HISTOPATHOLOGICAL ANALYSIS OF SCALY SKIN LESIONS OF NON-INFECTIOUS ETIOLOGY

Dissertation submitted to

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In partial fulfillment of the requirement for the degree of

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M.D. (PATHOLOGY)

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DEPARTMENT OF PATHOLOGY

CHENNAI MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE

TRICHY – 621 105.

DECLARATION

I solemnly declare that the dissertation entitled **"HISTOPATHOLOGICAL ANALYSIS OF SCALY SKIN LESIONS OF NON INFECTIOUS ETIOLOGY"** is a bonafide research work done by me in the Department of Pathology at Chennai Medical college Hospital & Research centre, Trichy during the period from July 2014 to July 31st 2016 under the guidance and supervision of DR. S. PRIYA BANTHAVI MD., Associate Professor, Department of Pathology, CMCH&RC, Trichy.

This dissertation is submitted to the Tamilnadu Dr. M.G.R.Medical University, Chennai towards the partial fulfillment of the requirement for the award of M.D., Degree (Branch III) in Pathology.

I have not submitted this dissertation on any previous occasion to any university for the award of any degree.

Place: Trichy

Date: DR.MANIMEGALAI.S

CERTIFICATE

This is to certify that the dissertation entitled **"HISTOPATHOLOGICAL ANALYSIS OF SCALY SKIN LESIONS OF NONINFECTIOUS ETIOLOGY"** is a record of bonafide work done by Dr. S. MANIMEGALAI, postgraduate student in the Department of Pathology, during the course of the study (2014-2017), Chennai Medical College Hospital and Research Centre, Trichy under the supervision and guidance of Dr. S. PRIYA BANTHAVI, MD.

This dissertation is a record of authentic work done by the candidate, **Dr. MANIMEGALAI. S;** this work was carried out by the candidate herself under my supervision.

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The research proposal submitted **Dr Manimegalai**. S - I year Post Graduate , Department of Pathology, Chennai Medical College, was discussed and analyzed by the Institutional Ethics Committee of the CMCH&RC. The committee approved the research project subject to existing rules and regulations.

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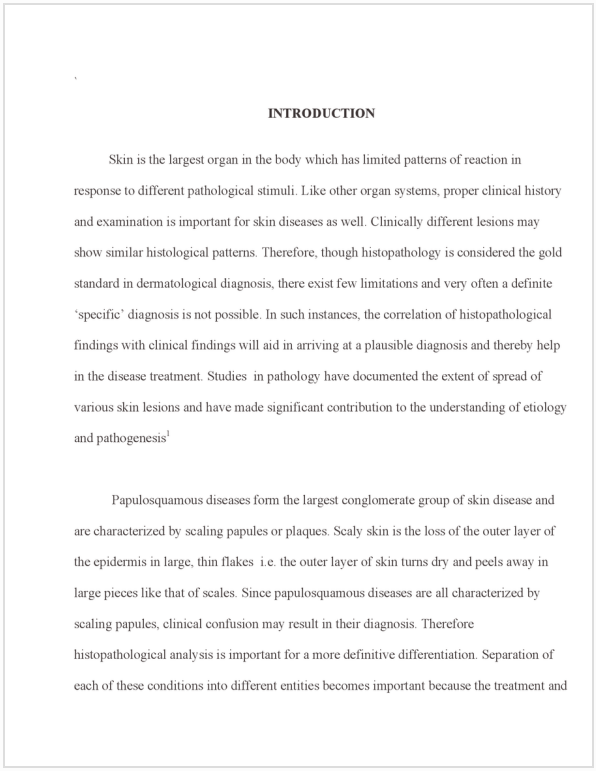
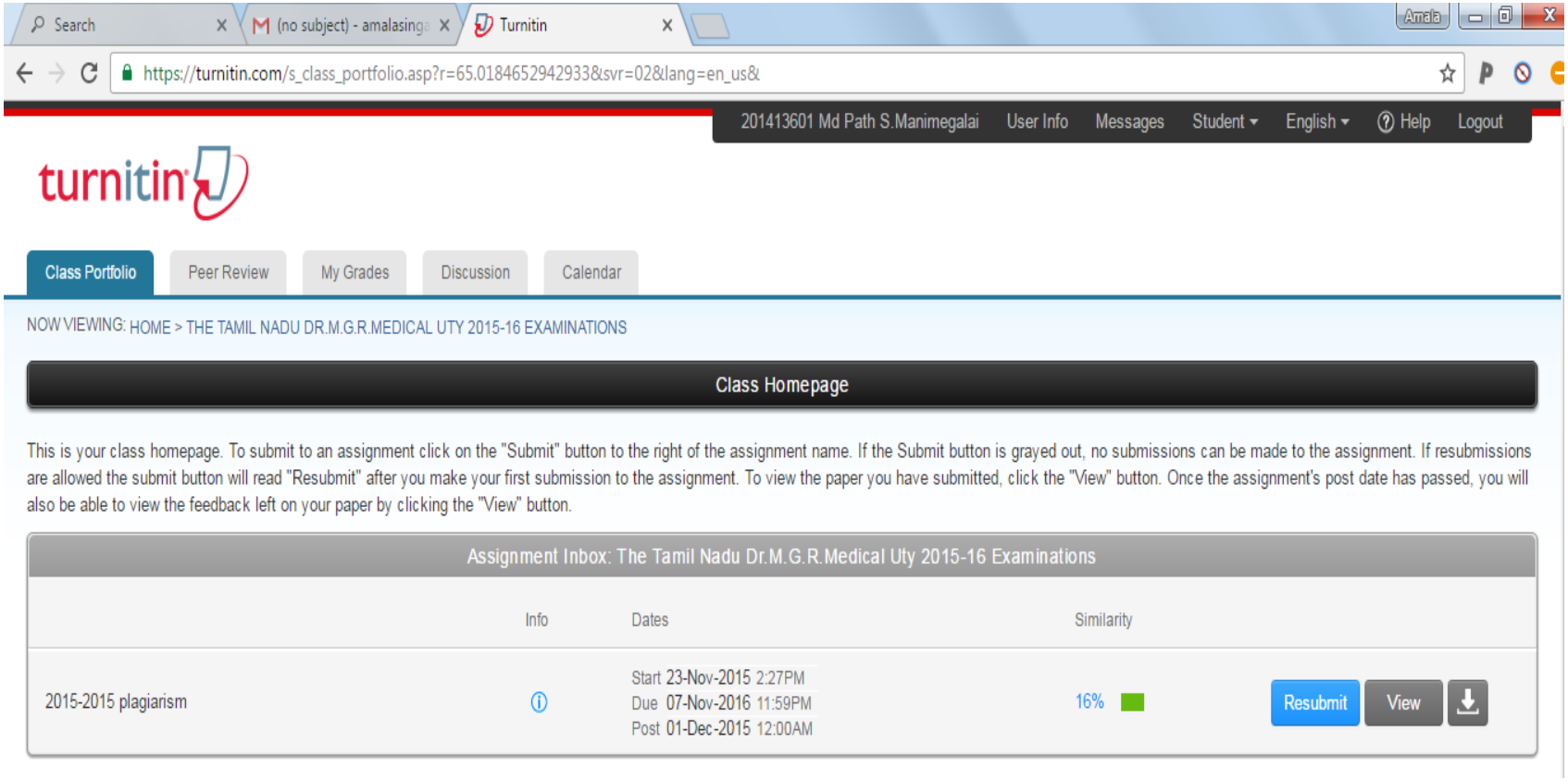


INTRODUCTION

Skin is the largest organ in the body which has limited patterns of reaction in response to different pathological stimuli. Like other organ systems, proper clinical history and examination is important for skin diseases as well. Clinically different lesions may show similar histological patterns. Therefore, though histopathology is considered the gold standard in dermatological diagnosis, there exist few limitations and very often a definite 'specific' diagnosis is not possible. In such instances, the correlation of histopathological findings with clinical findings will aid in arriving at a plausible diagnosis and thereby help in the disease treatment. Studies in pathology have documented the extent of spread of various skin lesions and have made significant contribution to the understanding of etiology and pathogenesis[1]

Papulosquamous diseases form the largest conglomerate group of skin disease and are characterized by scaling papules or plaques. Scaly skin is the loss of the outer layer of the epidermis in large, thin flakes i.e. the outer layer of skin turns dry and peels away in large pieces like that of scales. Since papulosquamous diseases are all characterized by scaling papules, clinical confusion may result in their diagnosis. Therefore histopathological analysis is important for a more definitive differentiation. Separation of each of these conditions into different entities becomes important because the treatment and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LIST OF ABBREVIATIONS

VEGF	Vascular Endothelial Growth Factor
TNF	Tumor Necrosis Factor
IL	Interleukin
IFN	Interferon
CD	Cluster Differentiation
UV	Ultraviolet
MHC	Major Histocompatibility Complex
HHV	Human Herpes Virus
NGF	Nerve Growth Factor
AD	Allergic Dermatitis
IHC	Immunohistochemistry
LPP	Large Plaque Parapsoriasis

Introduction

INTRODUCTION

Skin is the largest organ in the body which has limited patterns of reaction in response to different pathological stimuli. Like other organ systems, proper clinical history and examination is important for skin diseases as well. Clinically different lesions may show similar histological patterns. Therefore, though histopathology is considered the gold standard in dermatological diagnosis, there exist few limitations and very often a definite 'specific' diagnosis is not possible. In such instances, the correlation of histopathological findings with clinical findings will aid in arriving at a plausible diagnosis and thereby help in the disease treatment. Studies in pathology have documented the extent of spread of various skin lesions and have made significant contribution to the understanding of etiology and pathogenesis[1]

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important because the treatment and prognosis is disease-specific. These lesions can also be associated with hypo and hyperpigmentation.

The papulosquamous group of diseases include psoriasis, parapsoriasis, lichen planus, lichen nitidus, prurigo simplex,prurigo nodularis, pityriasis rosea, pityriasis rubra pilaris and many more. Certain conditions, like psoriasis mimic diverse dermatological conditions as they present with numerous clinical variants leading to diagnostic dilemma for the clinician. In such cases histopathological diagnosis will help the dermatologist in instituting proper therapy and can vary the prognosis significantly.

Aim and Objectives

AIMS AND OBJECTIVES

AIM OF STUDY

To study the age and sex distribution and histopathological spectrum of non-infectious scaly skin lesions.

OBJECTIVES OF THE STUDY

1) To analyse the histopathological spectrum of clinically diagnosed non-infectious scaly lesions in our institution.

2) To assess the various scaly skin lesions in patients subjected to biopsy for this study in relation to the age and sex distribution.

3) To correlate the histopathological diagnosis with the clinical diagnosis.

4) To study the vascular changes in the papillary dermis of biopsies reported histopathologically as psoriasis vulgaris using an immunohistochemical marker.

