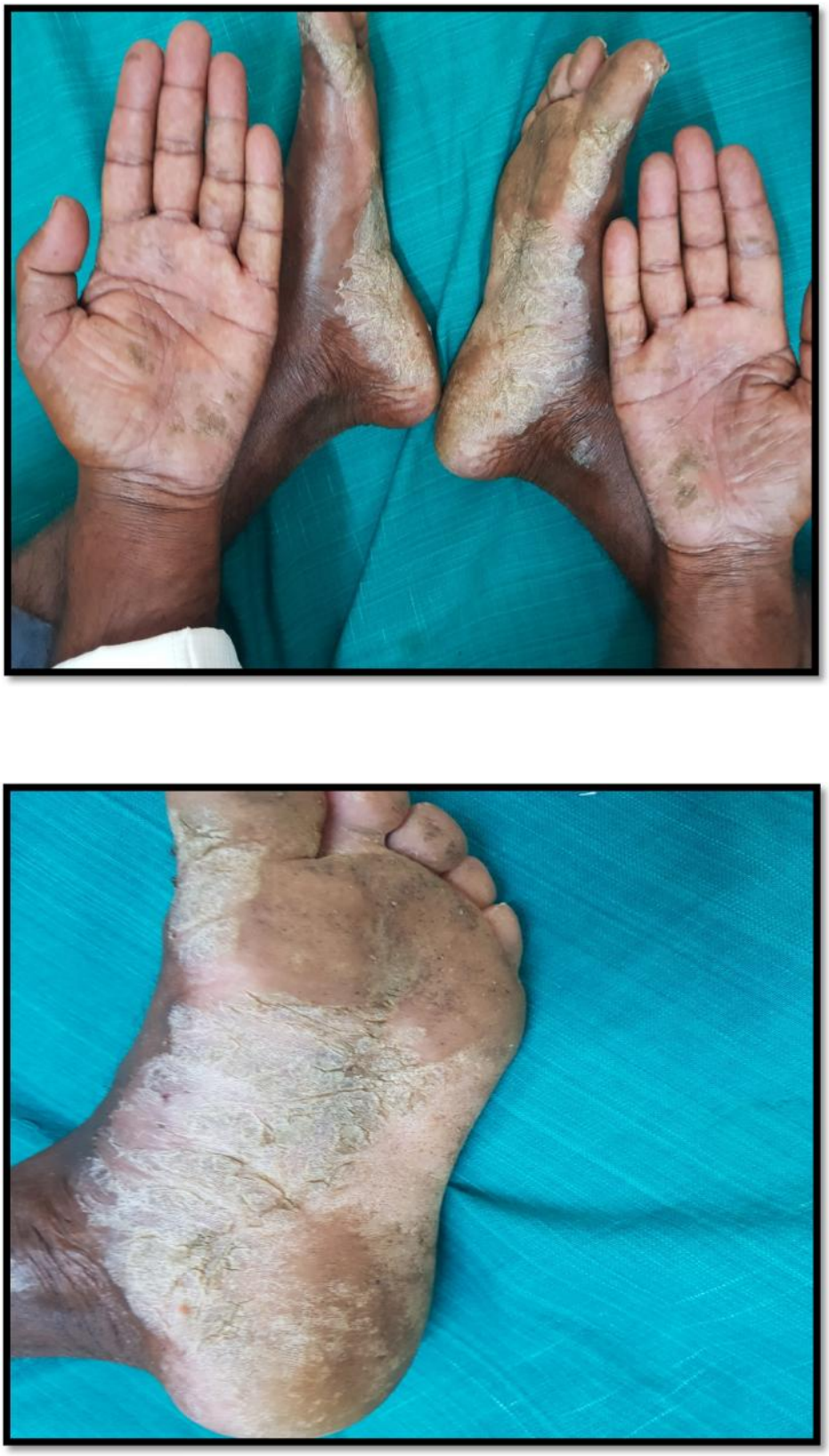

Figure 18- Palmoplantar keratoderma secondary to psoriasis

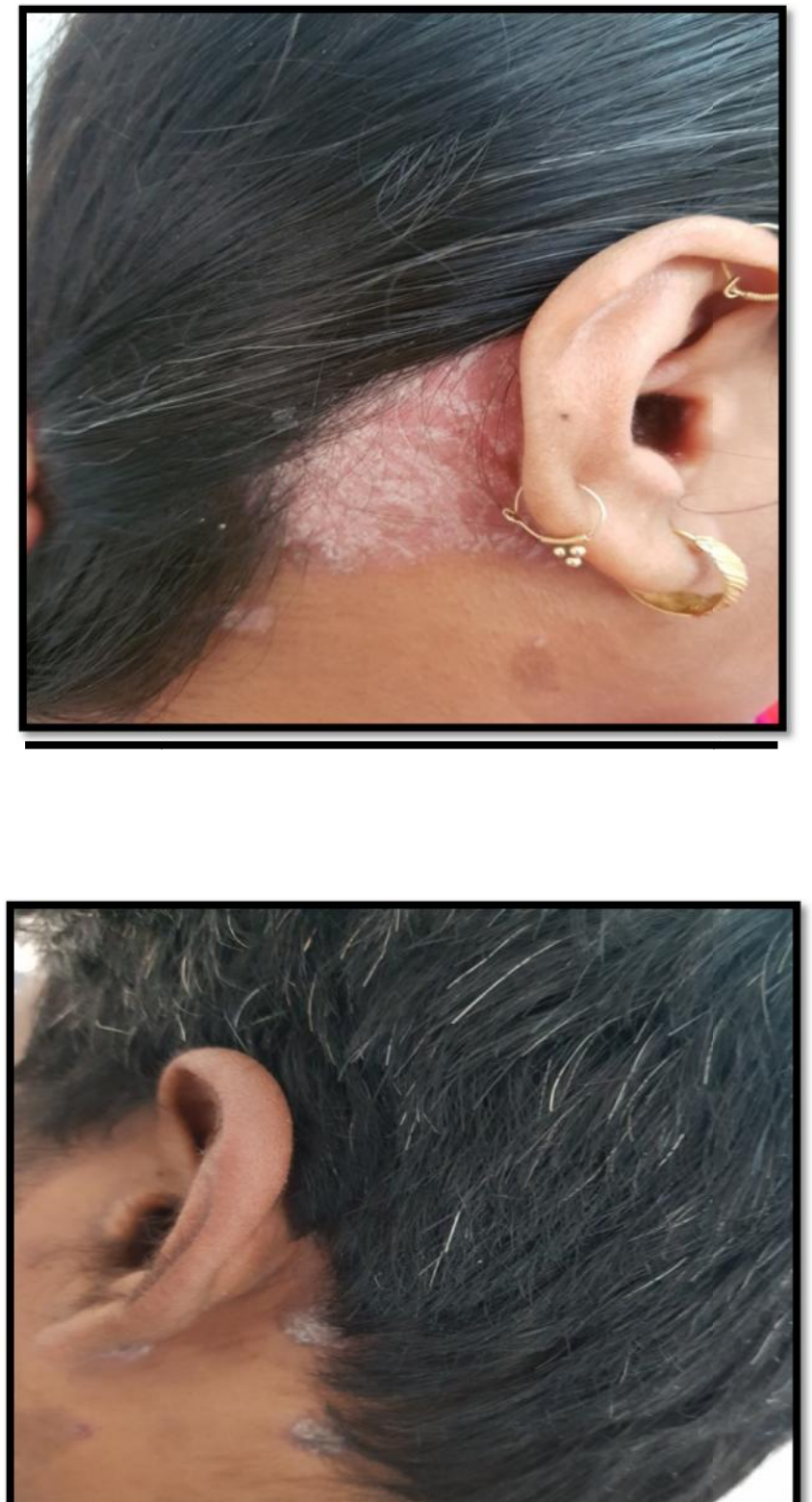

Figure 19 - Scalp psoriasis

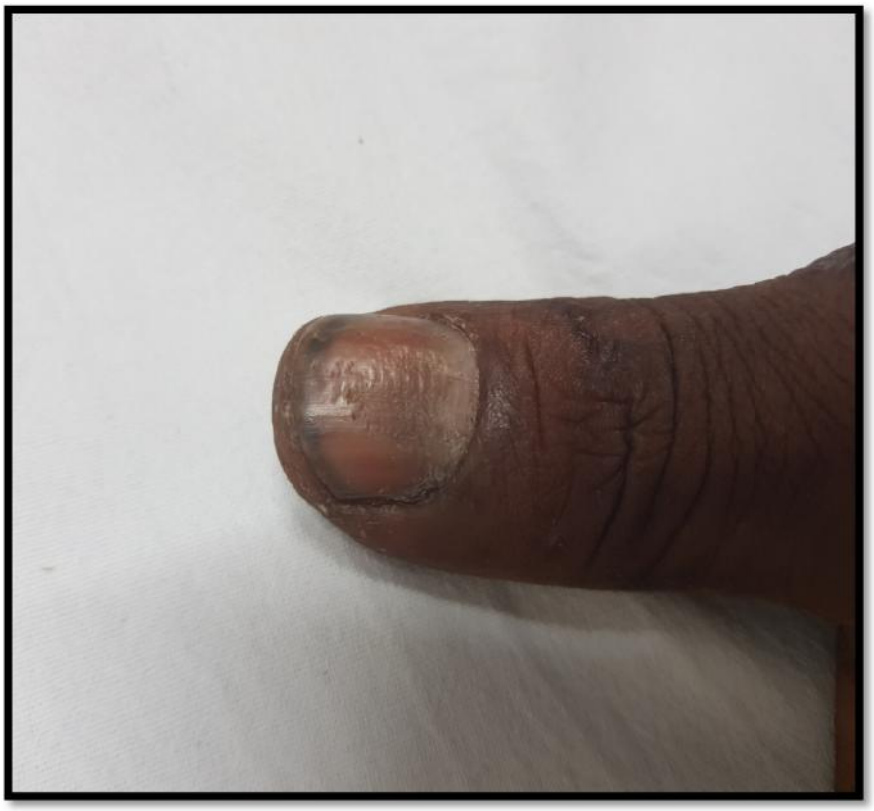

Figure 20 - Nail pits

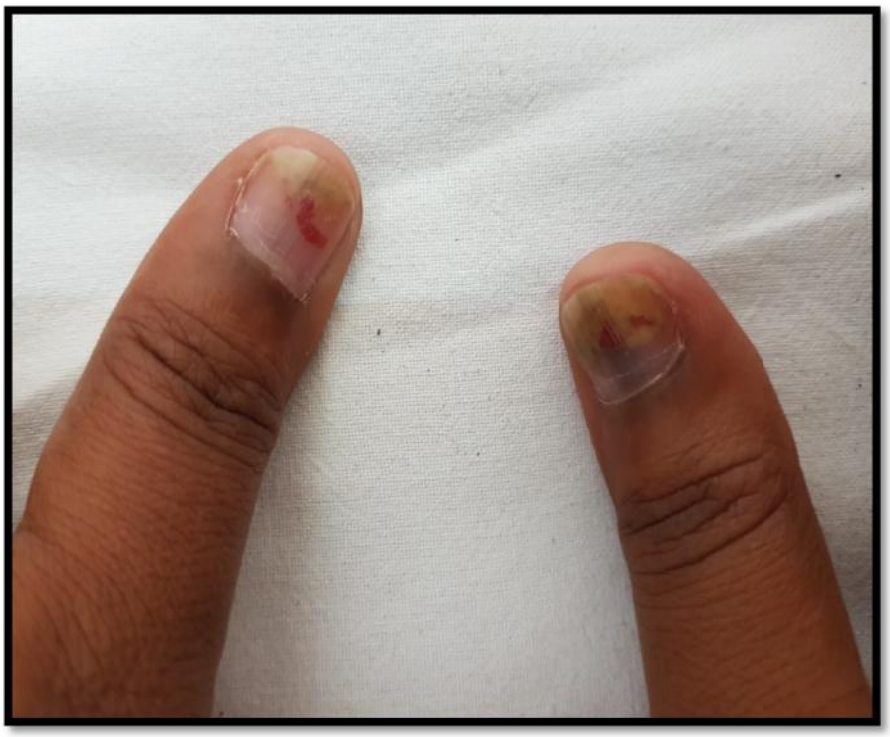

Figure 21- Onycholysis

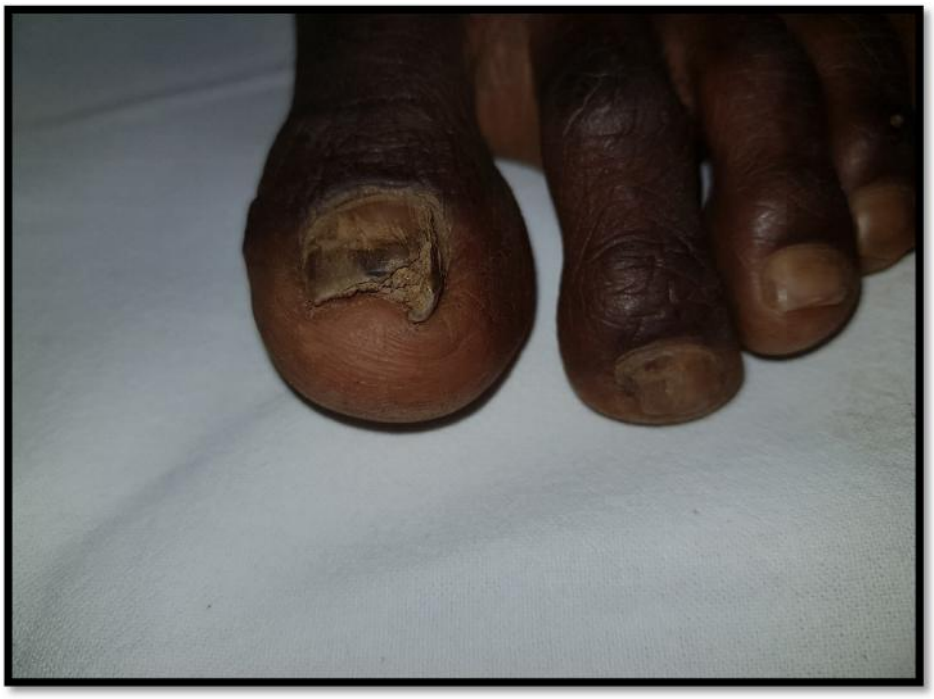

Figure 22 - Subungual hyperkeratosis

ANNEXURES IV - MASTER CHART

Serial no.	Age/Sex	Duration of psoriasis(yr)	HTN	DM	PASI	TYPE OF PSORIASIS	WC(inc)	BP(mm of Hg)	FBS(mg/dl)	TG(mg/dl)	HDL(mg/dl)	Metabolic syndrome
1	51yrs/M	20	-	-	10.8	CPP	36	140/90	119	86	27	+
