CORRELATION OF HEMATOLOGICAL MARKERS IN PSORIASIS WITH THE SEVERITY OF THE DISEASE

BY

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ABSTRACT

Background: Psoriasis is a chronic and recurrent disease that affects the skin, joints, and nails, characterised as an immune-mediated disorder with systemic involvement. This can lead to changes in the haemotological parameters as well. The psoriasis area and severity index (PASI) is a tool that assesses the severity of psoriasis lesions and the therapeutic response of the patients. Various studies have shown correlation between the PASI and haematological parameters.

Aim: To assess the haematological parameters- platelet-lymphocyte ratio (PLR), platelet distribution width (PDW), neutrophil-lymphocyte ratio (NLR), red cell distribution width (RDW), mean platelet volume (MPV) and plataelet count (PLT) in psoriasis patients. These values will also be correlated with the disease severity.

Materials and methods: A cross-sectional case-control study was conducted from May 2023 to January 2025 on 260 psoriasis patients and controls. The study was done at the Department of Dermatology, Shri BM Patil Medical College Hospital and Research Center, Vijayapura. Complete hemogram was done for all the study participants and the parameters were correlated with PASI in the cases. Additionally, the parameters were compared with that of controls.

Results: PLT and NLR ($p = 0.045$ and $p = 0.002$ respectively) had a statistically significant positive correlation with PASI. On comparison of cases to controls, a statistically significant elevation was seen in the MPV ($p = 0.012$) and NLR ($p = 0.012$) in cases.

Conclusion: NLR, PLT and MPV can be used as cost-effective and affordable laboratory markers in the assessment of disease severity in psoriasis, in conjunction with PASI (NLR, PLT).

LIST OF ABBREVIATIONS:

PsV	Psoriasis vulgaris
PP	Pustular psoriasis
PsoEry	Psoriatic erythroderma
PASI	Psoriasis area and severity index
IL-17	Interleukin -17
NLR	Neutrophil- to -lymphocyte ratio
PL	Platelet- to -lymphocyte ratio
MPV	Mean platelet volume
RDW	Red cell distribution width
PDW	Platelet distribution width
MHC	Major histocompatibility complex
HLA	Human leucocyte antigen
GWAS	Genome-wide association studies
TCR	T-cell receptor
AMP	Anti-microbial peptide
pDCs	Plasmacytoid dendritic cells
PLA2G4D	Phospholipid A2 group IVD

PsV	Psoriasis vulgaris
PP	Pustular psoriasis
PsoEry	Psoriatic erythroderma
PASI	Psoriasis area and severity index
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PDW	Platelet distribution width
MHC	Major histocompatibility complex
HLA	Human leucocyte antigen
GWAS	Genome-wide association studies
TCR	T-cell receptor
AMP	Anti-microbial peptide
pDCs	Plasmacytoid dendritic cells
PLA2G4D	Phospholipid A2 group IVD

PsA	Psoriatic arthritis
RA	Rheumatoid arthritis
BSA	Body surface area
HPA	Hypothalamic- pituitary- adrenal
MTX	Methotrexate
PUVA	Psoralen plus UVA
TNF	Tumour necrosis factor
JAK	Janus kinase
PDE4	Phophodiesterase 4
H	Head
UL	Upper limb
T	Trunk
LL	Lower limb
WBC	White blood cells
Hb	Haemoglobin
CRP	C- reactive protein
SD	Standard deviation
IQR	Interquartile range
PPP	Palmoplantar psoriasis

SCALP Pso	Scalp psoriasis
Guttate Pso	Guttate psoriasis
Plantar Pso	Plantar psoriasis
FOLLICULAR Pso	Follicular psoriasis
Palmar Pso	Palmar psoriasis
Scalp Pso	Scalp psoriasis
Flex Pso	Flexural psorasis
GPP	Generalized pustular psoriasis
NET	Neutrophil extracellular traps

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INTRODUCTION:

Psoriasis is a chronic, immune-mediated condition that impacts the skin, nails, and occasionally the joints. [1] Psoriasis is categorized into on the basis of age of onset, morphology and sites of involvement. The Psoriasis Area and Severity Index (PASI) is a commonly utilised scoring system for evaluating disease severity among individuals with psoriasis. [2] It evaluates the degree of erythema, induration, and desquamation in the impacted regions of the body. [2] Although its use among clinicians is highly variable, its utilisation is limited. Achieving uniformity in diagnosing and assessing the severity of psoriasis remains a significant challenge.[2]

Systemic inflammation is pivotal in the pathogenesis of psoriasis, with numerous mediators involved, such as interleukin 1 and IL-17.[3] Despite advances in understanding the pathophysiology of psoriasis, there is a pressing need to identify and treat improved markers for the severity and outcomes of psoriasis.[4] Two promising candidates are the neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR). [5] They are cost-effective, readily quantifiable, and reliable indicators of subclinical inflammation.[5] PLR and NLR have been recognised as possible prognostic indicators in various chronic inflammatory diseases and malignancies.[5]

Recent studies have explored the relationship between PLR, NLR, and the severity of psoriasis.[6] Platelet activation is integral to immunological inflammatory processes, with the mean platelet volume (MPV) acting as a significant indictator of platelet activation.[5] Red blood cell distribution width (RDW), quantifies the variability in red blood cell size, it has been documented to be increased in patients with psoriasis.[7] The influence of psychosocial

comorbidities in psoriasis patients underscores the necessity for comprehensive treatment approaches. These should encompass medical care psychological support and education about psoriasis vulgaris to address the overall burden of the condition.[8] Therefore, identification of haematological parameters as markers for disease severity could facilitate timely interventions to improve patient care.

Although numerous markers related to psoriasis have been studied, there has not been any definitive biomarker identified until date. Studies comparing haematological parameters such as NLR, PLR, RDW, PDW, PLT and MPV in psoriasis patients remain unexplored. The prospective relevance of these haematological indicators as a cost-effective approach for evaluating the disease severity in psoriasis is yet to be explored.[9] This study holds epidemiological significance for improving the management and assessing the prognosis of psoriasis vulgaris patients in India.

AIMS AND OBJECTIVES:

1. To assess the haematological parameters—PLT, PDW, RDW, NLR, PLR, and MPV, in individuals with psoriasis and correlate the values of each with disease severity.
2. To compare the haematological parameters of cases to controls.

REVIEW OF LITERATURE:

EPIDEMIOLOGY:

Around 2–3% of the world population is affected by psoriasis.[10]

Owing to the immense diversity of India, prevalence rates can differ across regions based on genetic and environmental factors.[1] Research conducted in North India, primarily in hospital populations, has estimated the prevalence of psoriasis in adult dermatologic patients (Table1).[10] Psoriasis is predominantly observed in males, with peak onset 30-40 years of life.

EPIDERMIOLOGICAL STUDIES OF PSORIASIS IN ADULTS IN INDIA						
	Okhandiar et. al. 1963	Bedi et al.; 1997	Kaur et al; 1986	Bedi et al.; 1995	Kaur et al.;1997	Asokan et al; 2011
Number of patients	3573	162	782	530	1220	275
Prevalence (percentage of total dermatological outpatients)	1.02	0.8	1.4	2.8	2.3	-
M:F ratio	2.46 : 1	2.5 : 1	2.3 : 1	2.4 : 1	2.03 : 1	2.9 : 1
Peak age of onset	Third and fourth decade	Third and fourth decade	-	Third and fourth decade	-	38.9 + -14.5 years
Mean age of males and females	Comparable	Reduced in females	Reduced in females	-	Reduced in females	Males→ 40.3+-13.4 years" "Females- 34.7+-16.4

Table 1: Epidemiological study of psoriasis in adults in India. [10]

A single large-scale Northern Indian study estimated paediatric psoriasis prevalence at 0.0002%.[10] The age of onset for boys is peaked betweeen 6 and 10 years. For girls, it is between 11 and 15 years.[10] A family history of psoriasis can be found in 9.8–28% of children.[10] Psoriatic arthritis usually occurs between 35 and 50 years, with no marked predilection for either sex.[10] Psoriasis precedes the onset of psoriatic arthritis in about 70% patients.[11] In 15% of patients, arthritis occurs more than one year before the onset of psoriasis and remaining 15%, both diseases occur within one year of one another.[10]

The prevalence and incidence of psoriatic arthritis have been estimated to range from 3.0–23.1 per 100,000 annually and 1–420 per 100,000 individuals, respectively and similar results have been reported in Western nations and China.[12,13] A systematic review by Prey et al. showed that psoriatic arthritis affected up to 24% of patients suffering from psoriasis.[14] Data of this sort are not available for Indian people as of yet. In children, arthritis can be antecedent to psoriasis in 50% of cases, with an average age of onset at 9–10 years and showing a female preponderance.[15]

Pathogenesis

Psoriasis is a complex disease with a significant genetic component, evidenced by familial clustering and the identification of susceptibility loci across many chromosomes.The MHC region on-chromosome 6p21, (PSORS1) is one of the most critical loci.[16] PSORS1 encodes the human leukocyte antigen-C (HLA-C) gene.[16] The HLA- Cw6 allele is linked to psoriasis. It indicates an earlier age of onset. It has been suggested that Cw6-positive women may develop psoriasis earlier than Cw6-positive men, though this pattern is not observed in Cw6-negative individuals.[17]

The activation of the immune system in psoriasis was initially suggested by evidence of increased inflammatory infiltrates in psoriatic lesions.[18] Further evidence corroborating this concept was derived from cases where psoriasis improved following bone marrow transplants or immunosuppressive treatments, including cyclosporine and methotrexate.[19]

Research has demonstrated that these infiltrates consist predominantly of CD4+ and CD8+ T lymphocytes.[18] A definitive link between T-cell activation and psoriasis was established by targeted therapies.[20] The selective suppression of activated T cells using the fusion protein DAB389IL-2 reinforced their classification as pathogens.[18,20] Clinical studies have demonstrated that immunomodulatory therapies, such as abatacept (a CTLA-4 Ig fusion protein), alefacept (a CD2-targeted LFA-3/Fc fusion protein), and efalizumab (an anti-CD11a monoclonal antibody), effectively reverse psoriasis, thereby reinforcing the characterisation of the disease as T-cell-mediated. [18,20]

Genome-wide association studies (GWAS) have identified several loci associated with psoriasis susceptibility, with HLA-C*06:02 emerging as the most significant. To date, over 63 susceptibility loci have been discovered in European populations, yet these account for only about 50% of psoriasis heritability.[21] The lack of expanded T-cell receptor (TCR) clones and the inability to identify consistent autoantigens initially cast doubt on the classification of psoriasis as a classical autoimmune disorder.[21] Recent findings of psoriasis-associated autoantigens, however, have resuscitated this hypothesis.[21]

Psoriasis autoantigens

Lande et al. reported that autoreactive CD4+ and CD8+ T cells recognising LL-

37/cathelicidin (cationic antimicrobial peptide) was detected in 75% of patients with moderate and severe psoriasis.[22] Upragulation of LL-37 expression was seen in psoriatic plaques, and it was correlated with disease severity.[18] This peptide interacts with nucleic acids (DNA/RNA) released during skin injury, activating plasmacytoid dendritic cells (pDCs) and myeloid dendritic cells (mDCs) through toll-like receptors.[18]
This stimulates augmented production of interferon-alpha (IFN-α) and pro-inflammatory cytokines TNF and IL-6.[18] AMPs like human β-defensin 3 (hBD3) , lysozyme, human β-defensin 2 (hBD2), and have also been involved in such pathways.[18]

Another autoantigen identified is ADAMTS-like protein 5 (ADAMTSL5), which was reported by Arakawa et al.[23]This secreted zinc metalloprotease-related protein is melanocyte expressed and has been reported as an autoimmune target in HLA-C*06:02–positive patients.[23] It stimulates IL-17–producing T cells, further propagating psoriatic inflammation.[23] Further studies indicate that keratinocytes and dendritic cells are also involved in antigen presentation of ADAMTSL5.[24]

Phospholipase A2 group IVD (PLA2G4D) has recently been identified as a potential autoantigen in psoriasis.[25] In contrast to other phospholipase enzymes, PLA2G4D is upregulated specifically in psoriatic plaques and plays a role in the presentation of neolipid antigens through CD1a-bearing dendritic cells.[26] These antigens induce autoreactive T-cell activation, resulting in elevated IFN-γ and IL-17A production.[26] PLA2G4D co-localizes with mast cell tryptase, and mast cell–derived exosomes can deliver neolipid antigens to antigen-presenting cells.[26] This finding highlights the potential role of non-peptide lipid antigens in psoriasis, alongside peptide autoantigens such as ADAMTSL5 and LL-37.[26]

Risk factors

In psoriasis, genetic imprinting of the transmitting parent's sex is intriguing.[27] Research demonstrates that birth weight of the child is affected by the gender of the affected parents, with children of the affected fathers exhibiting greater weight than those of the affected mothers.[27] Furthermore, disease expression seems more common when the father is affected.[27]Some scientists suggest that the genes involved in this imprinting can predispose to enhanced disease activity in men, though evidence on this topic is still scarce.[27]

As noted above, psoriasis arises as a result of an intricate interplay between genetic susceptibility and extrinsic stimuli. Extrinsic stimuli include trauma, psychological tension, infections such as streptococcus, HIV, and various viruses, as well as certain drugs like lithium, antimalarials, interferon, beta-blockers, NSAIDs, and the abrupt withdrawal of high-dose steroids.[28] A potential association exists between hepatitis C virus infection and psoriasis. Stressful life events have long been implicated as a trigger for psoriasis, with one report to show an increased risk in women with high stress in the preceding year.[29]

Alcohol is another potential risk factor, often associated with moderate-to-severe psoriasis.[28] Excessive alcohol consumption appears to worsen disease progression and treatment outcomes. Patients with psoriasis, regardless of gender, generally exhibit higher alcohol consumption compared to control groups; however, statistical significance has been established solely for male patients. [28]

Smoking contributes to the development of psoriasis, exhibiting a stronger cumulative association in women.[30] One study, though, found smoking to be a risk factor solely for

men.[30] Recent evidence establishes smoking as a risk factor for psoriasis, demonstrating no significant differences between younger or older women and even across genders.[30] Other research points to a positive correlation with adult passive smoking exposureand increased risk for ex-smokers, especially men.[30]

Clinical classification of psoriasis

Psoriasis is divided into two broad types: pustular and non-pustular forms.[31]

1) Non-pustular forms[31]

Psoriasis vulgaris (early onset and late onset)

Erythrodermic psoriasis

Palmoplantar psoriasis

Guttate psoriasis

Inverse psoriasis

Psoriatic arthritis

2) Pustular psoriasis

Generalized pustular psoriasis (von Zumbusch type)[31]

Impetigo herpetiformis (pustular psoriasis in pregnancy)

Localized types of pustular psoriasis:

Palmoplantar (pustular) psoriasis (Barber type)

Acrodermatitis continua of Hallopeau

Psoriasis vulgaris

Psoriasis vulgaris represents the predominant clinical type, constituting nearly 90% of cases. The characteristic lesions are plaques which are erythematous with distinct borders and a typical silvery-white scale.[1] Lesions symmetrically involve extensors sites like the elbows, knees, sacral area, and scalp. The preference for extensor involvement could be associated with friction or trauma.[31]

Scraping of psoriatic plaques with a dull scalpel removes layers of white, waxy scales, which have the appearance of candle wax—a wax spot phenomenon. More scraping unmasks a thin, moist layer named as “Bulkeley’s membrane.” Additional scraping unearths pinpoint bleeding points, which are described as "Auspitz sign," showing papillary capillary involvement. Surrounding healing plaques, a hypopigmented macular ring, the "Woronoff ring," can occur, possibly due to prostaglandin decrease during healing.[3233]

Guttate psoriasis

This type primarily manifests in the younger age group as small, raindrop-shaped erythematous lesions,which commonly occurs post streptococcal infections. [31] It has a strong association with the HLA-Cw6 gene. High antistreptolysin titers are common. The lesions typically resolve spontaneously within three to four months or they can sometimes they can evolve into psoriatic plaques.[31]

Erythrodermic psoriasis

This type of psoriasis is marked by extensive erythema affecting around 90% of the body surface area.[34] It is linked with profound systemic consequences, such as hypothermia, loss of protein, and possible organ failure. It can arise as a complication of psoriasis vulgaris or as an independent development. The patient can present with dramatic changes in the nails, lymphadenopathy, and intense pruritus. Because of the high risk of cardiovascular or septic shock, careful monitoring is necessary.[31,34]

Palmoplantar psoriasis

This variant of psoriasis involves the palms and soles, especially the thenar areas. Squamous lesions are the hallmark, occasionally with a keratoderma-like appearance.[31,34]
Unlike in other forms, Auspitz and wax spot phenomena do not occur.[3431]

Psoriatic arthritis (PsA)

Psoriatic arthritis (PsA) has a prevalence of 5.4–7% among individuals with psoriasis, reaching 30–40% in cases of severe pustular psoriasis.[35] It typically starts in after 30 years and has a male-to-female ratio of 1:1.[35]
80% of PsA patients have involvement of the nail.[31]Moll and Wright decribed the five classic subtypes of psoriatic arthritis.[35]

Types of Psoriatic arthritis:

Classical PsA: Involves distal interphalangeal joints, frequently involving the nails.

Asymmetric oligoarticular Arthritis: The most typical form, involving major and minor joints in an asymmetric distribution.[32]

Symmetric polyarthritis: Similar to rheumatoid arthritis (RA) but unlike it by occurring more often distally in interphalangeal joints.[32]

Arthritis mutilans: A severe deforming form with progressive phalangeal osteolysis and potential sacroiliitis.[32]

Spondylitic PsA: Only infrequently occurs in isolation; frequently associated with peripheral arthritis and asymmetric sacroiliac joint disease. Prognosis is usually superior to ankylosing spondylitis. [32]

Inverse psoriasis

This variant occurs in skin creases (flexural areas) as discrete, shiny red, non-squamous plaques. [32] It occurs more frequently in obese persons and usually co-occurs with seborrheic dermatitis. The usual treatments might be less responsive for this variant.[36]

Generalized pustular psoriasis

Generalized pustular psoriasis (GPP) is characterized by the presence of extensive sterile pustules on an erythematous background. [31] Systemic symptoms accompany GPP such as-malaise, fever and polyarthralgia.[31] GPP occur spontaneously or as a response to stimuli such as steroid withdrawal, hypocalcemia, or

irritant therapy. If left untreated, it can be fatal.[32]

Impetigo herpetiformis

This is an uncommon variant known as generalised pustular psoriasis of pregnancy, which typically manifests during the third trimester or following delivery..[31,32] Impetigo intially starts over the flexural areas as erythematous area with pustules and later disseminates to other regions.[37] Formation of subungual pustules may lead to nail changes such as onycholysis. Patients can experience burning, itching and emanation of a malodour.[37] Accompanying systemic manifestations also occur such as fever, weakness, chills, nausea, and vomiting.[37] Complications of impetigo herpetiformis include- malnutrition, hypoalbuminemia, hypocalcaemia, dehydration and electrolyte imbalance.[32]

Localized pustular psoriasis

Localized pustular psoriasis is divided into two subtypes: Palmoplantar pustulosis and Acrodermatitis continua of Hallopeau.[37]

Palmoplantar pustulosis (PPP):

PPP is a subtype of localizaed pustular psoriasis which is chronic and recurrent. It is more common in women and associated with a positive family history of PPP.[38] PPP starts as tiny pustules over an erythematous background localized to the palms and soles, particularly to the thenar and hypothenar regions.[38] The precise aetiology remains unclear; however,

underlying contact sensitivity appears to be a significant factor.[38]

Several provoking factors such as smoking, tonsillitis, humidity and heat can precipitate the condition.