Review of Literature

REVIEW OF LITERATURE

The skin is the largest organ of the body, accounting for about 15% of the total body weight in adult humans[2]. The skin has many types of cells which are helpful in mechanical support, photoprotection, immunosurveillance, nutrient metabolism and repair[3]. It consists of two main layers, epidermis and dermis along with a third layer termed subcutis[4]. These layers depend on each other functionally.

On a section, the dermal–epidermal junction is undulated with ridges of the epidermis, known as rete ridges which project into the dermis. The mechanical support for the epidermis is provided by the dermo-epidermal junction, which acts as a partial barrier against exchange of cells and large molecules[5,6]. The epidermal layer is composed primarily of keratinocytes along with few langerhan cells, melanocytes, neuroendocrine cells and unmyelinated axons. It is separated from the dermis by a structurally and chemically complex basement membrane[7].

Dermis consists of neural cells, endothelial cells, macrophages and fibroblasts enveloped within a matrix of collagen and glycosaminoglycans. Adnexal structures extend from the epidermis into the dermis. Adnexal structures consists of specialized cells required for

temperature regulation, hair growth and epithelial renewal. The dermis rests on subcutis, composed of adipose tissue, which acts as a shock absorber and thermal insulator[8].

EMBRYOLOGY

The skin arises by the juxtaposition of two major embryological elements: the prospective epidermis, which originates from the surface area of the early gastrula, and the prospective mesoderm, which is brought into contact with the inner surface of the epidermis during gastrulation[9,10]. The mesoderm not only provides the dermis but is also essential for triggering the differentiation of the epidermal structures, like the hair follicle[11]. The development of the epidermis and its appendages relies on specific initiation signals. Communication between the Notch and Wnt (wingless-related) signalling pathways, with β-catenin, Lef1 and Notch peptide trigger the epidermal formation[12]. Following the trigger, by 3 weeks of gestation it forms a single layer of undifferentiated, glycogen-filled ectodermal cells[13.] They differentiate focally into two layers, the superficial periderm and the basal stratum germinativum by about 5 weeks of gestational age[14.] The intervening layer called stratum intermedium develops between the periderm and stratum germinativum by 10 weeks. The periderm consists of large cells immersed in the amniotic fluid which bulges out from the epidermal

surface. By 19 weeks, the periderm begins to flatten and several layers of intermediate cells are formed. (Fig 1.)

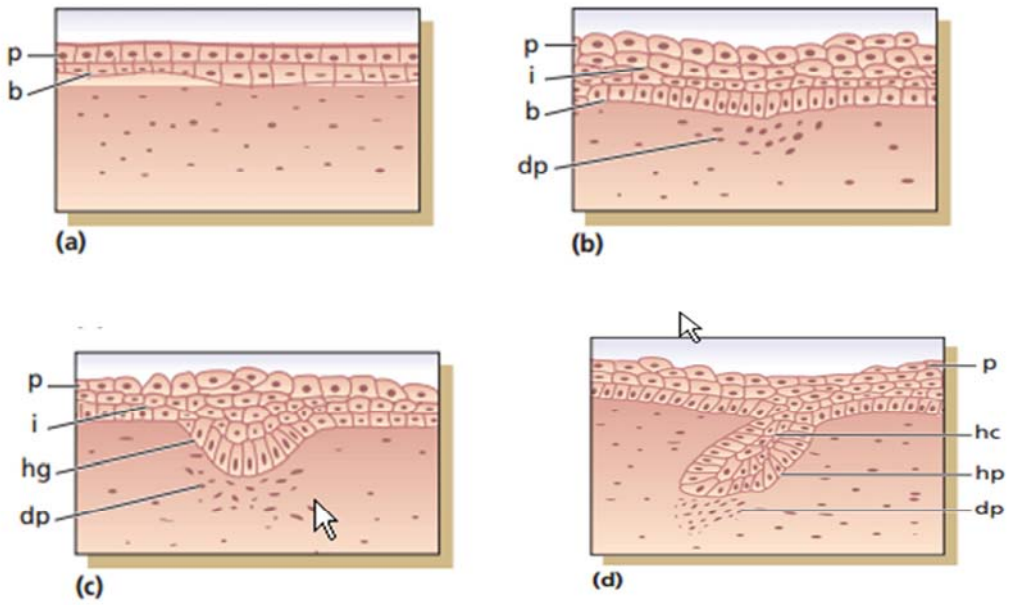


FIG 1. EMBRYOLOGY OF SKIN (a). At 4 weeks the periderm (p) and germinative layer (b) clearly seen. (b) At 11 weeks, The epidermis is made up of cuboidal basal cells (b), and stratum intermedium (i) appears above them. The periderm (p) consists of a single cell layer with mesenchyme cells (dp) aggregating below a presumptive hair follicle. (c) Hair germ (hg) stage. Basal cells become columnar and starts growing downwards. (d) Hair peg (hp) stage. Cells which form 'hair canal' (hc) in later stages[15].

Around 23 weeks, keratohyaline granules are found in association with stratum intermedium. Keratinocytes are almost fully formed just beneath the stratum corneum, whereas periderm cells have shed.

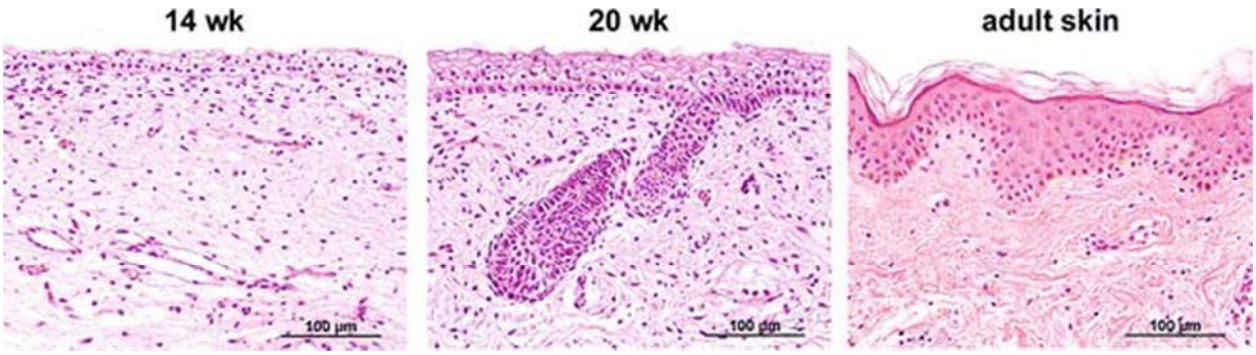


FIG 2. INTEGUMENTARY HISTOLOGY[16].

HISTOLOGY (Fig. 2)

Keratinocytes and dendritic cells principally constitute the epidermis. The epidermis is divided into five layers (Fig. 3)

I. Stratum basalis

II. Stratum spinosum

III. Stratum granulosum

IV. Stratum lucidum

V. Stratum corneum

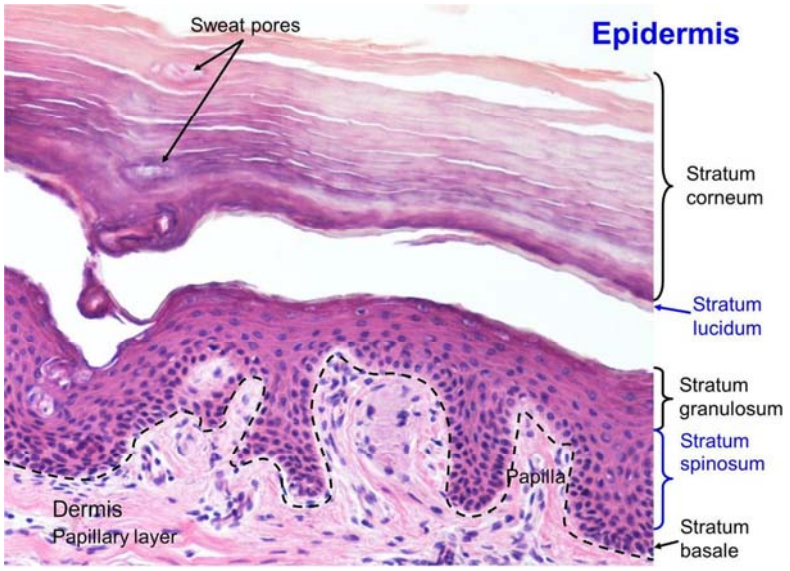


FIG 3. LAYERS OF EPIDERMIS[17]

Stratum basalis

It helps in regenerating other layer of epidermis by repeated mitotic division. It is composed of single layer of columnar cells which lie with their long axis perpendicular to the dividing line present between the epidermis and dermis. Desmosomes or intercellular bridges helps in connecting the basal cells with overlying squamous cells whereas hemidesmosomes attaches the basal cells with underlying basement membrane.

Stratum spinosum

It is also called as prickle cell layer and consists of polyhedral keratinocytes which is usually of five to ten layers thick. These cells contain large pale staining nuclei and prominent nucleoli, which

indicates active protein synthesis. The cytoplasm contains intermediate filament called cytokeratin that accumulate to form tonofibrils[18]. The adhesion of two adjacent keratinocytes is mediated by desmosomes, which is formed by convergence of the tonofibrils.

Stratum granulosum

It is also called as granular cell layer composed of flattened keratinocytes, since it loses its polyhedral shape. The cytoplasm of the granular layer contain dense basophilic keratohyaline granules, which contain proteins rich in sulfur containing amino acids such as cysteine and involucrin that interact with tonofibrils. The keratinocytes of granular layer lose their nuclei and cytoplasm as it progresses towards the surface. This results in the formation of keratin mass, which constitutes the surface covering of skin.

Stratum corneum

It is also called as keratin layer and composed of anucleate sheets and flakes of keratin. In formalin fixed specimen, keratin layer exhibit a basket-weave pattern due to the presence of large intra-cellular spaces. These spaces are due to inadequate fixation and subsequent removal of the soluble constituents by water, ethanol and xylene during processing.

Stratum lucidum

The lowest portion of the stratum corneum appears as a thin homogenous eosinophilic zone called stratum lucidum. It is more pronounced in palms and soles.

Basement membrane zone

It is located at the junction between the basal surface of basal keratinocytes and the underlying dermis. It is composed of lamina lucida, lamina densa and fibroreticular lamina. The fibro reticular lamina contains type VII collagen that connects the lamina densa to type IV collagen in the dermis and fibrillin microfilaments that connects to dermal elastic fibres.

Melanocytes

Melanocytes are found scattered in the basal layer having a small dark staining nucleus and clear cytoplasm. The average number of melanocytes to basal cells is one of ten cells in the basal layer. Melanosomes produce melanin from the amino acid tyrosine within specific cytoplasmic organelles called melanosomes. When exposed to ultraviolet light the melanosomes, which is situated over the nucleus, like a cap will deposit melanin and produce protective effect. Melanosomes are transferred to keratinocytes through cytoplasmic process of melanocytes. Melanocytes are abundant in areas, which are most exposed to light.

