"ONE YEAR HOSPITAL BASED OBSERVATIONAL STUDY OF SERUM CALCIUM LEVEL IN PSORIASIS PATIENTS ATTENDING KLE DR PRABHAKAR KORE HOSPITAL &MEDICAL RESEARCH CENTRE, BELAGAVI"

BY

REG NO.BT0117002

Dissertation

Submitted to the
KAHER, Belagavi, Karnataka
In partial fulfillment
of the requirements for the degree of

M.D.

in

DEPARTMENT OF DERMATOLOGY,

VENEREOLOGY AND LEPROSY

DEPARTMENT OF DERMATOLOGY,
VENEREOLOGY AND LEPROSY,
JAWAHARLAL NEHRU MEDICAL COLLEGE,
BELAGAVI, KARNATAKA.

APRIL – 2020

KLE ACADEMY OF HIGHER EDUCATION AND RESEARCH, BELAGAVI, KARNATAKA

Endorsement

This is to certify that the dissertation entitled **"ONE YEAR HOSPITAL BASED OBSERVATIONAL STUDY OF SERUM CALCIUM LEVEL IN PSORIASIS PATIENTS ATTENDING KLE DR PRABHAKAR KORE HOSPITAL &MEDICAL RESEARCH CENTRE, BELAGAVI"** is a bonafide research work done by **REG NO.BT0117002.**

Dr.B.S.MANJUNATHSWAMY$_{MD,DVD}$
Professor and Head,
Department of Dermatology,
Venereology and Leprosy
J. N. Medical College,
Nehru Nagar, Belagavi – 10

Date:
Place: Belagavi

Dr. N. S. Mahantshetti $_{MD}$
Principal,
J. N. Medical College,
Nehru Nagar, Belagavi – 10

Date:
Place: Belagavi

PLAGIARISM ACCEPTED LETTER

JAWAHARLAL NEHRU MEDICAL COLLEGE
(A constituent unit of KLE Academy of Higher Education & Research Deemed-to-be University)
Accredited 'A' Grade by NAAC (2nd Cycle) Placed in Category "A" by MHRD (GoI)
Nehru Nagar, Belagavi-590 010, Karnataka-India

Website : http/www.jnmc.edu
E-Mail : Principal@jnmc.edu
Office :+91-(0)831 2471350
FAX :+91 (0)831-2470759

Ref. No. :MDC/PG/2254 Date :20-9-2019

To,

REG NO.BT0117002

Sub: Acceptance Letter

Sir/Madam,

The softcopy of thesis entitled ""ONE YEAR HOSPITAL BASED OBSERVATIONAL STUDY OF SERUM CALCIUM LEVEL IN PSORIASIS PATIENTS ATTENDING KLE DR. PRABHAKAR KORE HOSPITAL & MRC, BELAGAVI"" has been submitted for Anti-Plagiarism check through Turnitin software. The scan has been carried out and the scanned output reveals a match percentage of 7% (Seven percentage) which is within the acceptable limits of 10% as per the guidelines given by UGC.

Thanking you,

Yours sincerely,

BNDCM

Coordinator
Department of Skin & VD,
J. N. M. C. Belagavi.

Dr. Bhavana R. Doshi
Consultant Dermatologist
MAH 20050000457KTK
KLES Dr. Prabhakar Kore Hospital &
MRC, Belagavi.

Chairman,
Antiplagiarism Committee

KAHER BELAGAVI J.N. MEDICAL COLLEGE

LIST OF ABBREVIATIONS USED

OPD	-	Out patient department
HLA	-	Human leukocyte antigen
IL	-	Interleukin
Th cell	-	T- helper cell
APC	-	Antigen presenting cell
TNF-α	-	Tumor necrosis factor
TGF-β	-	Transforming growth factor beta
ICAM-1	-	Intercellular adhesion molecule
VEGF	-	Vascular endothelial growth factor
IFN-ϒ	-	Interferon gamma
NK T CELL	-	Natural killer T cell
GM-CSF	-	Granulocyte monocyte colony stimulating factor
CXCL	-	Chemokine ligand
LCs	-	Langerhan cells
DCs	-	Dendritic cells
PDCs	-	Palsmacytoid dendritic cells
INOS	-	Inducible nitric oxide synthase
URTI	-	Upper respiratory tract infection
ACE	-	Angiotensin converting enzyme
PAMP	-	Pathogen associated molecular pattern
DAMP	-	Danger associated molecular pattern
NOD	-	Nucleotide binding oligomerization domain like
AMP	-	Anti microbial peptide
TCR	-	T cell receptor

UV	-	Ultra violet
ESR	-	Erythrocyte sedimentation rate
PASI	-	Psoriasis area severity index
Eh, Eu, El	-	Erythema of head, upper limb, lower limb
Lh, Lu, Ll	-	Induration of head, upper limb, lower limb
Dh, Du, Dl	-	Desquamation of head, upper limb, lower limb
GI	-	Gastrointestinal
ICU	-	Intensive care unit
SD	-	Standard deviation
Chr plq	-	Chronic plaque
Ery	-	Erythrodermic psoriasis
GUTT	-	Guttate psoriasis
PPK	-	Palmoplantar keratoderma secondary to Psoriasis
PUST	-	Pustular psoriasis

ABSTRACT

Introduction:

Psoriasis is a papulosquamous disease of the skin, which has genetic and environmental risk factors. The disease duration varies with remissions and exacerbations. Keratinocytes proliferation and maturation is regulated by intracellular calcium. Decreased serum calcium leads to exacerbation of the psoriatic lesions as shown in many studies. On basis of this, vitamin D and calcium are recommended. Hence this study was undertaken to observe serum calcium level in patients of psoriasis.

Methodology:

This study was conducted within duration of twelve months (January 2018 to December 2018) in the Department of Dermatology, Venereology and Leprosy in tertiary care hospital. The study was a hospital-based cross sectional study involving 60 patients. A written consent form was obtained. Subjects of both sex of age group 15-65 years with psoriasis were included. Venous sample was taken at enrollment visit, serum total calcium, serum albumin were measured. Statistical analysis of the data was done using chi square and student “t” test and using correlation method. An ethical committee clearance was obtained prior to the start of the study.

Results:

In our study majority were males 46(76.67%) and females were 14(23.37%). chronic plaque psoriasis was commonest type (85%).Hypocalcemia was seen in 14(23.33%) patients, commonest in severe psoriasis. Duration of disease and PASI were statistically significant in relation to hypocalcemia.

Conclusion:

Estimation of serum calcium should be considered in all patients with severe psoriasis and longer duration of disease, as hypocalcemia may be risk factor for severe psoriasis.

Keywords- Psoriasis, PASI, hypocalcemia, disease duration

CONTENTS

LIST OF TABLES

LIST OF CHARTS

LIST OF FIGURES

INTRODUCTION

Psoriasis is a papulosquamous disease of the skin, which has genetic and environmental risk factors. The disease duration varies with remissions and exacerbatons.[1]

Psoriasis incidence is 2-3% all over world and presents with erythematous plaques in symmetrical distribution with white scales.[2]

Keratinocytes proliferation and maturation is regulated by intracellular calcium[3].

Psoriasis cases with severity show alteration in systemic calcium metabolism.

Patients with pustular psoriasis of von Zumbush have shown mild hypocalcemia. [4]

Various studies have reported psoriasis exacerbation in primary and surgical hypoparathyroidism patients[5].

Decreased serum calcium leads to exacerbation of the psoriatic lesions shown in many studies. On basis of this, vitamin D and calcium are recommended[6].

Topical vitamin-D analogue, calcipotriene application improved lesions of psoriatic patients[6].

OBJECTIVES

To measure serum calcium level in psoriasis patients attending dermatology department in tertiary care hospital, Belgavi.

REVIEW OF LITERATURE

HISTORY

The word 'lepra' was used for various cutaneous disorders namely psoriasis, vitiligo, eczema and alopecia areata[7].The Aurelius Cornelius Celsus roman sage was the first who described psoriasis.[8] Galen was the first person to coin the word psoriasis and Robert Willan (1808)[9] described a term, Lepra vulgaris, which was psoriasis variant. In 1841, Hebra distinguished clinical features of psoriasis from those of leprosy.

EPIDEMIOLOGY

The psoriasis prevalence all over world is 2-3%.[10] Psoriasis prevalence in India is 0.44-2.8 percent; males are more affected than females, seen in third to fourth decade.[11]Psoriasis occurs commonest at two age groups, the first one among 15–20 years of age and a second one among 55–60 years.

There are two types of psoriasis on basis of HLA typing. Type I psoriasis has HLA CW6 association, shows positive family history. Seen in patients below 40 years of age, it is commonest (75%) type. Severe and recurrent type, has poor prognosis. Type II psoriasis which is sporadic type, has no HLA association. Occurs later in life, has good prognosis[12].

Psoriasis occurs before arthritis in 70% of patients; in other 15%, arthritis occurs one year prior to the onset of psoriasis and in another 15% of cases are seen below one year of their duration [13].

ETIOPATHOGENESIS

Psoriasis is a multifactorial skin disease, occurs secondary to genetic and environmental factors. Psoriasis results from combined factors such as genetic susceptibility, skin barrier defect and alteration in innate and adaptive immunity.[14]

ROLE OF GENETIC FACTORS

Chromosome 6p has PSORS1 which is involved in inheritance of the psoriasis. HLA-C06* gene situated on PSORS1 region is responsible for antigenic presentation, which shows the role of adaptive immunity in psoriasis.[15] The chromosome 6p locus has corneodesmin, which plays role in expression of mature keratinocytes and is considered as risk factor for psoriasis development.[16, 17, 18]

IL-23 (IL-23A, IL-12B and IL-23R) pathway, Th2 pathway (IL-4 and IL-13) and nuclear factor (NF) kappa beta signaling involved in psoriasis are present in different genes [19, 20].

Psoriasin gene situated in the epidermal differentiation complex attributable to psoriasis pathogenesis. The protein kinase C pathway leads to psoriasin expression. The psoriasin downregualation clearly shows that psoriasin acts as mediator in functioning of involucrin, desmoglein 1, transglutaminase 1 and CD24 in differentiation process.[21]The above points clearly indicate psoriasin gene over expression contributes to the abnormality in differentiation process of the psoriatic epidermis.[21]

ROLE OF ADAPTIVE AND INNATE IMMUNITY

T CELL FUNCTION

T cells are involved in initiation and maintenance of the psoriatic lesions. Helper T cells are CD 4, suppressor cells are CD 8 positive. T cells require antigen presenting cells (APCs) for processing peptide fragments on the APC surface.[22]

T CELL ACTIVATION

T cells activation occurs in three steps: attachment (I); antigen specific activation (signal 1) (II); specific Cell to cell interaction (signal2) (III). T lymphocytes are attached to adhesion molecules of

antigen presenting cell [22].

Antigen specific activation

T cell–antigen presenting cell interaction occurs through their surface adhesion molecules. The antigen stimulation leads to naïve T cell transformation into an activated T cell that may be converted into a memory T cell later. Naive T-cells get converted into any of the inflammatory cells Th1, Th2, Th17 or T regulatory cells depending on TNF-α, TGF-β and IL-6 [23]. The activated T cells enter the circulation and migrate to inflammatory site in skin and produce Th1-Th2-Th17 imbalance [24].

Cytokine Mediators

Psoriasis pathogenesis involves a network of various cytokines like TNF-α, IFN-γ, IL-1, IL-2, IL-8, IL-12, IL-17, IL-22, and IL-23 [25].

TNF-α:

- IL-8, ICAM-1, TGF-α, β-defensins production through keratinocytes stimulation.
- VEGF secretion through endothelial cell stimulation.
- Causes keratinocyte proliferation, increased cytokine secretion by macrophages[25,26,27]

IFN-γ:

- ICAM-1stimulation ,located on keratinocytes and endothelial cells.
- Causes mobilisation of the of T lymphocytes into lesional epidermis
- Enhances APC activity, TNF-α release by phagocytes and stimulation of TNF-α receptors [27, 28, 29.]
- IL-2 regulates the TNF-α and IFN-γ production. [30]
- TNF-α stimulates keratinocytes to produce IL-6 ,which Mediates T-cell activation, proliferation of keratinocyte and acute phase inflammation. [30]
- IL-8 is increased secondary to stimulation of TNF-α. [30]
- Keratinocytes produce β-defensins, anti microbial peptides and IL-8 under the influence of IL-17. [30]
- Keratinocytes, Macrophages and mast cells, produce VEGF which leads to dermal angiogenesis.[30]

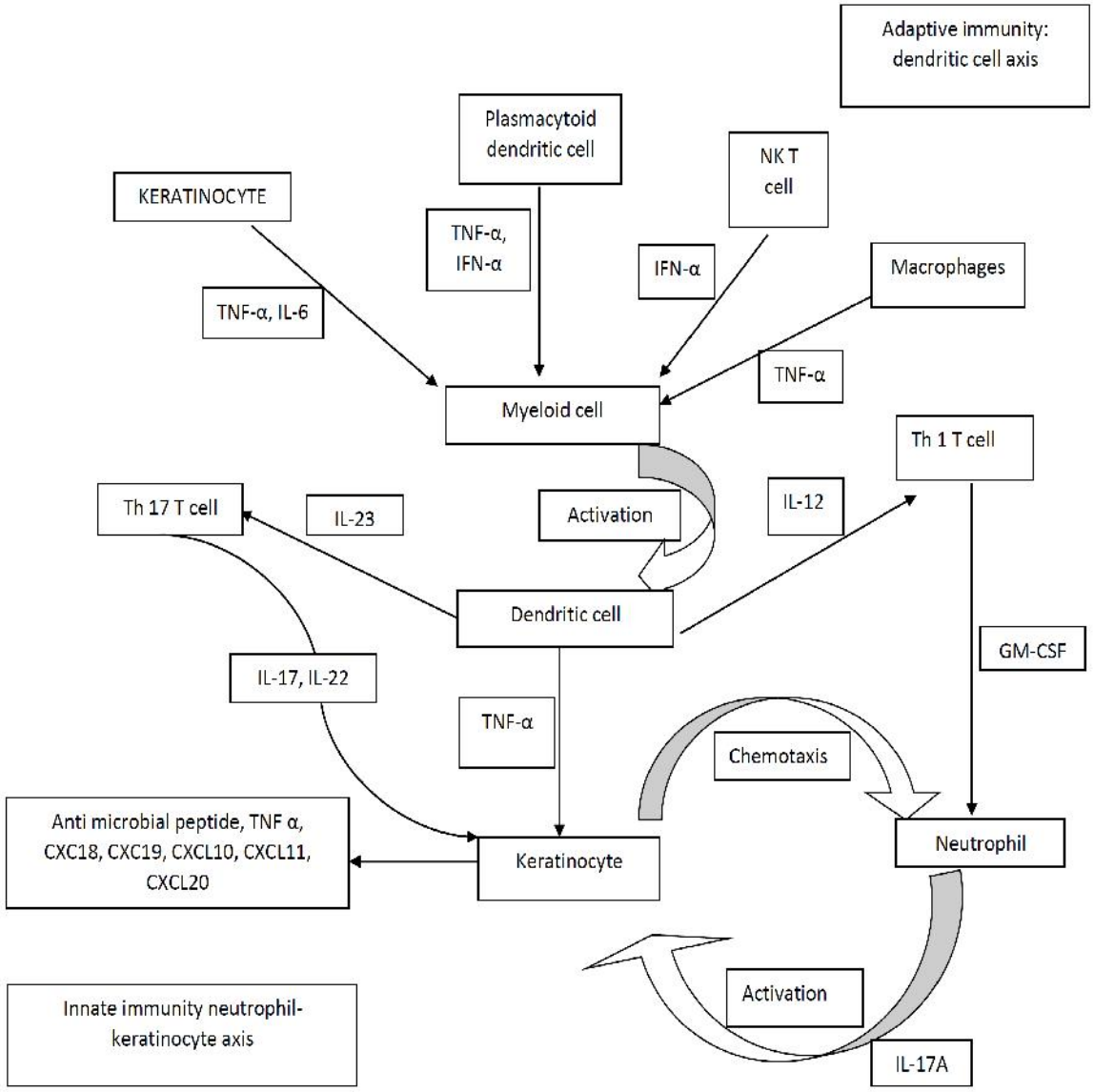


Figure 1- Cytokine mediators

T Cells:

The T cells consist of CD4+ and CD8+ subtypes, more commonly of the memory phenotype (CD45RO+). These cells show the cutaneous lymphocyte antigen (CLA) expression, a ligand for E-selectin present on skin capillaries. It provides a pathway for cells to skin.

CD8+ T cells are more in the epidermis, whereas CD4+ T cells are more in the upper dermis.[31]

The cytokine of psoriatic lesions contain interferon (IFN)-ϒ. This suggests CD4 cell predominance towards Th-1 and CD8+ cells towards T cytotoxic1 (Tc1).

Two other subtypes of CD4+ T cells under stimulation of IL-23 are characterized by IL-17 (Th17 cells) and/or IL-22 (Th22 cells) production, which are involved in maintenance of chronic inflammation in psoriasis.[31]

The majority of CD4 T cells are Th1, 20% of it produce IL-17 (Th17) and15% produce IL-22 (Th22). CD 8+ T cells which secrete IFN-γ (Tc1), IL-17 (Tc17), and IL-22 (Tc22) are seen in psoriasis.[31]

Dendritic Cells:

Langerhans cells are naive dendritic cells. LCs are markedly decreased in uninvolved psoriatic epidermis in comparison to normal skin, especially in type I (early onset) psoriasis.

Dermal DCs are immature dendritic cells, psoriasis lesions show increased and matured dermal DC. There are three types of myeloid-derived (CD11c+) DCs are seen in psoriasis lesions. The first one is the population of “resident” dermal DCs,

seen in normal skin as well. These CD11c+/CD1c+ DC account for 10%–15% of DCs in the lesions of psoriasis. The second population consists of mature DCs which show CD83 positivity. The DCs are seen in Clusters with T cells in the dermis, mature DCs are involved in chronic T- cell activation.