Acrodermatitis continua of Hallopeau:

Acrodermatitis continua of Hallopeau is a rare variant, characterized by the eruption of sterile pustules over the one or more digits. [39] It is marked by a chronic clinical course. Other names are-, acrodermatitis perstans, acrodermatitis continua suppurativa, dermatitis perstans, dermatitis repens and acropustulosis.[40,39] Infections or local trauma can be an inciting factor although, neurological and inflammatory causes have also been documented.[41]

TREATMENT:

Therapeutic targets encompass the enhancement of skin, joint, and nail lesions, alongside the improvement of quality of life in patients.[42] The standard treatment for psoriasis often comprises topical and systemic therapies. [42] Patients with mild disease with <10% of body surface area (BSA) are treated with topical medicaments. [43] Topical therapies for psoriasis consist of corticosteroids, calcineurin inhibitors, vitamin D3 analogues, and others such as emollients, salicylic acid, coal tar and anthralin (irritants avoided in unstable forms of psoriasis).[43] Advanced disease warrants systemic therapy. Age, gender, pregnancy status etc. are some of the factors to be taken into consideration while choosing the appropriate systemic treatment.[42]

Treatment options:

1. Corticosteroids

Corticosteroids are prescribed as the primary treatment for mild-to-moderate psoriasis.[32] Corticosteroids reduce alleviates the inflammation and itching by cell turnover by suppressing the immunological response.[32] It is given as monotherapy or in conjunction with systemic agents.[44]

Low-potency formulations should be utilised in delicate areas such as the intertriginous areas and the face.[44] Few adverse effects are- epidermal atrophy, telangiectasia, open- angle glaucoma. Clobetasol propionate (0.05%), betamethasone dipropionate and valerate (0.1%-1%), and mometasone furoate, halobetasol proprionate (0.05%) are most commonly used topical corticosteroids.[44]

2. Vitamin D Analogues

Calcitriol and calcipotriene are good long-term substitutes for corticosteroids.[45] They bind to the cytoplasmic vitamin-D receptor, which induces nuclear transcription of genes that regulate cell differentiation and inflammatory processes.[45] Although usually safe, they can induce perilesional erythema and irritation and, in a few instances, increase serum and urine calcium levels. Not more than 100 grams should be applied weekly.[45] Calcitriol is more effective, but calcipotriene is more established.[45] Combination therapy with corticosteroids has been more effective than monotherapy.[45]

3. Dithranol (anthralin)

Dithranol (1,8-dihydroxy-9-anthrone) is a natural compound derived from the bark of the araroba tree, which is indigenous to South America.[43] Dithranol is available in the form of creams, ointments and pastes. Dithranol is approved for chronic plaque psoriasis.[43] It can be used alongside UVB phototherapy with positive outcomes (the Ingram regimen). Commonly encountered adverse effects are- contact dermatitis (irritant) and discolouration of clothing, skin, hair, and nails.[43] Anthralin exhibits antiproliferative effects on human keratinocytes as well as strong antiinflammatory properties.

Traditional anthralin treatment begins with low concentrations (0.05%–0.1%) mixed in petrolatum or zinc paste and applied once each day.[45]

4. Coal Tar

The utilisation of tar for treating dermatological conditions dates back over 2000 years.[43] The mechanism of action is not clear. Owing to its inherent chemical complexity, coal tar lacks pharmacological standardization.[43]

Coal tar is available for use as creams, ointments, and pastes and the concentrations range from 5% to 20%.[32] Adverse effects include-Occasionally, sensitivity that can result in folliculitis, an undesirable odour and appearance, which may cause staining on textiles. [32] It also has carcinogenic effects.[32]

5. Retinoids

Acitretin is an oral second-generation retinoid employed in the management of moderate to severe psoriasis.[42] It is the drug of choice in pustular psoriasis and also used in conjunction with other topical treatments. Owing to the teratogenic properties of acitretin, women are counselled to avoid pregnancy for three years after discontinuing the medication..[42]

6. Methotrexate

Methotrexate is an antimetabolite that is approved for the treatment of psoriasis and psoriatic arthritis.[46] It must be used with caution in women of childbearing age group as it is a teratogen. Serial monitoring must be done at each visit with complete hemogram and liver function tests to watch for toxicity. [46] The dosage of methotrexate is 0.3 mg/kg/week. In psoriasis patients, it can be given subcutaneously or orally. [46] Patients must be supplemented with folic acid while on treatment with methotrexate. [46]

7. Cyclosporine

Cyclosporine is an immunosuppressive agent derived from the soil fungus Tolypocladium inflatum gams. [32] It acts against T-lymphocytes and IL-2. It is FDA approved for severe, recalcitrant and disabling psoriasis. [32] Dosing is 3-5 mg/kg/day and the common adverse effects are renal dysfunction and hypertension. [32]

8. Phototherapy

Reserved for those with severe or resistant cases or patients failing to respond to topical agents or with psoriasis with ≥20% of body surface area, phototherapy activates apoptosis and augments IL-10 expression by keratinocytes.[42] Ultraviolet B (UVB) therapy utilising anthralin (Ingram regimen) or coal tar (Goeckerman therapy) is efficacious in moderate-to-severe psoriasis.[42] Psoralen plus ultraviolet A (PUVA) is very effective but poses a high risk of skin cancer. Adverse effects include burns, erythema, pruritus, xerosis, and dermal ageing.[42]

Newer therapies- Biologicals and targeted agents-

Biological therapy consists of medications that are proteins generated by living entities to focus on particular aspects of the inflammation cascade, such as antibodies directed at cell surface markers, cytokines, and adhesion molecules.[47]

Tumor necrosis factor alpha (TNF-alpha) inhibitors

They inhibit TNF-α, an important inflammatory mediator in psoriasis. TNF-α activates innate and acquired immune responses thereby causing chronic inflammation, keratinocyte proliferation and tissue damage.[48] The following are the TNF-α inhibitors used in psoriasis:

Infliximab: It is a chimeric monoclonal antibody made up of a murine variable region and a human IgG1-alpha constant region. [48] It neutralizes both soluble and transmembrane TNF-

alpha molecules through binding. [48] It is authorised for adults for the treatment of psoriasis, psoriatic arthritis, ankylosing spondylitis and rheumatoid arthritis.[48]

Etanercept

It is a recombinant human TNF-α receptor protein. It binds to both soluble and membrane-bound TNF-α, as well as tumour necrosis factor-β.[48] It is currently approved for the management of moderate-to-severe plaque psoriasis in adults and children. [48] Additionally, it is also approved for use in psoriatic arthritis, ankylosing spondylitis, juvenile rheumatoid arthritis, rheumatoid arthritis. Dosage for psoriasis- 50 mg subcutaneously twice weekly for the first 12 weeks, followed by 50 mg weekly.[48]

Adalimumab, Certolizumab pegol, and **Golimumab** are other biologicals that provide further TNF-α blockade with varying efficacy and adverse effect profiles.

Interleukin-23 and Interleukin-12 inhibitors inhibit cytokines that are crucial to T-cell development.

Ustekinumab: It is a synthetic monoclonal antibody that inhibits the interaction with the receptors after binding to the p40 component of IL-12 and IL-23.[32] Ustekinumab inhibits IL-12 which is essential for Th1 differentiation. However, it has a more significant suppressive impact on IL-23. IL-23 promotes persistent inflammation driven by Th17 and Th22 cells.[32]

Guselkumab specifically inhibits IL-23, hence decreasing the production of IL-22, IL-17F,

and IL-17A.[48]

IL-17 Pathway Inhibitors

Brodalumab: Binds to IL-17RA, suppressing IL-17A/F signaling. FDA approved for moderate-to-severe psoriasis; linked to increased suicidal thinking.[47,48]

Secukinumab and Ixekizumab: IL-17A monoclonal antibodies effective for moderate-to-severe cases. Side effects are upper respiratory infections and neutropenia.[47,48]

Other drugs:

Tofacitinib: It belongs to a group of drugs called as Janus kinase (JAK) inhibitors. It is approved for the treatment of psoriatic arthritis; it works by inhibiting the Th17 differentiation.[49]

Alefacept: Alefacept inhibits T-cell activation and induces apoptosis. It can be administered taken intramuscularly or intravenously on a weekly basis. [50] Adverse effects are- lymphopenia and hepatotoxicity.[50]

Apremilast: It belongs to phosphodiesterase-4 (PDE4) inhibitor group of drugs. [51] Apremilast acts via increasing the cAMP levels and thereby curbs inflammation. [51] The approved indications are- psoriasis and psoriatic arthritis. Gastrointestinal disturbances and weight reduction are few of the adverse effects.[51]

PASI

Fredriksson and Pettersson created the PASI in the year 1978 to evaluate the therapeutic effect of retinoids on psoriasis. [52] For calculation of PASI, the body is divided into 4 regions- the head and neck (H), trunk (T), lower limbs (LL) and upper limbs.[52] The amount of affected area in each region is assigned a number scoring (A) which determines the extent of body involvement: 1 (0–9 percent) 2 (10–29%), 3 (30–49%), 4 (50–69%), 5 (70–89%), or 6 (90–100%).[53] The severity of three parameters in each region i.e. → erythema (E), thickness/induration (I) and desquamation (D) is quantified using a 5-point scale. It is given the following scores: 0-none, 1-mild, 2- moderate, 3- severe or 4- extremely severe.[52] The final PASI score varies from 0 to 72 and is determined by the formula: PASI = 0.1 (EH + IH + HH) AH + 0.2 (EUL + IUL + HUL) AUL + 0.3 (ET + IT + HT) AT + 0.4 (ELL + ILL + HLL) ALL [52]

	Thickness. 0- 4	Scaling. 0- 4	Erythema. 0 - 4	x Area (0-6)	Total score
Head(H)	a	b	c	d (a+b+c)	X 0.1 = A
Upper limbs(UL)	e	f	g	h (e+f+g)	X 0.2 =B
Trunk(T)	i	j	k	l (i+j+k)	X 0.3 =C
Lower limbs(LL)	m	n	o	p (m+n+o)	X 0.4 =D
					PASI =(A+B+C+D)

Severity: 0→ none; 1-mild ; 2→ moderate ; 3→severe; 4→very severe. 1→ <10% involvement; 2 →10–29%; 3 → 30–49%; 4→ 50–69%; 5→ 70–89%; and 6→ >90%

Table 2. PASI score calculation

Body surface area (BSA):

The predominant method for estimating the body surface area of psoriatic plaques is the "rule of nines."The Rule of Nines was initially developed to assess burn surface area. The method utilises exact percentages for certain body regions as follows: [52]

Head & neck: 9%

Each arm : 9%

Each leg (anterior + posterior): 9%

Each of the 4 quadrants of the trunk: 9%

Genitalia: 1%

Alternatively, BSA may be estimated by the handprint method in which one "handprint" (the palm and fingers of the patient) approximates 1% of the total BSA.[52]

Wang et al.[6] conducted a study that concluded that patient with psoriasis had elevated total white blood cell (WBC), neutrophil-to-lymphocyte ratio (NLR), neutrophils, PLT and PLR along with reduced red blood cell (RBC) counts and haemoglobin (Hb) levels compared to healthy controls.[6] Although, the study did not show association between PLR, NLR and PASI.[6]

Research done by Arunadevi et al.[54] demonstrated that white blood cell and neutrophil counts were raised in psoriasis patients when compared to healthy controls.[54] Additionally, NLR was markedly elevated in the patient group and wasi it linked to the severity and duration of psoriasis.[54]

Raghavan et al.[7] demonstrated that the mean values for RDW, MPV and PLT were significantly different between cases and controls.[7] In both genders, MPV and RDW were elevated, however PLT was reduced in comparison to controls.[7] In male patients, MPV and RDW showed a statistically significant correlation with PASI.[7]

Hammad et al.[5] suggested that NLR and PLR may serve as indicators for systemic inflammation in psoriasis.[5] The elevation of NLR is more significantly affected by the duration

of the disease than by its severity.[5] PLR and MPV can be utilised to assess the severity of psoriasis vulgaris and to evaluate patient follow-up.[5]

A study done by Özkur et al.[55] evaluated the levels of PLT and MPV in psoriasis patients also correlated them with the disease severity.[55] PLT and MPV were markedly elevated in psoriasis patients compared to controls. [55] The platelet count showed a positive correlation with PASI score.[55]

Nageen et al.[56] conducted a study to determine the significance of RDW, MPV and PLT as haematological indicators for evaluating the severity of psoriasis.[56] Their findings indicated that RDW, MPV and PLT assissted in evaluating the severity of psoriasis to a certain degree.[56] They concluded that RDW, MPV, and PLT could augment the PASI score in evaluating disease severity in patients with psoriasis.[56]

Balevi et al.[57] examined the correlation between PLR, NLR, RDW and PASI in psoriasis patients on treatment.[57] A reduction in RDW and an elevation in MPV were observed, alongside a decline in PASI values.[57] This study highlighted the significance of RDW and MPV in assessing clinical progression and treatment response in the first three months of therapy. [57]

Asahina et al.[58] conducted a study to assess the clinical relevance of PLR, NLR and MPV in Japanese patients suffering from plaque-type psoriasis and PsA.[58] Following 12-months of therapy with biologics, both NLR and PLR exhibited a rapid decline alongside a reduction in CRP, regardless of the specific biologics administered. [58]

Kim et al.[59] carried out a retrospective study to evaluate PLR and NLR as inflammatory biomarkers in patients with psoriasis and PsA.[59] They observed a positive correlation of PLT, PLR, and NLR with increased PASI.[59]

A study by Polat et al.[60] assessed the correlation of PLR and NLR to disease activity in patients with persistent plaque psoriasis.[60] They concluded that both PLR and NLR were considerably elevated in these patients. [60]

A study conducted by Liu et al.[61] showed that MPV, PLT and packed cell volume are considerably elevated in psoriasis patients, while MPV, PLT, and PDW [61] exhibit a modest correlation with PASI.[61]

MATERIALS AND METHODS:

SOURCE OF DATA:

Patients presenting to outpatient Department of Dermatology, Venerology and Leprosy at Shri BM Patil Medical College Hospital and Research Centre, Vijayapura.

Period of study: The study was conducted from May 2023 to January 2025.

Study design: A hospital based cross-sectional case-control study.

Sample size: With an anticipated Mean± SD of RDW in control 13.66±1.21 and in psoriasis vulgaris patients 15.16±3.88, the study required a minimum sample size of 250 per group (i.e. a total sample size of 500, assuming equal group sizes) to achieve a power of 99% and a level of significance of 5% (two sided), for detecting a true difference(d) in means between two groups. Sample size was calculated using the following formula:

$$N = 2\left[\frac{(Z_{\propto} + z_{\beta}) * S}{d}\right]^2$$

$Z_{\propto}$- Level of significance=95%

Z_{β}- power of the study=99%

d= clinically significant difference between two parameters ; SD= Common standard deviation

COLLECTION OF DATA:

Inclusion criteria:

All patients with typical clinical features of psoriasis vulgaris, except pustular variants irrespective of age, gender and ongoing or past topical treatment were included in the study.

Exclusion criteria:

• Patients with other co-existing chronic inflammatory disorders like rheumatoid arthritis or systemic lupus erythematosus.

• Patients with hematological diseases.

• Patients with active infections.

• Patients with history of smoking.

• Patients who received systemic therapy for psoriasis in the last 12 weeks.

METHODOLOGY:

After recruitment of the patients, details of the present illness-including the time of onset, duration of the lesion in each case, history of current or previous treatment and past history, was recorded in the proforma.

After the initial clinical history taking, examination of the lesions with respect to morphology, distribution, severity and body surface area was done. The severity of psoriasis was then, assessed using the PASI score.

- The scoring was subjectively assessed and measured in the range between 0-72.
- After obtaining patient consent, a blood sample of 5 ml was collected in a plain tube, and the sample was allowed to clot at room temperature.
- The sample was sent for complete hemogram, for assessment of hematological parameters.
- NLR was calculated by dividing the total number of neutrophils by the total number of lymphocytes.
- PLR was calculated by dividing the total number of platelets by the total number of neutrophils.
- RDW, MPV, PDW and PLT were obtained from the laboratory report.
- These values were represented in tables, percentages, diagrams and their values were correlated with the severity and duration of psoriasis.

STATISTICAL ANALYSIS:

- The data obtained was entered in a Microsoft Excel sheet, and statistical analysis was performed using the software JMP®, Version 16. SAS Institute Inc., Cary, NC, 1989-2021 and Python, Version 3.10.4. Python Software Foundation, Wilmington, DE, 1991-2022.
- Results were presented as Mean(Median), Inter-quartile range (IQR), counts, percentages and diagrams.
- For not normally distributed variables Mann Whitney U test was used.

- Pearson's/Spearman's correlation was used to find the correlation between quantitative variables.
- $p<0.05$ was considered statistically significant. All statistical tests were performed as two-tailed.

ETHICAL CLEARANCE:

Patients were recruited after obtaining ethical clearance was from BLDE University for the study- **BLDE(DU)/IEC/901/2023-24.**

RESULTS:

A hospital-based cross-sectional case-control study, was conducted from May 2023 to January 2025. A total of 260 patients diagnosed with different subtypes of psoriasis were enrolled in the study. An equal number of age and gender-matched controls were also incorporated.

Distribution of age:

The median age of the psoriasis patients was 37 years. The majority of patients belonged to the age group of 31-40 years (67,25.77%), followed by 41-50 years (51,19.62%), 21-30 years (43,16.54%), 51-60 years (36, 13.85%) and least patients belonged to the age group of more than 60 years (32, 12.31%), 11-20 years (25, 9.62%) and 1-10 years (6, 2.31%) as mentioned in the figure and table below:

Sr No	Age Group	Cases (Number, %)
1	1-10	6 (2.31%)
2	11-20	25 (9.62%)
3	21-30	43 (16.54%)
4	31-40	67 (25.77%)
5	41-50	51 (19.62%)
6	51-60	36 (13.85%)
7	>60	32 (12.31%)

Table 3: Age distribution in cases.