Langerhan cells

These cells are derived from the bone marrow and are present in all layers of epidermis and upper dermis,but more pronounced in the prickle cell layer. These cells have irregularly lobulated nuclei and have clear cytoplasm. Cytoplasm contains birbeck granules and the cytoplasmic process extends and insinuate between keratinocytes of all the layers. Langerhan cell play an important role in contact sensitization and immuno-surveillance against viral infections and neoplasms of the skin.

Merkel cells

These cells are present in the basal layer of epidermis, oral mucosa and in the bulge region of hair follicle. It is a touch receptor and contain neuroendocrine type membrane bound vesicles in their cytoplasm. They are rounded cells with pale staining cytoplasm and have round pale stained nuclei. They are frequent and recognised immunocytochemically. Merkel cells are distinguished from keratinocytes by cytokeratin 18 specific antibodies[19].

Skin appendages

The surface epithelium extends as a downgrowth into the developing subepithelial layers of mesoderm to become dermis and subcutis. Dermis has many skin appendages.

The skin appendages include:

1. Hair follicles
2. Sebaceous glands
3. Eccrine sweat glands
4. Apocrine glands

Hair follicles

The hair follicle has a complex structure. It has thin cylindrical structures composed of organized arrangement of keratin. The hair follicle has three parts, lower portion-extending from the base of the follicle to the insertion of the arrector pili muscle. Middle portion or Isthmus- extending from the insertion of the arrector pili to the entrance of the sebaceous duct. Upper portion or Infundibulum-extending from the entrance of the sebaceous duct to the follicular orifice. Hair follicle consists of five concentric layers of epithelial cells.There is a bulbous expansion at the base called hair bulb which encloses the hair papilla. The hair shaft consists of medulla, cortex, cuticle, internal root sheath and external root sheath. The external root sheath is separated from the sheath of connective tissue by a thick specialized basement membrane called glassy membrane.

The growth of the hair is cyclical and exhibits three phases.

1) Phase of active growth (Anagen)
2) Phase of involution (Catagen)
3) Resting phase (Telogen)

The body hair is fine and soft in infancy, childhood and females known as vellus hair, whereas the scalp has coarser hair called as terminal hair. The vellus hair is replaced by terminal hair due to the development of male sex hormone production at puberty. Hair growth averages about 0.4mm daily.

Sebaceous glands

At the time of birth, sebaceous glands are well developed because of maternal hormones. They undergo atrophy after few months. At puberty sebaceous glands are increased, as a result of increased androgen secretion. Sebaceous glands has many lobules that connects into the common excretory duct lined by stratified squamous epithelium. Two forms of sebaceous glands are encountered. Most of the sebaceous glands are associated with the hair follicle and are located at the junction between its upper and lower two third, developing as a lateral protrusion from the hair follicle. In few areas, sebaceous glands open directly onto the skin surface and are independent of hair follicles. These forms are seen in eyelids, nipple, areola and in the buccal and labial mucosa. One or more sebaceous

glands surrounds each hair follicle and these glands lie within the fibrous sheath provided by the outgrowth of external root sheath. The arrector pili muscle which consists of bundles of smooth muscle fibres has one end inserted into the sheath of the follicle at a point below the sebaceous glands and the other end inserted into the dermal papillary area beneath the epidermis. Each hair follicle with its associated sebaceous glands and arrector pili muscle together form the pilosebaceous unit. The peripheral layer of the sebaceous lobule is composed of cuboidal, deeply basophilic cells that contain no lipid droplets, whereas the centrally located cells has more lipid droplets that can be detected using fat stain. The acinar cells presenting towards the duct has increased lipid content, that causes distension and degeneration leading to discharging their contents called sebum. This process is called holocrine secretion. The sebum provides waterproofing of the skin and hair follicle.

Eccrine sweat glands

Eccrine sweat glands are distributed throughout the skin but are more frequent on the palms, soles, forehead and axilla. During the second trimester of intrauterine life they arise as downgrowth from the epidermis. Around the junction between the dermis and subcutis eccrine glands are located. The secretory gland components is lined by inner layer of large columnar cells, having central oval nucleus and pale staining cytoplasm admixed with smaller dark staining cells. Outer layer composed of

discontinuous, contractile myoepithelial cells that are spindle shaped arranged with their long axis parallel with the long axis of the coiled tubular gland. The secretions formed from the glands are passed into the coiled eccrine duct. The ductal portion is divided into intradermal and intraepidermal portion. The intraepidermal portion of the duct is called acrosyringium. The duct is lined by double layer of small, cuboidal and deeply basophilic epithelial cells with the prominent microvilli. The luminal surface has a characteristic eosinophilic appearance called the cuticle. These eccrine glands synthesise a thin watery liquid called sweat, that is passed along the eccrine duct and deposited on the skin surface. Sweat contains sodium, chloride ions, urea and some small molecular weight metabolites. It is considered as the important component of the thermoregulatory system,it helps by lowering the body temperature through the evaporation of sweat. Eccrine glands are highlighted by immunohistochemical stains for S-100 protein and carcinoembryonic antigen.

Apocrine glands

Apocrine glands are predominantly seen in the areola of the breast, axilla and genital regions. These glands are large and produce viscid, milky secretion,that is secreted into an adjacent hair follicle. The gland appears coiled having widely dilated lumen. The cells lining the glands are cuboidal and have an eosinophilic cytoplasm. They have a discontinuous

myoepithelial layer between the base of the secretory cells and basement membrane.

Dermis

The thickness of the dermal and subcutaneous layer corresponds to the thickness of the skin. The dermis has a superficial papillary dermis and a deep reticular dermis. In the normal dermis lymphocytes, mast cells and macrophages are scarcely present, whereas in many skin diseases it appears to be increased.

The epidermal layer is devoid of blood and lymphatic supply. The vascular supply of the upper part of the dermis and superficial appendages is either by the superficial plexus or subpapillary plexus. Subcutis, deeper aspect of the dermis and the deep dermal appendages are supplied by deep plexus or cutaneous plexus. In case of inflammation, several inflammatory mediators VEGF-A, TNF-α, IL-6, and IL-1β, activates blood endothelial cells leading to increased blood flow as a consequence of vessel dilation and edema formation due to increased permeability of the blood vessels.

Nerve supply

Efferent nervous system- It is formed by the sympathetic component of autonomic nervous system which is of non-myelinated fibres. It supplies blood vessels and dermal appendages, particularly to arrector pili muscle and dermal appendages.

Afferent nervous system- It is composed of both myelinated and non-myelinated fibres. It helps in the perception of cutaneous sensation by transmitting impulses from various nerve endings. The specialised nerve endings in the skin are Meissner's, Pacinian and Ruffini's corpuscles. Meissner’s corpuscles are responsible for touch sensation. These corpuscles are more prominent in the papillary dermis of the pulps of the fingers and toes, and soles and palms. Pacinian corpuscles helps in the detection of deep pressure and vibration. They are found deep in the subcutis and are more numerous in palms and soles.Ruffini’s corpuscles are mechanoreceptors and are seen in the soles.

Non-infectious erythematous papulosquamous diseases of skin

Skin lesions clinically present as hypopigmented or hyperpigmented macules, papules, nodules or patches. Each clinical presentation has different histopathological features, hence understanding the histopathology is important for confirmation of the clinical diagnosis. Because of the frequency of occurrence, papulosquamous disease acquires considerable importance. Like other organ systems clinical history and examination is necessary for diagnosis of skin disease. Because all papulosquamous diseases are characterized by similar morphological characteristics it is feasible to consider them as a group.

This group of diseases include

Psoriasis,

Lichen planus and lichenoid reactions,

Pityriasis rosea,

Pityriasis rubra pilaris,

Prurigo nodularis,

Parapsoriasis

Erythema annulare centrifugum

Lichen planopilaris,

Lichen nitidus and

Seborrheic dermatitis

PSORIASIS

Psoriasis, the word derived from 'Psora' in Greek which means 'the itch'. Psoriasis is characterised by chronic relapsing lesions and has variable clinical features. It causes significant morbidity by affecting 1-3% of the world population[20]. Psoriasis can affect all age groups, but the onset is usually between 20-30 years of age and sex incidence appears to be equal in males and females. Genetic predisposition plays an important role in disease expression. One-third of patients with the disease have at least one first degree relative with the disease.It has been divided into Psoriasis Vulgaris, Generalized Pustular Psoriasis, and localized Pustular Psoriasis. Psoriasis vulgaris is the commonest type of psoriasis, accounting for 90%

of all cases[21]. Most of the patients present with red or salmon pink plaques that are covered by silver-white scales. These plaques are well- delineated from the surrounding normal skin and occur most commonly on the extensor aspects of elbows and knees, on the scalp, lumbosacral region and umbilicus. The distribution of these plaques appear to be symmetrical. Koebner phenomenon, in which new lesions develop at sites of trauma or pressure is characteristic of acute inflammatory psoriasis.

Children and adolescents may develop papules,which are less than 1cm in diameter that erupt on the trunk about 2 weeks after a β-haemolytic streptococcal infection such as tonsillitis,pharyngitis or a viral exanthem,such lesions are termed Guttate Psoriasis[22]. It is self-limiting and resolves within 3–4 months of onset, although its long-term prognosis is unknown.

Generalised pustular psoriasis also called as von Zumbusch psoriasis, is characterised by small, monomorphic, sterile pustules that develop on painful, inflamed skin. Generalised pustular psoriasis is precipitated by intercurrent infection and abrupt withdrawal of systemic and, on occasion, ultrapotent topical corticosteroids.

Co-morbidities - Psoriatic nail disease occurs in 50% of patients with psoriasis. They are characterised by pitting, onycholysis (nail plate separation), oil spots (orange-yellow sub-ungual discolouration)and

dystrophy. Studies suggest that psoriasis is associated with systemic disorders including Crohn's disease, Diabetes mellitus, Metabolic syndrome and depression[23] due to the presence of circulating proinflammatory factors and endothelial activation.It has been reported that moderate to severe form of psoriasis is associated with cardiovascular diseases[24.]

Pathogenesis

Psoriasis was thought to be a disease primarily due to dysfunctional epidermal keratinocytes until the late 1970[25]. But further clinical and basic research observation indicates that the activation of lymphocytes play an important role in the pathogenesis of psoriasis.The successful treatment of patients with psoriasis using cyclosporine A, an immunosuppressive agent that inhibits T-cell proliferation and cytokine production, was the first clinical evidence to suggest the potential role of T- lymphocytes in the pathogenesis of Psoriasis[26].