The third population is the inflammatory DCs. These cells are involved in IL-23 production and Th17differentiation.[31]

Plasmacytoid DCs (pDCs) are insignificant presenters of antigens to T cells, pDCs are increased in both uninvolved and involved psoriatic skin, but activated only in involved skin.[31]They control inflammation and involved in connecting innate with adaptive immunity, on activation produce IFN-ϒ.[31]

Macrophages:

Macrophages are seen in more number in initial and developing psoriasis lesions. These active cells could be involved in creating holes in the basement membrane of epidermis. Involved in TNFα, iNOS, and IL-23 production.[31]

Keratinocytes:

As explained in above diagram, keratinocytes are producers of cytokines TNFα, IL- 6, chemokines CXC18, CXCL10, CXCL11-20, as well as innate immunity mediators.[31]

ENVIRONMENTAL FACTORS

Disease presentation can occur secondary to environmental factors. In an individual with genetic susceptibility, physical trauma, psychological stress, drugs and infections may initiate the disease.[32]

Local Factors

Skin injury sites can develop psoriasis lesions. The Koebner phenomenon is the induction of lesions at sites of traumatic skin. The injuries may be of physical, chemical, mechanical, allergic or of any other nature. This can be elicited at sites of sunburn, operation wounds, vaccination and other skin lesions.[33]

Seasonal Variation

Winter season exacerbation seen in most of individuals (89% in one study).[34,35,36] Sunlight improves in many and exacerbates psoriasis in few. [37]

Emotional Stress

Stress may aggravate psoriasis. Some patients with psoriasis who had stress shown abnormal hypothalamic–adrenal axis response. Neurogenic inflammation mediated by substance P occurs secondary to increased beta endorphin in psoriasis skin.[38]

Infections

URTI secondary to streptococci may aggravate an attack of guttate psoriasis. [39-41]

This is common in children and is associated with increased antistreptolysin 'O' titre.

Drugs

Many drugs induce or aggravate psoriasis, such as beta-blockers, lithium, antimalarials, imiquimod, interferons α and γ, and ACE inhibitors.[42-48]

Alcohol and Smoking

Heavy drinking exacerbates pre-existing psoriasis. Smoking 20 cigarettes or more per day is associated with risk of severe psoriasis.

Obesity

The obesity prevalence is twice of that in the normal population or those with other types of skin disease. Severe psoriasis is seen in obese patients

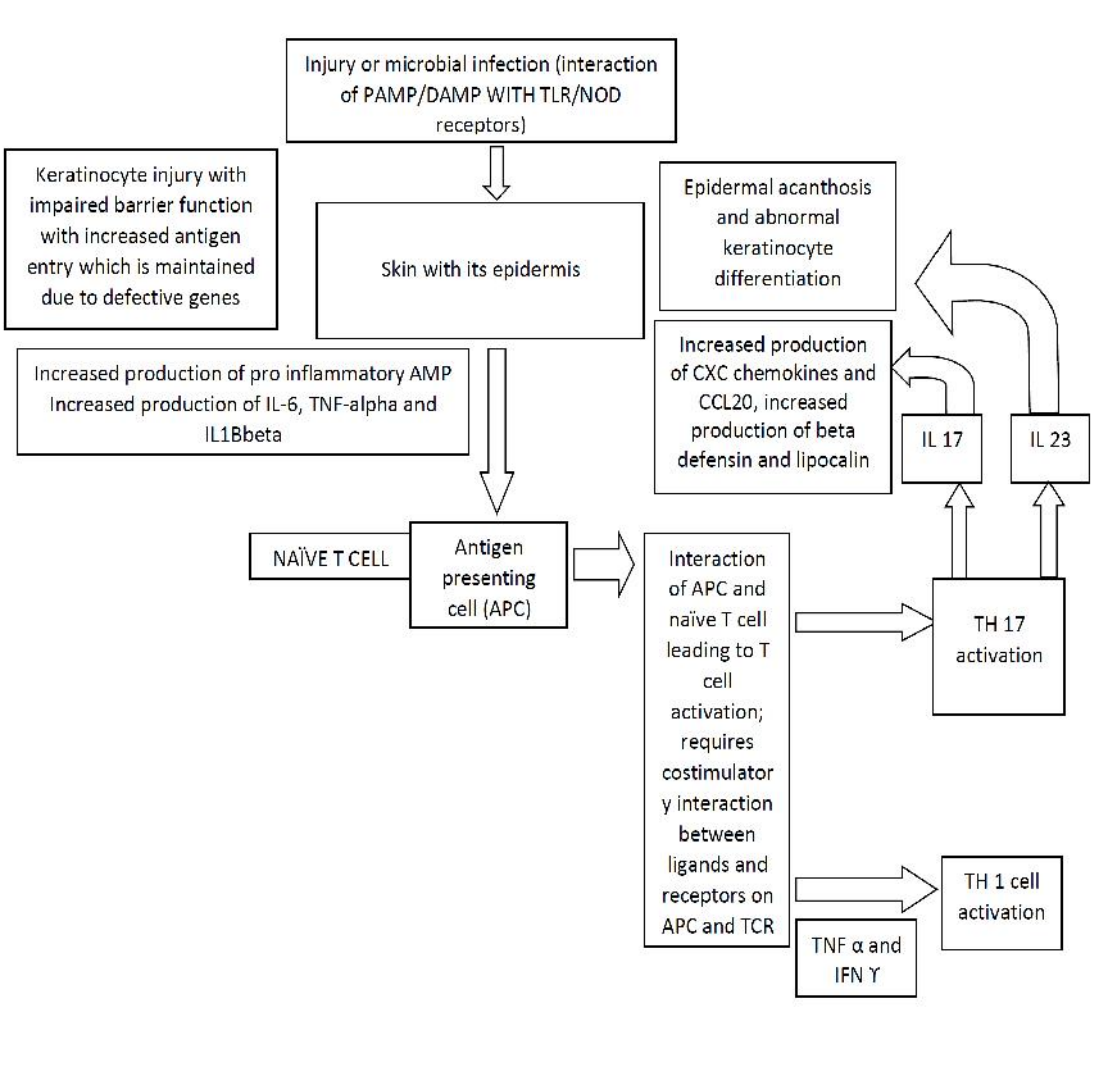


FIGURE2: PATHOGENESIS OF PSORIAISIS

Following injury to keratinocytes secondary to trauma, infection, there will be interaction between PAMP and nod like receptors/toll-like receptors of the keratinocytes. Keratinocytes are activated, which leads to production of cytokines TNFα, IL-6 and IL-1β and expression of antimicrobial peptides cathelecidin LL-37,Human beta defensin 12.

The cytokine production leads to recruitment of antigen presenting cells, which along with antigen migrate to regional lymph node. There will be interactivity between naïve T cell and antigen presenting cell. There will be T cell activation secondary to costimulatory molecules.

Majority of -T cells are TH-1 cells and there also will be activation of TH-17 and TH-22 cells, TH-17 cells produce interleukin IL-17 and IL -23.

IL-17 leads to production of antimicrobial peptides and lipocalin, IL-23 leads to acanthosis and undifferentiated keratinocytes.

These both TH17 and TH-22 cells are involved in maintenance of chronic activation of inflammation.

TH-1 cells produce IFN-ϒand other cytokines which lead to recruitment and activation of antigen presenting cells. They also cause acanthosis and parakeratosis of epidermis.

CALCIUM ROLE IN SKIN AND PSORIASIS

Calcium gradient across cell leads calcium to play role of internal messenger for signals outside cell. Calcium outside cell is 10000 times more than inside the cell, free intracellular calcium is functional form[49].

The studies demonstrated that calcium gradient is present in epidermis, calcium in basal, spinous layer is lower than serum calcium whereas calcium in granular layer is very high. [50]Factors Involved in gradient maintenance are unknown. Calcium concentration plays role in differentiation of keratinocytes. [51]

Calcium gradient is required for terminal differentiation. It provides an explanation for occurrence of gradient of calcium in vivo. Gradient loss was seen in vivo in psoriasis, indicating enhanced proliferation. [52] The increase in extracellular calcium concentration prior to intracellular calcium is necessary for keratinocytes maturation. [53-54]

CLINICAL FEATURES

Psoriasis presents as a well-defined raised red plaque with a white surface scales. Size varies from papules to plaques. Pin point bleeds are seen on removal of scale secondary to damaged dilated capillaries below (Auspitz sign).[55]

Psoriasis will have bilateral symmetric distribution. Koebner phenomenon is the occurrence of psoriasis at trauma sites on uninvolved skin and is an all-or-none phenomenon (i.e. if there is development at one site, all other sites will also develop). The Koebner phenomenon develops 7–14 days after injury. [56]

CLASSIFICATION

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.

Regional variations in psoriasis: Scalp, face, eyes, body flexures, scrotum, napkin area, palms and soles. [57]

GUTTATE (ERUPTIVE) PSORIASIS

Guttate psoriasis (Latin word, means “a drop”) presents with small (0.5–1.5 cm in diameter) papules distributed over the trunk and proximal extremities. It is seen more commonly in children and adolescents. This type of psoriasis has the association with HLACw6; guttate psoriasis occurs simultaneously or proceeded by streptococcal throat infection.[58]

CHRONIC PLAQUE PSORIASIS

Psoriasis vulgaris is commonest type of psoriasis, seen in 90% of patients.erythematous plaque in symmetrical distribution present over elbow, knees, back and genitals.

Lesions may coalesce to form plaques with borders similar to a map (psoriasis geographica).

Coalescing of several plaques, results in lateral expansion of lesions giving circinate appearance (psoriasis gyrata).

Ring-like lesions with central clearance may be seen in few patients (annular psoriasis).

Cone or limpet shape lesions are seen in rupoid psoriasis.

Concave lesions with hyperkeratosis similar to oyster shell appearance are seen in ostraceous type of psoriasis.

Large plaques with thick scales over the lower limb are seen in elephantine psoriasis.

A hypopigmented ring (Woronoff ring) around psoriatic lesions is associated with response to treatment. [59]

ERYTHRODERMIC PSORIASIS

Erythrodermic psoriasis seen in 1–2% of patients[60,61]. It involves greater than 90% body surface area, erythroderma secondary to psoriasis seen in 25% of patients.[62]

Erythroderma in psoriasis may undergo chronicity secondary to unstablity or slow progression of psoriasis.The acute form may get exacerbated by systemic illness, alcoholism, antimalarials, topical irritants, UV radiation or by sudden discontinuation of systemic corticosteroids and immunosuppressants.

The patient will be febrile. Pitting oedema will be seen. Itching is severe. More than 90% of skin may be affected and the clinical features of psoriasis will be absent. Skin failure, including sepsis, hypothermia or hyperthermia, hypoalbuminaemia, anaemia, dehydration and cardiac failure are complications seen.[63]

PUSTULAR PSORIASIS

When plaque surface contain superficial, sterile pustules, it is referred as pustular psoriasis. Treatment with coal tar, anthralin (in unstable psoriasis), potent steroids or progesterone exacerbate pustular psoriasis. infection, pregnancy and hypocalcemia can exacerbate it. Pustular psoriasis is classified into a localized type and a generalized type. [64,65]

Generalized pustular psoriasis again classified into:[57]

i. Von Zumbusch type
ii. Annular pustular type
iii. Pustular psoriasis of pregnancy
iv. Exanthematic Type

Localized pustular psoriasis has been further classified into:[57]

a) Pustulosis palmaris et plantaris

b) Acrodermatitis continua of Hallopeau.

PSORIASIS UNGUIS

Nail involvement seen in patients with prolonged disease, more body surface area and in psoriatic arthritis patients. The fingernails involvement is more common than the toenails. The nail involvement is higher in arthritis [57]. Different nail changes have been seen with different portion nail involvement.[66]

Proximal matrix: Pitting, onychorrhexis,

Intermediate matrix: Leukonychia

Distal matrix: Focal onycholysis, splinter hemorhages, erythema of the lunula

Nail bed; "Oil drop" sign or "salmon patch,"subungual hyperkeratosis, onycholysis,

Hyponychium: Subungual hyperkeratosis, onycholysis

MUCOUS MEMBRANE PSORIASIS

Mucosal involvement not so common in psoriasis.[67,68] Usually restricted to pustular and erythrodermic types, mostly seen on the buccal mucosa and dorsum of the tongue. Geographic tongue (glossitis areata migrans),seen in pustular psoriasis causes loss of filiform papillae. [31]

PSORIATIC ARTHRITIS

Psoriatic arthritis is an inflammatory arthritis seen in psoriasis patients, negative for rheumatoid factor[69].Arthritis seen in 5% - 10% of patients of psoriasis. It has genetical role with HLA B27, DR3, A26 and B38 positivity. It is seen in between the ages of 30-55. [57]

Psoriatic arthritis five types are seen: [70]

I. Asymmetrical DIP joint involvement (16%)
II. Arthritis mutilans (5%)
III. Symmetrical polyarthritis-like rheumatoid arthritis (15%)
IV. Oligoarthritis (70%)
V. Ankylosing spondylitis (5%)

SCALP PSORIASIS

The psoriasis plaques will have well defined border whereas seborrheic dermatitis will have ill defined border with diffuse involvement in most cases. Plaques in psoriasis may extend 2-5 cm beyond hair line (‘corona psoriatica’). [57]

PALMOPLANTAR PSORIASIS

Palmoplantar lesions show well defined border with fissure in many cases, scaling will be less. Three types of lesions can be seen at these sites: diffuse hyperkeratotic plaques, erythematous patches or plaques filled with minute superficial pustules. [57]

Childhood psoriasis

Children will have thin, soft lesions, less scales and more pruritis, Plaque type is commonly seen. Erythroderma, arthropathy, and localized and generalized pustular psoriasis are rarely seen. Guttate psoriasis seen commonly in children which is exacerbated by URTI.[71]

Inverse Psoriasis;

Inverse psoriasis occurs in skin folds, patient presents with well defined plaques in symmetrical distribution with absence of scales. They may show secondary changes such as fissures, maceration and complains of itching, burning sensation.[72]

Psoriasis in pregnancy

Impetigo herpetiformis is seen in pregnancy third trimester. Recurrences are seen in future pregnancies. Patients present with erythematous plaques filled with pustules, initially in flexures later involving allover the body. Symptoms such as fever, chills, malaise, diarrhea, nausea, and arthralgia will be present.[73]

Lab findings show leucocytosis with neutrophilia, increased ESR, anemia, and hypoalbuminemia. Hypocalcemia seen most commonly, the drug of choice is systemic corticosteroids. Calcium, fluid and electrolytes corrections also should be done.[73]

HISTOPATHOLOGY OF PSORIASIS

Histopathology shows epidermis findings are hyperkeratosis with parakeratosis and Munro's micro abscess (in stratum corneum), spongiform pustule of

Kogoj (in spinosum layer). Suprapapillary thinning with elongated rete ridges. Dermis shows dilated capillaries with peri vascular lymphocytic infiltrate. [74]

PASI Score for the Evaluation of Psoriasis [57].

Four body sites namely head, upper limb, trunk and lower limb are evaluated for three parameters erythema, induration and desquamation. The scoring of each done using severity scale of 0 to 4 where, 0 = nil, 1 = mild, 2 = moderate, 3 = severe and 4 = very severe.

The scores of each parameter are added and they are multiplied by area percentage involvement as follows 1 = less than 10% area, 2 = 10%–29%, 3 = 30%–49%, 4 = 50%–69%, 5 = 70%–89% and 6 = 90%.' The four body regions (head, upper limbs, trunk, and lower limbs) constitute 10%, 20%, 30% and 40% of the BSA respectively, which are given 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 will be between 0 and 72.

Overview of Calcium

INTRODUCTION

Calcium plays role in formation and metabolism of bone. Calcium hydroxyapatite (Ca_{10} $[PO_4]_6[OH]_2$) forms major percentage of calcium seen in bones and teeth. Calcium involved vessel contraction and dilatation, contraction of muscles, conduction of nerve impulses, intracellular signaling, secretion of hormones. Bone tissue contain majority of calcium, whenever in need of body mobilized through bone remodeling.[75]

SOURCES OF CALCIUM

Food

Dairy products; milk, yogurt, and cheese are rich in calcium.[75]

METABOLISM OF CALCIUM

ABSORPTION

Calcium absorption across intestine occurs by both active transport and passive diffusion.

Calcitriol and intestinal vitamin D receptor are necessary for active transport. [75]

CELLULAR AND INTRACELLULAR CALCIUM METABOLISM

Bone, renal, and GI cells are involved in determination of blood calcium. The transport mechanisms may be across or through the cell.[76-78]

The transport through cell is passive. There will be transport, exchange of other molecules namely sodium, potassium, chloride, hydrogen and bicarbonate.[79,80]

Biological functions of calcium:[81]

- Cell signaling
- Nerve conduction
- Muscle contraction
- Blood clotting
- co-factor along with enzymes
- Secretion

Calcium distribution in body:[82]

- Blood calcium – 10mgs (8.5-10.5)/100 mls
- Ionized – 3.5 mgs
- Non ionized – 6.5 mgs

REGARDING SERUM CALCIUM ESTIMATION

Conditions leading to hypoalbuminemia will decline the serum total calcium, "corrected" calcium is measure of the total calcium when the albumin was normal. This adjustment was first introduced in 1971.[83]

In general, measurement of active serum calcium by estimating total calcium is less costly and independent of sample transportation variation.[84]

Ionized calcium is the reliable marker of calcium and considered as the gold standard, is independent of serum albumin. The test is costly; value varies with pH, alteration in values with delayed analysis. [85]

Experts advised to calculate adjusted calcium for albumin, by using the formula devised by Payne et al corrected calcium (mmol/L) = total calcium (mmol/L) + 0.02 [40 (g/L) – albumin (g/L)][86,87].

The sensitivity of 5% and17% was seen using Payne's albumin-adjusted calcium formula for hypo- and hypercalcemia in a trauma ICU study. [88]

Clase et al studied different albumin-adjusted calcium formulas in relation to ionized calcium in hemodialysis patients, it was found adjusted calcium using Payne's formula was in less agreement with ionized calcium than the unadjusted total calcium.[89]

The calcium level division as high, normal or low, both the unadjusted total calcium and the albumin-adjusted calcium formula is moderately agreeable with ionized calcium, while Payne's albumin adjusted formula had less agreement .[90]

Hypocalcaemia[91]

Hypocalcemia presentation can vary from asymptomatic lab finding to life threatening complication.

Clinical features of hypocalcemia

Ectodermal changes

- Xerosis of skin
- Coarse hair
- Brittle nails
- Hair loss
- Hypoplastic enamel
- Shortened premolar roots
- Thickened lamina dura
- Delayed tooth eruption
- Atopic eczema
- Psoriasis
- Pustular psoriasis of pregnancy

METHODOLOGY

This study was conducted within duration of twelve months (January 2018 to December 2018) in the Department of Dermatology, Venereology and Leprosy in tertiary care hospital. The study was a hospital-based observational study.

- ***Sample size:*** The study was a non-randomized single-arm observational study. Hence, based on previous records of patients having psoriasis who had attended the outpatient department of Dermatology, Venereology and Leprosy in the previous year, a sample size of 70 was calculated. However, the total number of patients attending the OPD during the study period was 60; hence a sample size of 60 was studied.
- ***Ethical clearance:*** Clearance was taken from the Ethical Committee of the institute.

INCLUSION CRITERIA:

Subjects of both sex of age group 15-65 years with psoriasis, attending dermatology OPD.

EXCLUSION CRITERIA:

1. Patients with impaired renal function and renal failure
2. Patiens taking nephrotoxic drugs for long term illness
3. Patients on vitamins and minerals therapy
4. Patients of psoriasis with hypertension on ca2+ channel blockers.