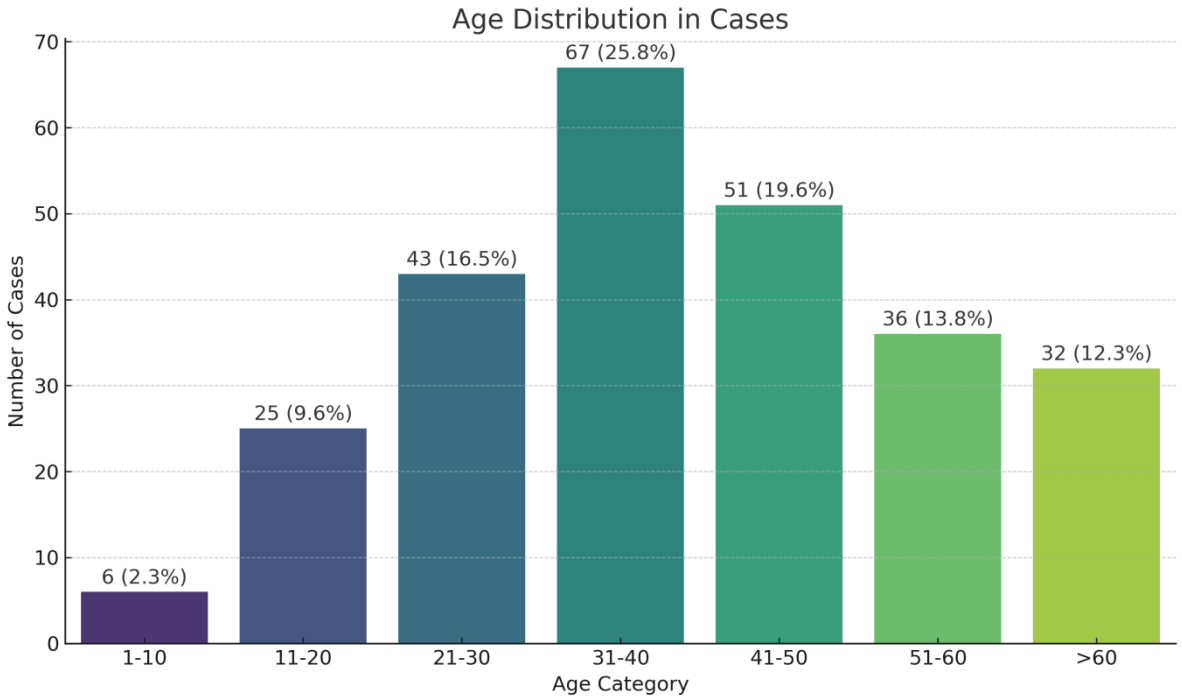


Figure 1: Age distribution in cases

Distribution of gender:

Among the 260 psoriasis patients, 178 (68.46%) were males and 82 (31.54%) were females and equal distribution was also noted in controls. Distribution of cases is depicted in the table given below:

Gender	**Cases (n, %)**
Male	178 (68.46%)
Female	82 (31.54%)
Total	260 (100%)

Table 4: Gender distribution in cases.

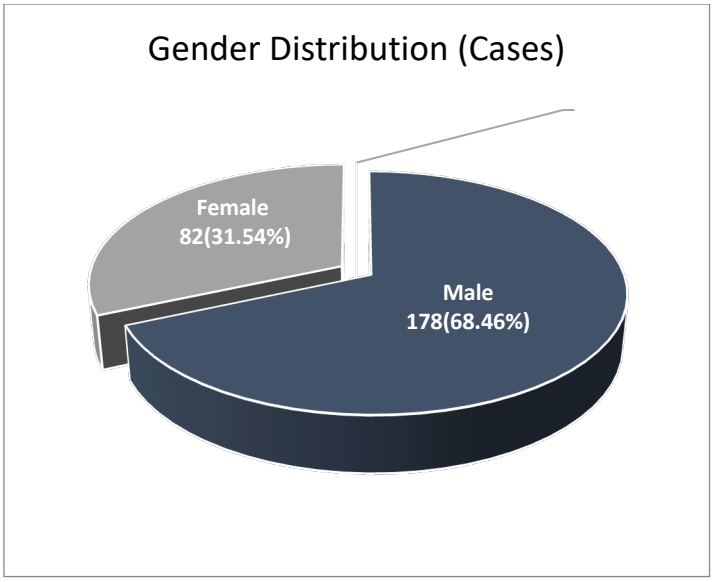


Figure 2: Gender distribution in cases

Distribution of subtypes of psoriasis:

The distribution of the various subtypes of the psoriasis cases and the percentages were as follows: Psoriasis vulgaris (PsoV)- 209 (80.4%), palmoplantar psoriasis (PPP)- 21 (8.1%), scalp psoriasis (scalp pso)- 11 (4.2%), Guttate psoriasis (Guttate Pso)- 6 (2.3%), Plantar psoriasis (Plantar Pso)- 4, (1.5%), Erythrodermic psoriasis (Pso Ery)- 3(1.2%), Follicular psoriasis (Follicular Pso)- 2 (0.8%), Palmar psoriasis (Palmar Pso)- 1 (0.4%), Psoriasis vulgaris+ scalp psoriasis (PsoV+Scalp Pso)- 1 (0.4%), Psoriasis vulgaris + Psoriatic arthritis (PsoV+PsA), Flexural psoriasis (Flex Pso)- 1(0.4%). The following diagram represents the distribution of the subtypes of psoriasis in the cases:

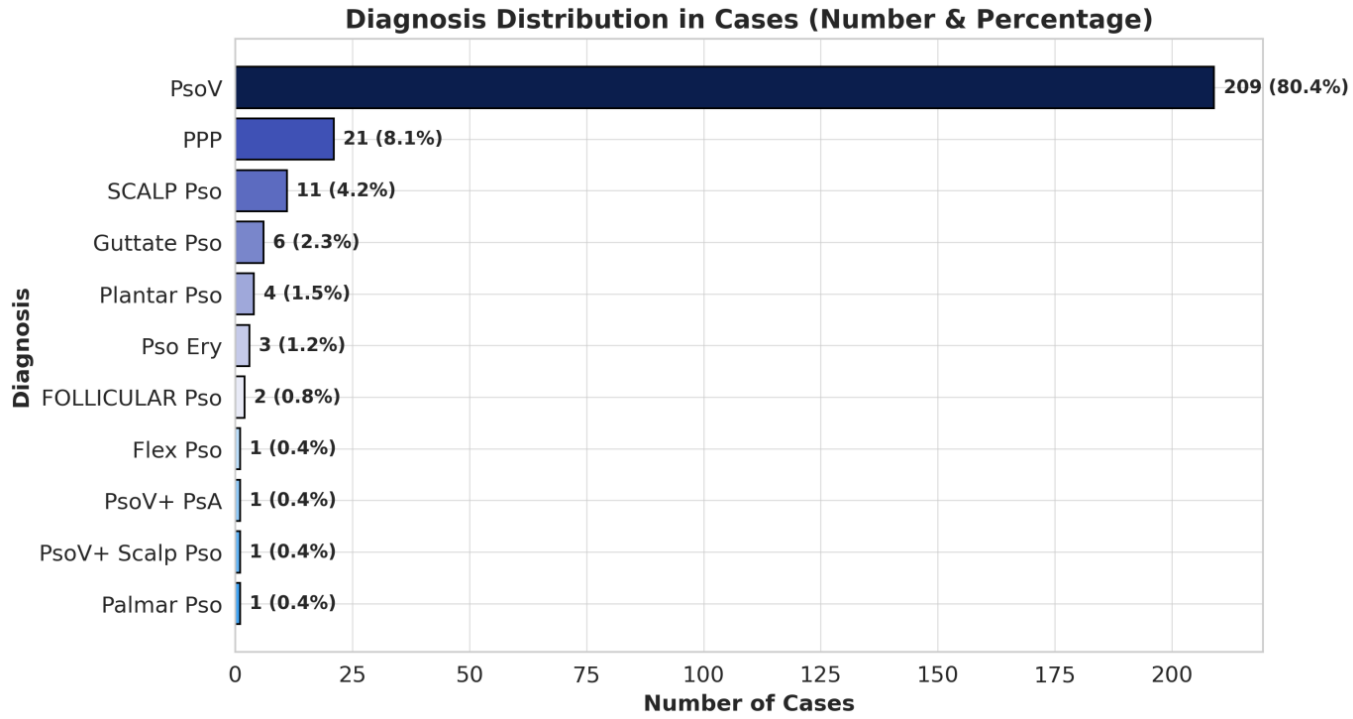


Figure 3: Distribution of psoriasis subtypes.

Correlation of PASI score with haematological parameters to assess severity of psoriasis.

The table given below shows the various haematological parameters- PLT, NLR, PLR,RDW, PDW and MPV and their correlation with PASI score. The median PASI score was 9. For PLT and NLR, Spearman's correlation coefficient was used due to their unequal variance calculated using Levene's test and non-parametric distribution. For other parameters, Pearson's correlation coefficient was used; p value < 0.05 was considered as statistically significant. For PLT, Spearman correlation was weakly positive and for NLR, it was weak to moderately positive with both having p-value of 0.045 and 0.002 respectively which is **statistically significant**. Although with PLR, PASI showed a negligible correlation, no meaningful correlation of RDW with PASI, and a weak negative correlation of PDW and MPV with PASI.

	Parameter	Correlation Coefficient (r)	p-value	Correlation
1	PLT	0.124	**0.045***	**Statistically significant**
2	NLR	0.19	**0.002***	**Statistically significant**
3	PLR	0.06	0.362	Negligible correlation
4	RDW	0.02	0.75	Not meaningful
5	PDW	-0.08	0.178	Weak negative
6	MPV	-0.05	0.445	Weak negative

For PLT and NLR, Spearman's correlation coefficient was used due to their unequal variance calculated using Levene's test and non-parametric distribution. For other parameters, Pearson's correlation coefficient was used. *p value < 0.05 was considered as statistically significant.

Table 5: Correlation of PASI score with hematological parameters

PASI correlation with haematological parameters.

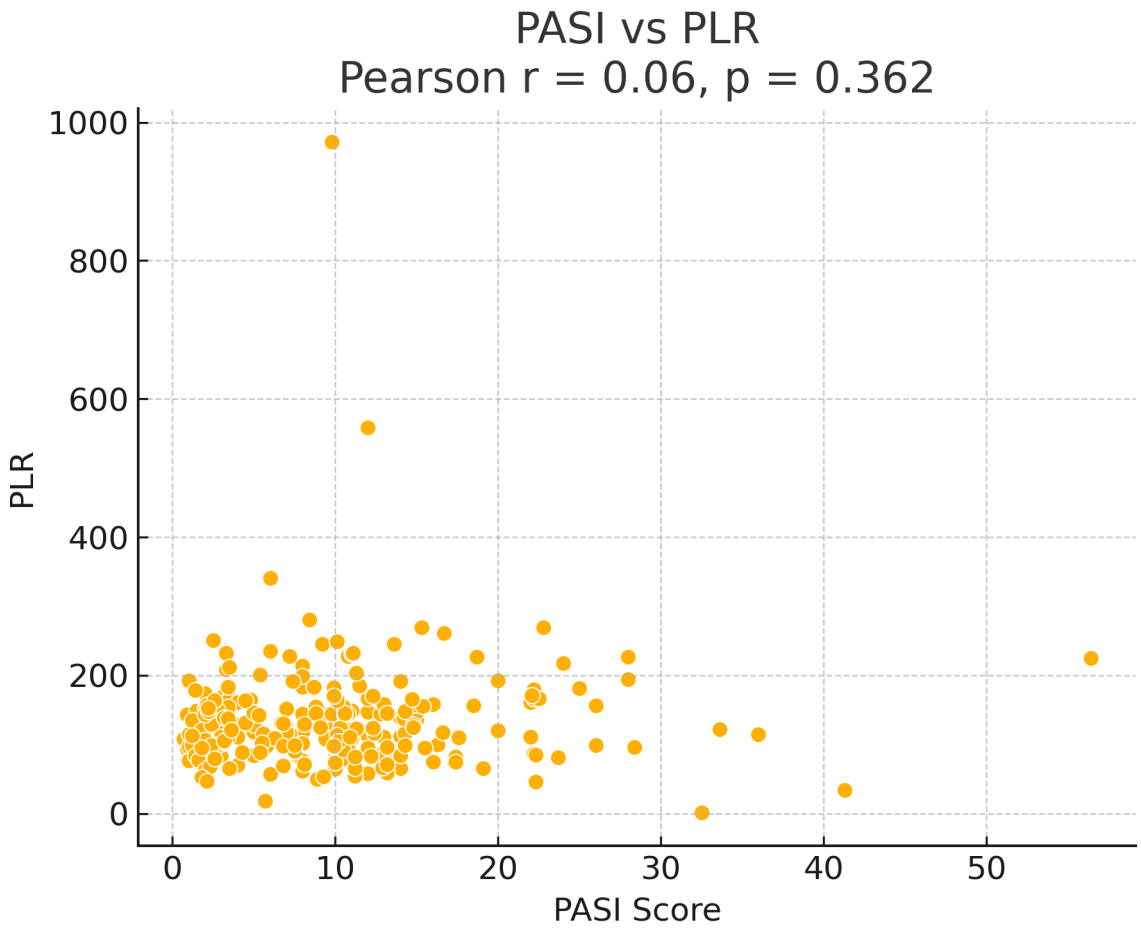


Figure 4: Scatter diagram showing PASI correlation with PLR.

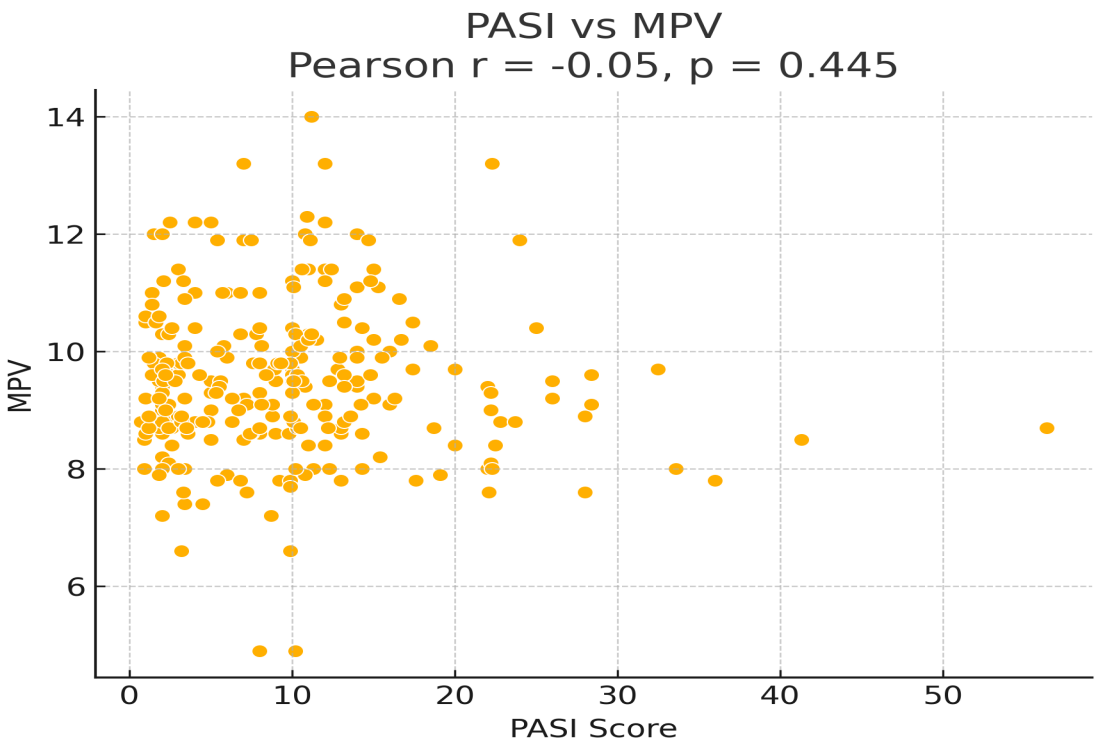


Figure 5: Scatter diagram showing PASI correlation with MPV.

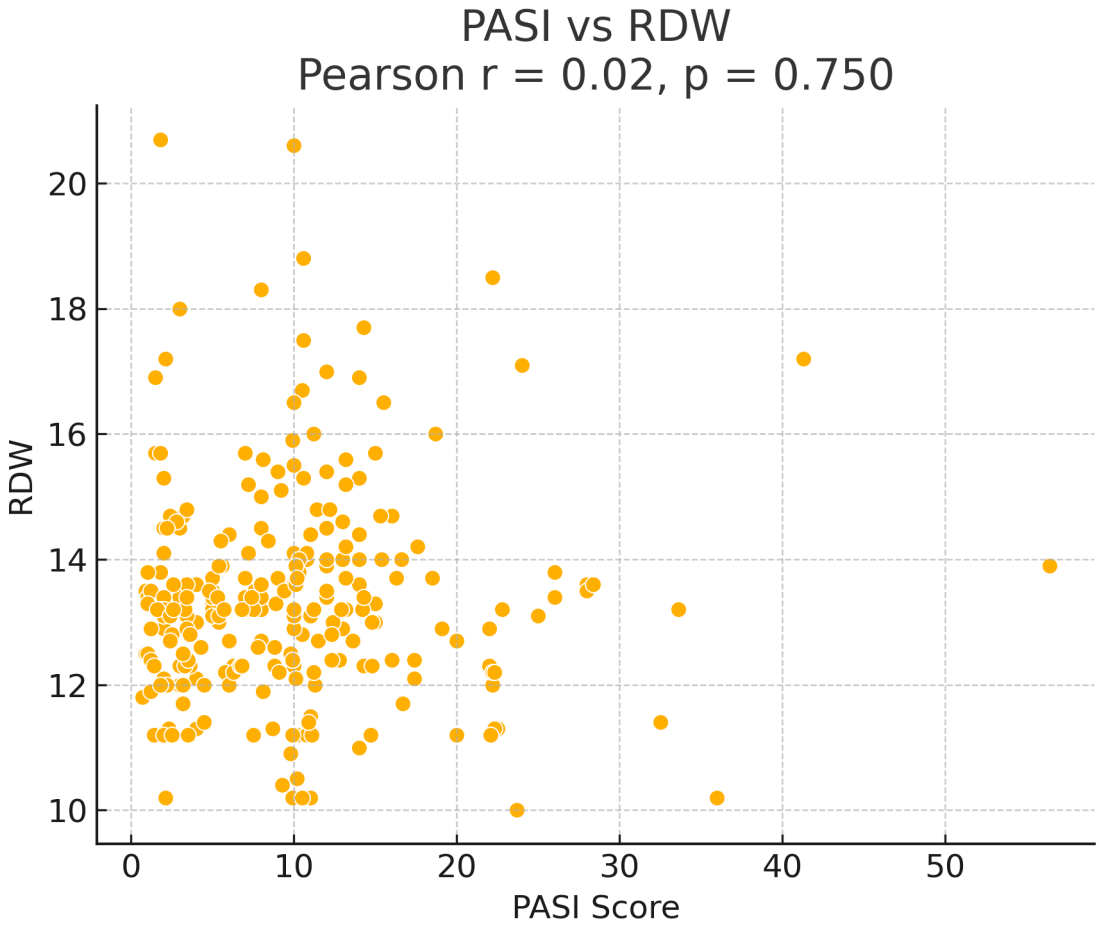


Figure 6: Scatter diagram showing PASI correlation with RDW.