The main pathogenic features of psoriasis are:

Abnormal keratinocyte differentiation

Hyperproliferation of keratinocytes

Infiltration of inflammatory components into the skin[27]

The first two features are related to the normal wound healing process. The inflammatory component infiltration is caused by variety of

cytokines, immune and inflammatory modulators, which are released by keratinocytes[27]. The amino acid sequences from streptococcal M-protein share the sequences with keratin 17, therefore an epitope on keratin 17 may be a target for autoreactive lymphocytes in psoriasis[28,29.] Patients with psoriatic lesions has $CD4^+$ T-lymphocytes and $CD8^+$ T-lymphocytes in the papillary dermis and epidermis. Cytokines such as interleukin-2 (IL-2), tumor necrosis factor-alpha (TNF) and gamma-interferon (IFN) are produced by activation of lymphocytes[30]. These cytokines stimulate keratinocytes to produce interleukin-8, which is a potent T-lymphocyte and neutrophil chemoattractant resulting in the formation of Munro's microabscesses.

Recent studies show that the pathogens carrying foreign antigens first activate the dendritic cells and macrophages to release IL-23, IL-1b and other pro-inflammatory cytokines. These cytokines activate dermal T cells and secrete abundant IL-17, further promoting the conventional acquired immune responses. IL-17, IL-22 and TNF act on keratinocytes and induce keratinocyte hyperproliferation[31]

Histopathology-

The early psoriatic lesions show elongation and dilatation of blood vessels of the papillary dermis with associated edema and lymphocytic infiltrate around the vessels (perivascular cuffing)[32.] Vessels are dilated

and tortuous, with some neutrophils in their lumen[33]. Lymphocytes and neutrophils extends to the lower portion of the epidermis, where spongiosis develops[34.] Later focal changes occurs in the epidermis which include parakeratosis, Munro's microabscess, hypogranulosis, acanthosis, elongation of the rete ridges, thinning of suprapapillary plates, Kogoj micropustules (aggregation of neutrophils in the stratum spinosum) and basal cell vacuolisation[35]. (Fig. 4)

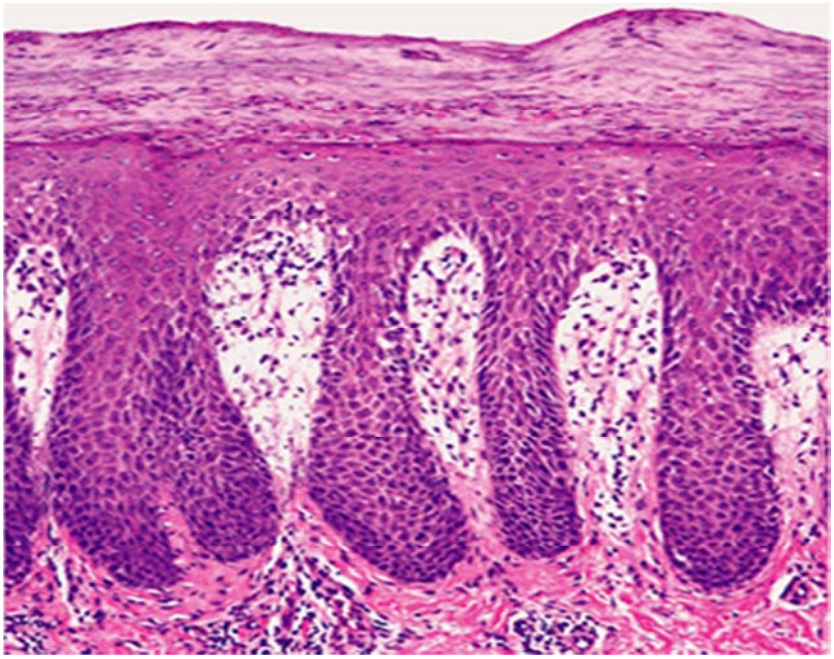


FIG 4: HISTOPATHOLOGY OF PSORIASIS

Role of immunohistochemistry in psoriasis

Recently, many studies are carried out by the dermatologists and pathologists to determine more criterias which would be significant in determining the course of psoriasis and also the outcome of treatment modalities, based on immunohistochemistry[36] . Depending on the form of

psoriasis and severity of the pathological processes, there is difference in the expression of immunohistochemical markers involved in cell proliferation, vascularization, apoptosis and inflammation. The immunohistochemical markers studied and of significance include CD3, CD68, Ki-67, VEGF, CD34, P63 and S100.

CD34 in psoriasis

CD34 reflects the level of angiogenesis in psoriasis and it has been postulated that it may not only be a cofactor but also an inducer of psoriasis development[37]. Studies show that the expression of CD34 is higher in psoriatic lesions than in normal skin or psoriasiform lesion. The newer immunopathogenetic concepts proposed has led to the emergence of new psoriasis treatment modalities that target the immune cells and molecules which induce and maintain the clinical changes seen in psoriatic plaques. One researched treatment strategy involves the inactivation of secreted effector cytokines like the Tumour necrosis factor α (TNFα),which appears to be a critical cytokine for many of the clinical features of psoriasis, including keratinocyte hyperproliferation, endothelial cell regulation, and recruitment/effector function of memory T cellsSeveral anti-effector drugs and anti-angiogenesis targeted therapy (CD34-early angiogenesis marker) have been suggested and some used successfully to treat psoriasis and Psoriatic arthritis.

LICHEN PLANUS

It is a subacute or chronic dermatosis involving skin, mucous membranes, hair follicles and nails. The prevelance of lichen planus in the total population is probably lower than the estimated 0.5–1.0 %[39]**.** It is characterized by pruritic violaceous papules most commonly on the extremities of middle-aged adults. It has a self-limited course ranging from several months to years, but it may last indefinitely[40]. Oral lesions of lichen planus are frequently found[41.] Nails are involved in about 10% of cases and show roughening and longitudinal ridging[42.] Physical factors such as thermal irritation or UV irradiation can result in an acute exacerbation of Lichen planus[43] . Based on morphology and distribution of the lesions, numerous variants of lichen planus can be distinguished from the classical form.A biopsy for histopathological examination is recommended to confirm the clinical diagnosis and to exclude epithelial atypia and signs of malignancy[44 .]

Clinical variants-

Annular lichen planus

Hypertrophic lichen planus

Atrophic lichen planus

Ulcerative lichen planus

Bullous lichen planus

Lichen planus pemphigoides

Lichen planus pigmentosus

Erythrodermic lichen planus

Inverse lichen planus

Linear lichen planus

Follicular lichen planus

Actinic lichen planus

Pathogenesis

The etiology of lichen planus remains unknown[45]. Recent studies have shown that lichen planus represents a cell-mediated immune response in the epidermis in a genetically predisposed individual to an induced antigenic change[46]. CD8+ infiltrates in the lesional skin recognize a MHC Class I antigen called Lichen planus specific antigen. These antigens are detected by indirect immunofluorescence[47].

Histopathology- the characteristic features of lichen planus include

Compact orthokeratosis

Wedge-shaped hypergranulosis

Irregular acanthosis

Vacuolar alteration of the basal layer

Band-like lymphocytic infiltrate in the upper dermis[48]

In the lower epidermis and in the papillary dermis necrotic keratinocytes are present and they are called as colloid, hyaline or Civatte bodies. (Fig. 5)

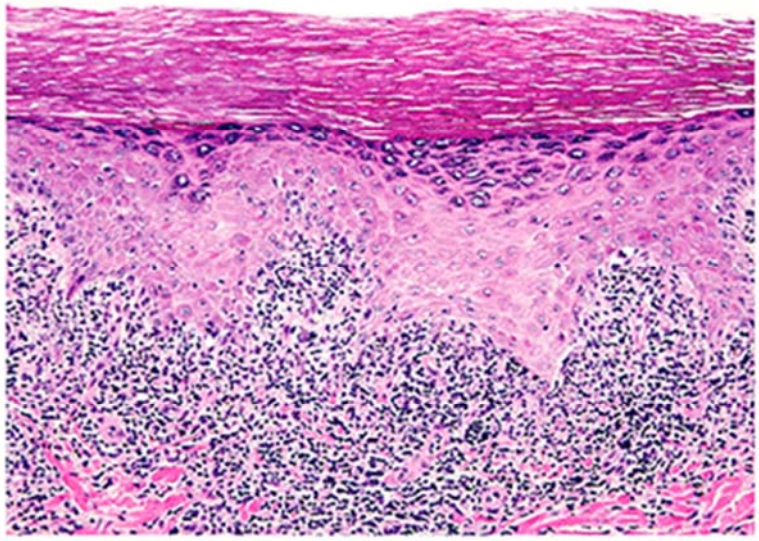


FIG 5: HISTOPATHOLOGY OF LICHEN PLANUS

Lichen nitidus

It is an uncommon inflammatory skin disease, predominantly occuring in children[49] . Lichen nitidus is distinguished from Lichen planus, based on its clinical and histologic pattern. Lichen nitidus may accompany clinical variants of Lichen planus and both conditions may occur together in the same patient[50]. Clinically, it presents as dome- shaped, shiny, asymptomatic papules measuring 2–5 mm in diameter.

Histopathology-

Lichen nitidus is characterised by well-circumscribed,mixed-cell granulomatous infiltrate composed of lymphocytes, histiocytes, occasional

epithelioid and Langhans cells in the upper dermis. There is downward enlargement of the epidermal rete ridges which surrounds the focal inflammatory infiltrate (resembling a claw clutching a ball)[51,52]. There is suprapapillary thinning along with vacuolar alteration of the basal layer and focal parakeratosis. (Fig. 6)

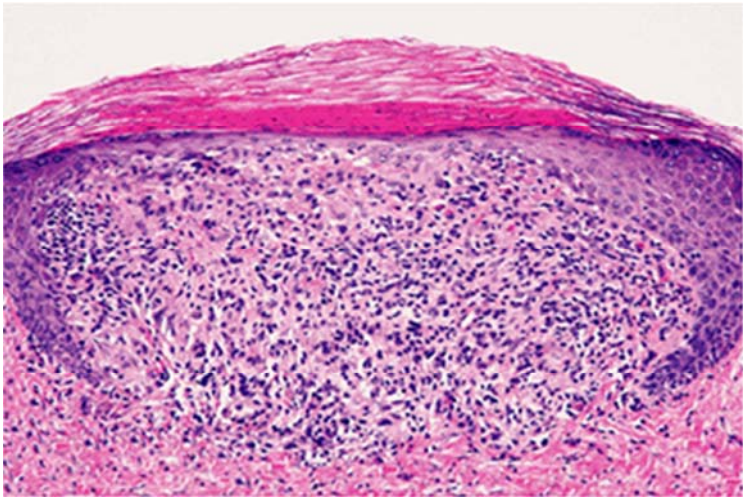


FIG 6: HISTOPATHOLOGY OF LICHEN NITIDUS

LICHEN STRIATUS

It is an uncommon, self-limiting, linear dermatosis. It usually affects children, especially girls between 5-15 years of age[53.] There is an increased incidence of lichen striatus in those with family history of atopy (asthma, allergic rhinitis, atopic dermatitis)[54]. Triggering factors include vaccines, pregnancy, stress, drugs, skin trauma and contact dermatitis[55].