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 chi square and student “t” test and using correlation method.

Psoriatic patients attending dermatology out patients and inpatients are selected for study, they are subjected to detailed history and clinical examination includes skin, nail and mucosa and joints (if involved).

Biochemical parameters measured are - serum calcium and serum albumin Patients were diagnosed with hypocalcemia if serum calcium is less than 8.5mg/dl with normal range being 8.5-10 mg/dl. Patient’s serum albumin measured with normal range being 4-5.5mg/dl.

Venous samples were taken at the enrolment visit of all patients for measuring serum total calcium and albumin level.

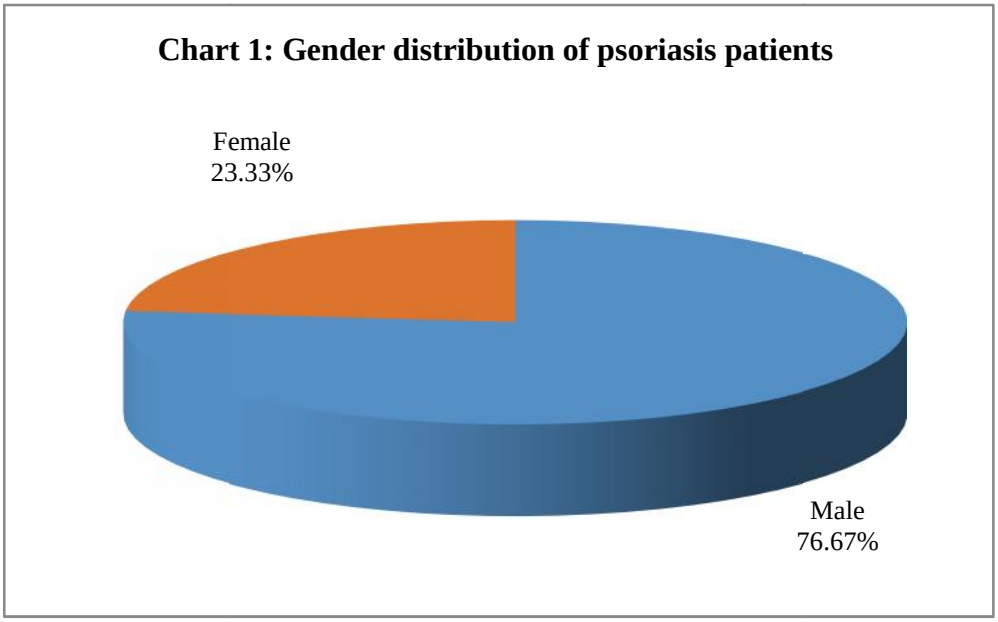
RESULTS

Table 1 : Gender distribution of psoriasis patients

Gender	No of psoriasis patients	% of psoriasis patients
Male	46	76.67
Female	14	23.33
Total	60	100.00

The above table shows total number of patients with male and female distribution, male patients were predominantly seen (76.67%).

Chart 1: Gender distribution of psoriasis patients



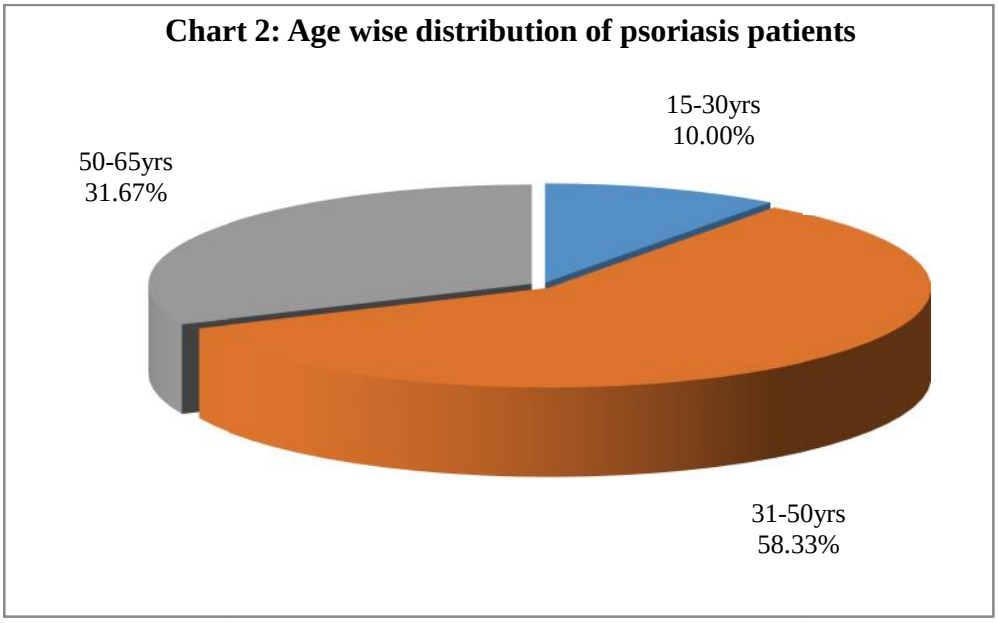
The above pie chart shows gender distribution among patients, male patients represent larger portion (76.67%).

Table 2 : Age wise distribution of psoriasis patients

Age groups	No of psoriasis patients	% of psoriasis patients
15-30yrs	6	10.00
31-50yrs	35	58.33
50-65yrs	19	31.67
Total	60	100.00
Mean age	44.80	
SD age	12.35	

Sixty patients between ages of 15 to 65 years were included in study; mean age of patients was 44.80. Patients in age group of 31-50 years were commonest (58.33%) followed by 50-65 years (31.67%).

Chart 2: Age wise distribution of psoriasis patients

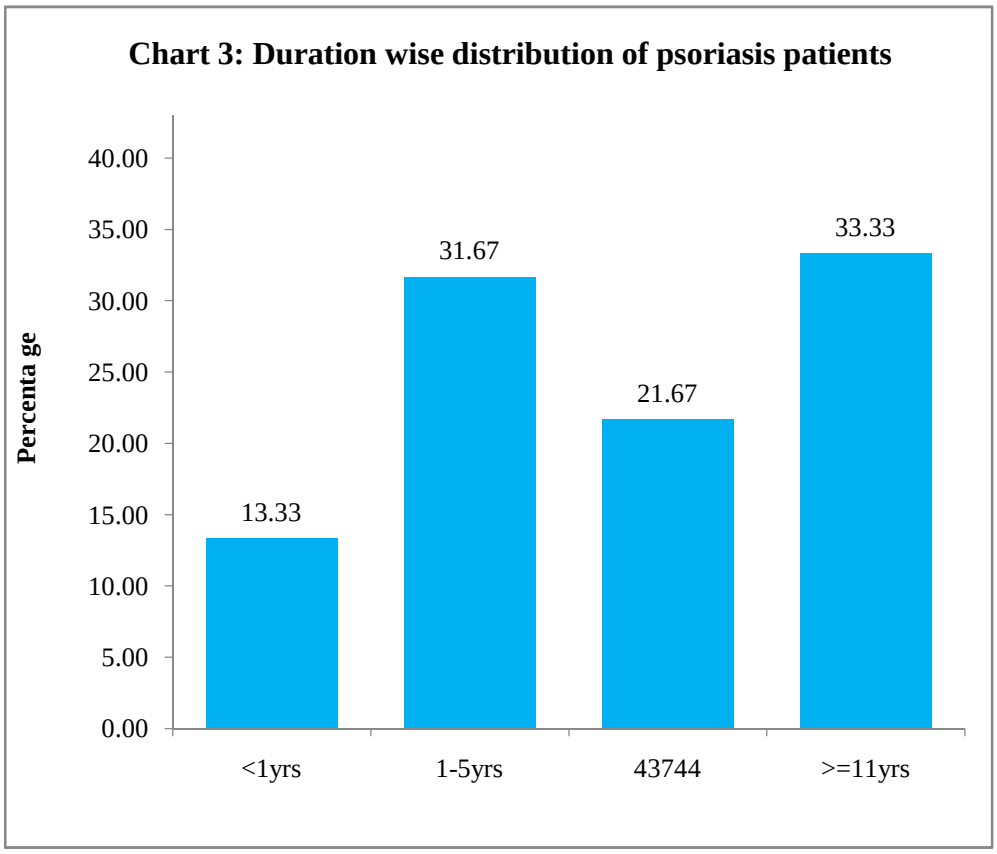


The above pie chart shows age wise distribution of patients. Patients between age group of 31-50 years were commonest (58.33%) followed by patients in age group of 51-65 years (31.67%).

Table 3 : Duration wise distribution of psoriasis patients

Duration	No of psoriasis patients	% of psoriasis patients
<1yrs	8	13.33
1-5yrs	19	31.67
6-10	13	21.67
>=11yrs	20	33.33
Total	60	100.00
Mean	9.24	
SD	8.29	

The above table shows duration wise distribution of psoriasis patients. Mean duration of disease seen in patients was 9.24 years. Patients with disease duration more than or equal to 11 years were commonest (33.33%) followed by patients with duration between 1-5 years (31.67%).

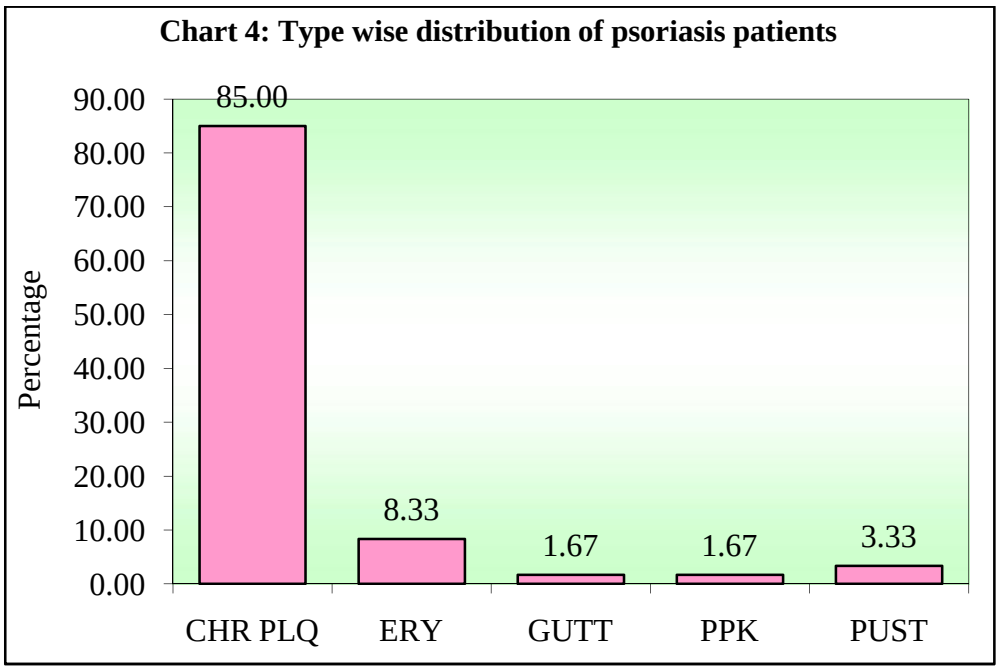


The above bar diagram represent duration psoriasis in patients, Disease duration more than or equal to 11 years were commonest (33.33%) followed by patients with duration between 1-5 years (31.67%).

Table 4 : Type wise distribution of psoriasis patients

Types	No of psoriasis patients	% of psoriasis patients
CHR PLQ	51	85.00
ERY	5	8.33
GUTT	1	1.67
PPK	1	1.67
PUST	2	3.33
Total	60	100.00

The above table shows different types of psoriasis in patients, chronic plaque type of psoriasis was commonest (85%) followed by erythrodermic type of psoriasis (8.33%).guttate type and palmoplantar type were least commonly seen(1.67%).



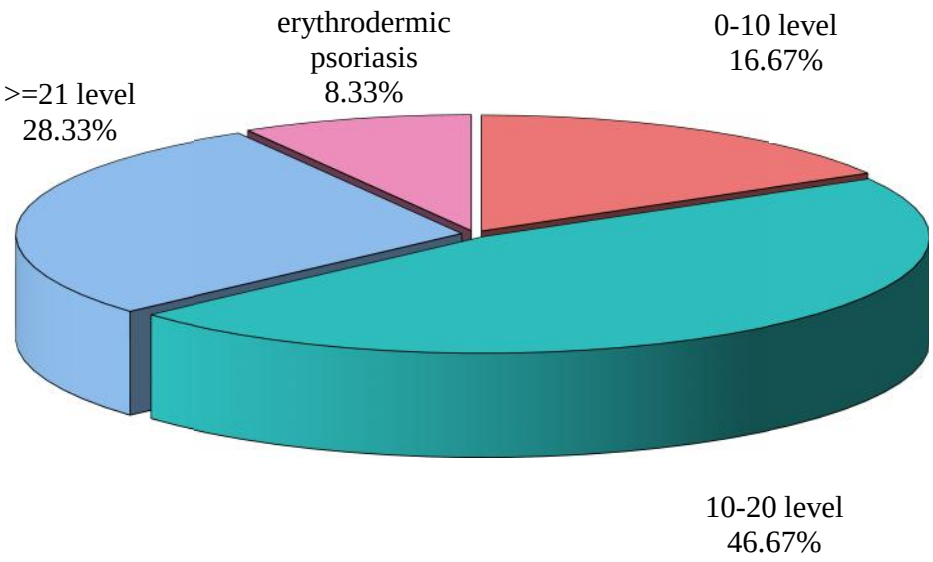
The above bar diagram shows types of psoriasis, chronic plaque type was commonest (85%) followed by erythrodermic type of psoriasis (8.33%).

Table 5 : PASI levels distribution of psoriasis patients

PASI levels	No of psoriasis patients	% of psoriasis patients
0-10 level	10	18.18
10-20 level	28	46.67
>=21 level	17	28.33
Erythrodermic psoriasis	5	8.33
Total	60	100.00
Mean	16.16	
SD	6.84	

The above table shows PASI score of patients. Patients on basis of PASI score were divided into three groups excluding erythrodermic psoriasis, PASI <10, between10-20 and >= 21. Mean PASI score was 16.16. Patients with PASI score between10-20 were commonest (46.67%) followed by patients with PASI>=21 (28.33%).

Chart 5: PASI levels distribution of psoriasis patients

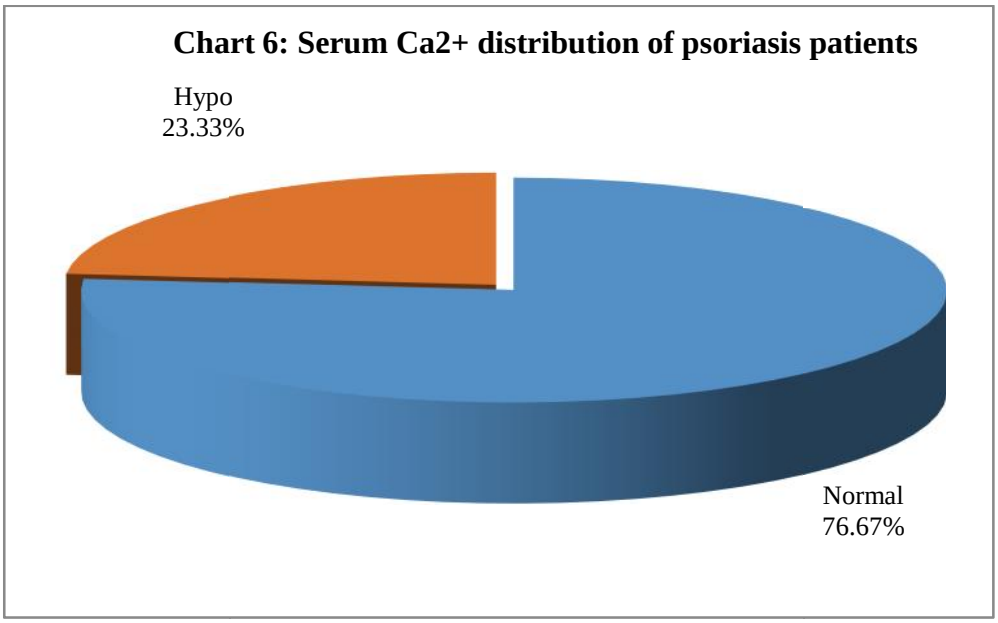


The above pie chart shows PASI score of patients, patients with PASI score between 10-20 were commonest (46.67%) followed by patients with PASI score >= 21 (28.33%).

Table 6 : Serum Ca2+ distribution of psoriasis patients

Serum Ca2+	No of psoriasis patients	% of psoriasis patients
Normal	46	83.64
Hypo	14	23.33
Total	60	100.00
Mean	8.95	
SD	0.56	

The above table shows serum calcium level in psoriasis patients. Normal serum calcium level range was (8.5-10mg/dl). Mean serum calcium seen among all patients was 8.95, normal calcium level was seen in 46 patients (83.64%) followed by hypocalcemia which seen in 14 patients (23.33%). No hypercalcemia was observed.

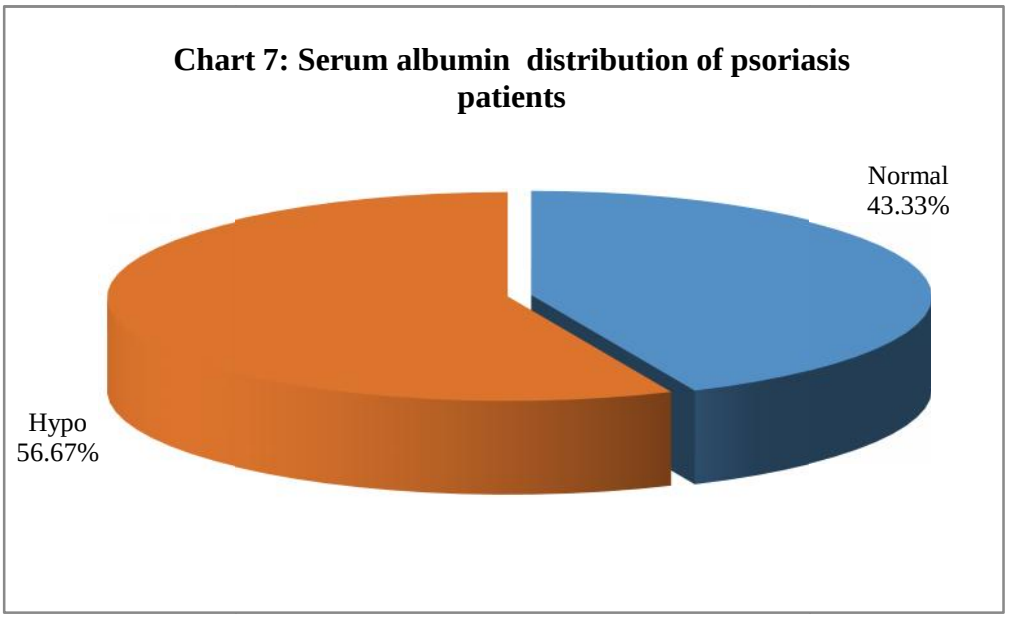


The above pie chart shows serum calcium in psoriasis patients, normocalcemia was seen more commonly (76.67%) than the hypocalcemia (23.33%). No hypercalcmia was observed.