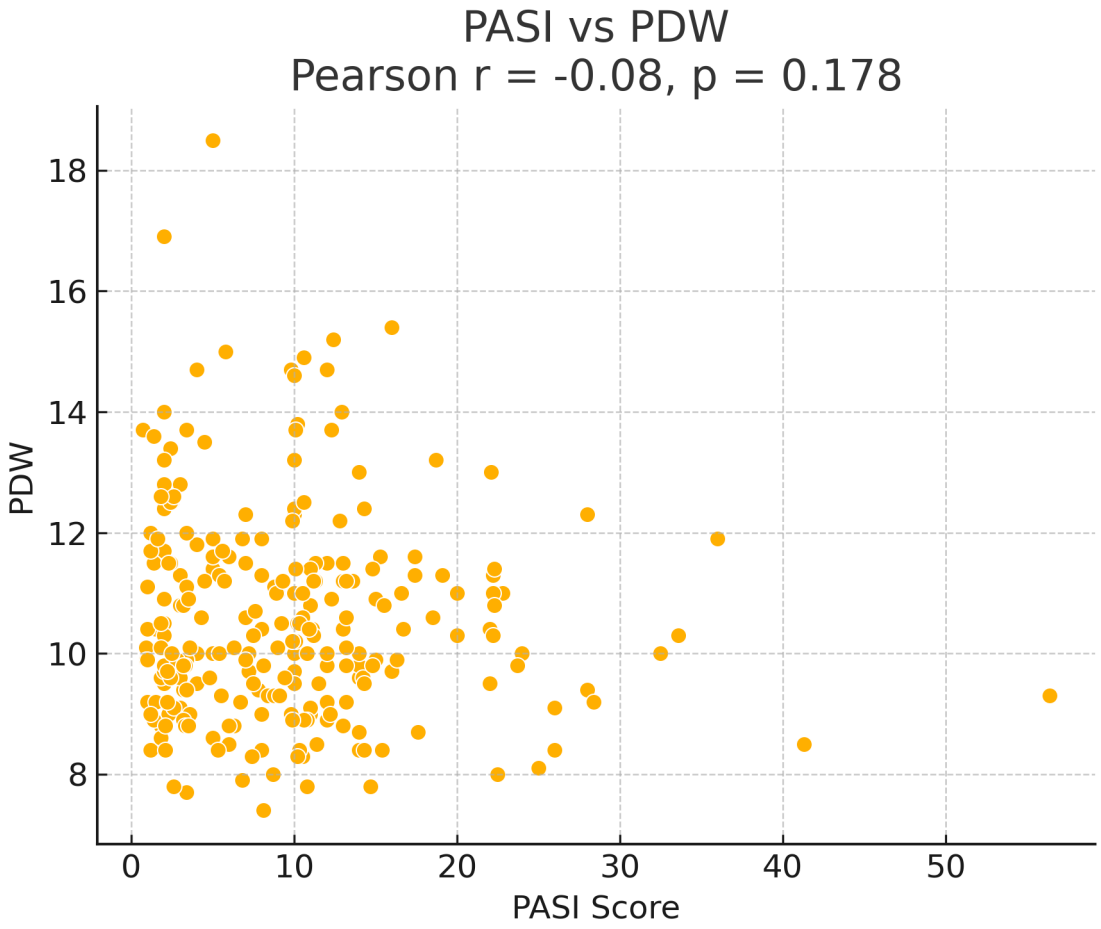


Figure 7: Scatter diagram showing PASI correlation with PDW.

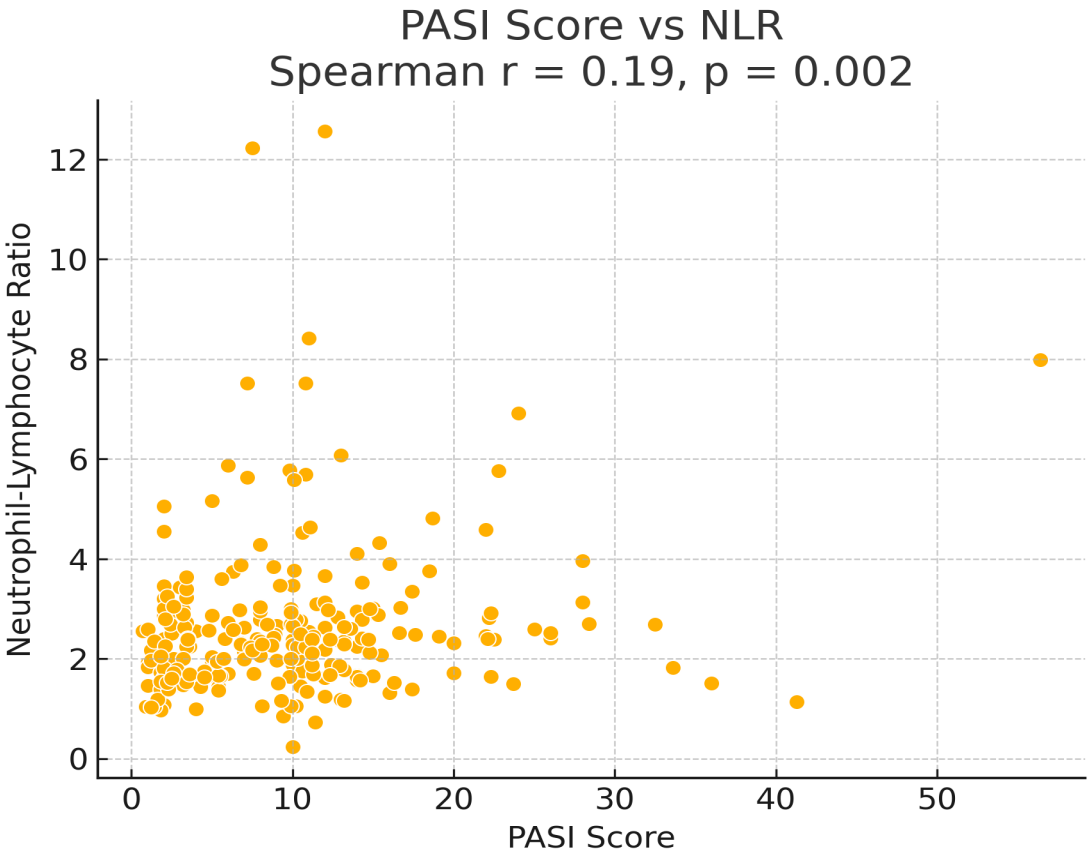


Figure 8: Scatter diagram showing PASI correlation with NLR.

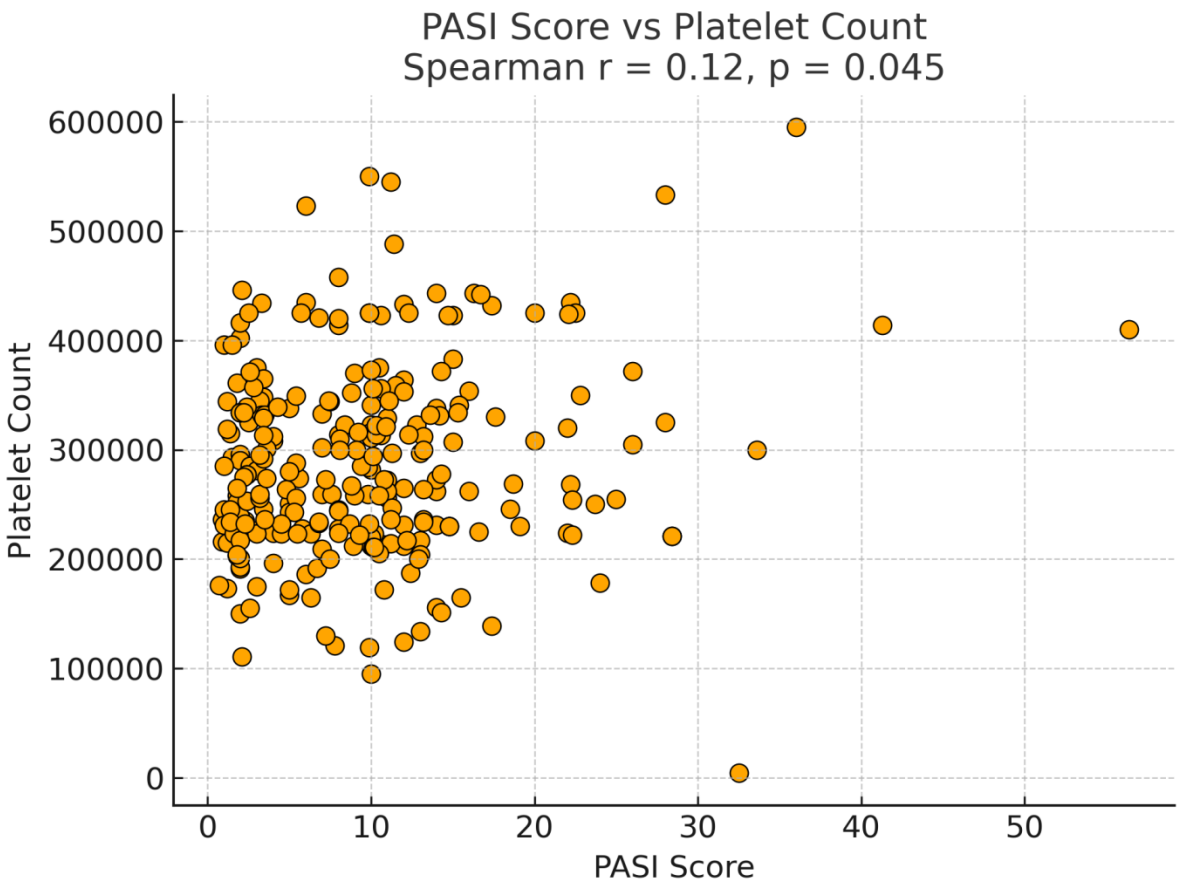


Figure 9: Scatter diagram showing PASI correlation with PLT.

Comparison of haematological parameters between cases and controls.

The median values and interquartile ranges of the haematological parameters of cases against controls and its comparison was done by Mann-Whitney U test. The NLR and MPV had a **statistically significant** increase in the values as compared to that of the controls with a p-value of 0.012 for both. The values of the cases against controls with its significance is depicted in the table below

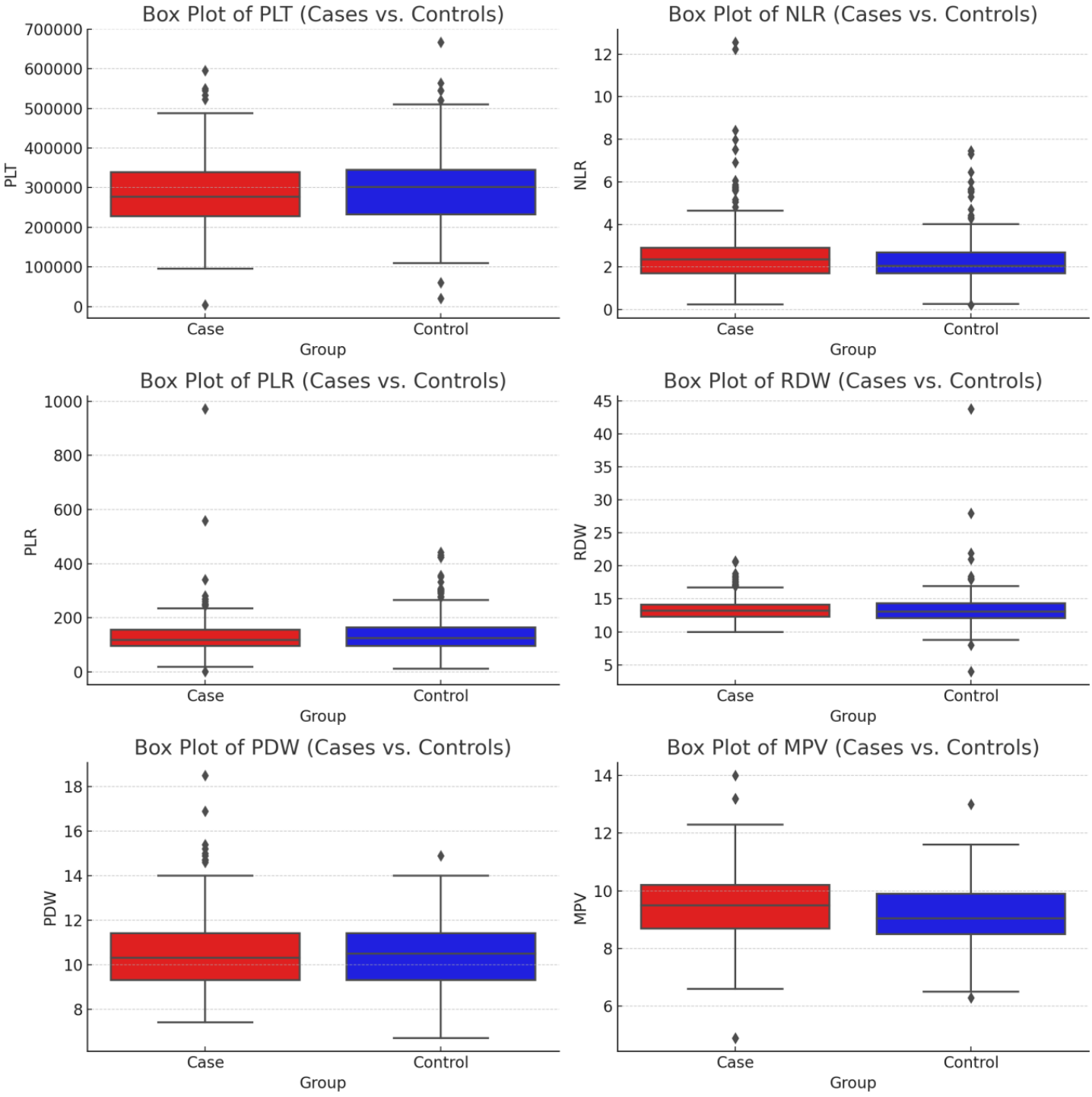
Sr. No.	Parameter	Cases (Median, IQR)	Controls (Median, IQR)	p-value	Significance
1	PLT	277500.0 (228000.0 - 339500.0)	301500.0 (232750.0 - 345250.0)	0.090	Not significant
2	NLR	2.35 (1.72 - 2.90)	2.05 (1.71 - 2.69)	**0.012***	**Statistically significant**
3	PLR	117.4 (95.8 - 154.9)	125.2 (95.2 - 164.7)	0.199	Not significant
4	RDW	13.2 (12.3 - 14.1)	13.05 (12.1 - 14.3)	0.189	Not significant
5	PDW	10.3 (9.3 - 11.4)	10.5 (9.3 - 11.4)	0.770	Not significant
6	MPV	9.5 (8.7 - 10.2)	9.1 (8.5 - 9.9)	**0.012***	**Statistically significant**

*p-value < 0.05 was considered as statistically significant.

Table 6: Comparison of study parameters between cases and controls (Mann-Whitney U Test)

Box plots showing comparison of study parameters between cases and controls

Figure 10: Comparison of haematological parameters between cases and controls.



DISCUSSION:

Psoriasis is a skin disease characterized not only by cutaneous manifestations, but it is also implicated in the systemic changes due to the inflammation.[6]

Certain research indicate a correlation between psoriasis and comorbidities like cardiovascular disease, metabolic syndrome, and diabetes.[62] There can be periods of remission interspersed with exacerbation of the disease depending upon certain triggers like alcohol intake, certain medications like antihypertensives. [63,64]

The PASI score evaluates severity of the condition with respect to morphology of the lesions. The calculation is based on the surface area of lesion distribution throughout the head, upper limbs, trunk, and lower limbs, factoring in erythema, induration, and scaling.[52]

So far, there has not been any gold standard haematological marker to strongly indicate the disease severity. Even though several studies[5,54,55,59] have investigated the relationship between illness severity and several of the aforementioned claboratory parameters, there has not been a consistent marker made available for the same. Additionally, the markers have been non-specific, i.e. elevation in infections, haematological disorders and smokers, which cannot represent an accurate measure of elevation with regard to the flare of psoriasis.

This study aimed to determine the correlation between the PASI as a clinical assessment tool and various haematological parameters, including PLT, RDW, PDW, NLR, PLR and MPV.

The number of male patients were (187, 68.4%) higher than the females (82, 31.5%) in our study which is an accurate replication of the epidemiological data available with respect to the gender distribution of psoriasis.

The majority of patients in our study (67, 25.7%) were aged between 31-40 years, followed by those aged 41-50 years (51, 19.6%). The smallest proportion of patients was in the age group of 1-10 years, indicating that psoriasis is less prevalent among children. Psoriasis mostly affects the middle aged population and it is also more commonly seen in men.

The predominant subtype of psoriasis observed in the patients was psoriasis vulgaris (209, 80.4%) followed by palmoplantar psoriasis (21, 8.1%), scalp psoriasis (11,4.2%) and the least common diagnoses among the cases were- flexural psoriasis (1,0.4%), followed by psoriasis vulgaris along with psoriatic arthritis (1, 0.4%), psoriasis vulgaris along with scalp psoriasis (1, 0.4%), palmar psoriasis (1, 0.4%) and follicular psoriasis (2,0.8%). Psoriasis vulgaris was the most common subtype, and follicular psoriasis was less commonly seen.

Our study found a statistically significant positive correlation between NLR and PASI score, aligning with the findings of a study done by Arunadevi et. al.[54] which also showed NLR to be significantly raised in patients as compared to controls along with the correation with PASI.

A studies conducted by Hammad et. al.[5] and Wang et.al[6] demonstrated an increase in the NLR of cases compared to controls.

The correlation between NLR and PASI, in our study is attributable to the role of neutrophils in psoriasis.[65] Neutrophils are abundantly deposited in the epidermis and dermis of the affected skin.[65]

Tumour necrosis factor alpha, interleukin-8 and interferon gamma are responsible for the priming and higher levels of neutrphils which contribute to the process of inflammation.[5] Inflammatory processes also cause differences in the various cell lineages of the heamatopoietic system.

In psoriasis, there is a possible reduction in lymphocytes owing to a large number of lymphocytes actively migrating to the affected areas with active ongoing inflammation, thereby causing pooling in the lesional skin rather than accumulating in the peripheral circulation.[66] This could be another potential reason for the subsequent rise in NLR in psoriasis patients in our study.

The study identified a statistically significant positive correlation between PLT and PASI. This aligns with research conducted by Özkur et. al.[55] where they established a positive correlation of PASI with PLT.

Evidence indicates that platelets are crucial in the pathogenesis of psoriasis, as activated platelets enhance leukocyte migration to the skin and release various pro-inflammatory cytokines.[67]

Platelets are activated excessively via endothelial system and contribute to the inflammation caused in psoriasis.[68] Increased platelet aggregation may serve as a potential risk factor for atherosclerosis, ischaemic heart disease, and stroke in patients with psoriasis due to endothelial damage.[69]

There was also weak negative correlation (not statistically significant) noted in PDW and MPV with PASI, and no significant correlation of PASI with PLR and RDW.

Our study demonstrated that the levels of NLR and MPV were significantly higher in cases compared to controls.

Wang et. al.[6], in their study, demonstrated a similar elevation in the NLR in psoriasis patients as compared to the controls. Neutrophils are significant contributors to the pathogenesis of psoriasis.[6] Neutrophil extracellular traps (NET) are a mesh like array of DNA released to bind pathogenic microbes.[70]

A study done by Hu et. al.[65] demonstrated a proportional increase in NET levels in lesional skin and peripheral blood of psoriasis patients,[65] which directly correlated with disease severity,[65] in contrast to the absence of NETs in eczema patients within the same study, following appropriate staining and visualisation via confocal microscopy.[65] Additionally, negative finding of lack of NETosis was found in patients with less severe cutaneous lesions, in patients with PASI of less than 8.[65]

This supports the finding of increased NLR in our study, as explained by the potential increase in neutrophils in disease state compared to the controls suggesting the role of neutrophils in the ongoing systemic inflammation. Psoriasis patients exhibit a low incidence of cutaneous infections.[71] This is due to the fact that there is increased accumulation of NETs in the psoriatic skin lesions[65] resulting from rise in neutrophil levels in the affected patients.

A retrospective study conducted by Polat et. al.[72] demonstrated a significant increase in NLR levels in patients compared to the control group, aligning with the findings of our study, and showed a positive correlation with PASI.[72]

The findings related to NLR in our study indicate that NLR is a dependable haematological marker for evaluating disease severity in psoriasis patients. Additionally, it is a feasible, easily available and affordable test for patients.