Clinically the lesion begins as small erythematous, raised scaly papules usually seen on the extremity or around the trunk following Blaschko's lines.

Histopathology shows focal parakeratosis, acanthosis and spongiosis. The papillary dermis shows band-like distribution of inflammatory infiltrate, which extends into the lower portion of the epidermis. The characteristic feature in lichen striatus is the presence of inflammatory infiltrate centered around the deep dermal vascular plexus and adnexae, especially perieccrine gland involvement[56]. (Fig. 7)

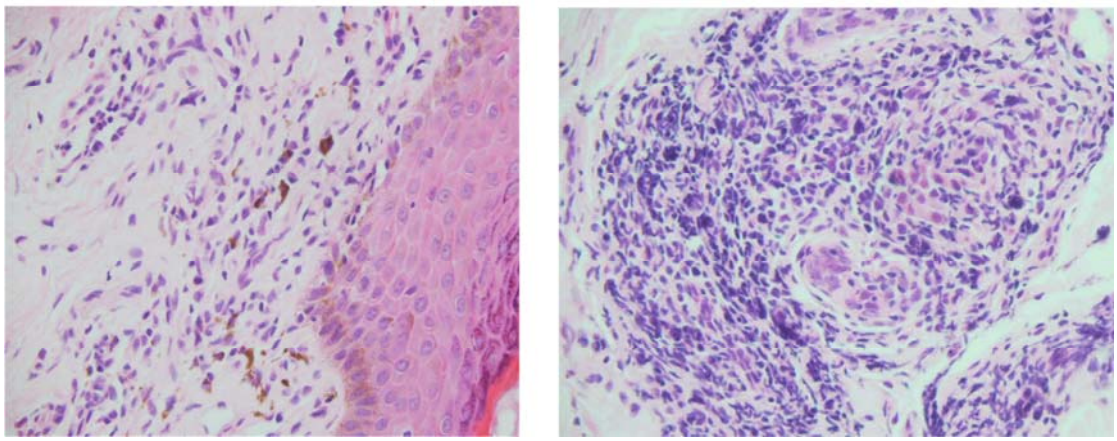


FIG 7 : HISTOPATHOLOGY OF LICHEN STRIATUS[57]

PARAPSORIASIS

It is a group of uncommon dermatoses, characterized by erythematous and scaly patches of variable size which run a chronic course and are resistant to treatment[58]. Parapsoriasis occurs mainly in adults and it is considered to be a cutaneous lymphoproliferative disorder[59] .The current

classification includes three entities: **large plaque parapsoriasis** (LPP), **small plaque parapsoriasis** (SPP) and **Pityriasis lichenoides**[60].

Small plaque parapsoriasis is known also as Xanthoerythroderma perstans of Crocker and digitate dermatoses[61]. It presents as scaly, elongated, finger print-like patches measuring 1 to 5 cm in diameter which are symmetrically distributed over the trunk and the proximal portions of the extremities. It has a chronic course, and tends to persist. It is a benign disorder without the potential of transformation into Mycosis fungoides[62].

Large plaque parapsoriasis occurs in middle-aged and older people, with slight male preponderance and no racial and geographical predilection. According to its clinical presentation,it is classified as Poikilodermatous and retiform pattern[63]. It presents as erythematous, round or irregularly shaped scaly patches measuring more than 5cms. It is regarded as the benign end of the Mycosis fungoides disease spectrum and may progress to definite MF approximately 10% per decade[64] in one studies and upto 35% in other studies[65].The progression to Mycosis fungoides often takes place over many years, hence the need of prolonged and careful follow-up in all cases of LPP.

Histopathology shows elongated mounds of parakeratosis with collections of plasma above a basket-weave cornified layer along with spongiosis, exocytosis of lymphocytes and mild acanthosis[66]. There is a

mild superficial perivascular lymphocytic infiltrate that in the papillary dermis[67]. (Fig. 8)

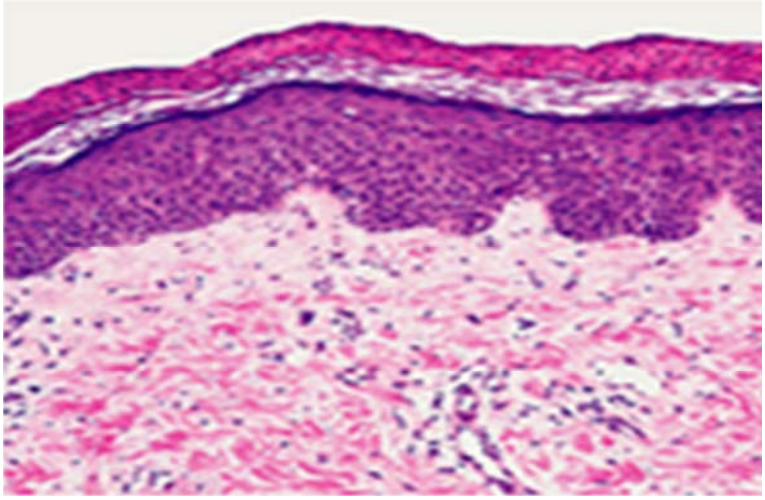


FIG 8: HISTOPATHOLOGY OF PARAPSORIASIS

Pityriasis rosea

It is a self-limiting disease with specific skin rash and rare systemic symptoms[68]. It is of sudden onset and occurs between 10-35 years of age[69]. The etiology is still unknown, although a viral etiology such as human herpesvirus 7 (HHV-7) is suspected[70]. But, subsequent study results showed no consistent increase of human herpesvirus7 levels in affected patients compared with control patients[71,72]. Positive staining with pan T lymphocyte marker CD3 supports the association with cellular immunity[73].

Clinically, the initial lesion present as a herald patch. Herald patch is a 2–10 cm oval or round patch which is erythematous, slightly elevated from the skin, and is located on the trunk[74]. The initial lesion is followed by secondary eruptions, which are oval, pink macular eruptions[73]. There

are several clinical presentations which includes papular, vesicular, urticarial, purpuric, and recurrent forms[75].

Histopathology shows focal parakeratosis, irregular acanthosis, decreased or absent granular layer and spongiosis. Papillary dermis show extravasation of erythrocytes which sometimes extends into the overlying epidermis[76]. The dermis show superficial perivascular infiltrate consists of lymphocytes, occasional eosinophils and histiocytes. These lymphocytes extend into the epidermis called exocytosis[77]. (Fig. 9)

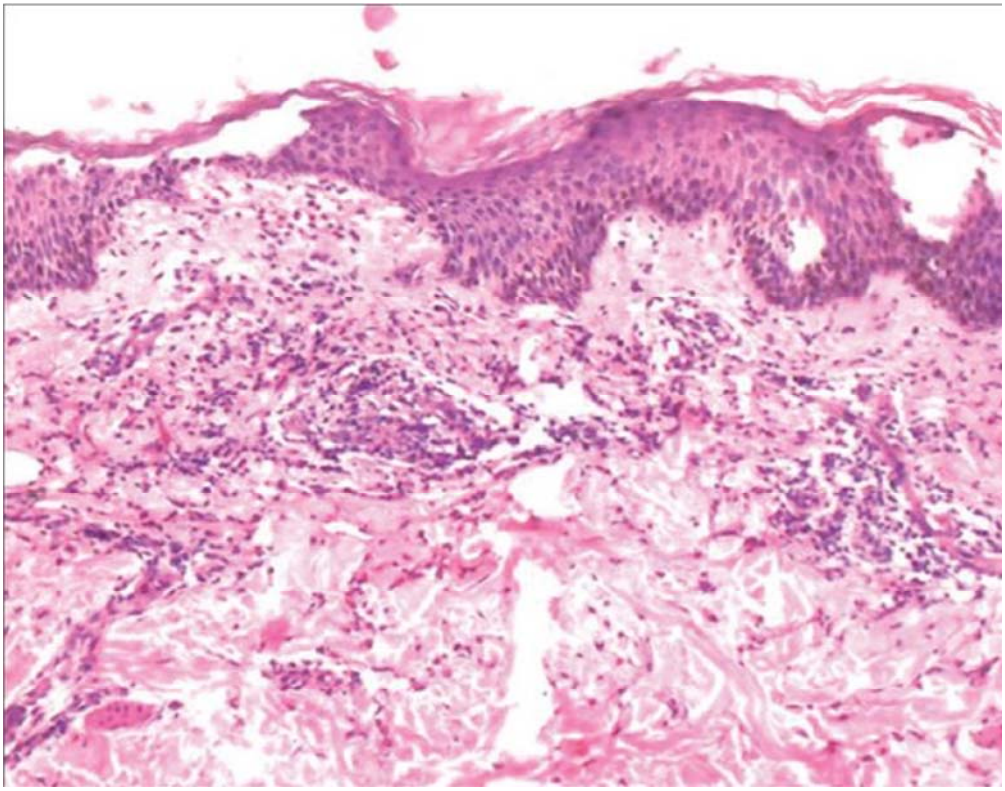


FIG 9: HISTOPATHOLOGY OF PITYRIASIS ROSEA

PITYRIASIS RUBRA PILARIS

It is a rare group of hyperkeratotic, papulosquamous disease that can be acquired or inherited[78]. It occurs equally in men and women and its incidence is about 1 in 50,000 in India[79]. The familial form of Pityriasis rubra pilaris is inherited as an autosomal dominant trait. Pityriasis rubra pilaris is classified into five types on the basis of age of onset, clinical features, and prognosis by Griffiths. They are

Classic adult type I,

Atypical adult type II,

Classic juvenile type III,

Circumscribed juvenile type IV and

Atypical juvenile type V[80].

Clinically it is characterised by the appearance of keratotic follicular papules, well-demarcated salmon-colored erythematous plaques covered with fine powdery scales and palmoplantar keratoderma. These plaques frequently contain islands of normal appearing skin.