Table 7 : Serum albumin distribution of psoriasis patients

Serum albumin	No of psoriasis patients	% of psoriasis patients
Normal	26	47.27
Hypo	34	56.67
Total	60	100.00
Mean	3.95	
SD	0.45	

The above table shows serum albumin level in patients, normal range being (4-5.5g/dl).Mean serum albumin level was 3.95; low albumin level was seen in 56.67% followed by normal albumin level in 47.27%.



The above pie chart shows serum albumin level in patients, hypoalbuminemia was commonest (56.67%) followed by normal albumin level (43.33%).

Table 8 : Joint and Nail Involvement distribution of psoriasis patients

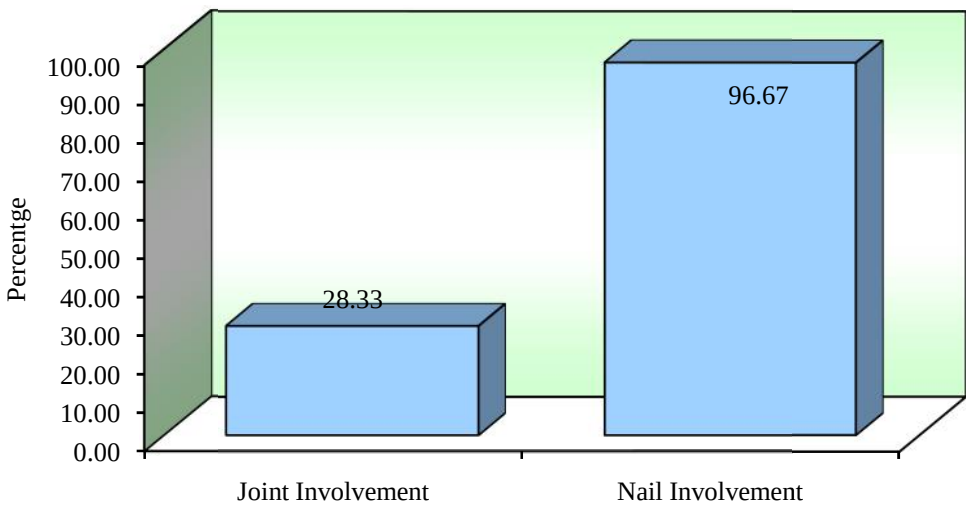
	No of psoriasis patients	% of psoriasis patients
Joint Involvement		
Absent	43	78.18
Present	17	28.33
Nail Involvement		
Absent	2	3.64
Present	58	96.67
Total	60	100.00

The above table shows Joint and nails involvement of psoriasis patients.

Joint involvement was seen in 17 patients (28.33%); knee joint involvement was commonest followed by ankle joint.

Nail involvement was seen in 58 patients (96.67%), pitting nail finding was commonest finding followed by subungual hyperkeratosis.

Chart 8: Joint and Nail Involvement distribution of psoriasis patients

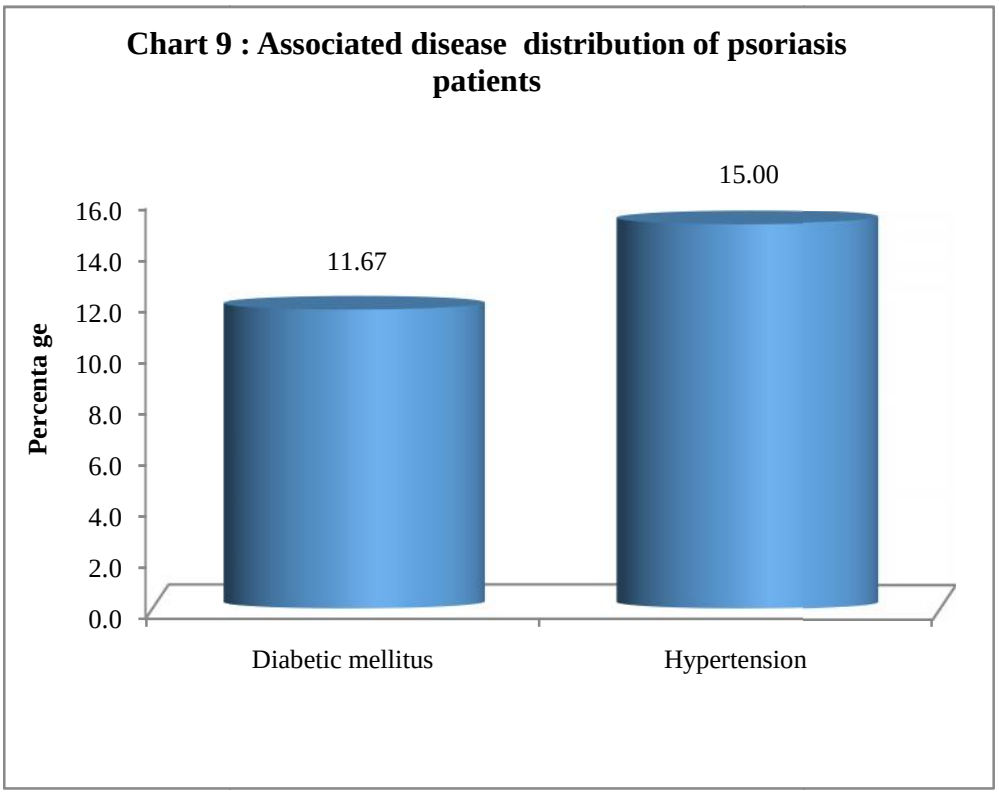


The above bar diagram represents joint and nail involvement in psoriasis patients. Joint involvement was less commonly seen (28.33%).Nail involvement was more commonly seen (96.67%).

Table 9 : Associated disease distribution of psoriasis patients

Diseases	No of psoriasis patients	% of psoriasis patients
Diabetic mellitus		
Absent	53	96.36
Present	7	11.67
Hypertension		
Absent	51	92.73
Present	9	15.00
Total	60	100.00

The above table shows associated disease diabetes mellitus and hypertension among all patients. Diabetes mellitus was seen in 7 patients (11.67%) and hypertension in 9 patients (15%).There was no overlap between diabetes and hypertension in patients.



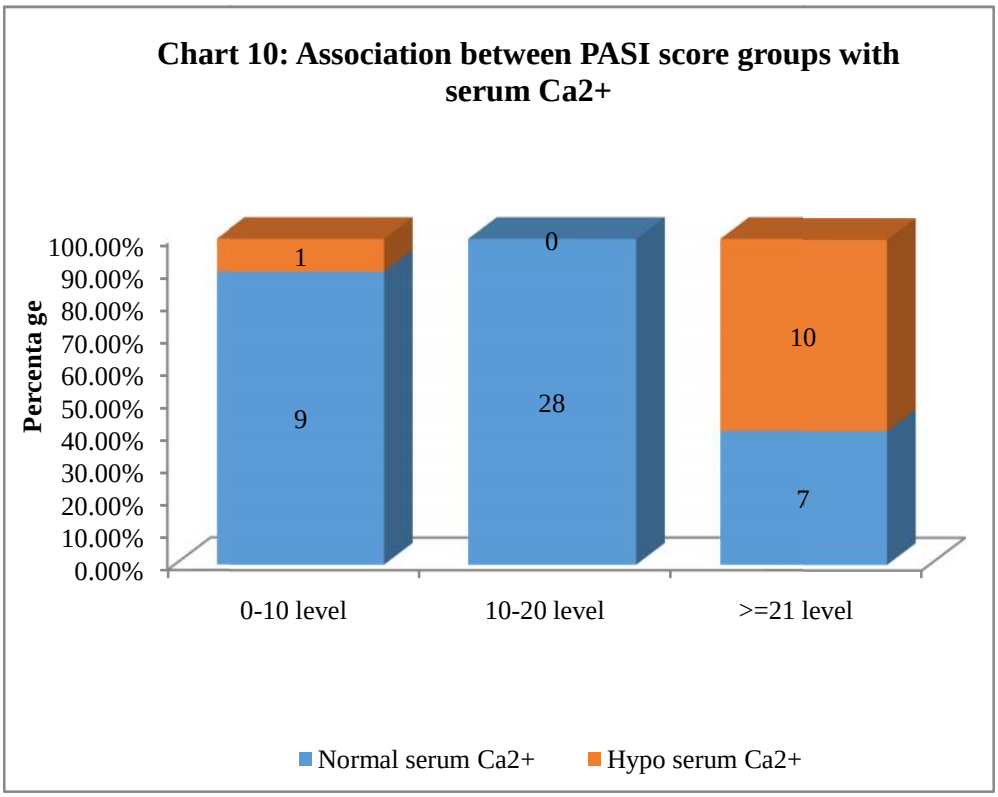
The above bar diagram shows Diabetes mellitus and Hypertension seen in psoriasis patients, diabetes mellitus and hypertension were seen less commonly representing 11.67%and 15% respectively.

Table 10 : Association between PASI score groups with serum Ca2+

PASI levels	Normal serum Ca2+	%	Hypo serum Ca2+	%	Total
0-10 level	9	90.00	1	10.00	10
10-20 level	28	100.00	0	0.00	28
>=21 level	7	41.18	10	58.82	17
Total	44	80.00	11	20.00	55
Chi-square=23.640 P = 0.0001*					

*p<0.05

The above table shows association between PASI score and serum calcium. Patients with PASI score< 10 were 10 (9 were normocalcemic and 1 was hypocalcemic), with PASI score between10-20 were 28(all had normal calcium level) and patients with PASI score >=21were 17 (hypocalcemia seen in 10 patients, normal calcium in7). There was positive association between PASI score and hypocalcemia with significant ‘P’ value of 0.0001.



The above bar diagram shows association between PASI score and serum calcium. There was Positive association between PASI score and hypocalcemia.

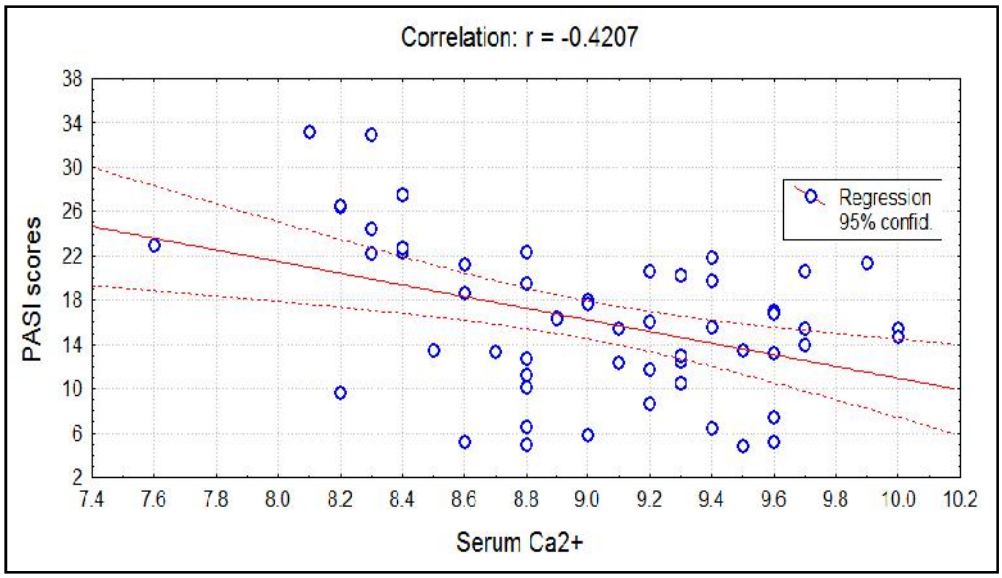
Table 11 : Correlation between PASI scores with serum Ca2+ scores by Karl Pearson's correlation coefficient method

Variables	Correlation between PASI scores with		
	r-value	t-value	p-value
Serum Ca2+	-0.4207	-3.3761	0.0014*

*p<0.05

The above table shows correlation between PASI score and serum calcium, showing negative correlation with significant P- value. Suggesting higher PASI score lower will be calcium.

Chart 11: Scatter diagram of correlation between PASI scores with serum Ca2+ scores



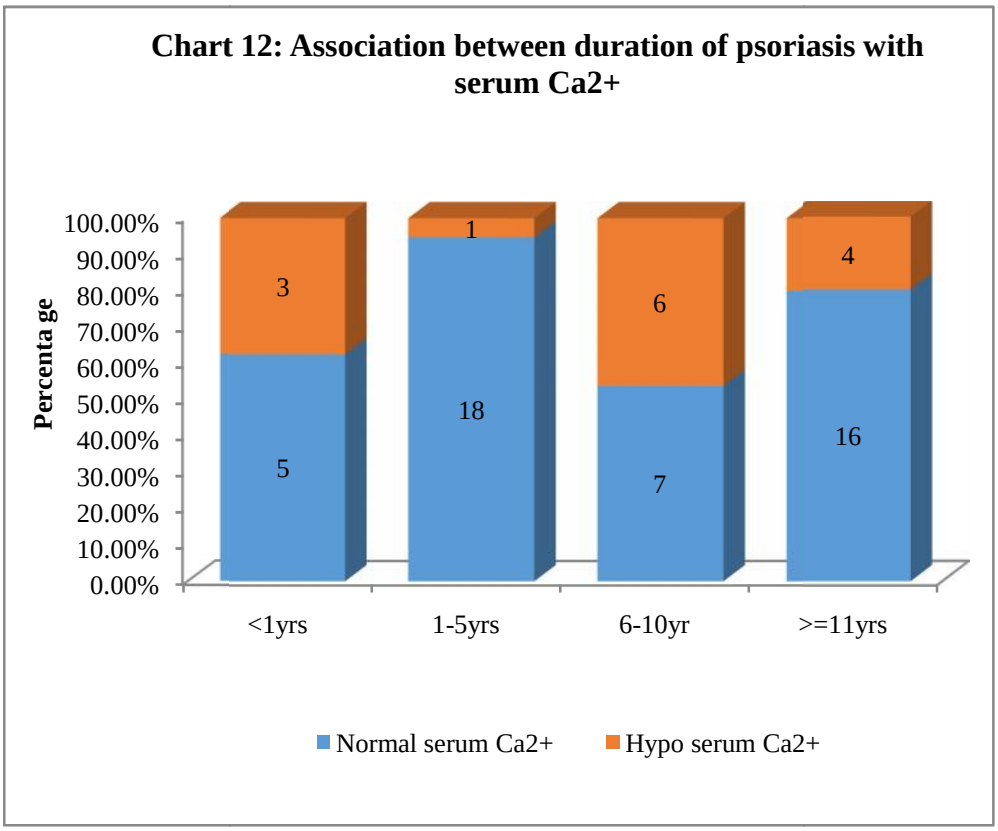
The above figure shows scatter diagram of correlation. In the above diagram hypocalcemia was commonest in patients with PASI score > 21, normal calcium was seen in patients with PASI score< 20.

Table 12 : Association between duration of psoriasis with serum Ca2+

Duration	Normal serum Ca2+	%	Hypo serum Ca2+	%	Total
<1yrs	5	62.50	3	37.50	8
1-5yrs	18	94.74	1	5.26	19
6-10yrs	7	53.85	6	46.15	13
>=11yrs	16	80.00	4	20.00	20
Total	46	76.67	14	23.33	60
Chi-square= 8.274 P = 0.0410*					

*p<0.05

The above table shows association between duration of psoriasis and serum calcium. There was positive association between duration of disease and hypocalcemia with significant P value (0.0410).



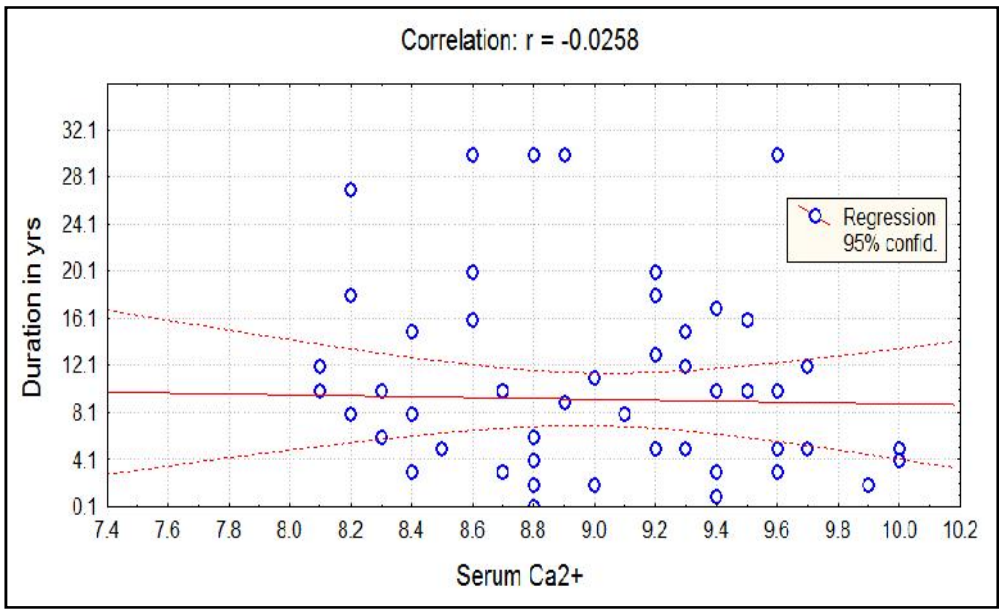
The above bar diagram shows association between duration of psoriasis and serum calcium. Most Patients with disease duration <5 years had normal calcium, whereas hypocalcemia was common in patients with disease >5 years.

Table 13 : Correlation between duration of psoriasis with serum Ca2+ scores by Karl Pearson's correlation coefficient method

Variables	Correlation between duration of psoriasis with		
	r-value	t-value	p-value
Serum Ca2+	-0.0258	-0.1963	0.8450

The above table shows correlation between duration of psoriasis and serum calcium. There was negative correlation found with insignificant P value (>0.005).