Furthermore, a statistically significant increase in MPV was observed in psoriasis patients compared to the control group. Platelets play a significant role in the pathogenesis of psoriasis, characterised by increased platelet aggregation and chronic inflammatory activity, which results in heightened platelet activation.[73] This leads to an increased size of the platelets.[73] Mean platelet volume is a metric that quantifies the average size of platelets in the bloodstream. This serves as a measure of platelet activity and aggregation potential.[74] A high mean platelet volume (MPV) is considered an indicator of inflammation, thrombosis, atherosclerosis, and various diseases associated with a systemic high inflammatory state.[75]

A cross-sectional study conducted by Kim et al.[73] concluded that MPV was significantly elevated in patients with psoriasis compared to controls.[73] The values of PLT, PLR, RDW and PDW showed no significant differences between the cases and controls in their study. [73]

Inflammatory markers such as NLR, PLT, and MPV may serve as indicators of systemic inflammation linked to psoriasis, as evidenced by their correlation with PASI in the cases of NLR and PLT. NLR serves as a direct indicator of disease severity.

However, there were a few limitations in our study such as :-

1. Recruitment of all cases irrespective of mild, moderate or severely affected patients which can dilute certain values.
2. Lack of subgrouping of the patients according to the level of PASI i.e. PASI < 10 and PASI > 10.
3. No follow up assessments of the patients done to assess the levels of the markers in response to treatment.
4. Single center study.

CONCLUSION:

Psoriasis is an inflammatory, papulosquamous disease affecting the skin and also the nails and joints. PASI helps guide the clinician on the efficacy of treatment and need for alternative treatment as a simple OPD assessment technique.

This was a hospital-based cross-sectional study conducted in North Karnataka, involving 260 psoriasis patients across all age groups and genders. An equivalent number of age and gender-matched controls, deemed apparently healthy, were recruited for the study.

The median age of the cases was 37 years, with the majority of patients falling within the 31-40 year age group. Of the total patients, 178 (68.4%) were male, while 82 (31.5%) were female.

On assessment of the subtypes of psoriasis, majority of the patients in our study were diagnosed with psoriasis vulgaris (209, 80.4%).

The values of PLT, NLR, PLR, MPV, RDW and PDW were correlated with PASI in the cases group. There was statistically significant positive correlation of NLR and PLT with PASI. Our study also showed the statistically significant rise of NLR and MPV in the cases in comparison with the controls.

In conclusion, the outcomes of this study appear to substantiate the hypothesis that NLR, PLT, and MPV serve as reliable, cost-effective, and readily accessible markers for indicating disease severity. This, in turn, indicates the degree of persistent systemic inflammation in individuals with psoriasis.

These indicators may also assist the clinician in evaluating treatment during follow-up assessments. Although, the precise clinical relevance of these parameters need to be studied using large scale, comprehensive and prospective trials in future.

SUMMARY:

A hospital-based cross-sectional case-control study was carried out in North Karnataka, from May 2023 to January 2025 where 260 psoriasis cases were recruited. An equal number of age and gender-matched controls were included as well, and the blood samples from these patients were analyzed for levels of haematological parameters namely, NLR, PLR, MPV, PLT, PDW and RDW. These parameters were then correlated with PASI in the cases. The values of the parameters were also compared between cases and controls group.

The salient features found in the study are as follows:

1. Psoriasis vulgaris was the most prevalent type of psoriasis.
2. Least commonly diagnosed subtypes were flexural psoriasis, followed by scalp psoriasis , palmar psoriasis and follicular psoriasis.
3. Majority of patients in the study, specifically 67 individuals, were in the age range of 31-40 years, followed by 51 individuals in the age group of 41-50 years.
4. The lowest number of patients was observed in the age group of 1-10 years.
5. Males were more in number than females.
6. All the demographics of the patient data accurately reflected the epidemiological trend of psoriasis.
7. PLT and NLR had a statistically significant positive correlation with PASI.
8. Other findings included weak negative correlation of PDW and MPV with PASI (not statistically significant)
9. There was no correlation of PLR and RDW with PASI.

10. The NLR and MPV exhibited a statistically significant increase in cases relative to controls.
11. No statistically significant differences were observed in the comparison of other measures such as PLR, PDW, RDW, and PLT between the patients and controls.

The findings in our study suggest that PLT and NLR can indicate disease severity in psoriasis in conjunction with PASI. In addition, NLR and MPV is also shown to be significantly elevated in psoriasis patients.

From these findings, we can infer that the above parameters in our study can be used to monitor disease activity in psoriasis patients.

Additionally, large-scale prospective studies are necessary to validate these laboratory parameters as reliable indicators for monitoring disease activity in psoriasis patients, thereby assisting clinicians in improving patient treatment.

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ETHICAL CLEARANCE CERTIFICATE

Azadi Ka Amrit Mahotsav

BLDE
(DEEMED TO BE UNIVERSITY)
Declared as Deemed to be University u/s 3 of UGC Act, 1956
Accredited with 'A' Grade by NAAC (Cycle-2)
The Constituent College
SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPURA

BLDE (DU)/IEC/ 901/2023-24 10/4/2023

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this University met on **Saturday, 18th March, 2023 at 11.30 a.m. in the CAL Laboratory, Dept. of Pharmacology,** scrutinizes the Synopsis/ Research Projects of Post Graduate Student / Under Graduate Student /Faculty members of this University /Ph.D. Student College from ethical clearance point of view. After scrutiny, the following original/ corrected and revised version synopsis of the thesis/ research projects has been accorded ethical clearance.

TITLE: "CORRELATION OF HEMATOLOGICAL MARKERS IN PSORIASIS WITH THE SEVERITY OF THE DISEASE".

NAME OF THE STUDENT/PRINCIPAL INVESTIGATOR: DR.VISHNAVI PRAN KANATH

NAME OF THE GUIDE: DR.AJIT B. JANAGOND , ASSOCIATE PROFESSOR, DEPT. OF DERMATOLOGY,VENEROLOGY AND LEPROSY.

Dr. Santoshkumar Jeevangi
Chairperson
IEC, BLDE (DU),
VIJAYAPURA
Chairman,
Institutional Ethical Committee,
BLDE (Deemed to be University)
Vijayapura

Dr.Akram A. Naikwadi
Member Secretary
IEC, BLDE (DU),
VIJAYAPURA
MEMBER SECRETARY
Institutional Ethics Committee
BLDE (Deemed to be University)
Vijayapura-586103. Karnataka

Following documents were placed before Ethical Committee for Scrutinization.

- Copy of Synopsis/Research Projects
- Copy of inform consent form
- Any other relevant document

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B.L.D.E. U'S SHRI B. M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, VIJAYAPURA.

Department of Dermatology, Venereology and Leprosy.

WRITTEN INFORMED RESEARCH CONSENT

TITLE OF THE PROJECT: -	CORRELATION OF HAEMATOLOGICAL MARKERS IN PSORIASIS WITH THE SEVERITY OF THE DISEASE
PG GUIDE: -	DR. AJIT B JANAGOND
PG STUDENT: -	DR. VISHNAVI PRAN

PURPOSE OF RESEARCH:

I have been informed that this project will determine the haematological parameters and the values will be used to correlate it with the disease activity.

BENEFITS:

I understand that my participation in this particular study will help the investigator to know the hematological parameters in patients with psoriasis vulgaris and its correlation with the disease activity and extent.

PROCEDURE: -

I understand that relevant history will be taken, and I will undergo a detailed clinical examination, after which treatment will be given.

RISK AND DISCOMFORTS: -

I understand that no risk is involved, and I will experience no discomfort during the clinical examination.

CONFIDENTIALITY: -

I understand that medical information produced by this study will become a part of my hospital records and will be subjected to the hospital's confidentiality and privacy regulation. Information of a sensitive personal nature will not be a part of the medical records but will be stored in the investigator's research file.

Suppose the data are used for publication in the medical literature or teaching purposes. No names will be used in that case, and other identifiers such as photographs and audio or videotapes will be used only with my special written permission. I understand I may see the photographs and videotapes and hear the audiotapes before giving this permission.

REQUEST FOR MORE INFORMATION: -

I understand that I may ask additional questions about the study anytime. Concerned, Dr. Vishnavi Pran is available to answer my questions or queries. I understand that I will be informed about any significant new findings discovered during the course of this study, which may influence my continued participation.

REFUSAL OR WITHDRAWAL OF PARTICIPATION: -

I fully understand that my participation is voluntary, and I may refuse to participate or withdraw consent and discontinue participation in this study at any time without prejudice. I also understand that Dr. Vishnavi Pran may terminate my participation in this study at any time after she has explained the reasons for doing so and has helped arrange for my continued care by my physician if this is appropriate.

INJURY STATEMENT: -

I understand that in the unlikely event of injury resulting directly from my participation in this study. If such an injury were promptly reported, then medical treatment would be available to me, but no further compensation will be provided. I understand that I am not waiving any of my legal rights by my agreement for my participation in this study.

I have explained to (patient's/relevant guardian's name) the purpose of the research, the procedures required, and the possible risks and benefits to the best of my ability in the patient's language.

_____ _____

Investigator/P. G. Guide Date

I confirm that ….....……(Name of the PG guide/chief researcher) has explained the research and study procedures I may undergo and the possible risks, discomforts, and benefits I may experience. I have read and understood this consent form. Therefore, I agree to consent to my participation as a subject in this research project.

_____ _____

Participant/Guardian Date

_____ _____

Witness to signature Date

B.L.D.E.U'S SHRI BM PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, VIJAYAPURA

Department of Dermatology, Venereology and Leprosy.

CASE PROFORMA

SCHEME OF CASE TAKING

GENERAL INFORMATION

Sl. No.: Date:

Name:I.P/O.P No:

Age: Hospital:

Sex:

Address:

Occupation:

PRESENTING COMPLAINTS AND DURATION:

HISTORY OF PRESENTING ILLNESS

- Skin lesions: 1. Onset – Sudden/ Gradual

  2. Duration

  3. Associated complaints:

     Redness/Burning/Scaling/Erosions/Exudation/Lichenification

- Site: Unilateral/Bilateral

- Distribution of lesions: Unilateral/Bilateral

•Associated features: Present/Absent

- Constitutional symptoms: Present/Absent
- Other symptoms

•History of similar complaints in the family:

Present/Absent

•History of associated diseases:

•Treatment history: Topical/Systemic

PAST HISTORY

-History of similar complaints in the past: Present/Absent

-Comorbidities:

PERSONAL HISTORY

•Diet: Veg/Non-veg

- Appetite: Normal/Poor
- Bowel/Bladder: Regular/Disturbed
- Sleep: Normal/Disturbed
- Habits:

Smoker/Alcoholic/Drug addiction/No habits

FAMILY HISTORY

GENERAL PHYSICAL EXAMINATION

- Built: Well/Moderate/Poor
- Nourishment: Well/Moderate/Poor

- Others: Pallor/Icterus/Cyanosis/Clubbing/Oedema/Lymphadenopathy

- Vital signs: Pulse rate: R.R:
  B.P: Temperature:

CUTANEOUS EXAMINATION

- Lesions:
- Sites:
- Borders: Regular/Irregular

- Side: Unilateral/Bilateral
- Body surface area (According to the rule of palm):
- Examination of mucous membranes:
  Oral

  Genital

- Others: Hair
  Genitals Nails

- Other cutaneous lesions elsewhere:

PSORIASIS AREA SEVERITY INDEX:

A . Degree of severity: Encircle the appropriate number

0- none 1-slight 2-moderate 3- severe 4- very severe

	HEAD	**TRUNK**	**UPPER LIMB**	**LOWER LIMB**
ERYTHEMA	**1 2 3 4**	**1 2 3 4**	**1 2 3 4**	**1 2 3 4**

INDURATION	**1 2 3 4**	**1 2 3 4**	**1 2 3 4**	**1 2 3 4**
SCALING	**1 2 3 4**	**1 2 3 4**	**1 2 3 4**	**1 2 3 4**
TOTAL (A1)				

2. BODY SURFACE AREA :

	HEAD	**TRUNK**	**UPPER LIMB**	**LOWER LIMB**
0:Nil, 1: 1-9% (1)				
2: 10-29% 3: 30-49% 4: 50-69% 5: 70-89% 6: 90-100%				
TOTAL (A1xB)= C				

D- AREA MULTIPLICATION FACTOR

C x D	x 0.1	**x 0.2**	**x 0.3**	**x 0.4**

4: TOTAL- HEAD+TRUNK+UPPER LIMB+LOWER LIMB

=

SYSTEMIC EXAMINATION

CVS:

CNS:

RS:

Per abdomen:

PROVISIONAL DIAGNOSIS:

INVESTIGATIONS:

Complete hemogram

FINAL DIAGNOSIS:

KEY TO MASTERCHART:

M- Male

F- Female

PLT- Platelet count

RDW- Red cell distribution width

PDW- Platelet distribution width

(fl)- femtoliters

PASI- Psoriasis assessment and severity index

MPV- Mean platelet volume

NLR- Neutrophil-lymphocyte ratio

PLR- Platelet-lymphocyte ratio

Pso Ery- Psoriatic erythroderma

PsoV- Psoriasis vulgaris

Guttate pso- Guttate psoriasis

Plantar pso- Plantar psoriasis

Palmar pso- Palmar psoriasis

FOLLICULAR Pso- Follicular psoriasis

SCALP pso- Scalp psoriasis

PPP- Palmoplantar psoriasis

Flex pso- Flexural psoriasis

Pso HKD- Psoriatic HKD

PsoV+PsA- Psoriasis vulgaris with psoriatic arthritis

PsoV+ Scalp Pso- Psoriasis vulgaris with scalp psoriasis

Master Chart

SNO.	Names	Age(years)	Sex	PLT	RDW(%)	PDW(fl)	Group	DIAGNOSIS (For Cases)	PASI	MPV(fl)	NLR	PLR
1	BABY SRIRAJ DONI	1	M	488000	14.8	8.5	Case	Pso Ery	11.4	8.0	0.7	59.3
2	BABY DANAMMA KARKUN	4	F	375000	12.8	8.3	Case	PsoV	10.5	9.9	1.5	79.2
3	SANVI SURESH	6	F	338000	14.0	9.6	Case	PsoV	14.0	12.0	1.7	65.8
4	ANANYA MAHAPATA HARIDAS	12	F	308000	13.0	10.0	Case	PsoV	4.0	11.0	1.6	128.3
5	SHANTAVEER NINGAPPA	13	M	293000	15.7	10.4	Case	Guttat e Pso	1.5	12.0	1.5	94.2
6	MUTTAPPA CHANDAPPA	13	M	272000	14.4	9.0	Case	PsoV	11.0	11.4	2.0	149.4
7	ARPITA BANSAL	15	F	323000	20.6	9.7	Case	PsoV	10.0	10.4	1.7	110.6
8	AJEETKUMAR CHAVAN	15	M	236000	13.5	10.1	Case	Plantar Pso	0.9	8.0	1.0	94.7
9	SIDDAPPA MADAR	15	M	372000	13.4	8.4	Case	PsoV	26.0	9.5	2.4	98.5
10	SOMANATH HOLASUR	17	M	196000	13.6	11.8	Case	PsoV	4.0	8.8	2.6	110.8
11	AVINASH C GUDIMANI	17	M	251000	13.5	10.0	Case	PsoV	5.0	9.5	1.7	135.8

12	SHRAVAN VIDHATE	17	M	316000	12.9	12.3	Case	PsoV	10.0	10.0	2.9	128.3
13	GOPAL CHAVAN	17	M	265000	13.4	9.8	Case	FOLLICULAR Pso	12.0	13.2	1.3	91.2
14	SUDEEP RATHOD	18	M	396000	13.4	9.2	Case	SCALP Pso	1.0	10.5	1.9	192.5
15	BAGAPPA KARANAL	18	M	348000	13.1	12.0	Case	PsoV	3.4	10.9	2.7	110.9
16	AMRUTHA MAHADEV GUJARI	20	F	173000	13.5	12.0	Case	SCALP Pso	1.2	8.9	1.0	97.1
17	POOJA B LAKUNDI	20	F	167000	13.7	11.4	Case	PsoV	5.0	12.2	5.2	127.7
18	PRAFULL P JOSHI	21	M	191000	12.9	10.5	Case	PsoV	2.0	8.2	1.1	91.7
19	KUMAR V HUGAR	21	M	259000	13.2	10.6	Case	PsoV	7.0	9.2	2.2	110.2
20	AQEEB SHAIKH	21	M	235000	13.2	9.0	Case	PPP	2.3	8.8	1.5	72.2
21	PAVITRA YELI	23	F	433000	15.4	9.2	Case	PsoV	12.0	11.4	3.7	147.5
22	NAINA KANNUR	23	F	383000	15.7	9.9	Case	PsoV	15.0	9.2	3.0	135.7
23	MAHANANDA CHALAWADI	24	F	312000	12.1	9.5	Case	PsoV	4.0	10.4	1.0	69.8
24	VAISHALI ALONI	24	F	296000	14.5	11.7	Case	PsoV	2.0	8.6	2.3	126.2
25	PRABHUGOUDA M RAREDDY	25	M	216000	12.5	9.9	Case	SCALP Pso	0.9	8.5	2.6	143.2
26	SHRADANAND TUKARAM	26	M	402000	13.2	10.3	Case	PsoV	2.0	9.0	2.4	148.1

27	HARISH MUTTAPANAVAR	27	M	165000	12.3	8.8	Case	PsoV	6.3	8.8	3.8	104.6
28	RAJASAB NADAF	27	M	262000	14.7	9.7	Case	PsoV	16.0	9.1	3.9	158.1
29	SUMITRA KAVALAGI	29	F	364000	13.9	10.0	Case	PsoV	12.0	11.2	2.2	166.2
30	SANGEETA HIREMATH	29	F	370000	13.7	11.0	Case	Guttate Pso	9.0	8.6	2.7	148.2
31	KRISHNAPPA APPANA MALALI	30	M	176000	11.8	13.7	Case	PsoV	0.7	8.8	2.6	108.6
32	SIDRAM KUMBAR	30	M	300000	13.2	10.3	Case	PsoV	33.6	8.0	1.8	122.1
33	YSHMEEN ALEEM	30	F	262000	14.4	9.8	Case	PsoV	14.0	11.1	1.6	114.0
34	SUJATA BELLANGADI	30	F	290000	13.4	12.4	Case	PsoV	2.0	9.4	2.8	104.8
35	ALLABAKSH MANGALOR	30	M	256000	11.5	9.1	Case	PsoV	11.0	10.3	2.3	125.6
36	BABU H M	30	M	228000	13.3	11.9	Case	PsoV	8.0	9.8	2.8	117.4
37	VASANTH KALLAPPA	30	M	273000	16.9	8.7	Case	PsoV	14.0	9.4	2.2	138.6
38	SUJATA ELANGATI	31	F	259000	10.9	14.7	Case	PsoV	9.8	7.8	5.8	971.9
39	VIRESH MANTHAL	32	M	416000	14.1	9.5	Case	PsoV	2.0	9.1	4.5	173.5
40	JYOTI SADASHIV GUDIMANI	33	F	297000	12.9	10.4	Case	PsoV	13.0	10.8	1.9	110.6
41	NAGESH J	34	M	245000	12.5	10.4	Case	PsoV	1.0	8.6	1.5	97.2
42	VINOD KATTIMANI	34	M	175000	12.3	9.1	Case	PPP	3.0	11.4	1.5	83.2
43	VIJAYAKUMAR LAKKUNDI	34	M	186000	12.7	11.6	Case	PsoV	6.0	9.9	1.7	57.0