2	42yrs/M	4	-	+(3yrs)	12.2	CPP	37	130/90	116	156	27	+
3	60yrs/M	3	-	+(1yrs)	3.2	Pustular	34	140/90	85	97	25	
4	60yrs/M	10	-	+(2yrs)	5.8	CPP	36	130/80	161	88	28	
5	33yrs/M	0.2	-	-	33	Erythroderma	41	136/90	100	111	37	+
6	43yrs/M	5	-	-	10.2	Erythroderma	37	130/70	94	99	33	
7	31yrs/M	1.5	-	-	9	CPP	40.5	138/90	125	145	44	+
8	50yrs/F	4	-	-	12.5	CPP	38	130/80	110	128	38	+
9	27yrs/F	0.6	-	-	8.4	CPP	40	120/70	84	90	30	
10	40yrs/M	10	+(2yrs)	-	11.2	CPP	38	140/90	100	353	28	+
11	42yrs/M	4	-	-	10.2	CPP	41.2	120/80	101	83	47	
12	15yrs/F	0.2	-	-	7.8	Guttate	28	120/80	86	97	54	
13	20yrs/F	10	-	-	6.2	CPP	30	110/70	74	52	41	
14	16yrs/M	1	-	-	5.3	CPP	32	118/72	90	46	28	
15	55yrs/M	1	-	-	13.2	CPP	40.6	160/100	108	163	30	+
16	22yrs/M	4	-	-	8.3	CPP	41	136/88	91	66	41	
17	55yrs/M	10	-	-	4.1	CPP	38	120/70	88	137	40	
18	58yrs/M	0.6	-	-	6.4	CPP	42	140/90	121	132	45	+
19	57yrs/M	18	+(6yrs)	-	8.8	CPP	41	136/86	95	142	31	+
20	51yrs/F	4	-	-	8.2	CPP	35	120/70	92	140	34	
21	43yrs/F	0.8	-	-	10.2	CPP	37	140/90	109	54	44	+
22	35yrs/M	1	-	-	4.3	PPK	39	120/70	92	163	35	
23	59yrs/M	3	+(2yrs)	+(1 1/2yrs)	9.6	CPP	38	140/90	121	109	27	+
24	30yrs/F	5	-	-	7.8	Pustular	36	140/90	99	137	30	+
25	36yrs/M	15	-	-	4.2	CPP	42	130/80	100	167	41	
26	48yrs/M	12	-	-	15	CPP	39	130/80	113	160	33	+
27	52yrs/M	10	-	-	11.4	CPP	38	110/70	90	84	49	
28	38yrs/F	22	-	-	33.8	CPP	34	110/80	137	134	32	
29	60yrs/M	15	-	-	15.6	CPP	36	160/100	110	165	32	+
30	52yrs/M	3	-	-	10.6	CPP	41	120/70	92	120	41	
31	31yrs/M	2	-	-	26	CPP	38	150/100	89	136	45	
32	33yrs/F	1	-	-	4.3	CPP,PA	35	130/80	90	113	29	
33	53yrs/M	10	-	-	13.2	CPP	46	136/88	102	147	38	+
34	59yrs/M	4	-	-	29	Erythroderma	44	110/70	121	89	17	+
35	39yrs/M	0.6	-	-	1.6	PPK	37	130/88	80	209	44	
36	33yrs/F	1	-	-	2.2	CPP	33	122/72	92	150	40	