Chart 13: Scatter diagram of correlation between duration of psoriasis with serum Ca2+ scores

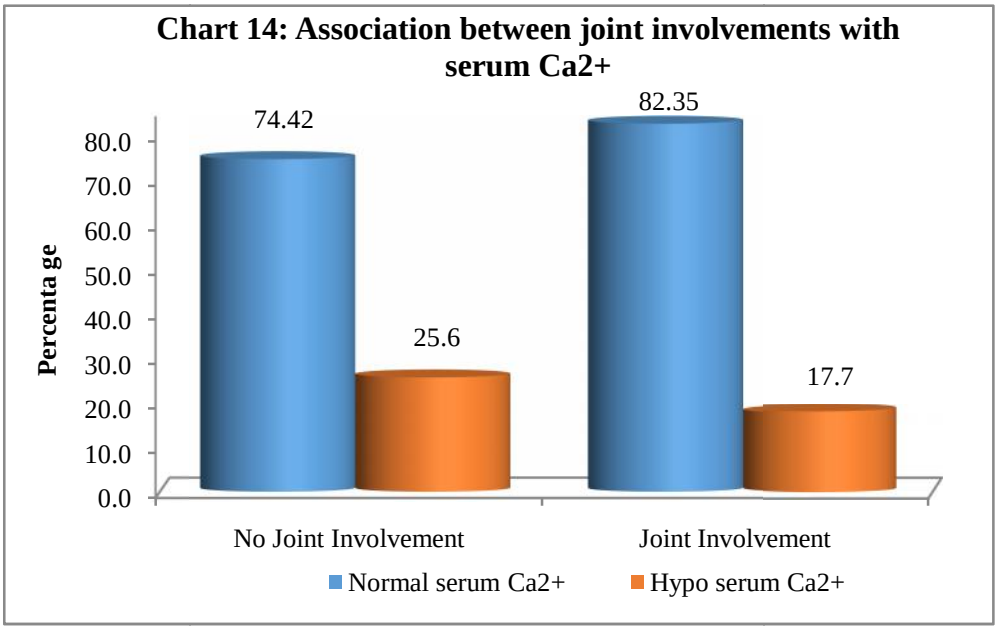


The above figure shows scatter diagram of correlation between duration of psoriasis and serum calcium. Most patients with normal serum calcium are around duration of 5 years, whereas most hypocalcemic patients were seen with duration more than 6years.

Table 14 : Association between joint involvements with serum Ca2+

Joint Involvement	Normal serum Ca2+	%	Hypo serum Ca2+	%	Total
Absent	32	74.42	11	25.58	43
Present	14	82.35	3	17.65	17
Total	46	76.67	14	23.33	60
Chi-square with Yates's correction = 0.1001 P = 0.7521					

The above table shows association between joint involvement and serum calcium level. There was no significant association between serum calcium and joint involvement with insignificant P value (>0.005).



The above bar diagram shows association between serum calcium and joint involvement. Joint involvement was seen in 82.35%of normal calcium patients and in 17.7 % low serum calcium of patients. There was no significant association found between joint involvement and serum calcium.

Table 15 : Comparison of PASI groups with serum Ca2+ score by one Way ANOVA

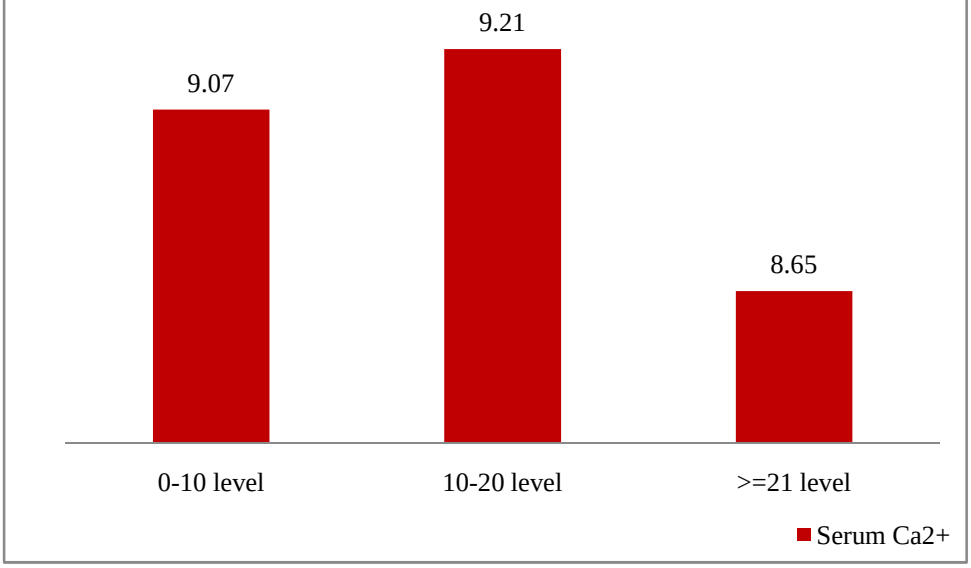
PASI levels	Serum Ca2+	
	Mean	**SD**
0-10 level	9.07	0.47
>=21 level	8.65	0.63
Total	9.01	0.55
F-value	6.6745	
P-value	0.0026*	
Pair wise comparison of PASI groups using Turkeys post Hoc procedure		
0-10 level vs. 10-20 level	p=0.7352	
0-10 level vs. >=21 level	p=0.0982	
10-20 level vs >=21 level	p=0.0019*	

*p<0.05

The above table shows comparison of PASI groups with serum calcium. Mean calcium level among patients with PASI <10 was 9.07, with PASI between10-20 was 9.21 and in PASI >21 was 8.65. ANOVA test shows positive association between hypocalcemia and PASI score with significant P value (<0.005).

The above table also shows pair wise comparisons of PASI score with serum calcium, comparison among different PASI group was done using Post Hoc test. P value was significant in comparison between moderate and severe psoriasis (<0.005).

Chart 15: Comparison of PASI groups with serum calcium scores



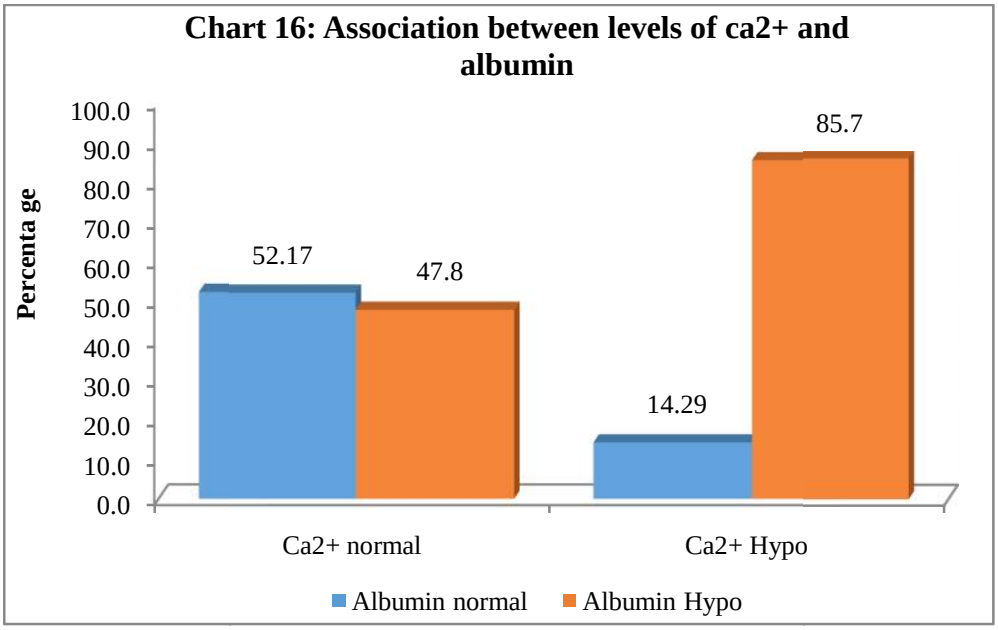
The above bar diagram shows comparison of mean value of serum calcium in different PASI groups. Showing low serum calcium among patients with PASI >=21

Table 16 : Association between levels of ca2+ and albumin

	Albumin normal	%	Albumin Hypo	%	Total
Ca2+ normal	24	52.17	22	47.83	46
Ca2+ Hypo	2	14.29	12	85.71	14
Total	26	43.33	34	56.67	60
Chi-square= 6.2752 P = 0.0120*					

*p<0.05

The above table shows association between serum calcium and serum albumin. There was positive association between low albumin and low calcium with significant P value (0.0120). Suggesting hypoalbuminemia is common finding among patients with hypocalcemia.

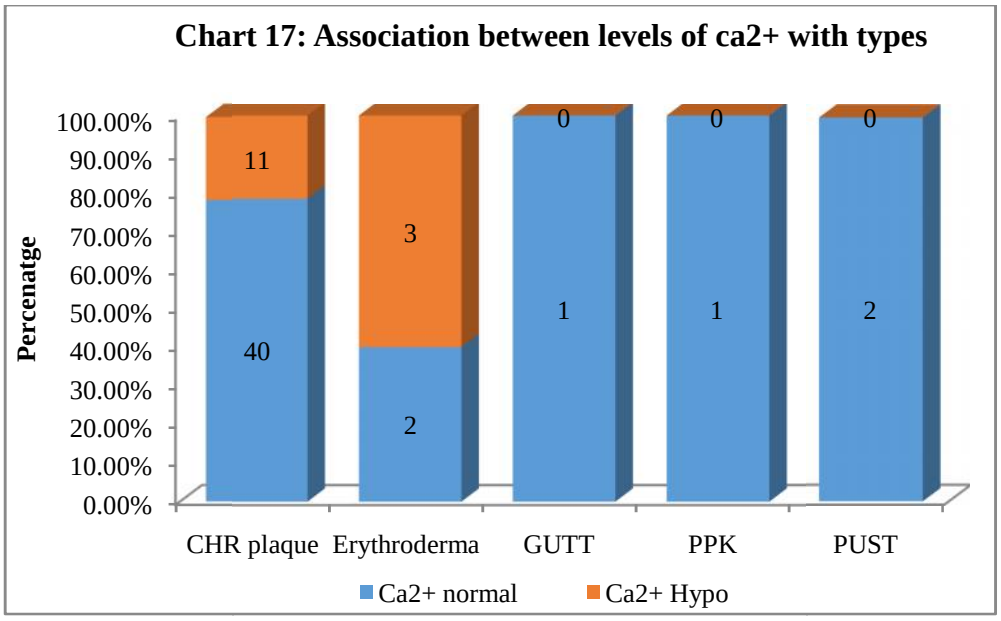


The above bar diagram shows hypoalbuminemia was seen commonly among patients with hypocalcemia (85.7%), In comparison to patients with normal serum calcium (47.8%).

Table 17 : Association between levels of ca2+ with types

TYPE	Ca2+ normal	%	Ca2+ Hypo	%	Total
CHR plaque	40	78.43	11	21.57	51
Erythroderma	2	40.00	3	60.00	5
GUTT	1	100.00	0	0.00	1
PPK	1	100.00	0	0.00	1
PUST	2	100.00	0	0.00	2
Total	46	76.67	14	23.33	60

The above table shows association of serum calcium with types of psoriasis. The above findings suggest erythrodermic type and chronic plaque with (PASI score >21 as mentioned in previous table)) show hypocalcemia.



The above bar diagram represents distribution of serum calcium level in all types of psoriasis patients. In the above diagram hypocalcemia was most common finding among erythrodermic type of psoriasis (60%), followed by chronic plaque type of psoriasis (21%).

DISCUSSION

The present study is a hospital- based cross sectional study conducted over a period of 12 months from January 2018 to December 2018 in the Department of Dermatology, Venereology and Leprosy in a tertiary care hospital, Belagavi.

Since there is a bimodal age on onset, the first peak at 15-20 years of age and a second one at 55-65 years, patients between ages of 15-65 years are included in this study.

In a study, a study of serum calcium and uric acid levels in Psoriasis, a hospital based observational study conducted on 100 patients carried by Bijina KD et al [92] the incidence of psoriasis in males was 74% and in females was 24%.

In a study, a hospital based cross sectional study on early and late onset psoriatic patients, performed on 40 patients of psoriasis and 40 controls carried by Deepti E et al [93]. The incidence of psoriasis in males was 57.5 % and in females was 42.5%.

In our study sixty patients were enrolled. Incidence of psoriasis in males was (76.67%) and in females was (23.33%).

In a study by Bijina KD et al [92] the age of the patients ranged from 10 years to 60 years. The majority of patients (28%) belonged to the age group of 31years to 40 years. Mean age of the patients included in study was 36.8 years.

In a study of clinical and biochemical correlation in patients of psoriasis in acute exacerbation by Neeraja puri et al [94] Mean age of patients was 38.46+3.28 year.

Maximum cases (22%) were seen in age group of 51-60 years followed by 20% in age group of 31-40 years.

In present study mean age of patients was 44.80 years; cases were commonest in age group of 31-50 years (58.33%) followed by patients in 50-65 years age group (31.65%).

In a study by Bijina KD et al [92] the duration of disease ranged from less than year to more than 6 years, with most patients had disease duration between 13 to 36 months (38%) followed by patients with disease duration between 0-12 months (36%).There was no correlation observed between duration of disease and hypocalcemia.

In present study the duration of disease ranged from less than a year to more than 6years. Most patients had disease duration >11 years (33.33%), patients with less than 1 year duration were least (13.33%). Duration of disease was significant in relation to occurrence of hypocalcemia with significant P value (<0.05), our study has shown positive association between disease duration and hypocalcemia.

In a study by H.H.Quadim et al [95] Hypocalcemia was seen in psoriasis vulgaris patients (14.2%), pustular psoriasis (50%), erythrodermic psoriasis (100%). Hypercalcemia was not observed, hypocalcemia frequency was high in severe types of psoriasis.

In a study by Bijina KD et al [92] hypocalcemia was seen in 39.45% of psoriasis vulgaris, all patients with pustular and erythrodermic psoriasis had hypocalcemia (100%), high frequency of hypocalcemia was seen in severe types of psoriasis.

In our study hypocalcemia was seen in 21.57% psoriasis vulgaris patients, 60% of erythrodermic Psoriasis patients, no hypocalcemia was observed in pustular psoriasis. Hypocalcemia frequency was high in severe psoriasis, which includes patients with PASI >21and erythrodermic psoriasis.

In a study by Bijina KD et al [92] 16 out of 58 patients with a PASI score 0-15 had hypocalcemia (27.6%), 18 out of 38 patients with PASI 16-30 had hypocalcemia (47.4%) and all Four Patients with PASI score of 31 to 45 had hypocalcemia (100%). There was positive association between hypocalcemia and PASI score with significant P value (<0.005).

In a study of correlation of serum calcium with severity of psoriasis conducted by Sunil Chaudari et al [96] patients were divided into mild PASI<10, moderate 10-20 and severe >21 psoriasis. Serum calcium level among mild (9.0+0.20), moderate (8.93+0.24) and in severe psoriasis (8.98+0.22), ANOVA test analysis test showed positive association between hypocalcemia and PASI score with significant P value (0.001).

In our study 1 out of 9 patients with PASI SCORE <10 had hypocalcaemia (10%), no patients out of 28 with PASI score between10-20 had hypocalcemia (0%) and 10 out of 17 patients with PASI >21 had hypocalcaemia (58.82%). Positive Association between hypocalcemia and PASI score with significant P value (<0.005) was found. Suggesting higher the PASI score lower will be serum calcium.

In a study carried by Sunil Chaudari et al [96] comparison of P value using post-Hoc test among different severity levels was done in cases of psoriasis, P value of comparison between mild and moderate form of disease, among mild and severe form

of disease and among moderate and severe form of disease was statistically insignificant with P value (>0.05) in all groups.

In our study, Post Hoc test done to compare calcium among different PASI group, P value comparison among mild and moderate psoriasis, mild and severe psoriasis was statistically Insignificant (P>0.005) but P value in moderate and severe psoriasis was statistically significant (0.0019). The values found in our study need to be reconfirmed in future studies.

In a study by H.H.Quadim et al [95] hypocalcemia was seen in 37 cases (37.2%), among these hypocalcemic patient's low serum albumin was observed in 24 cases (64.9%) and all patients in Control group had normal serum albumin level. Hypocalcemic patients with low albumin level needed correction to get corrected calcium, in spite of correction only few patients had normal calcium and was practically negligible.

In our study hypocalcemia was seen in 14 patients, 12 patients had low serum albumin level (85.71%). There was association between hypocalcaemia and hypoalbuminemia with significant P value (0.012). In this study no calcium correction was done as according to Steen, Oren et al [90] study unadjusted calcium had better agreement than adjusted calcium with ionized calcium.

This finding needs to be studied in further studies as there was only one study to compare calcium association with albumin.

In a study carried out by H.H.Quadim et al [95] total number of psoriasis vulgaris with arthritis were 14 (14.2%) of all cases, among these 57.1 % of patients

had hypocalcemia and 42.9% had normal calcium. There was association found between hypocalcemia and psoriatic arthritis with significant P value (<0.005).

In our study there were 17 patients with psoriasis vulgaris with arthritis constituting 23.33% of Cases, among these 82.35% had normal serum calcium and 17.65% had hypocalcemia. There was no association between hypocalcemia and psoriatic arthritis with P value (>0.05).

CONCLUSION

Study was done to observe serum calcium in patients of psoriasis, based on findings following conclusions made. Hypocalcemia was seen in severe psoriasis (PASI>21 and erythrodermic type) and with longer duration of disease.

To conclude measurement of serum calcium level should be considered in all patients with severe psoriasis, with longer duration of disease, as hypocalcemia may be a risk factor for severe psoriasis.

SUMMARY

A total of 60 psoriasis patients attending tertiary care hospital were included in study.

In the present study:

- 46 (76.67%) patients were males and 14 (23.33%) were females.
- Patients with Age group between 31-50 years were 35 (58.33%) commonest followed by 51-65 years in which 19 (31.67%) were seen.
- Patients with duration >11 years were 20 (33.33%) commonest followed by patients between 1-5 years in which 19 (31.67%) were seen.
- Chronic plaque psoriasis was commonest type seen in 51 (85%) patients.
- Patients with PASI score between10-20 were commonest 28 (46.67%) followed by patients with PASI >21 17 (28.33%).
- Normal calcium was seen in 46 (76.67%) patients, was commonest among patients with PASI score between10-20 (100%).
- Normal albumin level was seen in 26 (46.27%), hypoalbuminemia was seen in 34 (56.67%) of patients.
- Hypocalcemia was seen in 14 (23.33%) patients, was commonest in severe types of psoriasis [PASI score >21 (58.82%), erythrodermic psoriasis (60%)].
- Hypoalbuminemia was associated finding seen in high frequency among patients with Hypocalcemia (85.71%).
- Hypocalcemia was commonest in patients with disease duration between 6-10 years (46.15%).

- Nail changes were seen in 58 (96.67%) patients, joint involvement was seen in 17 (28.33%) patients.
- Diabetes mellitus was seen in 7 (11.67%) patients, hypertension was seen in 9 (15%) patients.
- Among 17 patients with joint involvement hypocalcemia was seen in 3 (17.65%) patients with insignificant association.
- Comparison of calcium among different PASI group shows significant P value among moderate and severe psoriasis ($p<0.005$).