44	PADMANNA DEVARNAVADAGI	35	M	172000	13.3	18.5	Case	PsoV	5.0	9.3	2.9	84.2
45	SALIM I BAGAWAN	35	M	325000	12.8	12.5	Case	PsoV	2.5	9.6	2.5	102.1
46	SHARANAWWA NANDAYAL	35	F	314000	10.5	13.8	Case	PsoV	10.2	10.3	2.0	102.0
47	SANGANGOUDA PATIL	35	M	308000	12.7	11.0	Case	PsoV	20.0	9.7	2.3	192.2
48	PADMAVATI TIPPANATAGI	35	F	228000	12.2	15.0	Case	PsoV	5.8	10.1	2.4	97.9
49	SUBASH	36	M	359000	12.7	9.5	Case	PsoV	11.5	10.2	3.1	184.9
50	PADMANNA	36	M	124000	13.5	14.7	Case	PsoV	12.0	9.1	2.6	58.0
51	RAJU PAWAR	36	M	414000	12.7	9.0	Case	PsoV	8.0	4.9	4.3	214.2
52	SUBASH SADASHIV HADAPAD	37	M	320000	12.3	9.5	Case	PsoV	22.0	9.4	4.6	160.6
53	PRASHANTH DHANYAL	38	M	258000	13.2	8.8	Case	PsoV	1.8	8.7	1.0	81.4
54	SHASHIKALA	38	F	231000	13.6	10.0	Case	PsoV	14.0	10.0	1.6	84.0
55	VASUDEV AGNIHOTRI	38	M	311000	13.1	10.0	Case	PsoV	10.0	9.6	2.4	143.8
56	SHASHIKALA TADALAGI	38	F	259000	13.5	10.7	Case	PsoV	7.6	9.8	1.7	97.0
57	JAGADISH SURESH	38	M	282000	13.2	12.4	Case	PsoV	10.0	9.7	1.7	103.4
58	MANJUNATH BASANNA	38	M	221000	13.6	9.2	Case	PsoV	28.4	9.6	2.7	96.4
59	SALIM IMAMSAB	38	M	121000	12.6	9.4	Case	PsoV	7.8	10.3	2.4	81.1
60	SUNANDHA KRISHNA	39	F	243000	13.2	11.9	Case	PsoV	5.0	9.5	1.7	118.7
61	PRASHANTH PATIL	39	M	373000	15.5	9.5	Case	PsoV	10.0	9.6	2.3	147.6

62	SANGAPPA KATTEPANAVAR	39	M	361000	13.8	9.6	Case	PsoV	1.8	9.9	1.4	91.8
63	SUBASH ISHWAR RATHOD	40	M	252000	13.2	10.5	Case	PsoV	1.8	9.5	1.8	104.1
64	BASAMMA ASHOK MELINKERI	40	F	302000	13.7	12.3	Case	PsoV	7.0	8.5	2.0	123.8
65	PAVITRA L E	40	F	458000	18.3	10.4	Case	PsoV	8.0	9.3	3.0	144.2
66	DEVAREDDI DESAI	40	M	305000	13.8	9.1	Case	PsoV	26.0	9.2	2.5	156.6
67	SHASHIBAI CHAHVAN	40	F	338000	13.4	11.6	Case	PsoV	5.0	9.0	1.9	128.5
68	MUTTAPPA BHIMAPPA CHAVAN	42	M	301000	12.3	9.0	Case	PsoV	3.6	8.6	2.3	164.4
69	HANAMANTH RINDUSA	42	M	353000	14.0	11.5	Case	PsoV	12.0	8.9	12.6	558.4
70	BHRATI SHEKAYYA	42	F	262000	13.1	10.8	Case	PsoV	11.0	8.4	2.5	109.2
71	KAZAHUSSEN KARIMSAB	43	M	134000	14.6	11.2	Case	PsoV	13.0	8.6	6.1	89.8
72	RAMESH VIJAPUR	45	M	273000	14.1	10.0	Case	PsoV	7.2	9.1	7.5	227.3
73	SHANTAVVA KAMATGI	45	F	285000	13.3	9.9	Case	Palmar Pso	1.0	9.2	2.6	115.0
74	MARUTI SADASHIV	45	M	285000	14.6	9.1	Case	PsoV + Scalp Pso	2.6	8.7	2.0	107.3
75	MAHADEV HEGGOND	45	M	396000	16.9	9.2	Case	SCALP Pso	1.5	9.8	2.3	148.8
76	SALIYA S JAKATI	45	F	420000	15.0	10.4	Case	PsoV	8.0	9.8	2.3	183.6

77	SIDARAY NEELAPPA BIRADAR	46	M	255000	13.1	8.1	Case	PsoV	25.0	10.4	2.6	181.8
78	INDUMATHI HIREMATH	47	F	231000	13.8	11.1	Case	PPP	1.0	10.6	1.8	77.1
79	HANAMAWWA BAJANTRI	47	F	281000	14.5	11.3	Case	Guttate Pso	3.0	8.9	3.4	124.8
80	MANJUNATH BANGARSHETTI	47	M	150000	13.2	9.7	Case	PsoV	2.0	7.2	3.2	95.0
81	VEENA VINAYAK	47	F	315000	12.3	11.5	Case	PsoV	1.4	9.6	2.0	101.9
82	SHANKAR SIDRAY CHIMAD	47	M	523000	14.4	8.5	Case	PsoV + PsA	6.0	7.9	2.7	234.8
83	LALEMASHAK H BAGAWAN	47	M	224000	12.9	10.4	Case	PsoV	22.0	8.0	2.5	111.1
84	KASTURI	48	F	330000	14.2	8.7	Case	PsoV	17.6	7.8	2.5	109.8
85	CHANDU G WALIKAR	48	M	193000	13.1	16.9	Case	PsoV	2.0	8.8	3.5	112.1
86	CHIDANAND KOLKAR	48	M	274000	13.9	11.7	Case	PsoV	5.6	9.5	3.6	115.4
87	SHIVANAND PANCHAYYA SWAMI	49	M	344000	12.4	8.4	Case	PPP	1.2	9.9	2.2	134.8
88	KAUSARBANU KOLHAR	50	F	215000	11.9	11.7	Case	PsoV	1.2	8.7	2.0	98.6
89	SURESH APPASAB SALAGAR	50	M	226000	13.4	12.8	Case	PsoV	3.0	9.6	2.9	102.7
90	REVAPPA MADAR	50	M	258000	15.4	10.1	Case	PsoV	9.0	9.5	2.0	136.7
91	MALLIKARJUN YARAGALL	50	M	231000	14.5	8.9	Case	PsoV	12.0	8.4	1.6	112.8

92	KASHIBAI	50	F	410000	13.9	9.3	Case	PsoV	56.4	8.7	8.0	225.4
93	SHAINAZ BEGUM	52	F	277000	12.7	11.5	Case	Pso HKD	2.4	8.1	3.1	143.5
94	REVANSIDDAPPA BIRADAR	53	M	130000	15.2	9.7	Case	PsoV	7.2	7.6	5.6	115.9
95	DATTAPRASAD PATTAR	53	M	345000	14.7	9.4	Case	PsoV	3.2	6.6	3.0	171.5
96	ASHOK GURUBASAPPA JADAR	53	M	246000	13.6	13.7	Case	PsoV	3.4	10.1	3.2	147.3
97	PARASHURAM MUNJI	53	M	323000	12.4	12.2	Case	PsoV	12.8	9.7	2.8	144.2
98	LEENA R K RAGHA	53	F	217000	14.0	11.5	Case	PsoV	13.0	7.8	2.3	158.7
99	ANNAPPA YALLAPPA	54	M	209000	15.7	11.5	Case	PsoV	7.0	11.9	2.6	117.4
100	SHANKREPPA DODDABASAPPA	55	M	239000	12.1	10.9	Case	PsoV	2.0	8.0	2.9	110.9
101	AMEENSAB JAMADAR	55	M	95000	14.1	14.6	Case	PsoV	10.0	9.5	3.5	89.5
102	S S BEKKERI	56	M	375000	18.0	9.6	Case	Pso HKD	3.0	9.8	1.5	111.1
103	RAMCHANDAR SINGE	56	M	325000	13.6	12.3	Case	PsoV	28.0	8.9	4.0	194.7
104	NABISAB MALLI	57	M	307000	13.3	10.9	Case	PsoV	15.0	10.2	1.7	146.8
105	SHAMU RATHOD	58	M	245000	12.3	13.6	Case	PsoV	1.4	11.0	1.0	84.5
106	PARVATI SHIVANNA WALIKAR	58	F	223000	13.2	11.9	Case	PsoV	1.6	10.5	1.2	77.7
107	GURABASAPPA NALAWAD	58	M	288000	13.9	10.0	Case	PsoV	5.4	11.9	1.4	201.2

108	GURURAJ SAJJAN	58	M	204000	12.9	8.8	Case	PsoV	13.0	8.7	1.2	81.9
109	SUNANDA SURYAKANT	60	F	365000	12.9	9.9	Case	PPP	3.4	7.4	3.4	162.6
110	KHAJASAB BABSAB	60	M	178000	17.1	10.0	Case	PsoV	24.0	11.9	6.9	217.3
111	M N BILAGI	61	M	203000	20.7	8.6	Case	PsoV	1.8	7.9	1.7	120.9
112	SUBACHANDRA NAVI	63	M	264000	13.5	9.6	Case	PsoV	4.8	8.8	2.6	164.4
113	CHANDRASHEKAR D DESAI	68	M	217000	14.1	14.0	Case	Pso HKD	2.0	9.3	1.6	62.7
114	MD HANIF INAMADAR	69	M	253000	13.1	13.4	Case	Pso HKD	2.4	8.7	2.8	100.6
115	SHARIFA HASANSAB	73	F	349000	13.0	10.0	Case	Pso HKD	5.4	7.8	1.4	142.4
116	PHAKIRAPPA MADAR	82	M	201000	15.3	12.8	Case	PPP	2.0	10.3	5.1	106.0
117	SUJATHA BASAVARAJ PATTAR	50	F	245000	13.2	11.3	Case	PsoV	8.0	11.0	3.0	198.9
118	PRAVEEN PATTAR	34	M	224000	11.3	14.7	Case	Plantar Pso	4.0	12.2	1.6	160.7
119	VINOD MANTOOR	32	M	341000	12.3	10.0	Case	PsoV	10.0	11.2	1.9	97.2
120	BHIMAPPA MADHABAVI	59	M	292000	12.4	7.7	Case	PsoV	3.4	8.0	2.2	154.6
121	BASAPPA MANTUR	50	M	156000	15.3	8.4	Case	PsoV	14.0	9.9	4.1	112.3
122	SAVITRI	30	F	213000	14.0	7.8	Case	PsoV	10.8	12.0	5.7	144.3
123	UDAYKUMAR BASAVARAJ BANDAKERI	16	M	435000	12.0	8.8	Case	PsoV	6.0	11.0	5.9	340.7

124	SURESH MUJAGOND	38	M	334000	14.5	9.8	Case	PsoV	2.0	12.0	3.0	152.3
125	GOVIND RAJA KAMREDDY	38	M	423000	13.0	10.9	Case	PsoV	15.0	11.4	2.9	157.3
126	VISHNU DODDAMANI	33	M	314000	14.5	11.3	Case	PsoV	8.0	10.4	2.3	76.3
127	SHIVANAND SWAMI	50	M	223000	12.0	10.8	Case	PPP	3.0	8.0	1.5	100.9
128	SHANKARLING SINKHED	17	M	356000	15.3	12.5	Case	PsoV	10.6	9.5	1.8	99.5
129	NINGAMMA PATIL	43	F	223000	12.0	11.2	Case	PsoV	4.5	8.8	1.7	131.4
130	PRABHU C YARAGAL	40	M	443000	11.0	13.0	Case	PsoV	14.0	9.5	2.9	191.2
131	SUBHASHA N HADIMANI	48	M	354000	12.4	15.4	Case	PsoV	16.0	10.0	1.3	74.8
132	ANOOP NAIKODI	21	M	333000	13.4	9.9	Case	FOLLICULAR Pso	7.0	13.2	2.6	151.8
133	ABDUL HANNAN	48	M	432000	12.4	11.6	Case	PsoV	17.4	10.5	1.4	81.8
134	ALISHA N BIJAPUR	9	F	312000	13.2	10.6	Case	Guttate Pso	13.2	10.9	1.8	69.5
135	VIJAYGOWDA	12	M	352000	12.3	11.1	Case	PsoV	8.8	8.9	3.8	154.8
136	RAYAPPA SAIDAPPA HARIJAN	58	M	212000	17.0	8.9	Case	PsoV	12.0	12.2	3.1	95.3
137	OM SAI S KANNUR	20	M	341000	14.0	8.4	Case	PsoV	15.4	8.2	4.3	155.3
138	VARUN YARANAL	21	M	223000	12.2	10.1	Case	PsoV	6.3	9.2	2.6	108.8
139	VISHAKA P VATSA	29	F	434000	12.3	9.8	Case	PPP	3.3	7.6	1.6	232.0

140	VIVEK REDDY	32	M	187000	13.0	15.2	Case	PsoV	12.4	11.4	1.9	116.1
141	NEELAKKA TAMMANNA BARAGAL	65	M	443000	13.7	9.9	Case	PsoV	16.3	9.2	1.5	100.0
142	ASHA SANK	56	F	278000	12.3	12.4	Case	PsoV	14.3	10.4	3.5	116.7
143	KAVYA BASAVARAJ	20	F	244000	13.4	8.4	Case	PsoV	8.0	8.6	2.1	101.3
144	SRUSHITA RAJUGOUD PATIL	15	F	280000	13.1	8.6	Case	PsoV	5.0	8.5	2.0	146.5
145	BASAVARAJ CHANDRAM MIRAGI	65	M	192000	12.3	9.2	Case	PsoV	6.7	9.0	3.0	131.5
146	SANGAMESH BALAGAR	22	M	256000	11.7	8.9	Case	SCALP Pso	3.2	8.8	1.5	103.6
147	JAGADEV G BIRADAR	47	M	277000	14.6	9.6	Case	PsoV	2.4	9.1	2.7	129.1
148	MADAN KUMAR	27	M	332000	13.2	8.8	Case	SCALP Pso	3.3	11.2	2.6	207.8
149	USAMAN J SANGAPUR	40	M	344000	13.2	9.5	Case	PsoV	7.5	8.6	12.2	90.1
150	SHOBHA SHIVARAJ MAGI	45	M	236000	13.7	9.2	Case	PsoV	13.2	8.8	1.2	59.5
151	AKSHAYREDDY PATIL	26	M	247000	16.0	11.5	Case	PsoV	11.3	8.0	1.7	122.9
152	SHARANAPPA GOUDA PATIL	81	M	334000	14.7	11.6	Case	PsoV	15.3	11.1	2.9	269.5
153	GOUDAPPAGOUDA BIRADAR	50	M	223000	14.3	9.3	Case	PsoV	5.5	9.4	1.7	102.3
154	MAMTAJ GULABARAGA	35	F	339000	14.7	12.5	Case	SCALP Pso	2.4	10.3	1.7	98.7

155	SHIVANAND OURASANG	28	M	295000	12.0	10.8	Case	SCALP Pso	3.2	9.8	2.0	105.4
156	RAMESH BHEEMASHANKAR VIJAPUR	47	M	273000	14.1	10.0	Case	PsoV	10.8	9.4	7.5	227.3
157	CHANBASAPPA KHADI	65	M	233000	13.2	7.9	Case	PsoV	6.8	7.8	2.3	69.2
158	BASAVARAJ SHIVANAND GANGANAGOUDAR	19	M	331000	13.2	9.6	Case	PsoV	14.2	9.1	1.6	137.9
159	MALLAMMA NINGAPPA HONNAKATTI	48	M	234000	11.2	8.9	Case	PsoV	1.4	10.8	2.4	178.6
160	SUNIL SRISHAIL DHANYAL	26	M	284000	12.5	9.0	Case	PsoV	9.8	8.6	1.6	144.3
161	VANITA SANTOSH SHIRKANHALLI	34	F	313000	17.5	14.9	Case	PsoV	10.6	11.4	2.2	142.7
162	TUKARAM MALLAPPA BAGALI	55	M	200000	13.2	14.0	Case	PsoV	12.9	9.9	1.9	66.1
163	BASAVARAJ SIDDANAGOUDA BASARKOD37M	37	M	212000	13.6	13.2	Case	PsoV	10.0	9.3	0.2	64.5
164	DHAMAVVA BASAPPA UMRANI	50	F	264000	14.2	10.1	Case	PsoV	13.2	9.6	2.4	145.3
165	GULAPPA KUMBAR	83	M	234000	12.3	11.9	Case	PsoV	6.8	10.3	2.3	98.0
166	GURANNA S MADHABHAVI	65	M	212000	13.3	11.0	Case	PsoV	8.9	9.7	2.5	50.1

167	SALIM INAMADAR	61	M	258000	16.7	10.6	Case	PsoV	10.5	10.1	2.8	154.6
168	EASHAN AMIT SETH	20	M	329000	10.2	11.4	Case	PsoV	11.0	10.2	8.4	231.1
169	MADIVALAPPA SANGANNA BINJALBHAVI	34	M	297000	12.0	11.2	Case	PsoV	11.3	9.1	2.5	203.3
170	SURESH NIMBAL	30	M	223000	11.2	10.5	Case	PsoV	10.2	4.9	2.8	111.2
171	SABAVVA	68	F	300000	15.6	9.8	Case	PsoV	13.2	9.4	2.3	70.6
172	NOORAIN MULLA	13	F	332000	12.4	8.8	Case	Pso HKD	3.5	8.7	2.4	211.8
173	ANILKUMAR D BADIGER	57	F	256000	13.1	11.3	Case	PsoV	5.4	10.0	1.7	88.3
174	VIDYA MOPAGAR	32	F	339000	12.6	10.6	Case	PsoV	4.3	9.6	1.4	88.9
175	PREETAM PANDURANG JAGADALE	43	M	232000	11.3	11.5	Case	PPP	2.3	9.8	1.4	68.4
176	M S KUMBAR	39	M	334000	11.2	13.2	Case	PsoV	2.0	9.7	1.8	143.8
177	BHIMASHI	61	M	323000	14.3	9.3	Case	PsoV	8.4	9.6	2.7	280.4
178	JAIPAL P MURAGUNDI	42	M	214000	13.2	10.3	Case	PsoV	11.2	10.3	1.9	54.2
179	BHAVANA GUTTEDAR	17	M	446000	17.2	8.8	Case	SCALP Pso	2.1	9.5	2.3	157.0
180	SHARANAWWA NANDAYAL	35	F	314000	13.8	10.5	Case	PsoV	10.3	9.6	2.0	102.0
181	DANAMMA KORI	49	F	329000	14.8	11.1	Case	PPP	3.4	9.9	3.6	183.1
182	VIJAYALAXMI PATIL	63	F	357000	14.6	9.9	Case	Plantar Pso	2.8	9.5	1.8	149.5
183	CHANDRAKANTH G CHANAL	65	M	414000	17.2	8.5	Case	Pso Ery	41.3	8.5	1.1	34.5