Histopathology shows alternating orthokeratosis and parakeratosis in both the vertical and horizontal directions, hypergranulosis, irregular acanthosis seen in the form of short and broad rete-ridges, thick suprapapillary plates and sparse to moderate lymphocytic perivascular infiltrate in the dermis [81]. (Fig. 10)

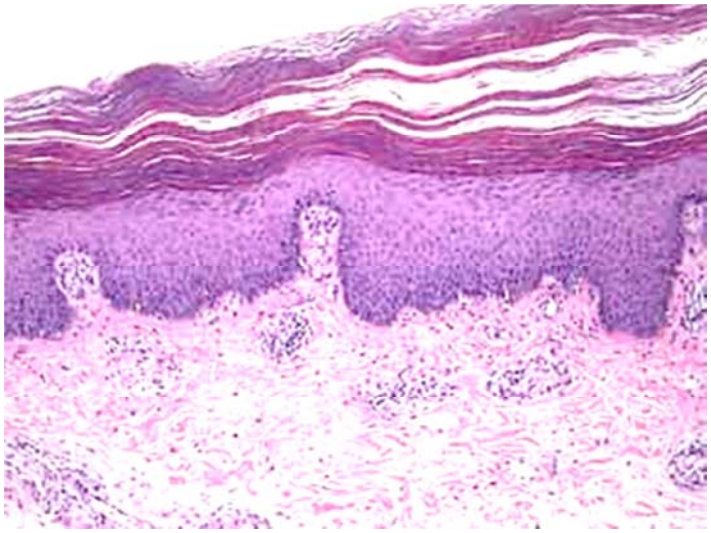


FIG 10: HISTOPATHOLOGY OF PITYRIASIS RUBRA PILARIS [82]

PRURIGO NODULARIS

It is a benign neurodermatitis of unknown etiology. It usually begins in middle age and women are more frequently affected. It presents symmetrically on the extensor surfaces of lower extremities. It represents a dermatological manifestation of repeated traumatic manipulation due to chronic pruritus. It is triggered by many conditions including arthropod bite reactions, psychiatric illness, hyperthyroidism, iron deficiency anemia, chronic liver disease, chronic kidney disease, depression, anxiety, leukemia and lymphoma. Mast cells play an important role in the pathogenesis of prurigo nodularis by increasing the expression of nerve growth factor (NGF). These factors produce neurohyperplasia.

Clinically it is characterised by firm hyperkeratotic, excoriated, pruritic papules or nodules of size 2-3 mm to 2 cm in diameter. It appears well demarcated, scaly and hyperpigmented.

Histopathology shows hyperkeratosis, parakeratosis, acanthosis and papillomatosis. There is elongation of the rete ridges along with a dense dermal lymphohistiocytic infiltrate. Dermis show hypertrophy of cutaneous nerves. (Fig. 11)

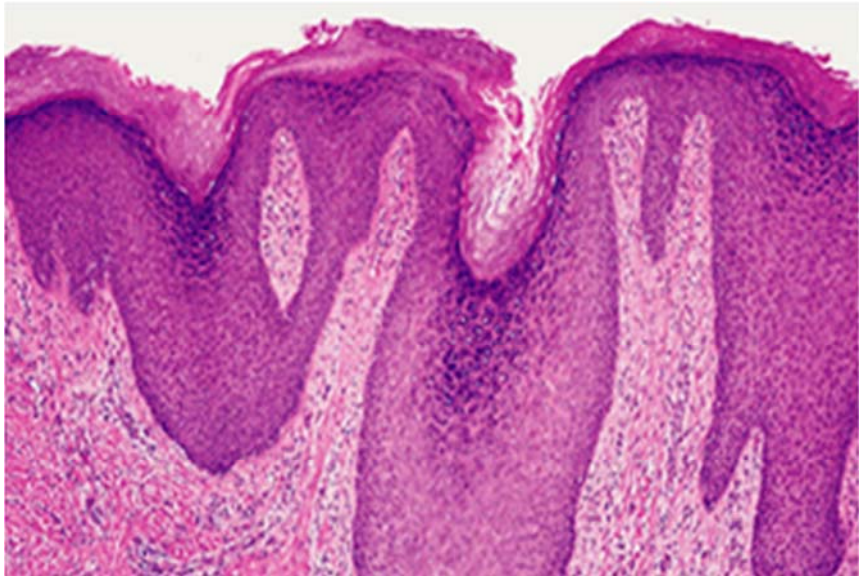


FIG 11: HISTOPATHOLOGY OF PRURIGO NODULARIS

ERYTHEMA ANNULARE CENTRIFUGUM

It is considered as a hypersensitivity reaction to an unknown stimulus that can be an infection, an infestation, carcinoma, blood dyscrasia, drug sensitivity, dysproteinemia, or immunological disturbances.It is one of the gyrate or figurate erythemas[84]. Clinically and

histologically it is subdivided into superficial and deep forms. Superficial variant is characterized by a peripheral trailing scale and has an indistinct border, whereas the deeper type is non-scaly and has a firm, indurated border[85].

Histopathology show focal spongiosis, parakeratosis, epidermal hyperplasia and papillary dermal edema. It is characterized by perivascular lymphocytic infiltrate with lmphocytes in a coat-sleeve appearance. In the deeper variant, there is involvement of the deeper dermal vasculature and dense perivascular infiltrate[86]. (Fig. 12)

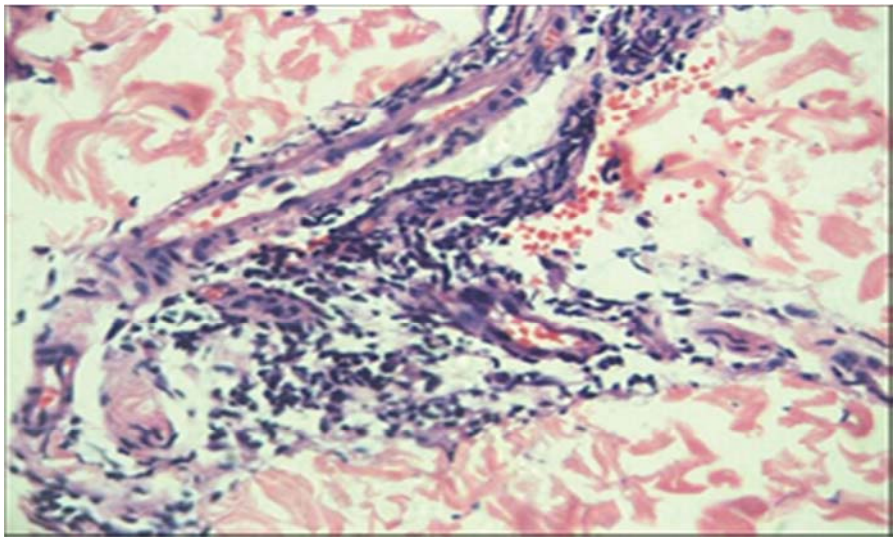


FIG 12: HISTOPATHOLOGY OF ERYTHEMA ANNULARE CENTRIFUGUM

PSORIASIFORM DERMATITIS

Psoriasiform dermatitis must be distinguished from psoriasis,since the treatment differs for both. The diseases falling under this category mimic psoriasis either clinically or histologically.

This category includes,

Seborrheic dermatitis,

Allergic dermatitis and

Lichen simplex chronicus

Seborrheic dermatitis

It is a common chronic inflammatory skin condition, characterized by poorly defined erythematous patches and scaling. It primarily affects sebum-rich areas, such as the scalp, face, upper chest, and back. 11.6% of the general population and up to 70% of infants in the first three months of life are affected by seborrheic dermatitis. The peak incidence is in the third and fourth decades of life. Patients with Parkinson's disease and in patients treated with certain psychotropic drugs such as haloperidol decanoate, lithium and chlorpromazine. It is one of the most common dermatoses seen in individuals infected with HIV.

Clinically it is characterized by the development of pruritic, erythematous patches with easily detachable, greasy large scales**.** It may be confused with psoriasis, atopic and contact dermatitis and erythrasma in adults and tinea capitis in children.

LICHEN SIMPLEX CHRONICUS

It is a chronic skin disease characterized by small, round itchy spots.Due to constant rubbing and scratching,the skin becomes thick and

leathery.It is also called as neurodermitis circumscripta. This pathology commonly involves the nape of neck, ankles, anogenital region and scalp.

Clinically it is characterised by single or multiple, slightly erythematous, scaly, well demarcated, hyperpigmented, lichenified, rough plaque. Histopathology reveals hyperkeratosis, hypergranulosis, acanthosis with thickening of collagen in the dermis.

ALLERGIC DERMATITIS

It is an immunologically complex chronic inflammatory disease characterised by inflammatory hypersensitivity to environmental triggers. Acute AD are associated with CD4 cells which infiltrates the epithelium. In AD many chemokines are upregulated,which helps in recruitment of inflammatory cells to the sites of injury. Histopathology reveals large number of mononuclear cells infiltrating predominantly the dermis with associated spongiosis in the epidermis.

Materials and Methods

MATERIALS AND METHODS

Study design: Hospital-based prospective observational study

Study area: Chennai Medical College Hospital and Research Centre, Trichy

Study period: July 2014 to July 2016 (Two years)

Study population: The skin biopsy of patients presenting with scaly, non-infectious lesions received in the Department of Pathology is microscopically analysed and evaluated.

Inclusion criteria:

Patients clinically diagnosed with non-infectious scaly lesions consenting for biopsy

Exclusion criteria:

Patients clinically diagnosed with infectious scaly lesions

Patients with non-infectious scaly lesions not consenting for biopsy

All non-scaly lesions

Sampling method and Sample size:

All patients attending the Dermatology OPD with H/O scaly skin lesions of any duration within the two year period (July 2014 to July

2016) will be registered for the study after applying the inclusion and exclusion criteria. Sample size will be according to the number of patients attending the Dermatology OPD within the above specified time frame.

Informed consent:

The participants will be recruited for the study after obtaining their written informed consent. The purpose and objectives of the study will be clearly explained in the local language to them while recruiting.

Data collection:

Clinical Data:

After obtaining the informed consent from the patient, the patient is examined by the dermatologist to identify the site, size, colour and distribution of the lesion/lesions**.**

Following the clinical examination and data collection in the department of Dermatology, lesional punch or excisional biopsy is done on the patient clinically diagnosed to have scaly skin lesion of non-infectious etiology.

Biopsy:

It is a technique used for microscopic evaluation to arrive at the diagnosis, from the lesion under study.The biopsy techniques are commonly employed are

Punch biopsy,

Superficial and deep shave biopsy,

Deep incisional biopsy,

Complete excision and

Curettage

Punch biopsy is the standard procedure for obtaining samples of inflammatory dermatoses. specimen obtained with a 4-mm biopsy punch is adequate for histologic study. A punch biopsy specimen can be squeezed gently out of its socket or carefully speared with the syringe needle. Immediately after removal it should be placed in fixative, to prevent autolysis.

The skin specimen biopsied is fixed in 10% formalin and sent to the department of Pathology.

GROSS EXAMINATION:

The skin specimen received should be given a proper gross description which should include tissue size, presence or absence of epidermis, color, presence and absence of hair and alterations to the epidermal surface. The tissue is then thinly sliced,processed and embedded in paraffin blocks, after which sections are cut and affixed on glass slides.The tissue sections are then subjected to hematoxylin and eosin staining,followed by mounting and proper labeling of the slides.The slides

are then subjected to meticulous microscopic examination by the reporting pathologist.

Tissue staining procedure on skin biopsies ::

Routinely, Haematoxylin and Eosin staining is done on the processed skin biopsy sections.