BIBLIOGRAPHY

1. Griffiths CEM, Barker JNWN. Psoriasis. In Burns T, Breathnach S, Cox N, Christophers G Eds. Rook's Textbook of Dermatology. 8th edition, Wiley-Blackwell Publication 2010:20.1 20.60.
2. Christophers E. Psoriasis – Epidemiology and clinical spectrum. Clin Exp Dermatol 2001; 26:314-20.
3. Lebwohl, M., J.P. Ortonne, P. Andres and P. Briantais, 2009. Calcitriol ointment 3microg/g is safe and effective over 52 weeks for the treatment of mild to moderate plaque psoriasis. Cutis, 83: 205-212.
4. Plavina. T, M. Hincapie, E. Wakshull, M. Subramanyam and W.S. Hancock, 2008. Increased plasma concentrations of cytoskeletal and Ca2+-binding proteins and their peptides in psoriasis patients. Clin. Chem, 54: 1805-1814.
5. Braun, G.S., M. Witt, V. Mayer, H. Schmid, 2007. Hypercalcemia caused by vitamin D3 analogs in psoriasis treatment. Int. J. Dermatol., 46: 1315-1317.
6. Noborio, R., K. Kobayashi, Y. Shintani and A. Morita, 2006. Comparison of the efficacy of calcipotriol and maxacalcitol in combination with narrow-band ultraviolet B therapy for the treatment of psoriasis vulgaris. Photodermatol. Photoimmunol. Photomed. 22: 262-264.
7. Glickman FS. Lepra, psora, psoriasis. J Am Acad Dermatol. 1986; 14:863–866.
8. Beheet PE. Psoriasis, a brief historical review. Arch Dermatol Syphilol. 1936; 33:327–334.
9. Farber EM, MeClintock RP Jr. A current review of psoriasis. Calif Med. 1968; 108:440–457.

10. Pariser DM, Bagel J, Gelfand JM, Korman NJ, Ritchlin CT, Strober BE, et al. National Psoriasis Foundation Clinical Consensus on Disease Severity. Arch Dermatol. 2007; 143:239–24.
11. Dogra S, Yadav S. Psoriasis in India: Prevalence and pattern. Indian J Dermatol Venereol Leprol. 2010; 76:595–601
12. Dhar S, Banerjee R, Agrawal N, Chatterjee S, Malakar R. Psoriasis in children: An insight. Indian J Dermatol 2011; 56:262-5.
13. Leonard DG, O'Duffy JD, Rogers RS. Prospective analysis of arthritis in hospitalized for psoriasis patients. Mayo Clin Proc 1978; 53:511-8.
14. Griffiths CE, Barker JN. Pathogenesis and clinical features of psoriasis. Lancet 2007; 370:263-71.
15. Trembath RC, Clough TL, Rosbotham JL. Identification of a major susceptibility gene locus on chromosome 6p and evidence for further disease loci revealed by a two stage genome-wide search in psoriasis. Human Mol Genet 1997; 6:813-20.
16. Krulig L, Farber FM, Gruneet C. Histocompatibility (HLA) antigens in psoriasis. Arch Dermatol. 1975; 111:857–860.
17. Bedi TR, Sengupta S, Sehgal S. Histocompatibility antigens in psoriasis. Indian J Dermatol Venereol Leprol. 1979; 45:21–23.
18. Ameen M, Barker JN. Genetics. A. Psoriasis. In: Gordon KB, Ruderman EM, editors. Psoriasis and psoriatic arthritis. 1st edn. New York; Springer, 2005. p. 3–12.
19. Nair RP, Ruether A, Stuart PE, Jenisch S, Tejasvi T, Hiremagalore R, *et al*. Polymorphisms of the IL12B and IL23R genes are associated with psoriasis. J Invest Dermatol 2008; 128:1653-61.

20. Nair RP, Duffin KC, Helms C, Ding J, Stuart PE, Goldgar D, *et al*. Genome-wide studies reveals association of psoriasis with IL-23 and NF-kB pathways. Nat Genet 2009; 41:199-204.
21. Anna-karin EKMANet al overexpression of psoriasin (S100A7) contributes dysregulated differentiation in psoriasis. Acta Derm Venereol 2017; 97: 441–448.
22. Mehlis SL, Gordon KB. The immunulogy of psoriasis and biological immunotherapy. J Am Acad Dermatol 2003; 49: (Suppl):S44-50.
23. Stockinger B, Veldhoen M. Differentiation and function of Th17 T cells. *Curr Opin Immunol* 2007;19:281-6.
24. Bettelli E, Carrier Y, Gao W, Korn T, Strom TB, Oukka M, *et al*. Reciprocal developmental pathways for the generation of pathogenic effector TH17 and regulatory T cells. Nature 2006;441:235-8.
25. Bonifati C, Ameglio F. Cytokines in psoriasis. Int J Dermatol 1999; 38:241-51.
26. Goffe B, Cather JC. Etanercept: An overview. J Am Acad Dermatol 2003;49:S105-11.
27. Nickoloff BJ, Karabin GD, Barker JN, Griffiths CE, Sarma V, Mitra RS, *et al*. Cellular localization of interleukin-8 and its inducer, tumor necrosis factor-alpha in psoriasis. Am J Pathol 1991;138:129-40.
28. Krueger JG. The immunologic basis for the treatment of psoriasis with new biologic agents. J Am Acad Dermatol 2002;46:1-23.
29. Barker JN, Sarma V, Mitra RS, Dixit VM, Nickoloff BJ. Marked synergism between tumour necrosis factor-alpha and interferon gamma in regulation of

keratinocyte-derived adhesion molecules and chemotactic factors. J Clin Investig 1990;85:605-8.

30. Ghosh A, Panda S. Recent understanding of the etiopathogenesis of psoriasis. Indian J Paediatr Dermatol 2017;18:1-8.
31. Johann E. Gudjonsson & James T. Elder. Fitzpatrik's Dermatology in General Medicine. 8th Edition. Chapter 18, Psoriasis;p.197-242
32. Basavaraj KH, Ashok NM, Rashmi R, Praveen TK. the drugs role in the induction and/or exacerbation of psoriasis. Int J Dermatol 2010;49:1351-61.
33. Reiss P. Psoriasis and adrenocortical function. Arch DermSyph.1949;59:78.
34. Lomholt G. Skin diseases prevalence in a population, a census study from the Faroe Islands. Danmed Bull. 1964;11:1–7.
35. Zlotogorski A.Psoriasis of the left elbow (correspondence). Australas J Dermatol. 1989; 30:106.
36. Baker H. Psoriasis: A review. Dermatologica. 1975; 150:16–25.
37. Zlotogorski A. Psoriasis of the left elbow (correspondence).Australas J Dermatol. 1989; 30:106
38. Glinski W, Brodecka H, Glinska–Ferenz M, et al. Neuropeptides in psoriasis: beta-endorphin possible role in the pathomechanism of the disease. Int J Dermatol. 1994; 33:356–360.
39. Norrlind R. The significance of infection in the organization of psoriasis. Acta Rheum Scand. 1955; 1:135.
40. Whyte HJ, Baughmann RD. Acute guttate psoriasis and streptococcal infection. Arch Dermatol. 1964; 89:350.
41. Pavithran K. Henoch–Schönlein purpura evolving into acute guttate psoriasis. Indian J Dermatol Venereol Leprol. 1990; 56: 147–149.

42. Pavithran K. Exacerbation of psoriasis by propranolol. Indian J Dermatol Venereol Leprol. 1981; 47:326–328
43. Lowe NJ, Ridgway HB. Generalized pustular psoriasis is precipitated by lithium carbonate. Arch Dermatol. 1978; 114:1788–1789.
44. Pavithran K. Exacerbation of psoriasis by ibuprofen. Indian J Dermatol Venereol Leprol. 1987; 53:372–373.
45. Ben–Chetrit E, Rubinow A. Exacerbation of psoriasis by ibuprofen. Cutis. 1986;38:45.
46. Roland MGM, Stevenson CJ. Exfoliative dermatitis and practolol.Lancet. 1972; 1:1130.
47. Leonard JC. Oxprenolol and a psoriasis-like eruption. Lancet. 1975; 1:630.
48. Jensen HA, Mikkelsen IH, Wadskor S, et al. Cutaneous reactions to propranolol (Inderal). Acta Med Scand. 1976; 199:363–367.
49. Rasmussen .h; the calcium messenger system. N engl j med 314:1094-1101, 1986
50. Menon GK, Grayson S, Elias PM: ionic calcium reservoirs in mammalians epidermis: ultra structural localization by ion- cytochemistry j invest dermatol 84: 504-512, 1985
51. Yuspa SH, Kilkenny A, Steiner PM, Roop DR: expression of murine epidermal differentiation marker is tightly regulated by extracellular calcium concentration in vitro j cell biol 109: 1207-1217, 1989
52. Menon GK, Elias PM: ultra structural localization of calcium in psoriatic and normal epidermis. Arch dermatol 127: 57-63, 1991.

53. Hennings H, Michael D, Cheng c, Steinert, Yuspa SH: calcium regulation of growth and differentiation of mouse epidermal cells in culture19: 245-254, 1980

54. Sharpe GR, Gilespi ji, Greenwell JR: an increase intracellular free calcium is an early event during differentiation of cultured human keratinocytes FEBS let 254:25-28, 1989

55. Fry L: *An Atlas of Psoriasis*. Carnforth, ParthenonPublishing, 1992

56. Weiss G, Shemer A, Trau H: The Koebner phenomenon: review of the literature. *J Eur Acad DermatolVenereol 16*(3):241-248, 2002

57. K. Pavithran, M. Karunakaran, Aparna Palit, Ragunatha S. IADVL Textbook of Dermatology. 3rd Edition. Chapter 30, Disorders of keratinization; p.995-1069.

58. Telfer NR et al: The role of streptococcal infection in the initiation of guttate psoriasis. *Arch Dermatol 128*(1):39-42, 1992

59. Fitzpatrick's Dermatology in General Medicine. 8th Edition. Chapter 18, Psoriasis; p.197-242

60. Goeckerman WH, O'Leary PA. Erythroderma psoriaticum: a review of 22 cases. *JAMA* 1932; 99:2102–5.

61. Rosenbach M, Hsu S, Korman NJ, *et al.* Treatment of erythrodermic psoriasis: From the medical board of the National Psoriasis Foundation. *J Am Acad Dermatol* 2010; 62:655–62.

62. Boyd AS, Menter A. Erythrodermic psoriasis. Precipitating factors, course, and prognosis in 50 patients. *J Am Acad Dermatol* 1989; 21:985–91

63. Green MS, Prystowsky JH, Cohen SR, *et al.* Infectious complications of erythrodermic psoriasis. *J Am Acad Dermatol* 1996; 34:911–14.

64. Braverman JM, Cohen I, O'Keefe E. Metabolic and ultrastructural studies in a patient with pustular psoriasis. Arch Dermatol. 1972; 105:189.

65. Shelley WB. Consultations in dermatology. Philadelphia: WB Saunders; 1972. p. 210.

66. Del Rosso JQ et al: Dermatologic diseases of the nail unit. In: *Nails: Therapy, Diagnosis, Surgery*, edited by RK Scher, CR Daniel. Philadelphia, W.B. Saunders, 1997, pp. 172-182.

67. Liao WC, Mutasim DF. Adult onset pityriasis rubra pilaris treatment with infliximab. Arch Dermatol. 2005; 141:423–5.

68. Babu M, Ramachandran P, Nair BKH. Psoriasis of the mucous membrane. Indian J Dermatol Venereol. 1967; 33:83–85.

69. Moll JHM, Wright V. Psoriatic arthritis. Semin Arth Rheumat. 1973; 3:55–78.

70. William D. James et al. Andrews Diseases of the Skin Clinical Dermatology. Psoriasis, seborrheic dermatitis, Palmoplantar Eruptions, Pustular Dermatitis and Erythroderma. 11th Edition. Chapter 10; p.196-210.

71. Dogra sunil, kaur indrajeet childhood psoriasis Indian dermatolvolume76 2010, 357-365.

72. Antonio guglielmetti, Rodrigo conlledo, J Bedoya inverse psoriasis involving genital skin folds: successful therapy with dapsone dermatol Ther .2012 dec 2(1):15

73. giono Antonio Vena, Nicoletta Cassano psoriasis in pregnancy: challenges and solutions psoriasis (auckl) 2015; 5:83-95

74. A. David Burden and Brian Kirby. Rook's Textbook of Dermatology. Psoriasis and related disorders. 9th Edition. Chapter 35; p.35.1-35.48.

75. Institute of Medicine (US) Committee to Review Dietary Reference Intakes for Vitamin D and Calcium; Ross AC, Taylor CL, Yaktine AL, et al., editors. Washington; 2011

76. Drezner, MK: Phosphorus Homeostasis and Related Disorders. In: Principles of Bone Biology, Second Edition, JP Bilezikian, LG Raisz, and GA Rodan (eds.); Chapter 22, pp.321-338, 2002.

77. Yanagawa N and Lee DBN: Renal Handling of Calcium and Phosphorus. In: Disorders of Bone and Mineral Metabolism, FL Coe and MJ Favus (eds.); Chapter 1, pp.3-40, 1992.

78. Favus MJ: Intestinal Absorption of Calcium, Magnesium, and Phosphorus. In: Disorder of Bone and Mineral Metabolism, FL Coe and MJ Favus (eds.); Chapter 3, pp. 57-82, 1992.

79. Reuss, L: Basic Mechanisms of Ion Transport. In: The Kidney, Third Edition, DW Seldin and G Giebisch (eds.); Chapter 3, pp.85-106, 2000.

80. Berger UV, Peng JB, and Hedier MA: The membrane transporter families in mammals. In: The Kidney, Third Edition, DW Seldin and G Giebisch (eds.); Chapter 4, pp. 107-138, 2000.

81. Deftos, LJ: Clinical Essentials of Calcium and Skeletal Metabolism, Professional Communication Inc, First Edition, pp. 1-208, 1998 (Published on-line at Medscape.com)

82. Shaker JL, Deftos L. Calcium and Phosphate Homeostasis. [Updated 2018 Jan 19]. In: Feingold KR, Anawalt B, Boyce A, et al., editors. Endotext

83. Orrell DH. Albumin as an aid to the interpretation of serum calcium. Clin Chim Acta. 1971; 35:483-9.

84. Toffaletti JG. Is ionized calcium always right and total calcium always wrong? CLN. 2011;37(9):8-10.

85. Bushinsky DA, Monk RD. Calcium. Lancet. 1998; 352:306-11.

86. Payne RB, Little AJ, Williams RB, Milner JR. Interpretation of serum calcium in patients with abnormal serum proteins. Br Med J. 1973; 4:643-6.

87. Longo D, Fauci A, Kasper D et al. Harrison's Principles of Internal Medicine.18th ed. McGraw-Hill Education, 2011.

88. Dickerson RN, Alexander KH, Minard G et al. Accuracy of methods to estimate ionized and "corrected" serum calcium concentrations in critically ill multiple trauma patients receiving specialized nutrition support.J Parenter Enteral Nutr. 2004; 28(3):133-41.

89. Clase CM, Norman GL, Beecroft ML, Churchill DN. Albumin-corrected calcium and ionized calcium in stable haemodialysis patients. Nephrol Dial Transplant. 2000; 15(1841):6.

90. Steen, Oren & Clase, Catherine & Don-Wauchope, Andrew. (2016). Corrected Calcium Formula in Routine Clinical Use Does Not Accurately Reflect Ionized Calcium in Hospital Patients. Canadian Journal of General Internal Medicine. 11. 14 - 21. 10.22374/cjgim.v11i3.150.

91. Schafer AL and Shoback D: Hypocalcemia: definition, etiology, pathogenesis, diagnosis and management. Primer on the Metabolic Bone Diseases and Disorders of Mineral Metabolism, C. J. Rosen (ed), John Wiley and Sons, Eighth Edition. pp 572-578, 2013.

92. Bijina KD A study of serum calcium and uric acid levels in Psoriasis Indian Journal of Clinical and Experimental Dermatology, October-December, 2018;4(4):342-345

93. Deepti E, Vijayakumar S .A Hospital based cross sectional study on early and late onset psoriatic patients Indian Journal of Pharmacy Practice Volume 5 Jul-Sep, 2012

94. Neeraja puri, BB Mahajan 2014 A study of clinical and biochemical correlation in patients of psoriasis in acute exacerbation .Journal of Pakistan Association of Dermatologists. 2014; 24 (3):236-240.

95. H.H Quadim, F Goforoushan, S B NEJAD and M Goldust, 2013. Studying the calcium level in patients suffering from psoriasis. Pakistan journal of Biological sciences, 16: 291-294.

96. Chaudhari S et al.correlation of serum calcium level with severity of psoriasis Int J Res Dermatol. 2018 Nov; 4(4):591-594

ANNEXURE -I

INFORMED CONSENT FORM

I.D.NO. | | | |

Title of the study: "ONE YEAR HOSPITAL BASED OBSERVATIONAL STUDY OF SERUM CALCIUM LEVEL IN PSORIASIS PATIENTS ATTENDING KLE DR PRABHAKAR KORE HOSPITAL &MEDICAL RESEARCH CENTRE, BELAGAVI" The study is conducted by **_____,** Post Graduate (M.D) student in Dermatology under the guidance of **_____,** Department of Dermatology, Venereology and Leprosy,JNMC, 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:

Hypocalcemia may be associated with psoriasis. Hence this study intends to find whether hypocalcemia is associated with psoriasis, which is measured by recording serum calcium and serum albumin. 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 and consent for the investigation like serum calcium and serum albumin, which requires drawing of 2 ml blood.

Risks and benefits:

While drawing of 2 ml blood for investigations like estimation of serum calcium and serum albumin, 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:

Your participation in this study is voluntary. Your decision whether or not to participate will neither affect the care of your current disease, nor your future relations with the doctor or the hospital. In the event if you suffer any physical injury as the result of your participation in this study, you may contact

In case you need further information regarding your rights as a study participant, you may please contact Dr. Roopa M Bellad, 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 orit 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- ETHICAL CLEARANCE LETTER

K.L.E.UNIVERSITY'S
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
(Accredited 'A' Grade by NAAC)

Website: http www.jnmc.edu
E-Mail : dome@jnmc.edu

Phone: (+91-(0)831 Office : 2471350
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref: MDC/DOME/ 28 **Date: 22/11/2017**

To,

REG NO.BT0117002

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"ONE YEAR HOSPITAL BASED OBSERVATIONAL STUDY OF SERUM CALCIUM LEVEL IN PSORIASIS PATIENTS ATTENDING KLES DR PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE, BELAGAVI"**, is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

(Dr. Arathi Darshan)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research.
J.N.Medical College, Belagavi.