184	RAVI BALU CHAVAN	34	M	243000	13.4	8.4	Case	PsoV	5.3	9.3	1.9	142.5
185	MALLANGOUDA BIRADAR	48	M	204000	15.7	10.1	Case	PsoV	1.8	9.2	1.5	52.4
186	CHANDRASHEKAR DANPPA SAVALSANG	49	M	275000	12.0	9.2	Case	PPP	2.2	9.0	3.3	147.7
187	SIDDAPPA NINGAPPA MADAR	13	M	372000	13.4	8.4	Case	PsoV	14.3	8.6	2.4	98.5
188	IRANNA GURUBASAPPA NINGANUR	69	M	259000	12.5	9.8	Case	Pso HKD	3.2	8.9	2.9	138.7
189	SHERIFA SIKKALAGAR	65	F	371000	13.2	7.8	Case	PPP	2.6	8.4	3.0	164.2
190	NAJIYA MAINUDDIN AJANAL	33	F	345000	13.4	8.3	Case	PsoV	7.4	8.6	2.2	191.9
191	VARUN YARANAL	21	M	267000	12.6	9.3	Case	PsoV	8.8	9.1	2.4	145.1
192	RAMANNA KARIYAPPA	60	M	313000	13.4	9.4	Case	Pso HKD	3.4	9.2	1.5	138.1
193	GEETA MIRAJI	43	F	322000	14.0	8.4	Case	PsoV	10.3	8.7	2.2	124.4
194	BALAMMA HONDIHAL	45	F	274000	12.8	10.1	Case	PPP	3.6	9.8	1.7	121.1
195	SHOBHA GOUDAR	28	F	285000	13.5	9.6	Case	PsoV	9.4	9.8	0.9	108.3
196	P G GIDAVEER	62	M	155000	13.6	12.6	Case	Plantar Pso	2.6	10.4	1.7	79.8
197	UMESH KOPPAD	45	M	265000	12.0	12.6	Case	PsoV	1.8	10.6	2.0	95.4
198	RAHUL KALAL	28	M	319000	12.9	9.0	Case	SCALP Pso	1.2	8.9	1.0	113.0
199	JENISHA LAIR	32	F	232000	11.3	8.0	Case	PsoV	8.7	7.2	2.3	183.6

200	YAMANU PUJARI	35	F	334000	14.5	9.7	Case	SCALP Pso	2.2	9.6	1.5	152.5
201	SAVITRI	30	F	172000	11.2	8.9	Case	PsoV	10.8	7.9	2.2	93.0
202	BASSANAGOUDA BIRADAR	51	M	151000	17.7	9.5	Case	PsoV	14.3	8.0	2.8	148.2
203	MALLAYYA NOORANDAYYA GURAVIN	59	M	316000	15.1	10.5	Case	PsoV	9.2	7.8	3.5	245.1
204	VISHWANATH	64	M	356000	13.9	13.7	Case	PsoV	10.1	8.8	3.8	249.3
205	MEHABOBI HUSENSAB SAIKH	65	F	423000	18.8	8.9	Case	PsoV	10.6	9.5	4.5	145.6
206	BABU SADEVA MORE	48	M	165000	16.5	10.8	Case	PsoV	15.5	9.9	2.1	94.8
207	CHANDRASHEKHAR B	23	M	224000	13.6	11.9	Case	PsoV	8.0	8.7	3.0	62.3
208	SIDDAMALLA RODAGI	40	M	230000	13.0	11.4	Case	PsoV	14.8	9.6	2.1	128.7
209	VASUDEV AGNIHOTRI	40	M	314000	12.8	10.9	Case	PsoV	12.3	9.5	1.7	124.0
210	SHEKAR	38	M	425000	11.2	10.3	Case	PsoV	20.0	8.4	1.7	120.5
211	SHANKAREMMA MINAJAGI	75	F	269000	16.0	13.2	Case	PsoV	18.7	8.7	4.8	227.0
212	RAMAPPA APPANA HANAGANDI	58	M	425000	18.5	11.3	Case	PsoV	22.2	8.1	2.4	166.8
213	PARNIKA AMBADAS BURA	21	F	533000	13.5	9.4	Case	Flex Pso	28.0	7.6	3.1	226.4
214	PRASANN	24	M	425000	12.4	8.9	Case	PsoV	9.9	6.6	2.7	168.5
215	REKHA PUJARI	38	F	246000	13.7	10.6	Case	PsoV	18.5	10.1	3.8	156.7

216	CHANDRAMMA	40	F	4250	11.4	10.0	Case	PsoV	32.5	9.7	2.7	1.7
217	RAMEEJ RAJA NADAF	24	M	425000	10.2	8.9	Case	PsoV	9.9	7.8	3.0	182.6
218	NINGAPPA BAPU PAMOJI	36	M	423000	11.2	7.8	Case	PsoV	14.7	11.9	2.4	166.0
219	KARTHIK	42	M	425000	12.4	13.7	Case	PsoV	12.3	8.0	2.4	170.3
220	IRAPPA HADAPAD	60	M	220000	16.5	11.0	Case	PsoV	10.0	9.5	2.7	73.8
221	APPASAB VITOBHA MORE	67	M	119000	15.9	12.2	Case	PsoV	9.9	9.8	2.9	96.0
222	TAISEEN ANVAR AVATI	22	F	332000	12.7	11.2	Case	PsoV	13.6	8.9	2.6	245.1
223	CHANDRABAGHA RAMAPPA BYAKOD	57	M	442000	11.7	10.4	Case	PsoV	16.7	10.2	3.0	260.8
224	MALLU S KEMASHETTI	42	M	421000	12.3	11.9	Case	PsoV	6.8	11.0	3.9	130.3
225	SHRISHAIL VANROTTI	56	M	234000	15.2	11.2	Case	PsoV	13.2	10.5	2.6	96.4
226	SHIVANAND SOMANATH SOMANAR	31	M	345000	11.2	10.4	Case	Guttate Pso	11.1	11.9	4.6	232.2
227	H S BAGALI	79	M	205000	10.2	11.0	Case	PsoV	10.5	8.7	2.5	92.8
228	MALLAMMA GURAPPA	55	F	232000	11.4	13.5	Case	Guttate Pso	4.5	7.4	1.6	163.5
229	SANGAPPA KUMBAR	60	M	200000	11.2	10.3	Case	PsoV	7.5	11.9	2.2	98.8
230	KAILASH PATEL	32	M	230000	12.9	11.3	Case	PsoV	19.1	7.9	2.4	65.5
231	ASHOK	32	M	350000	13.2	11.0	Case	PsoV	22.8	8.8	5.8	269.2
232	SOWMYA	36	F	268000	12.0	10.3	Case	PsoV	22.2	9.3	2.8	86.5

233	MANJUNATH	69	M	236000	11.2	10.9	Case	PsoV	3.5	8.7	2.4	65.3
234	SAHANA SANJU CHAVAN	7	F	595000	10.2	11.9	Case	Pso Ery	36.0	7.8	1.5	114.4
235	VITTAL SHIVARAI NAIKODE	63	M	236000	16.0	11.2	Case	PsoV	11.2	10.3	2.4	65.3
236	MARUTI K B	50	M	425000	13.2	11.2	Case	PsoV	5.7	11.0	2.0	18.0
237	CHANDRAWWA PARASHURAM BAGADE	39	F	425000	11.2	10.0	Case	PsoV	2.5	12.2	1.6	251.3
238	MAHAMAD ZAKI TARKAR	4	M	111000	10.2	8.4	Case	PsoV	2.1	11.2	2.8	46.7
239	SHANTA DEVI	37	F	310000	11.9	7.4	Case	PsoV	8.1	10.1	1.1	129.5
240	HAKIM SAHEEB	41	M	300000	12.2	9.3	Case	PsoV	9.1	9.8	1.5	124.7
241	GEETHA MUSTAQ	26	F	211000	13.6	10.2	Case	PsoV	10.1	11.1	1.1	113.9
242	SHRISHAIL HALAGAYA MATHAPATI	50	M	545000	12.2	10.3	Case	PsoV	11.2	14.0	2.1	82.6
243	RAJESH K	40	M	294000	12.1	11.4	Case	PsoV	10.1	9.5	5.6	163.3
244	IBRAHIM MUSHTAQ	34	M	211000	13.7	8.3	Case	PsoV	10.2	8.0	1.1	104.9
245	LALITA BAI	35	M	232000	11.2	10.2	Case	PsoV	9.9	7.7	1.1	97.6
246	SHARANBAS APPA	33	M	222000	10.4	11.2	Case	PsoV	9.3	9.8	1.2	53.9
247	SHANTABAI	65	F	321000	11.4	10.4	Case	PsoV	10.9	12.3	1.3	110.7
248	BABUGOUDA BIRADAR	59	M	435000	12.2	11.0	Case	PsoV	22.2	9.0	2.4	179.4
249	NAMRATA SAYAGAV	24	F	550000	12.4	10.2	Case	PsoV	9.9	8.9	2.0	170.3
250	K A MANAN	61	M	425000	11.3	8.0	Case	PsoV	22.5	8.4	2.4	166.8

251	SABU UPPAR	70	M	225000	14.0	11.0	Case	PsoV	16.6	10.9	2.5	117.3
252	RUKMINI	51	F	230000	12.3	9.8	Case	PsoV	14.8	11.2	3.0	125.1
253	BHIMANNA S MADAR	58	M	222000	11.3	10.8	Case	PsoV	22.3	13.2	1.6	46.1
254	SHARANAWWA	37	F	250000	10.0	9.8	Case	PsoV	23.7	8.8	1.5	81.2
255	YALLAPPA	53	M	424000	11.2	13.0	Case	PsoV	22.1	7.6	2.4	171.3
256	SABAVVA HANAMAPPA WALIKAR	69	F	300000	15.6	9.8	Case	PsoV	8.1	9.1	2.3	70.6
257	SHASHIKUMAR JOGUR	23	M	139000	12.1	11.3	Case	PsoV	17.4	9.7	3.4	74.6
258	MONISHA	27	F	254000	12.2	11.4	Case	PsoV	22.3	8.0	2.9	85.3
259	SHIVAGONDAPPA ALLAGI	80	M	217000	14.8	9.0	Case	PsoV	12.2	8.7	3.0	82.8
260	MANJUNATH BASANNA PATHAR	38	M	221000	13.6	9.2	Case	PsoV	28.4	9.1	2.7	96.4
261	LAXMI HIREMATH	6	F	422000	12.2	8.8	Control			8.9	1.4	131.1
262	FAIZAL LALEMASHAK MALAGADINNI	8	M	450000	12.7	7.9	Control			8.5	1.9	135.4
263	SANA HUSENSAB	8	F	422000	11.5	7.8	Control			8.6	1.7	134.6
264	ANIKET ANIL	9	M	468000	12.6	8.6	Control			7.4	1.3	124.5
265	ASMA SHEIKH	9	F	323000	12.3	6.8	Control			6.8	1.5	85.4
266	NETRA CHATTARKI	10	F	422000	11.5	7.0	Control			8.6	2.0	166.1
267	SAMPATH KENGAR	11	M	286000	13.2	13.1	Control			8.6	0.9	96.9
268	MASTER AJAY	12	M	256000	14.8	9.0	Control			7.1	0.8	68.2

269	NIKHIL MALLED	12	M	380000	13.4	10.3	Control			9.5	1.8	152.1
270	PREMAN M KAKHANDAKI	12	M	324000	13.6	9.6	Control			9.2	1.6	132.6
271	NOORAIN MULLA	13	M	421000	13.5	10.4	Control			8.2	5.3	289.9
272	AADARSH SAKHARE	13	M	237000	14.7	9.9	Control			6.5	2.4	105.8
273	MASTER JEEVAN BADAGI	14	M	235000	14.3	9.6	Control			9.3	0.3	79.7
274	MAHADEV AVATADE	16	M	308000	15.9	9.9	Control			8.3	1.0	114.5
275	VEDA KABADE	16	F	325000	13.6	9.7	Control			9.5	2.5	151.0
276	VINAY BADIGER	16	M	322000	12.4	9.8	Control			8.9	2.7	211.8
277	ALIHUSEN SHAKEELAH MED BIDIWALE	16	M	126000	12.4	12.3	Control			10.2	1.4	22.5
278	CHETAN ASKI	16	M	235000	14.0	8.6	Control			8.8	1.4	176.7
279	SRUJA UTTUR	17	F	133000	11.1	10.3	Control			8.7	1.8	36.8
280	VINODARAJ BHIMARAYA DODDAMANI	17	M	181000	12.7	11.0	Control			8.7	2.8	77.1
281	ANUSHRI KULKARNI	17	F	232000	12.6	9.9	Control			9.2	1.6	128.9
282	AISHWARYA TALAWAR	18	F	240000	15.6	11.7	Control			8.4	2.4	81.5
283	VIVEK RAJENDRA VALEPPU	18	M	212000	8.8	11.4	Control			8.8	2.0	164.6
284	BAHAMATARI NADAF	18	F	143000	14.0	11.2	Control			10.0	2.0	107.2

285	PRADEEP METI	19	M	296000	12.9	10.9	Control			9.2	1.4	122.8
286	PRIYANKA RATHOD	19	F	264000	14.5	10.7	Control			13.0	1.4	92.7
287	SOMANING K DEVAKATI	19	M	352000	12.4	10.3	Control			9.3	2.2	109.8
288	GANGADHAR KAMBALE	19	M	332000	12.6	14.9	Control			7.8	2.1	111.6
289	SONALI SHINDE	20	M	414000	15.9	9.4	Control			10.3	2.2	119.9
290	PARASAD VANARASE	20	M	287000	13.2	10.2	Control			9.7	2.1	147.2
291	POOJA CHAVAN	20	F	21000	12.2	11.4	Control			8.7	1.8	10.9
292	AJEET NIMBARAGI	21	M	150000	12.8	12.0	Control			10.5	2.1	50.5
293	PRABHUGOU DA PATIL	21	M	196000	12.6	10.3	Control			9.9	2.7	164.4
294	PRABHUGOU DA R PATIL	21	M	196000	14.2	13.0	Control			8.8	2.7	164.4
295	SHIVASHARA N CHAKADI	21	M	321000	13.2	9.9	Control			8.4	1.9	125.1
296	AMARSING KARE	21	F	257000	13.3	9.9	Control			9.4	1.9	109.9
297	ARATI PAWAR	21	F	276000	14.3	8.9	Control			10.2	1.5	88.5
298	NIMISHA MARIHAL	21	F	224000	12.0	8.3	Control			10.4	1.2	156.9
299	VARUN YARAMAL	21	M	267000	12.6	9.3	Control			11.0	2.4	145.1
300	RITISH RATHOD	22	M	243000	12.0	10.2	Control			8.8	1.5	80.0
301	SUDEEP APANGOUD	22	M	308000	14.0	10.3	Control			10.0	2.3	91.0

302	NANDINI KASHINATH	22	F	377000	14.7	10.2	Control			7.3	2.2	132.3
303	ANNAPURANA BUDHIYAL	22	F	331000	15.5	9.9	Control			9.2	1.8	90.1
304	RITESH TEJUSING RATHOD	22	M	243000	12.0	10.2	Control			9.3	1.5	80.0
305	NIRMALA DYAPUR	22	M	232000	11.2	13.0	Control			9.9	1.9	100.0
306	HEENA ALAMEL	23	F	272000	13.4	11.4	Control			9.0	2.4	133.4
307	SHRUSTI TILLIHAL	23	F	260000	13.2	12.8	Control			9.8	1.7	112.0
308	AJIT JADHAV	23	M	196000	13.6	12.7	Control			10.0	4.3	170.7
309	SHRISHAIL TAMBAKAD	23	M	203000	12.6	11.4	Control			9.7	2.0	81.3
310	MAHESH NANDARE	23	M	226000	15.8	11.8	Control			11.0	2.0	90.1
311	SANJANA R JAVALAGI	23	F	432000	13.2	10.7	Control			9.8	1.8	191.0
312	RAHUL SHIVANNA	24	M	339000	13.8	9.8	Control			11.0	1.9	135.0
313	SANGAMESH BAPUGOUDA	24	M	223000	11.0	10.0	Control			10.3	1.5	126.2
314	RANI BIJJARGI	24	F	332000	12.1	11.2	Control			7.7	1.8	193.0
315	VAISHNAVI BEERALDINNI	24	F	321000	14.1	8.5	Control			8.6	2.2	125.7
316	VANISHREE PAWAR	24	M	221000	12.3	13.2	Control			10.9	1.5	59.3
317	ASHWINI KATTIMANI	25	F	344000	13.0	11.8	Control			11.2	0.2	423.0
318	AKASH MALLIKARJUN JAMGOND	25	M	332000	11.2	9.9	Control			8.8	2.0	276.3

319	BASAVARAJ M HUNNUR	25	M	311000	16.8	9.3	Control			9.0	1.7	142.1
320	BHAVANI SHYAM UPPAR	26	F	244000	14.3	11.0	Control			8.8	1.8	79.9
321	SACHIN CHANDRAKANTH BIRADAR	26	M	342000	13.2	11.0	Control			11.4	6.5	308.4
322	SADIYA IMRAN KARAJGI	26	F	300000	12.3	12.2	Control			9.6	1.1	65.3
323	JYOTIK SAJJAN	26	F	314000	12.5	9.8	Control			10.8	1.6	73.4
324	AKASH KORI	26	M	302000	21.9	8.4	Control			8.1	1.4	146.7
325	SALMAN HYDER	27	M	188000	12.8	10.9	Control			8.9	1.8	105.5
326	SUNIL RATHOD	27	M	293000	12.3	9.4	Control			11.3	2.2	150.1
327	PALLAVI ANIL	28	M	330000	14.4	11.5	Control			10.1	1.5	91.7
328	PRAJWAL YANKACHI	28	M	222000	13.4	13.9	Control			8.6	2.5	93.3
329	ANNAPPA GURUPAD	29	M	311000	12.3	11.2	Control			9.0	2.7	144.5
330	SHILPA BAGALI	30	F	351000	14.5	9.2	Control			9.0	1.8	129.2
331	ROOPA CHAVAN	30	F	190000	13.9	13.5	Control			9.6	1.4	110.4
332	PARASHURAM TALWAR	30	M	324000	12.3	11.6	Control			9.7	2.3	122.7
333	CHANDA ASHPAK JATAGAR	30	F	356000	12.5	12.6	Control			8.3	0.9	112.0
334	PINAKI SARKAR	30	M	60000	11.2	10.9	Control			8.9	2.0	42.2

335	RIZWANA MUTTAVALI	31	F	312000	11.0	10.8	Control			11.0	1.6	126.3
336	HUSENABI MAKATHUM VALIKAR	31	F	400000	13.6	7.7	Control			8.2	2.6	123.7
337	JAGADEVI NAAD	31	F	348000	12.0	8.1	Control			8.5	2.4	143.8
338	BHERAPPA KASHIRAM NARUTI	31	M	357000	10.3	8.1	Control			9.8	1.8	111.7
339	VASANTH KALLAPPA MAJAGI	31	M	298000	13.6	8.0	Control			9.6	1.4	68.2
340	JAVEED MULLA	31	M	354000	11.2	11.0	Control			7.4	3.1	157.8
341	AYESHA JAHANGIRDAR	32	F	323000	13.1	8.9	Control			9.2	3.3	304.3
342	CHANDRAKALA NAIK	32	F	283000	11.9	11.0	Control			9.6	5.7	193.2
343	MAHADEVI TELI	32	F	290000	12.9	10.9	Control			8.0	4.4	171.0
344	ZAIBUN PATHAN	32	F	344000	13.1	10.4	Control			9.8	2.7	154.5
345	AKSHATA D MATH	32	M	232000	12.0	12.3	Control			8.4	1.3	59.7
346	HUSENBASHA IMAMASAB MULLA	32	M	347000	15.8	11.2	Control			9.2	1.6	101.0
347	ARUNKUMAR PARSHETTI	32	M	165000	13.1	13.5	Control			10.0	2.6	61.9
348	ASIF NADAF	32	M	434000	13.2	10.4	Control			10.2	3.0	332.5
349	SHIVANAND MUAGEPPA KABADAGI	32	M	223000	11.2	13.2	Control			9.2	2.1	164.3