The procedure is as follows:,

Chemical composition:

Erhlich's hematoxylin

Eosin 50

HEMATOXYLIN AND EOSIN STAIN – PROCEDURE

1. Deparaffinise the tissue sections in xylene for about 5 – 10 min
2. Subject the tissue section to water through reducing grades of alcohol (100% to 50%)
3. Keep it in hematoxylin for 15 to 20 minutes
4. Rinse it in tap water
5. Differentiate with 1% acid alcohol
6. For bluing - place in tap water for about 10 minutes
7. Counter stain by eosin 1-2 minutes
8. Rinse in water
9. Dehydration followed by clearing and mount it

All cases reported histopathologically as psoriasis and psoriasiform dermatitis were subjected to CD34 immunohistochemical marker staining, to study the presence of dermal vascular changes.The immunohistochemical staining on the lesional cases was done along with proven positive and negative control.

IMMUNOHISTOCHEMICAL (IHC) STAINING – PROCEDURE

1. Keep the slides in xylene I for 5 minutes.
2. Keep it in xylene II for 5 minutes.
3. Keep in alcohol I for 5 minutes.
4. Keep in alcohol II for 5 minutes.
5. Add 3% H_2O_2 for 30 minutes.
6. Place in running tap water for about 5 minutes.
7. Place in distilled water for 5 minutes.
8. Transfer the slides to citrate buffer for 10 minutes (pH-6.4).
9. Antigen retrieval by microwave processing for about 30 minutes.
10. Wash in tris buffer for 5 minutes – two times at pH 7.4.
11. Overnight incubation after adding primary antibody
12. Add the primary antibody and incubate.
13. Wash in tris buffer for 5 minutes – 2 times.
14. Treat with skimmed milk for 3 minutes.
15. Add the secondary antibody for 40 minutes.

16. Rinse in tris buffer -5 minutes (two times).
17. Add chromogen - 5 minutes.
18. Rinse it in distilled water.
19. Counterstaining done with Haematoxylin - one minute
20. Dehydrate, clean and mount

IMMUNOHISTOCHEMICAL STUDY

CD34 is a 110-kDa transmembrane glycoprotein expressed predominantly in endothelial cells and stem cells, it is also present on cells of the splenic marginal zone, dendritic interstitial cells around vessels, nerves, hair follicles, muscle bundles, and sweat glands in a variety of tissues and organs[38]. CD34 has been observed to act as a molecular "Teflon" which blocks mast cell, eosinophil and dendritic cell precursor adhesion, and facilitates the opening of vascular lumens.

CD 34 immunomarker, stains the endothelium of blood vessels. The CD34 staining intensity is evaluated by performing capillary counting in the 3 most highly vascularized areas selected under 40X field. Single or clusters of endothelial cells, with or without lumen are considered to be individual vessels. They are scored as mild, moderate and severe depending upon the number of vessels stained. The scoring pattern is tabulated as follows[87]

TABLE 1:

CD 34 SCORING

SCORE	INTENSITY
4-10	WEAK
11-20	MODERATE
21-28	STRONG

The interpretation of CD34 staining is by viewing the three highly vascularised areas in 400X field and counting the number of vessels. If the number of vessels stained are between 4-10,then it is categorized as weak, if it is between 11-20 then it is moderate, if it is between 21-28 it is categorized as strong.

Results

RESULTS

In the present study, a total of 51 biopsies taken from the study group of patients was studied in the Department of Pathology, Chennai Medical College Hospital and Research Centre, Trichy between July 2014 to July2016.

The objectives of the study include the age and sex distribution of patients clinically presenting with scaly skin lesions of non-infectious etiology which are tabulated in the Table 1 and Table 2 respectively.

TABLE 2:

AGE DISTRIBUTION OF PATIENTS STUDIED

Age in years	No. of patients	%
<10	1	2.0
10-20	9	17.6
21-30	13	25.5
31-40	5	9.8
41-50	11	21.6
51-60	5	9.8
>60	7	13.7
Total	51	100.0

Mean ± SD: 37.63±18.25

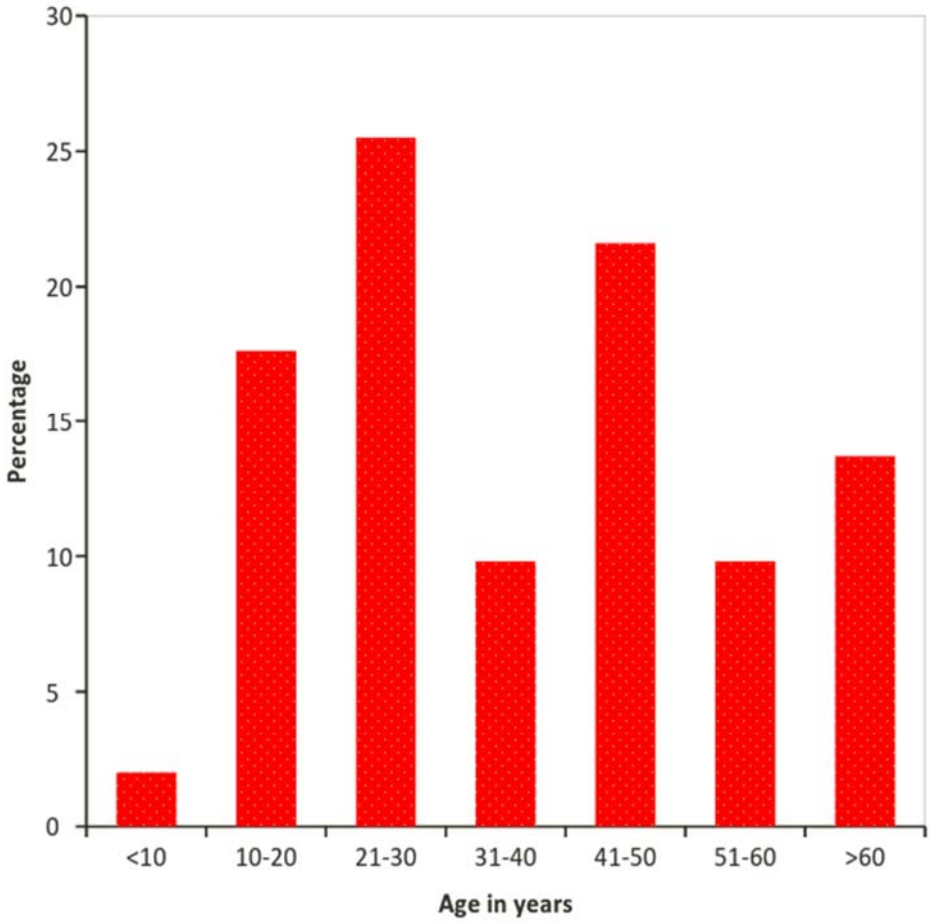


FIG 1: AGE DISTRIBUTION OF PATIENTS STUDIED

In the present study, maximum numbers of cases were found to be in second decade i.e., (21-40) years comprising of 25.5% of the study population. Minimum number of cases is found to in the age group of less than 10 years

TABLE 3:

GENDER DISTRIBUTION OF PATIENTS STUDIED

Gender	No. of patients	%
Female	25	49.0
Male	26	51.0
Total	51	100.0

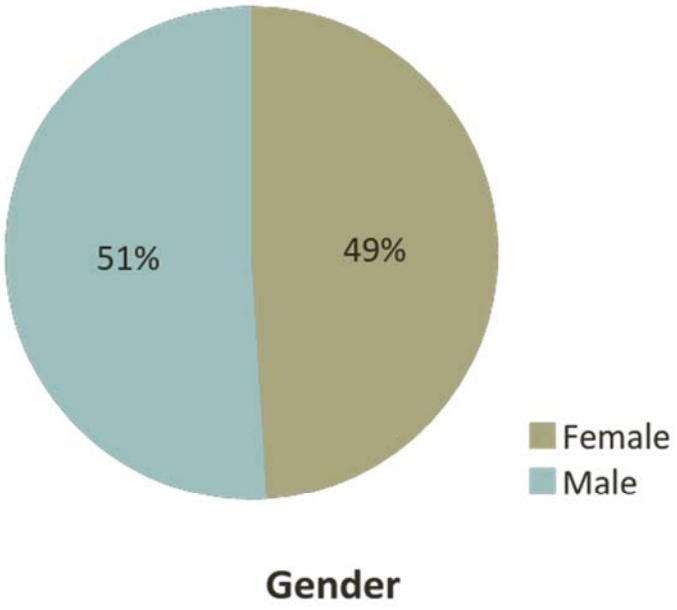


FIG: 2 SEX DISTRIBUTION OF SCALY LESIONS OF NON INFECTIOUS ETIOLOGY

The incidence of scaly skin lesions of noninfectious etiology in the present study shows 51% of the affected individuals are males and 49% are females

TABLE 4:

AGE AND SEX DISTRIBUTION OF SCALY SKIN DISEASE IN THE STUDY POPULATION

Age in years	Gender		Total
	Female	Male	
<10	1 (4%)	0 (0%)	1 (2%)
10-20	4 (16%)	5 (19.2%)	9 (17.6%)
21-30	7 (28%)	6 (23.1%)	13 (25.5%)
31-40	3 (12%)	2 (7.7%)	5 (9.8%)
41-50	6 (24%)	5 (19.2%)	11 (21.6%)
51-60	0 (0%)	5 (19.2%)	5 (9.8%)
>60	4 (16%)	3 (11.5%)	7 (13.7%)
Total	25 (100%)	26 (100%)	51 (100%)

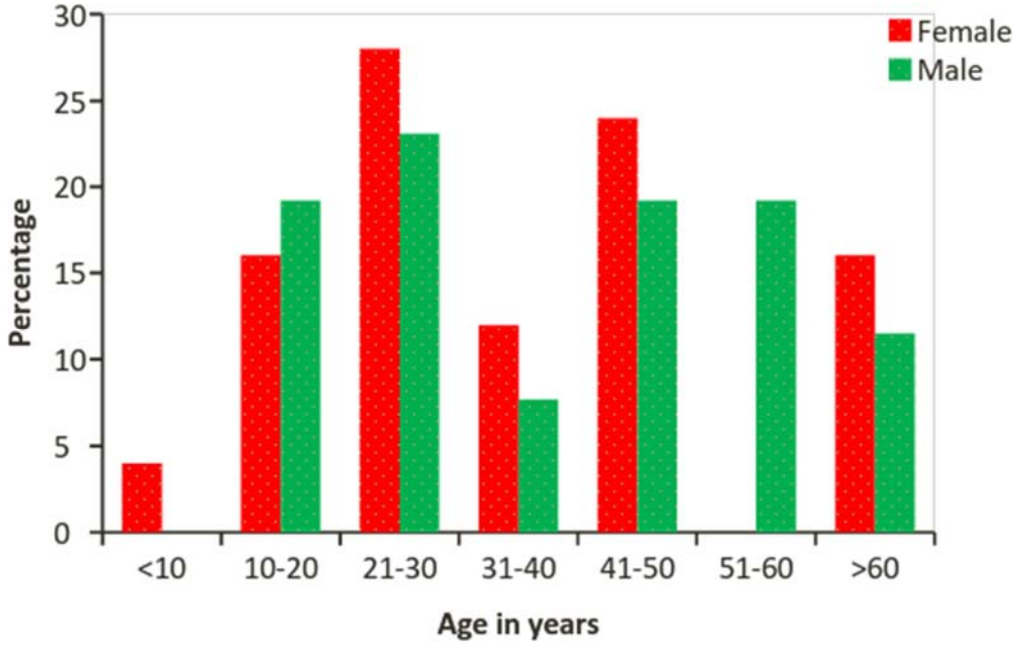


FIG 3: AGE AND SEX DISTRIBUTION OF SCALY SKIN LESION

In the present study,out of the 51 patients maximum number of cases (28%) are seen in females of the second decade (21-30years) and minimum number of cases (4%) are seen in young females of less than 10 years of age.