(Dr. Roopa M Bellad)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Researc
J.N.Medical College, Belagavi.

ANNEXURE-III

PROFORMA

"ONE YEAR HOSPITAL BASED OBSERVATIONAL STUDY OF SERUM CALCIUM LEVEL IN PSORIASIS PATIENTS ATTENDING KLE DR PRABHAKAR KORE HOSPITAL &MEDICAL RESEARCH CENTRE, BELAGAVI"

Case no OP No-

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	☐ Present	☐ Absent
6.	H/o joint involvement a) pain	☐ Present	☐ Absent
	b) Swelling	☐ 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 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 degree 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	☐ Stress
III.	Sleep:	☐ Adequate	☐ Disturbed
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

Weight- kg

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)Splinterhaemorrhages ☐ Present ☐ Absent

5)Oil drop ☐ Present ☐ Absent

6)Beau's lines ☐ Present ☐ Absent

7)Yellowdiscolouration ☐ 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

Serum calcium

Serum albumin

PASI SCORE-

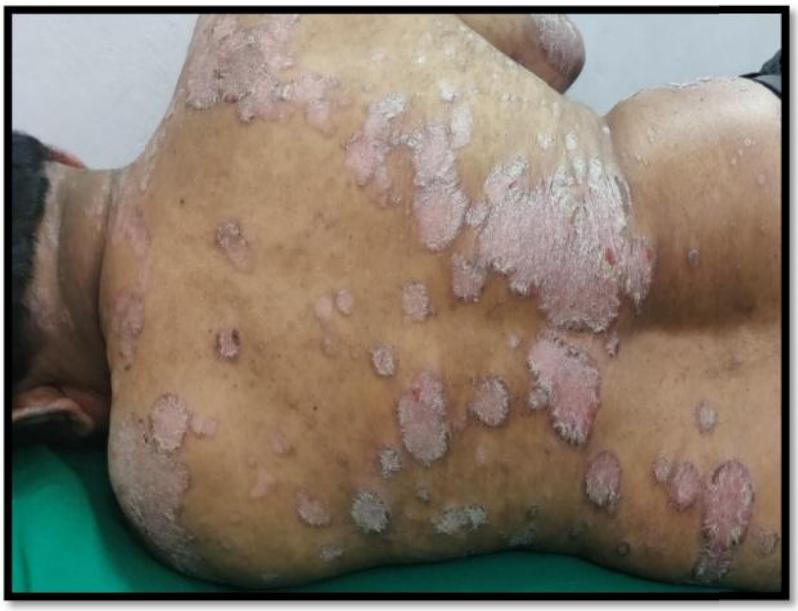
Scaling

Induration

Desquamation

Signature
Guide's signature

ANNEXURE IV – PHOTOGRAPHS



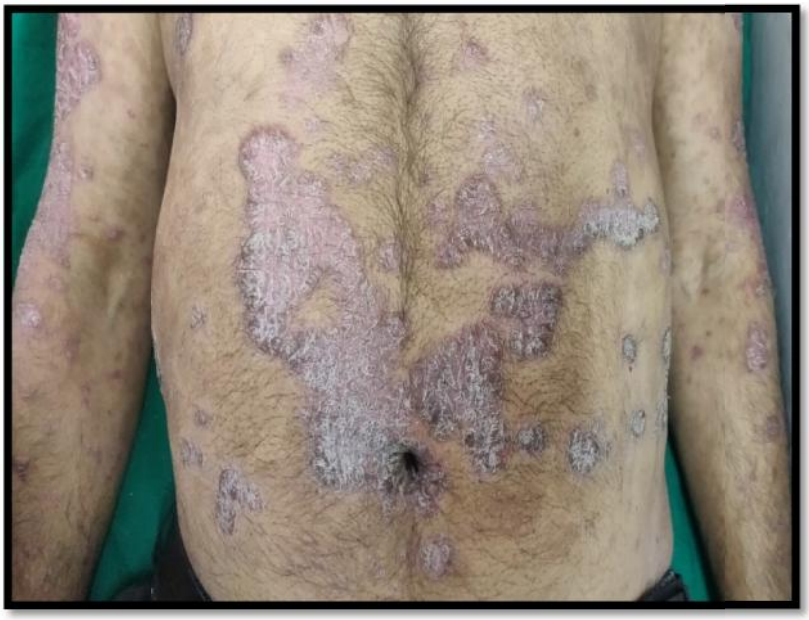


FIGURE 3: CHRONIC PLAQUE PSORIASIS

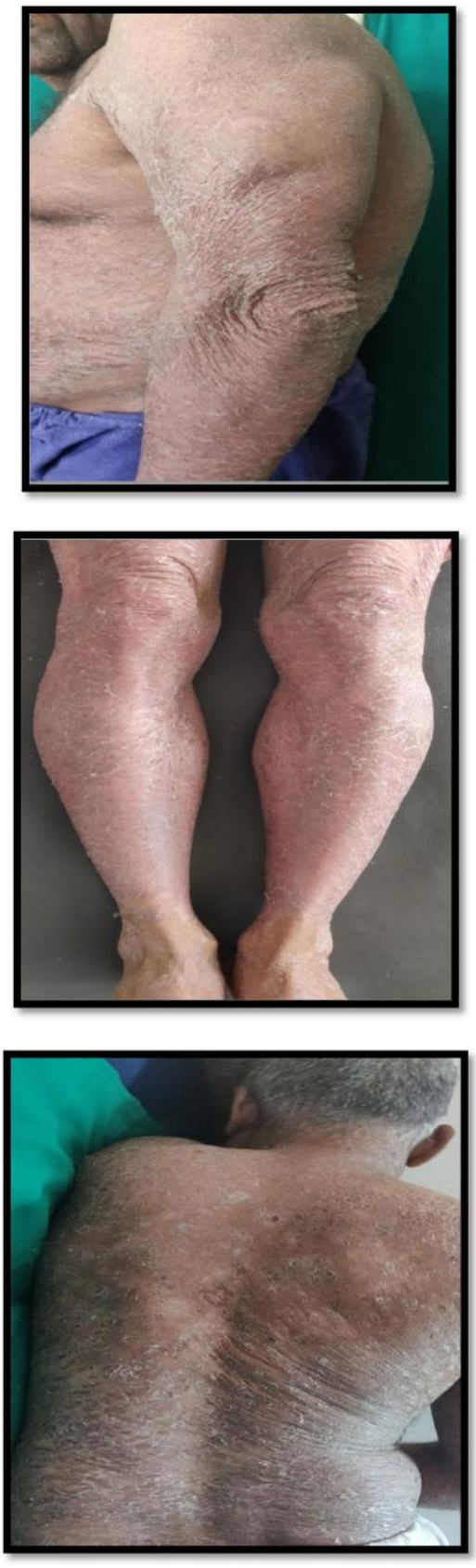
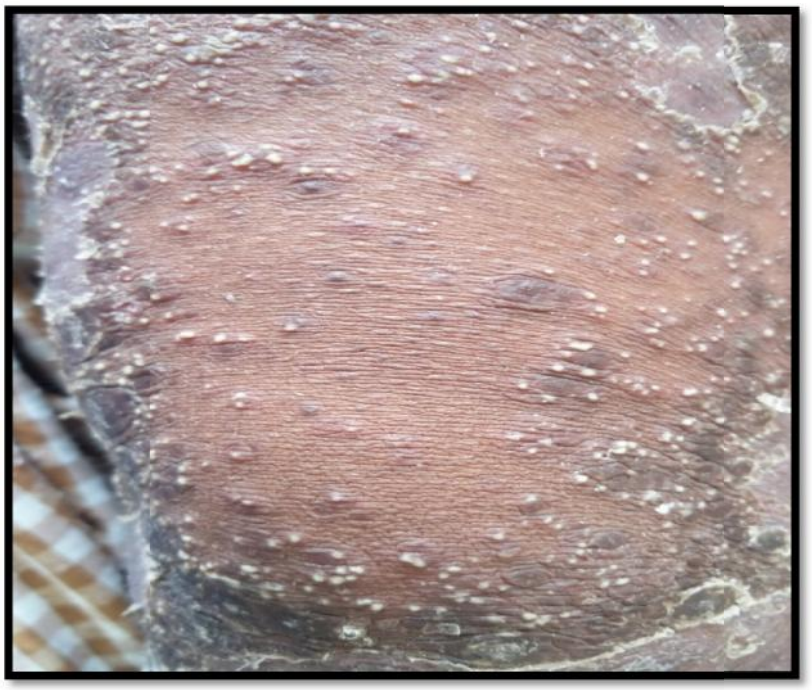