350	MAHESH KAKHANDAKI	32	M	267000	13.1	11.2	Control			8.8	3.9	200.5
351	JAYASHREE GANGADHAR BHUSERI	33	F	335000	11.1	11.0	Control			8.9	2.2	94.0
352	SAVITRI HAJISABH PRASAD	33	F	267000	13.5	13.8	Control			10.8	2.2	87.8
353	VIJAYLAXMI MUKARTAL	33	F	316000	14.3	10.4	Control			9.9	3.4	160.4
354	SUNIL KATABUR	33	M	334000	12.3	11.0	Control			11.6	3.0	165.3
355	SHEKHAR SIDHANNA DESHPANDE	33	M	212000	15.8	11.2	Control			10.6	1.7	59.5
356	MALLAPPA KAKKAMERI	33	M	435000	12.0	10.4	Control			11.2	1.9	181.7
357	SIDAPPA UPPAR	33	M	564000	4.0	10.4	Control			8.8	2.2	279.0
358	NAGESH J	34	M	212000	13.0	10.4	Control			8.5	2.1	124.1
359	ROOPA S SUMBAD	34	F	362000	13.0	11.0	Control			8.4	1.5	168.7
360	MENAKA MANJUNATH JUMANAL	34	F	351000	12.0	11.0	Control			8.2	3.0	251.3
361	SADDAM HUSSEIN WALIKAR	34	M	454000	14.0	10.0	Control			6.9	2.0	431.6
362	RAJU PAWAR	34	M	520000	13.0	9.8	Control			8.6	5.5	352.0
363	NAGARAJ SANGAM	34	M	172000	11.2	9.3	Control			11.1	3.0	125.9
364	UDAY HOTAGI	34	M	667000	12.0	11.2	Control			8.9	1.8	205.6
365	NITIN SHINDE	34	M	545000	15.0	11.0	Control			9.6	3.7	302.3

366	JAYASHREE KORAWAR	35	F	294000	16.2	9.3	Control			10.5	1.8	105.4
367	RUBINA MOMIN	35	F	356000	11.4	11.2	Control			9.8	3.2	159.9
368	GURURAJ B SHRIRAMGOND	35	M	299000	12.6	10.0	Control			8.9	2.0	122.9
369	YAMANU PUJARI	35	F	359000	14.5	9.7	Control			9.6	1.5	163.9
370	S G DODAMANI	35	F	177000	13.0	9.0	Control			9.5	2.9	227.9
371	BHIMASHANKAR NAGAPPA VARJAWAD	35	M	416000	13.3	8.1	Control			7.7	3.4	164.9
372	RAKESH MATH	35	M	189000	11.4	9.7	Control			10.6	1.8	73.5
373	NAGOJI HINGOLI	35	M	455000	13.0	8.9	Control			8.5	3.7	239.3
374	ASHOK LAMANI	35	M	332000	14.5	8.1	Control			9.8	2.1	105.9
375	SAHEBGOUDA HARANAL	35	M	321000	12.0	11.3	Control			8.0	2.3	82.8
376	PRABHAVATI KOLKAR	36	F	221000	13.2	11.3	Control			8.6	1.4	118.9
377	ROOPA HARGI	36	F	267000	14.6	13.8	Control			8.6	1.9	99.2
378	SAKAMMA	36	F	313000	14.5	10.5	Control			7.2	2.9	126.6
379	HEMA GANACHARI	36	M	212000	17.9	12.0	Control			8.8	2.1	91.8
380	SHIVALING REDDY	36	M	324000	15.9	10.0	Control			9.7	4.0	213.7
381	SHARANAPPA B PUJARI	36	M	326000	15.3	8.7	Control			8.8	7.5	294.4
382	VIJAYKUMAR	36	M	465000	13.3	12.4	Control			11.2	3.8	160.1

383	MANOJ BHOSALE	36	M	319000	11.8	10.5	Control			7.3	3.7	145.0
384	RAMESH GOUNDI	36	M	434000	14.7	12.0	Control			9.7	1.5	99.1
385	RAMAPPA MAHADEVAPPA KARI	36	M	232000	13.9	11.6	Control			7.6	2.7	74.1
386	REKHA KHANAPUR	37	F	294000	14.1	11.2	Control			8.2	1.8	103.9
387	CHANNAMMA KADABALAKATTI	37	F	520000	12.1	10.6	Control			7.7	1.9	119.4
388	JYOTI NAYAK	37	F	172000	16.4	12.6	Control			8.7	2.1	80.7
389	SHREEDEVI DALALI	37	F	338000	12.5	10.0	Control			9.6	3.0	119.5
390	SHRANAGOUDA BIRADAR	37	M	109000	12.6	8.7	Control			9.8	3.0	44.3
391	ABDULRAZAK KOLI	37	M	112000	11.5	11.2	Control			7.7	1.9	61.2
392	DEVENDRA NASHI	37	M	142000	15.3	14.0	Control			9.9	4.7	144.9
393	WASEEM ATTAR	37	M	208000	14.1	12.6	Control			9.6	2.0	67.9
394	MANJUNATH BASANNA PATTAR	38	M	221000	13.6	9.2	Control			9.1	2.7	96.4
395	TOUSIF MADHABHAVI	38	M	177000	12.8	10.0	Control			7.0	1.8	63.1
396	VIKAR RATNAPUR	38	M	416000	13.4	8.9	Control			9.0	1.7	120.1
397	ARAVIND KORADI	38	M	234000	12.5	12.3	Control			8.8	3.6	89.7
398	SUNIL SANDRIMANI	39	M	267000	12.3	11.6	Control			11.0	2.8	189.6

399	MAHESH MOSALAGI	39	M	434000	15.2	13.4	Control			6.7	3.8	139.1
400	KURSHID KOLAR	39	M	330000	14.7	8.7	Control			8.2	2.8	130.8
401	RAVINDAR KHAMBHALE	40	M	510000	15.2	11.0	Control			8.1	2.7	356.2
402	VINAYAK PADAGANNA VAR	41	M	200000	14.0	9.0	Control			6.3	2.2	135.5
403	FAYAZ MULLA	41	M	222000	12.3	8.7	Control			7.8	2.0	121.2
404	MAHANTAPP A	41	M	243000	11.4	8.0	Control			8.8	1.4	74.3
405	MAHADEVI HANAMANT	42	F	314000	15.8	9.2	Control			9.8	2.9	189.1
406	PRAKASH PAWAR	42	M	235000	12.0	11.9	Control			9.1	1.5	105.7
407	DEEPA MAHESH	42	F	263000	13.6	10.1	Control			6.5	1.7	161.5
408	R B PATIL	42	M	293000	11.2	7.9	Control			9.0	0.8	70.1
409	RENUKA NAIK	42	F	217000	10.5	8.6	Control			7.7	1.9	94.2
410	BHIMAPPA KORI	42	M	246000	16.5	10.9	Control			8.9	1.2	83.3
411	RUDRAPPA K HERALAGI	42	M	234000	13.2	11.3	Control			6.7	1.6	111.6
412	LALEETA MAHESH	43	F	489000	16.9	8.7	Control			9.3	1.0	95.4
413	RAVINDRA SINGH	43	M	211000	12.7	10.8	Control			8.3	1.0	79.4
414	RACHAPPA BIRADAR	43	M	432000	11.4	11.0	Control			9.9	0.9	108.0
415	ZAKEER ATTAR	43	M	299000	13.2	13.2	Control			9.0	2.0	142.9

416	UMESH DANDOTI	43	M	434000	16.7	7.8	Control			6.7	2.1	199.7
417	MAMATA JAIN	44	F	320000	12.4	11.0	Control			10.2	1.7	113.9
418	SUVARNA HIREMATH	45	F	366000	21.0	11.0	Control			9.2	1.3	153.8
419	GULAM HUSSAIN	45	M	290000	13.3	10.9	Control			9.4	1.7	156.6
420	Y B PATIL	45	M	326000	11.7	8.6	Control			10.3	3.1	203.8
421	SHREEDEVI S KONNUR	45	F	299000	15.5	11.5	Control			11.2	2.2	153.1
422	PRAVEEN A BARADOL	45	M	343000	10.2	10.4	Control			8.0	2.4	229.4
423	REVANASIDDA VANAJAKAR	45	M	211000	15.5	7.3	Control			10.8	1.6	89.0
424	RAFIYA SALIM MULLA	45	F	217000	13.4	13.7	Control			11.5	2.7	134.0
425	SUMITRA	45	F	284000	13.0	8.8	Control			7.8	2.3	95.6
426	ASHWINI NAMADEV PAWAR	45	F	335000	15.9	10.5	Control			9.9	1.8	113.6
427	ANAND KANNUR	45	M	324000	12.3	12.3	Control			8.9	3.0	237.5
428	MAHADEVAPPA SAIBANA BHOVI	45	M	221000	11.9	11.7	Control			8.8	2.5	115.6
429	VINOD JATTI	45	M	326000	13.2	6.8	Control			9.0	2.8	160.9
430	RUDRASWAMI GANACHARI	46	M	273000	12.5	11.4	Control			7.8	2.3	91.0
431	LALSAB DALAWAI	46	M	434000	12.8	7.4	Control			10.9	2.2	263.9
432	NATHIN JAGADE	46	M	343000	13.7	8.8	Control			11.3	2.5	244.2

433	GAMANABAI RATHOD	47	F	253000	11.9	11.5	Control			8.5	3.0	105.0
434	RACHAYYA D MATH	47	M	342000	13.0	9.9	Control			11.4	2.1	265.5
435	SHIVANAND SWAMI	47	M	545000	14.0	10.5	Control			10.0	2.0	230.0
436	SANDEEP WALI	47	M	217000	11.2	11.3	Control			8.8	2.0	103.4
437	SIDRAYA HACCHAD	47	M	283000	14.3	11.0	Control			8.6	2.3	167.5
438	BHIMASHAN KAR	47	M	334000	8.0	9.8	Control			11.0	2.9	165.6
439	PARVATI BASARAGAV	48	F	314000	18.1	11.2	Control			11.3	5.6	214.0
440	SOUMYA SANGANGOW DA PATIL	48	F	296000	12.7	10.7	Control			9.7	1.6	126.1
441	RAJASHEKAR SIDDAPPA PURANKI	48	M	334000	11.6	10.8	Control			11.2	1.7	171.4
442	SHAHMSHAD GULBARGA	48	F	432000	13.2	9.7	Control			9.3	1.7	142.1
443	IRANNA AGASAR	48	M	277000	11.4	11.1	Control			10.9	3.3	235.3
444	TIMAPPA MANUR	48	M	254000	10.7	10.0	Control			11.4	1.7	82.0
445	SHAKERA BEGUM	49	F	462000	14.5	10.9	Control			8.5	2.3	81.1
446	MAHADEV NAGUR	49	M	346000	11.3	10.6	Control			10.5	2.4	139.8
447	DURAGAPPA MANE	49	M	324000	10.8	9.5	Control			7.8	2.4	139.1
448	SHIVAYOGI GOKAVI	50	M	347000	28.0	8.9	Control			8.6	3.5	184.2

449	CHANDAPPA MADAR	50	M	255000	13.7	9.9	Control			9.3	5.6	257.2
450	AMBANNA POTE	50	M	265000	12.3	10.4	Control			9.0	1.3	110.0
451	TYAMANAPPA	50	M	333000	15.6	10.8	Control			6.8	2.2	105.5
452	NANDU BISTAGOND	50	M	314000	12.0	9.9	Control			8.5	1.7	140.5
453	BASAVARAJ BHEEMRAR BIRADAR	51	M	206000	13.1	12.1	Control			10.3	3.0	115.1
454	SHOBHA AKKI	52	F	301000	14.6	10.5	Control			9.8	1.9	82.5
455	JAGADISH BIRADAR	52	M	334000	11.0	12.0	Control			8.8	2.0	113.7
456	SUDHAKAR JUMANAL	52	M	322000	14.0	8.9	Control			11.0	1.9	114.3
457	PARAPPA HUGAR	52	M	233000	13.2	8.0	Control			11.0	2.8	104.6
458	LAXMAN CHAVAN	52	M	445000	18.0	9.7	Control			8.9	2.0	142.8
459	K RAJENDRA	52	M	294000	13.2	12.0	Control			7.7	2.0	125.0
460	SADIQ MULLA	53	M	246000	11.3	9.8	Control			7.6	3.0	102.5
461	BASANNA HORAKERI	53	M	365000	12.3	11.0	Control			8.0	2.9	188.7
462	GANGUBAI	54	F	447000	16.5	13.5	Control			8.1	2.6	117.9
463	ALIHUSEN INAMADAR	54	M	293000	12.7	11.7	Control			8.5	2.0	127.2
464	DAVALMALIK IMAMSAB	54	M	259000	14.3	12.3	Control			8.8	1.8	94.7
465	PARASHURAM INDI	54	M	309000	15.4	8.8	Control			9.6	1.9	102.7
466	RUDRAGOUDA BAGEWADI	55	M	378000	18.4	8.0	Control			8.9	2.9	205.9

467	SUVARNA S PATIL	55	F	302000	12.7	9.5	Control			7.5	2.0	114.4
468	PARAVATI KARENNAVAR	55	F	342000	12.0	12.0	Control			9.8	1.8	129.0
469	MANINGAPPA GOUDA BIRADAR	55	M	199000	15.3	9.2	Control			9.3	1.3	99.0
470	CHAITRA GANIYAR	55	F	344000	12.6	11.2	Control			9.8	2.1	97.6
471	LAXMAN KOLI	55	M	232000	11.2	13.5	Control			10.3	2.8	148.2
472	CHANDRAM PUJARI	55	M	324000	12.6	12.3	Control			9.9	2.4	126.7
473	VIJAYALAKXMI ASHOK MAILASHANKAR	56	F	268000	14.2	10.7	Control			9.5	2.7	120.3
474	RAMESH HUGAR	56	M	166000	14.3	11.0	Control			7.6	2.4	64.3
475	MALAKAPPA PATIL	56	M	275000	13.2	8.8	Control			11.0	1.6	80.9
476	OMPRAKASH B ARALIMATH	57	M	317000	12.3	10.5	Control			9.3	2.3	142.1
477	SUDESH JATTI	57	M	244000	11.9	12.4	Control			10.0	2.1	118.5
478	UDAY TUPPAD	57	M	364000	9.8	9.4	Control			9.4	2.4	199.7
479	VITTAL KANABUR	58	M	216000	13.6	11.2	Control			9.5	1.5	81.4
480	BASAPPA HADAPAD	58	M	240000	14.0	9.3	Control			8.5	3.8	107.9
481	BASAVARAJ P UPPALDINNI	58	M	232000	12.3	11.0	Control			9.6	2.4	107.1
482	N S BHUSNUR	58	M	282000	13.0	8.5	Control			8.6	1.7	116.1
483	CHANDRABAGH KENNUR	60	F	270000	12.0	8.7	Control			9.6	1.3	103.1

484	SULTANBEE RAJESAB MUJAWAR	60	F	343000	12.3	10.9	Control			8.0	1.4	115.9
485	DUNDAYYA MATH	60	M	265000	14.3	12.0	Control			9.4	2.6	130.8
486	SABUDDIN MELIMANI	60	M	345000	12.9	13.4	Control			8.7	2.2	136.3
487	CHANNAPPA HATTI	60	M	436000	11.3	11.7	Control			9.7	1.9	134.2
488	SHIVANAND TADAVALGI	60	M	182000	12.1	11.0	Control			9.0	2.8	143.2
489	UJJWALA RAMANNAVAR	61	F	381000	15.9	8.9	Control			9.7	3.0	254.3
490	SHANTABAI M PATIL	61	F	448000	14.4	9.0	Control			8.6	2.4	182.8
491	SHANTABAI M PATIL	61	F	400000	13.2	9.0	Control			8.8	2.4	163.2
492	CHANNAPPA HATTI	61	M	345000	13.4	9.9	Control			9.0	2.7	169.5
493	REVAYYA MATH	62	M	207000	15.0	11.3	Control			8.8	2.2	122.7
494	ZAITUN A LONI	62	F	287000	14.7	12.0	Control			10.0	1.3	71.0
495	NEELAMMA SIDDANATH	62	F	434000	14.2	10.0	Control			10.2	1.9	125.3
496	KALLAPPA BIRADAR	62	M	444000	12.6	9.8	Control			9.8	2.3	200.0
497	SHIVAPPA JANGAMSHETTI	62	M	434000	13.7	12.4	Control			11.1	2.4	294.3
498	ASHOK LAMANI	63	M	221000	14.0	12.0	Control			9.5	1.5	62.7
499	KASHINATH	63	M	267000	15.6	10.8	Control			11.3	1.9	78.2

500	BHIMASHANKAR HANJAGI	64	M	438000	12.1	11.7	Control			8.9	1.8	234.7
501	MALLAPPA IRAPPA NIDONI	64	M	156000	11.0	7.8	Control			6.7	1.7	84.8
502	S I SOUDI	64	M	143000	12.8	8.9	Control			11.6	2.2	78.1
503	V N ANGADI	65	M	178000	13.2	12.4	Control			9.0	1.8	93.3
504	RAMANNA JADAR	65	M	186000	11.9	6.7	Control			6.8	2.6	76.2
505	SOMANING GOJJI	65	M	409000	14.3	9.9	Control			8.4	2.8	198.3
506	GURANNA S MADHABHAVI	65	M	334000	11.0	13.0	Control			8.7	2.8	238.6
507	B H HIREMATH	65	M	213000	11.4	11.2	Control			9.6	1.8	65.6
508	SUNITA JAHAGIRADAR	66	F	126000	14.3	10.7	Control			9.9	1.8	73.6
509	MALASIDDA	68	M	306000	43.8	9.2	Control			9.1	6.0	442.4
510	KALLAPPA SANKH	68	M	143000	12.1	11.4	Control			8.8	1.7	41.6
511	SADANAND KORI	68	M	333000	11.1	10.9	Control			11.2	1.8	180.8
512	BASAPPA DUNDUAPPA ATHANI	69	M	432000	14.3	8.9	Control			9.6	1.7	176.3
513	ANIL H HONAWAD	70	M	455000	10.9	8.7	Control			8.6	1.9	297.4
514	A G MATH	72	M	179000	14.2	8.9	Control			9.1	2.1	140.7
515	BOURAMMA	72	F	262000	11.2	10.6	Control			7.6	2.1	82.1
516	NINGANGOUDA PATIL	79	M	277000	12.3	12.4	Control			8.5	2.2	112.1

517	KASTURIBAI TALAWAR	80	F	222000	11.2	7.5	Control			7.9	4.3	144.4
518	SIDRAM BHIMASHA YADAWAD	84	M	380000	14.5	10.6	Control			9.7	7.3	424.2
519	BASAPPA SIDDAPPA BANGI	85	M	434000	11.2	9.8	Control			8.1	2.3	301.3
520	PEERAPPA HOSAMANI	88	M	331000	12.5	8.7	Control			7.2	1.9	167.0

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