Serial no.	Age/Sex	Duration of psoriasis(yr)	HTN	DM	PASI	TYPE OF PSORIASIS	WC(inc)	BP(mm of Hg)	FBS(mg/dl)	TG(mg/dl)	HDL(mg/dl)	Metabolic syndrome
37	39yrs/M	3	-	-	6.2	CPP	41	110/70	120	142	44	
38	60yrs/F	1	-	-	7.2	CPP	34	126/82	78	74	42	
39	46yrs/F	10	-	-	4.1	CPP	42	130/80	82	100	38	
40	41yrs/M	20	-	-	5.2	CPP	41	140/90	80	128	37	+
41	38yrs/M	20	-	-	3.9	CPP	42	138/86	100	110	36	+
42	37yrs/F	0.4	-	-	2.2	PPK	35	120/70	88	140	35	
43	35yrs/M	0.3	-	+(2mth)	3.2	Guttate	43	130/80	110	186	31	+
44	38yrs/F	20	-	-	13.4	CPP	38	130/90	92	81	34	
45	60yrs/M	12	-	-	11.8	CPP	38	130/90	100	140	42	
46	29yrs/F	15	-	-	3.6	CPP	36	120/70	82	130	41	
47	19yrs/M	4	-	-	2.8	CPP	38	130/80	110	158	36	+
48	32yrs/M	10	-	-	6.4	CPP	41	120/70	92	59	32	
49	26yrs/F	10	-	-	5.2	CPP	36	130/70	86	110	43	
50	33yrs/M	11	-	-	5.4	CPP	38	120/90	92	120	45	
51	52yrs/M	10	-	-	3.4	PPK	37	120/70	78	110	48	
52	52yrs/M	10	-	-	4.2	CPP	36	130/80	90	120	44	
53	60yrs/M	18	-	-	6.4	CPP	36	130/80	104	142	51	
54	16yrs/F	5	-	-	10.2	CPP	30	100/60	90	93	15	
55	26yrs/F	10	-	-	5.2	CPP	38	120/70	92	110	36	
56	40yrs/M	3	-	-	14.9	CPP,PA	41	140/90	92	102	31	+
57	60yrs/M	30	-	-	7.2	CPP	38	130/80	102	120	34	
58	34yrs/M	10	-	-	6.2	CPP	37	134/80	92	139	26	
59	35yrs/F	8	-	-	6.2	CPP	37	120/70	82	140	34	
60	40yrs/M	15	-	-	1.2	CPP	42	130/80	94	160	36	+
61	44yrs/M	5	+(3yrs)	+(2yrs)	1.3	Scalp	43	140/86	120	130	35	+
62	38yrs/M	6	-	-	12.2	Erythroderma	37	130/70	87	134	35	
63	34yrs/F	1	-	-	6.2	CPP	34	110/70	90	120	34	
64	30yrs/F	2	-	-	3.8	CPP	38	120/70	94	132	38	
65	57yrs/M	5	-	-	2.1	PPK	42	120/70	90	162	44	+
66	41yrs/M	1	-	-	2	Scalp	37	130/70	110	110	40	
67	35yrs/F	0.6	-	-	1.4	Scalp	34	120/70	84	125	30	
68	18yrs/M	4	-	-	5.2	CPP	32	120/70	92	120	38	
69	45yrs/F	15	-	-	6.2	Pustular	38	130/70	109	140	39	+
70	50yrs/M	0.6	-	-	7.6	CPP	44	130/70	94	142	36	
71	44yrs/M	8	-	-	3.4	CPP	37	120/70	90	113	42	
72	32yrs/F	8	-	-	4.2	CPP	34	130/80	82	120	38	
73	30yrs/M	0.3	-	-	4.6	CPP	36	120/70	84	120	34	
74	50yrs/M	2	-	-	10.1	CPP	41	100/70	167	97	32	+
75	32yrs/M	4	-	-	5.4	PPK	35	120/70	84	126	36	