FIGURE 4: ERYTHRODERMIC PSORIASIS



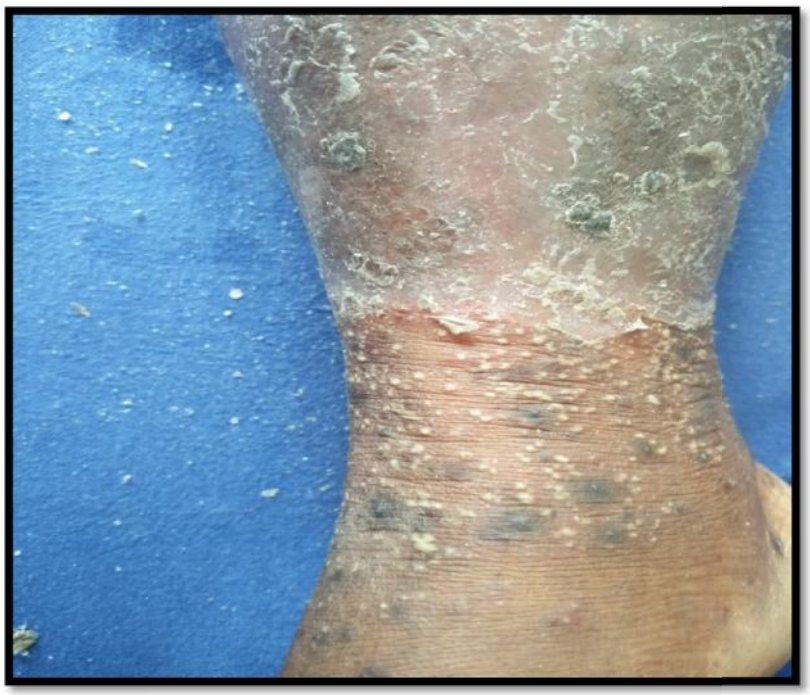


FIGURE: PUSTULAR PSORIASIS

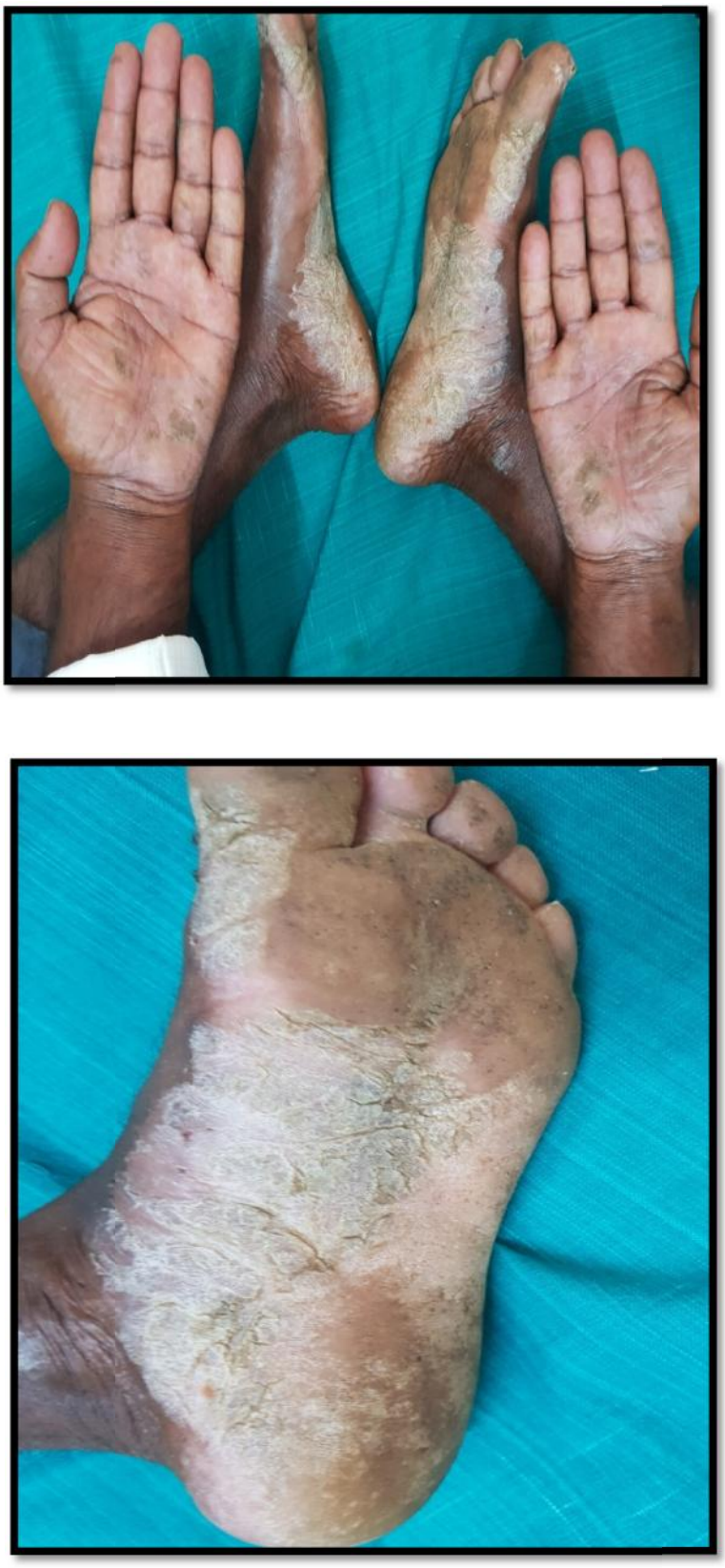


FIGURE 6: PALMOPLANTAR PSORIASIS

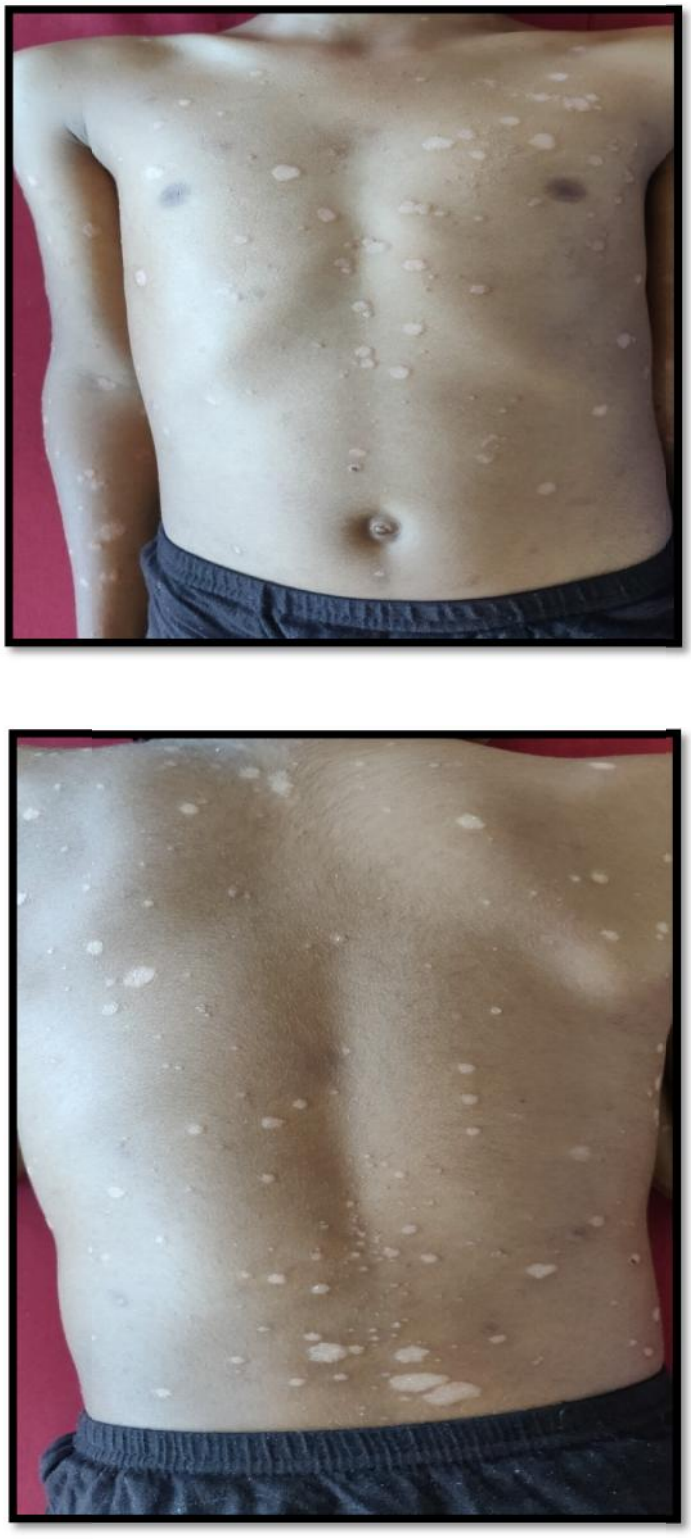


FIGURE 7: GUTTATE PSORIASIS

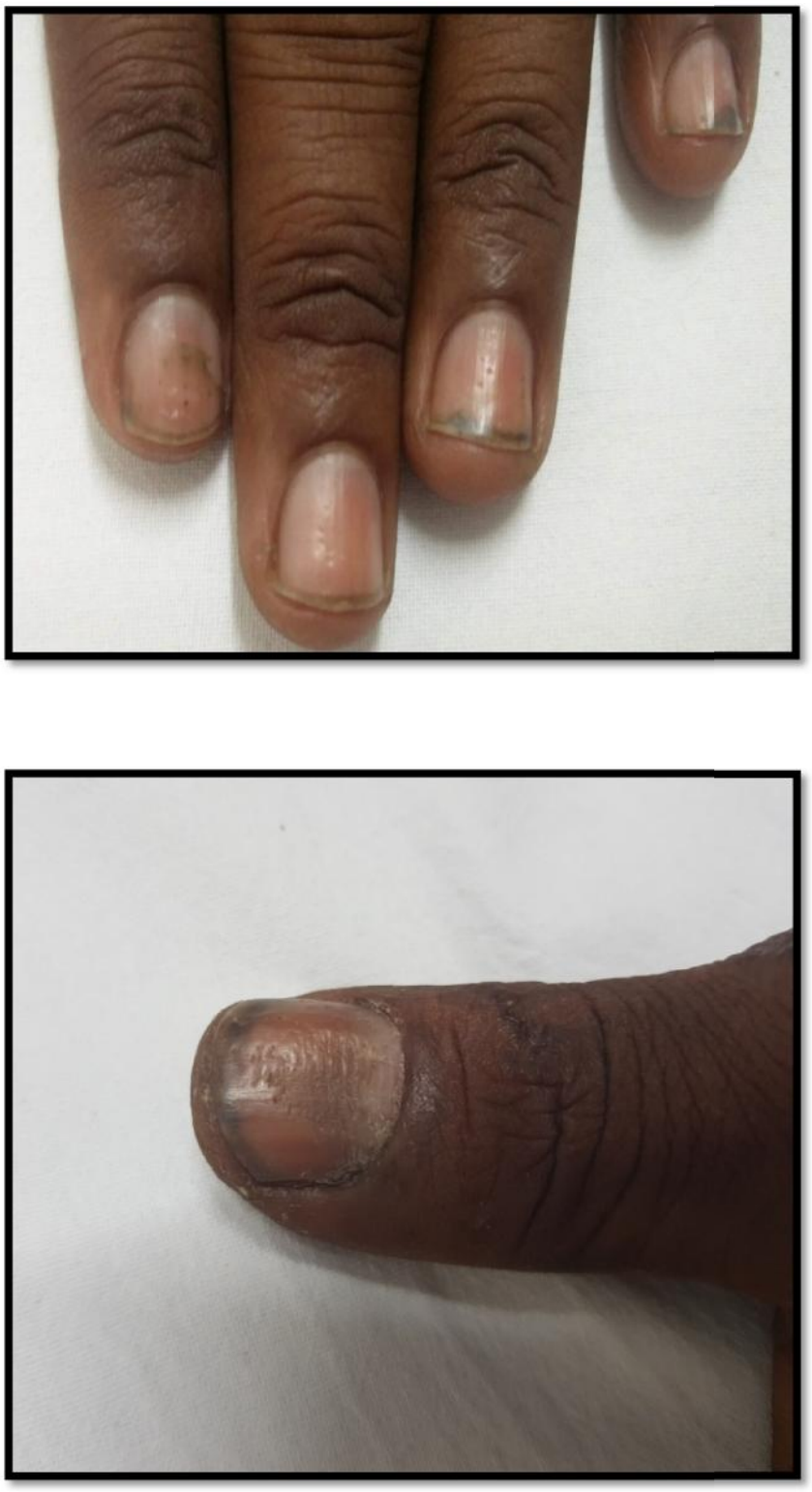


FIGURE 8 : NAIL PITS

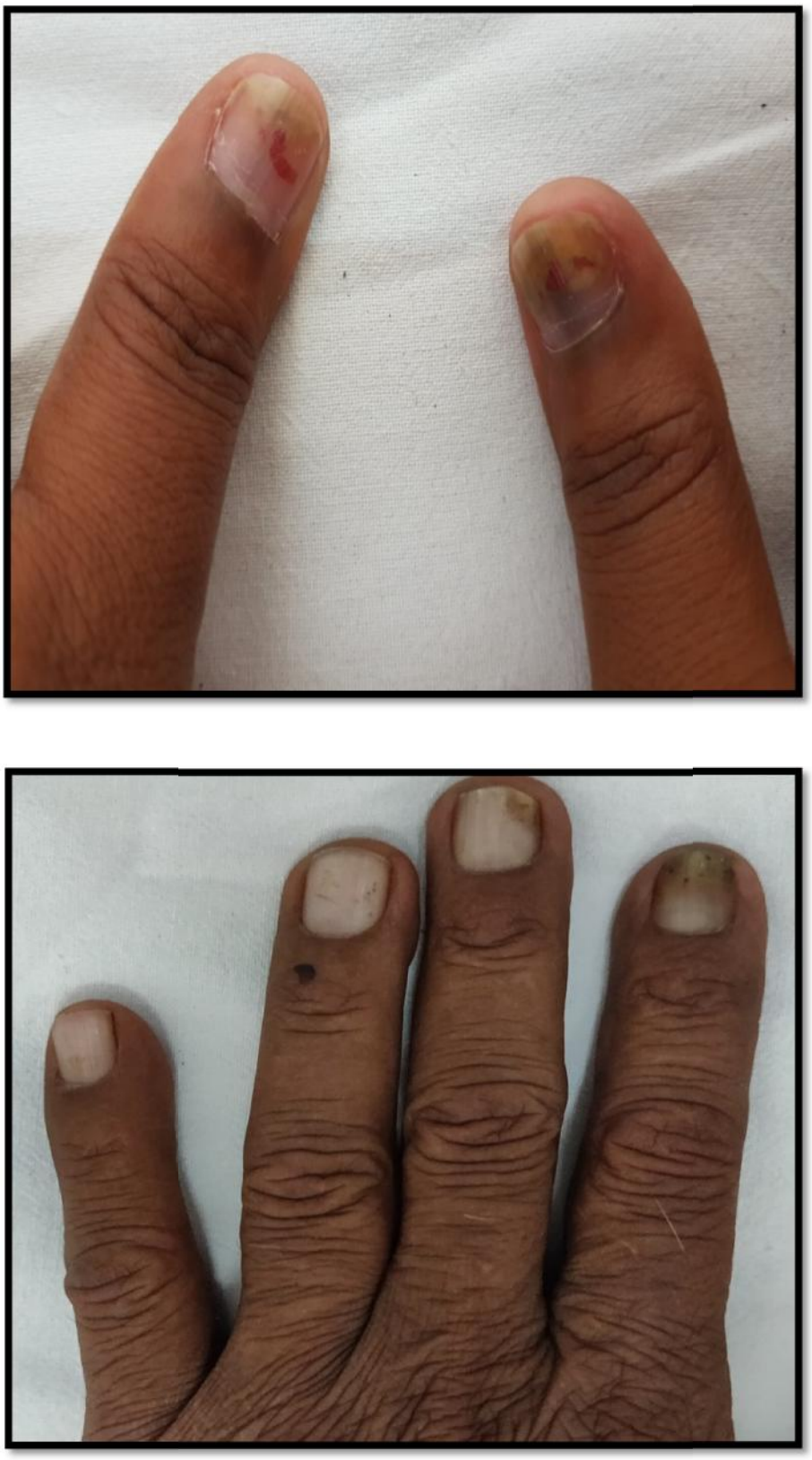


FIGURE 9: ONYCHOLYSIS

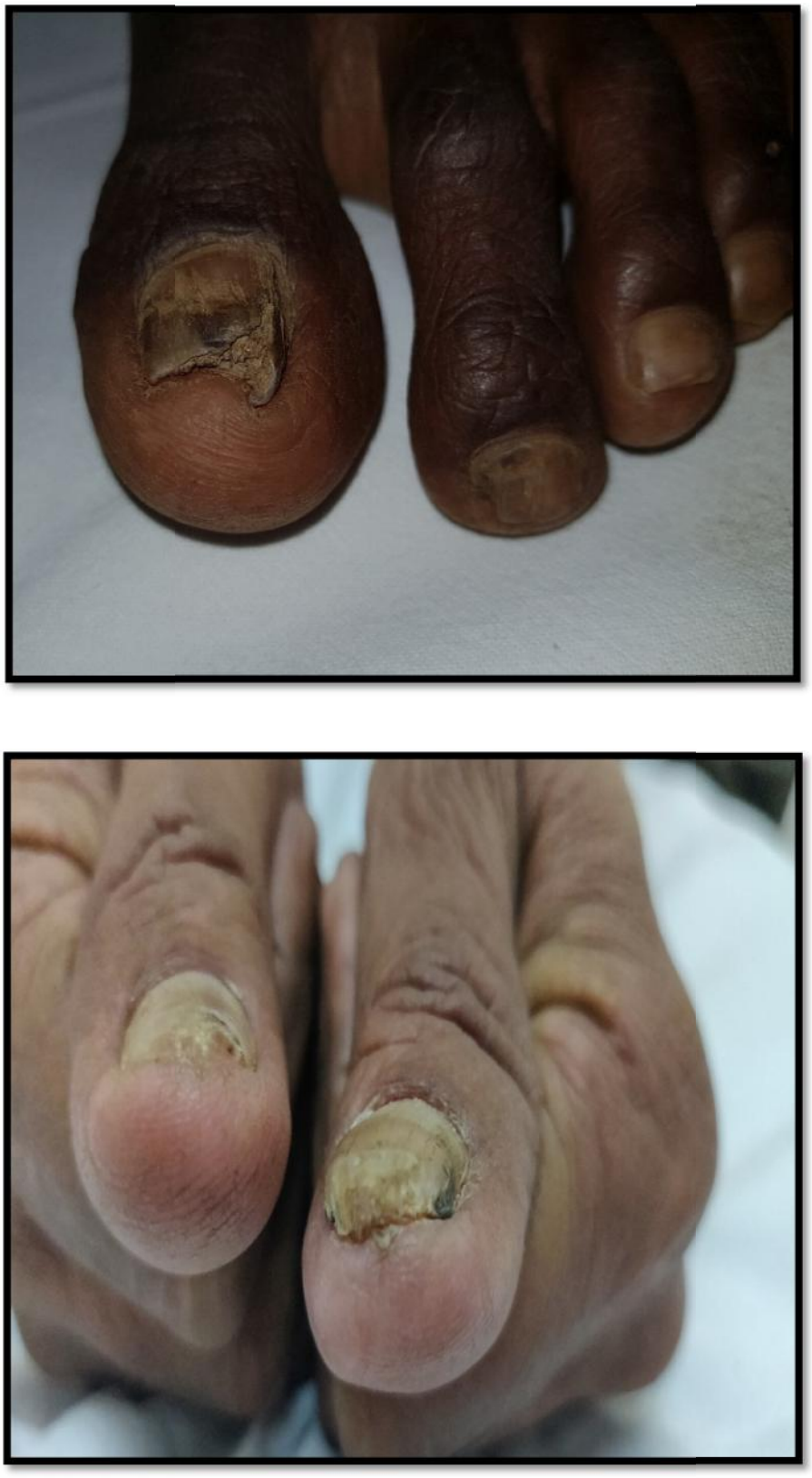


FIGURE 10 : SUBUNGUAL HYPERKERATOSIS

ANNEXURES V - MASTER CHART

sl no	AGE/SEX	DURN	TYPE	PASI			CA2+			ALB		JOINT INV	NAIL INV	DM	HTN
				0-10	10-20	>20	N	HYPO	HYPER	N	HYPO				
1	55/m	30YR	PPK	5.2			8.6				3.8	A	P	A	A
2	38/m	4MT	CHR PLQ			23		7.6			2.9	A	P	A	A
3	50/f	3MT	CHR PLQ		12.5		9.3				3.6	A	P	A	A
4	38/f	15YR	CHR PLQ			20.2	9.3				3.6	A	P	A	A
5	61/m	12YR	ERY					8.1			3.7	A	P	P	A
6	32/m	10 MT	CHR PLQ		19.5		8.8			4.6		A	P	A	A
7	35/f	6MT	PUST	5.8			9			4		A	P	A	A
8	35/m	2MT	GUTT	5			8.8				3.8	A	A	A	A
9	40/f	20YR	CHR PLQ			21.2	8.6				3.8	A	P	A	A
10	50/m	6YR	CHR PLQ		10.1		8.8				3.6	A	P	A	A
11	33/m	11YR	CHR PLQ		18		9				3.6	A	P	A	A
12	56/m	3YR	CHR PLQ		15.6		9.4				3.8	P	P	A	A
13	58/m	15YR	CHR PLQ			27.5		8.4			3.9	A	P	A	A
14	50/f	5YR	CHR PLQ		13.5		8.5				3.9	A	P	A	A
15	33/m	3YR	CHR PLQ		13.2		9.6				3.9	A	P	A	A
16	19/m	3MT	CHR PLQ		12.3		9.1				3.9	A	P	A	A
17	35/f	3YR	PUST		13.3		8.7				3.2	P	P	A	A
18	50/m	10YR	CHR PLQ			33.2		8.1			3.6	P	P	A	P
19	65/m	30YR	CHR PLQ	6.6			8.8				3.8	P	P	A	A
20	56/m	1YR	CHR PLQ	6.5			9.4				3.8	P	P	A	A
21	60/m	8YR	CHR PLQ		15.5		9.1			4.2		P	P	A	P
22	60/m	27YR	CHR PLQ	9.7				8.2			3.7	P	P	P	A
23	30/m	5YR	CHR PLQ			20.6	9.7			4.6		A	P	A	A
24	45/f	12YR	CHR PLQ		14		9.7			4.1		A	P	P	A
25	32/m	2YR	CHR PLQ			21.3	9.9			4.6		A	P	A	A
26	60/m	18YR	CHR PLQ		11.8		9.2				3.6	P	P	A	P
27	45/m	8YR	CHR PLQ			22.4		8.4			3.9	P	P	A	A
28	38/m	16YR	CHR PLQ		18.6		8.6				3.9	P	P	A	A
29	53/m	30YR	CHR PLQ	5.2			9.6			4.3		A	A	A	A
30	31/m	5YR	CHR PLQ		15.5		10			4.6		A	P	A	A

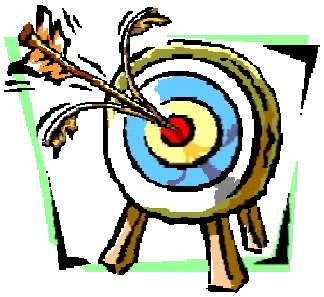
sl no	AGE/SEX	DURN	TYPE	PASI			CA2+			ALB		JOINT INV	NAIL INV	DM	HTN
				0-10	10-20	>20	N	HYPO	HYPER	N	HYPO				
31	43/m	10YR	CHR PLQ		19.8		9.4			4.1		A	P	A	A
32	33/f	5YR	CHR PLQ		17		9.6			4.5		A	P	A	A
33	62/m	6YR	CHR PLQ			33		8.3			3.8	A	P	A	A
34	65/m	20YR	CHR PLQ			20.6	9.2			4.3		A	P	A	A
35	33/m	17YR	CHR PLQ			21.8	9.4				3.8	A	P	A	A
36	55/f	2YR	CHR PLQ		12.7		8.8			4.3		A	P	P	P
37	28/m	10YR	CHR PLQ			22.2		8.3		4.6		A	P	A	A
38	27/f	8YR	CHR PLQ			26.4		8.2			3.9	A	P	A	A
39	15/m	2YR	CHR PLQ		17.7		9			4.2		A	P	A	A
40	48/m	10YR	CHR PLQ	4.8			9.5				3.6	A	P	A	A
41	30/m	4YR	CHR PLQ		14.7		10			4.6		A	P	A	A
42	65/m	2MT	CHR PLQ			24.5		8.3			3.1	A	P	A	P
43	58/f	6MT	ERY					8.2			3.7	A	P	A	P
44	37/m	9YR	CHR PLQ		16.4		8.9			4.9		P	P	A	A
45	37/f	5YR	CHR PLQ		13		9.3			4.3		A	P	A	A
46	51/m	8yr	CHR PLQ			26.5		8.2			3.7	A	P	A	A
47	42/f	12YR	CHR PLQ		15.4		9.7			4.2		P	P	A	A
48	49/m	5yr	CHR PLQ	7.4			9.6			4.4		A	P	A	A
49	50/f	4yr	CHR PLQ		11.2		8.8			4.2		A	P	A	A
50	50/m	18YR	ERY					8.2			3.8	A	P	A	P
51	52/m	12yr	CHR PLQ		10.5		9.3			4.2		P	P	A	P
52	65/m	3yr	CHR PLQ			22.7		8.4		4		A	P	A	P
53	41/m	16YR	CHR PLQ		13.5		9.5			4.8		P	P	P	A
54	44/m	13 YR	CHR PLQ		16		9.2			4.4		P	P	P	A
55	49/m	3YR	ERY				9.1				3.5	A	P	P	A
56	40/m	10YR	ERY				8.7				2.9	A	P	A	A
57	36/m	10 YR	CHR PLQ		16.8		9.6			4.1		A	P	A	A
58	43/m	5yrs	CHR PLQ	8.6			9.2			4.3		P	P	A	A
59	65/m	30YR	CHR PLQ		16.3		8.9				3.9	P	P	A	A
60	32/m	2yr	CHR PLQ			22.3	8.8				3.9	A	P	A	A

ANNEXURE-VI

KEY TO MASTER CHART

M	-	Males
F	-	Females
DM	-	Diabetes Mellitus
HTN	-	Hypertension
PASI	-	Psoriasis area severity index
DURN	-	Duration
CHR PLQ	-	Chronic plaque psoriasis
PPK	-	Palmoplantar keratoderma secondary to psoriasis
GUTT	-	Guttate psoriasis
ERY	-	Erythrodermic psoriasis
PUST	-	Pustular psoriasis
HYPO	-	Hypocalcemia, hypoalbuminemia
N	-	Normal
HYPER	-	Hypercalcemia

Introduction



Objectives

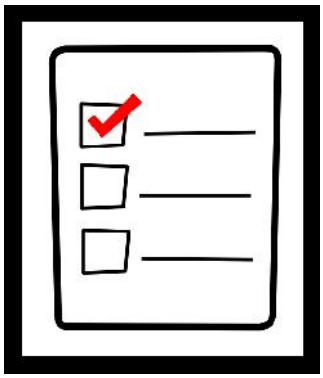
Review of Literature

Methodology

Results

Discussion

Conclusion

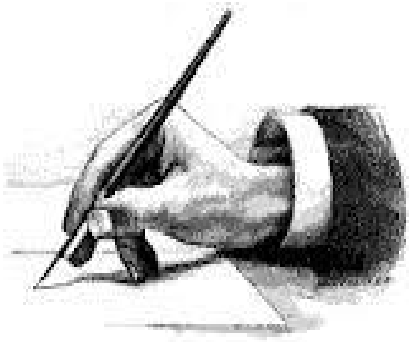


Limitations

Recommendations

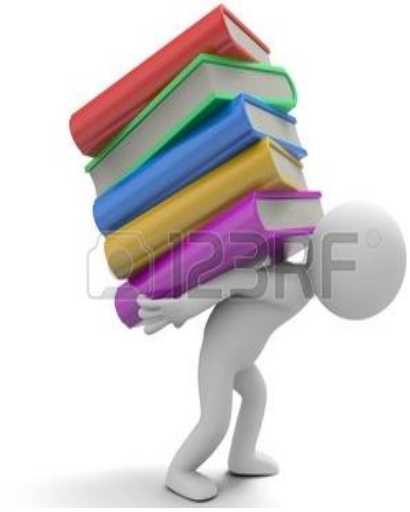
Summary

Bibliography



Annexure-I

Annexure-II



Annexure-III

Annexure-IV

Annexure-V