Serial no.	Age/Sex	Duration of psoriasis(yr)	HTN	DM	PASI	TYPE OF PSORIASIS	WC(inc)	BP(mm of Hg)	FBS(mg/dl)	TG(mg/dl)	HDL(mg/dl)	Metabolic syndrome
76	42yrs/F	5	-	-	4.2	CPP	38	130/70	92	134	27	
77	55yrs/M	10	-	-	5.2	CPP	34	110/80	78	143	38	
78	35yrs/F	10	-	-	18.6	Pustular, PA	37	130/70	112	96	16	
79	47yrs/M	15	-	-	12.2	CPP	32	120/70	134	156	32	
80	40yrs/M	15	-	-	15.2	CPP	36	140/80	116	256	25	+
81	35yrs/F	10	-	-	20.3	Pustular, PA	38	130/70	98	143	30	
82	30yrs/M	0.6	-	-	10.4	CPP	34	110/70	121	163	23	+
83	21yrs/M	0.3	-	-	4.3	CPP	41	130/70	82	172	35	
84	50yrs/M	20	-	-	16.8	CPP	34	110/70	94	138	34	
85	47yrs/M	1	-	-	2.6	PPK	34	130/70	83	120	32	
86	60yrs/M	20	+(7yrs)	+(6yrs)	17.9	Erythroderma	36	120/80	110	116	30	
87	46yrs/M	4	-	-	9.7	CPP	37	130/80	87	132	35	
88	25yrs/F	2	-	-	4.2	CPP	35	120/70	84	124	40	
89	46yrs/M	3	-	-	7.2	CPP	38	140/90	92	192	26	+
90	30yrs/M	4	-	-	6.3	CPP	34	130/70	88	141	36	
91	40yrs/M	20	-	+(4yrs)	3.2	CPP	36	110/70	92	120	37	
92	50yrs/F	20	+(5yrs)	-	7.8	CPP	35	132/90	110	119	26	+
93	52yrs/F	18	-	+(7yrs)	8.3	CPP	38	120/84	125	127	24	+
94	59yrs/M	22	+(1yr)	+(20yrs)	26.8	CPP,PA	34	136/90	225	131	34	+
95	40yrs/M	6	-	-	33.6	CPP	42	130/82	82	140	36	
96	30yrs/M	3	-	-	39.8	CPP	35	110/80	93	132	28	
97	17yrs/M	2	-	-	8.7	CPP	32	126/78	86	128	37	
98	46yrs/M	4	-	-	9.7	CPP	38	130/82	78	134	32	
99	25yrs/F	2	-	-	4.5	CPP	37	128/80	84	125	28	
100	30yrs/M	0.3	-	-	6.8	CPP	35	130/80	74	138	32	

ANNEXURE-V

KEY TO MASTER CHART

M	-	Males
F	-	Females
DM	-	Diabetes Mellitus
HTN	-	Hypertension
PASI	-	Psoriasis area severity index
CPP	-	Chronic plaque psoriasis
PPK	-	Palmoplantar keratoderma
PA	-	Psoriatic arthritis
WC	-	Waist circumference
FBS	-	Fasting blood sugar
BP	-	Blood pressure
TG	-	Triglyceride
HDL	-	High density lipoprotein