TABLE 5:

PRESENTING SYMPTOMS OF PATIENTS STUDIED

Complaints	Gender		Total
	Female	Male	
Scaly lesions	1 (4%)	0 (0%)	1 (2%)
Hyper pigmented lesions	14 (56%)	11 (42.3%)	25 (49%)
Hypopigmented lesions	10 (40%)	15 (57.7%)	25 (49%)
Total	25 (100%)	26 (100%)	51 (100%)

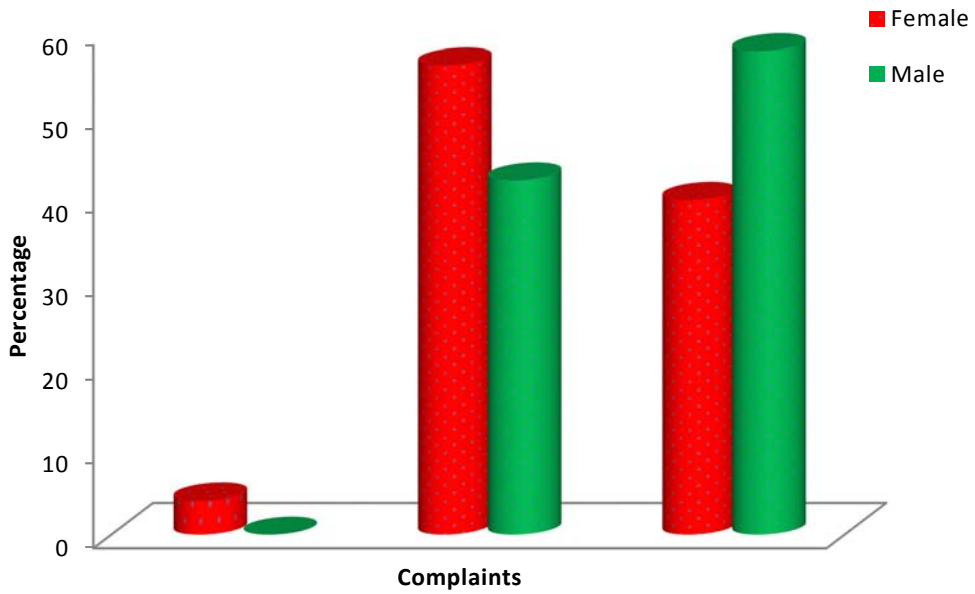


FIG 4:PRESENTING SYMPTOMS OF PATIENTS STUDIED

Out of 51 patients studied, 25 (49%) patients presented with hypopigmented lesions,25 (49%) patients presented with hyperpigmented

lesions and 1 (2%) patient presented with scaly lesion over normal appearing skin.Fifteen patients,who presented with hypopigmented lesions wee males (57.7%) and the rest (40%) were females. Out of the twenty five patients who presented with hyperpigmented lesions, 14 (56%) patients, were females and 11 (42.7 %) patients males. One female patient presented with a scaly lesion without pigment alteration.

TABLE 6:

THE SPECTRUM OF CLINICAL DIAGNOSIS WITH GENDER CO-RELATION IN THE STUDY POPULATION

Clinical Diagnosis	Gender		Total
	Female	Male	
Psoriasis	8 (32%)	15 (57.7%)	23 (45.1%)
Parapsoriasis	3 (12%)	1 (3.8%)	4 (7.8%)
Lichen planus	10 (40%)	6 (23.1%)	16 (31.4%)
Hypertrophic lichen planus	1 (4%)	1 (3.8%)	2 (3.9%)
Lichen nitidus	1 (4%)	1 (3.8%)	2 (3.9%)
Pustular psoriasis	1 (4%)	0 (0%)	1 (2%)
Erythrodermicpsoriasis	1 (4%)	0 (0%)	1 (2%)
Pityriasis rosea	0 (0%)	1 (3.8%)	1 (2%)
Pityriasis rotunda	0 (0%)	1 (3.8%)	1 (2%)
Total	25 (100%)	26 (100%)	51 (100%)

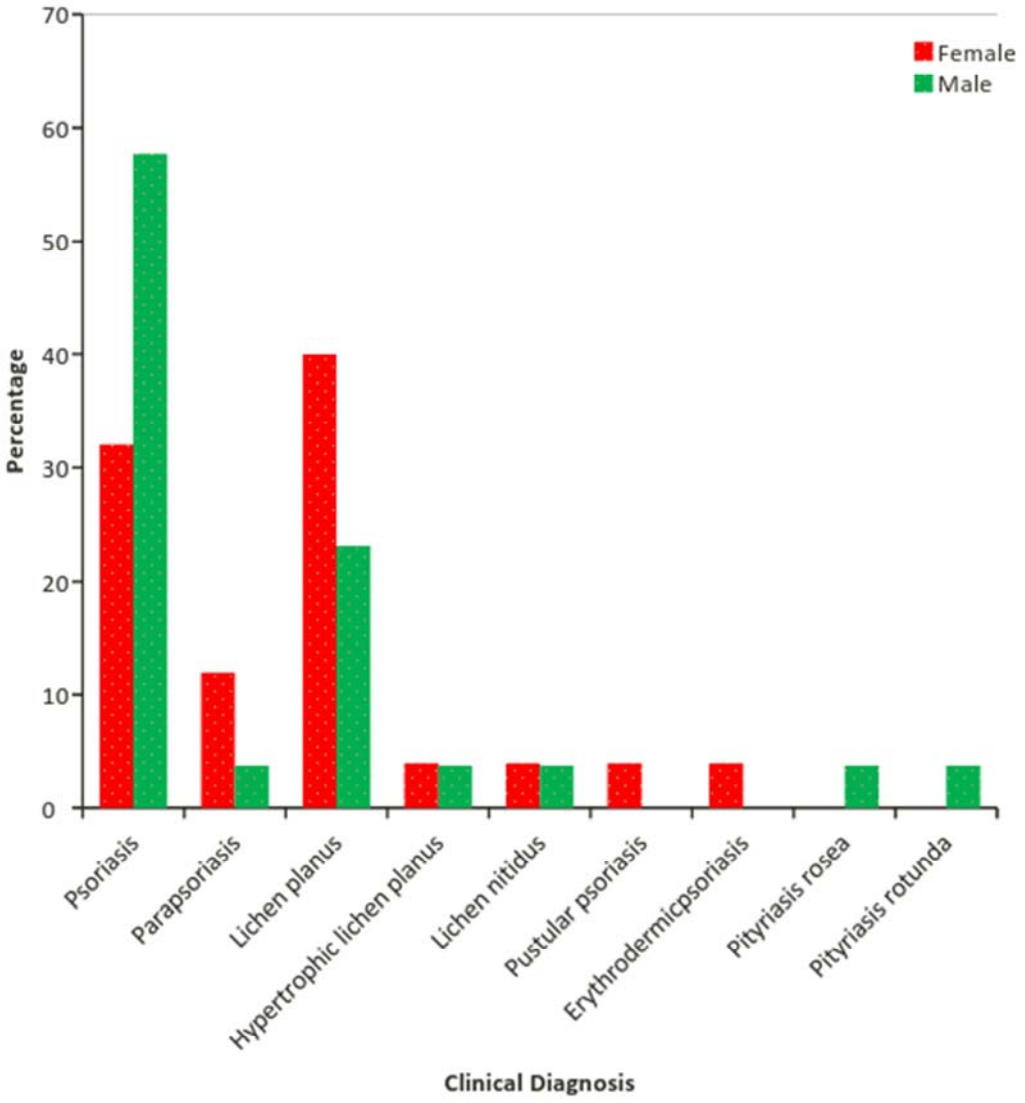


FIG 5: THE SPECTRUM OF CLINICAL DIAGNOSIS WITH GENDER CO-RELATION IN THE STUDY POPULATION

Out of 51 patients studied, 23 patients were clinically diagnosed as Psoriasis,16 patients as Lichen planus, 4 as Parapsoriasis, 2 cases as Hypertrophic lichen planus and lichen nitidus and 1 case each as Pustular psoriasis, erythrodermic psoriasis, pityriasis rosea and pityriasis rotunda.

TABLE 7:

AGE DISTRIBUTION OF PATIENTS STUDIED IN RELATION TO CLINICAL DIAGNOSIS

Age in years	Clinical Diagnosis									Total
	Psoriasis	Parapsoriasis	Lichen planus	Hypertrophic lichen planus	Lichen nitidus	Pustular psoriasis	Erythrodermicpsoriasis	Pityriasis rosea	Pityriasis rotunda	
<10	0 (0%)	1 (25%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (2%)
10-20	1 (4.3%)	0 (0%)	5 (31.3%)	1 (50%)	0 (0%)	0 (0%)	0 (0%)	1 (100%)	1 (100%)	9 (17.6%)
21-30	5 (21.7%)	1 (25%)	6 (37.5%)	0 (0%)	1 (50%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	13 (25.5%)
31-40	2 (8.7%)	0 (0%)	1 (6.3%)	0 (0%)	1 (50%)	0 (0%)	1 (100%)	0 (0%)	0 (0%)	5 (9.8%)
41-50	7 (30.4%)	1 (25%)	1 (6.3%)	1 (50%)	0 (0%)	1 (100%)	0 (0%)	0 (0%)	0 (0%)	11 (21.6%)
51-60	4 (17.4%)	1 (25%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	5 (9.8%)
>60	4 (17.4%)	0 (0%)	3 (18.8%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	7 (13.7%)
Total	23 (100%)	4 (100%)	16 (100%)	2 (100%)	2 (100%)	1 (100%)	1 (100%)	1 (100%)	1 (100%)	51 (100%)
Mean ± SD	46.13 ± 15.90	29.50 ± 21.14	31.94 ± 18.66	28.00 ± 24.04	31.50 ± 12.02	45.00 ± 0.00	35.00 ± 0.00	18.00 ± 0.00	12.00 ± 0.00	37.63 ± 18.25

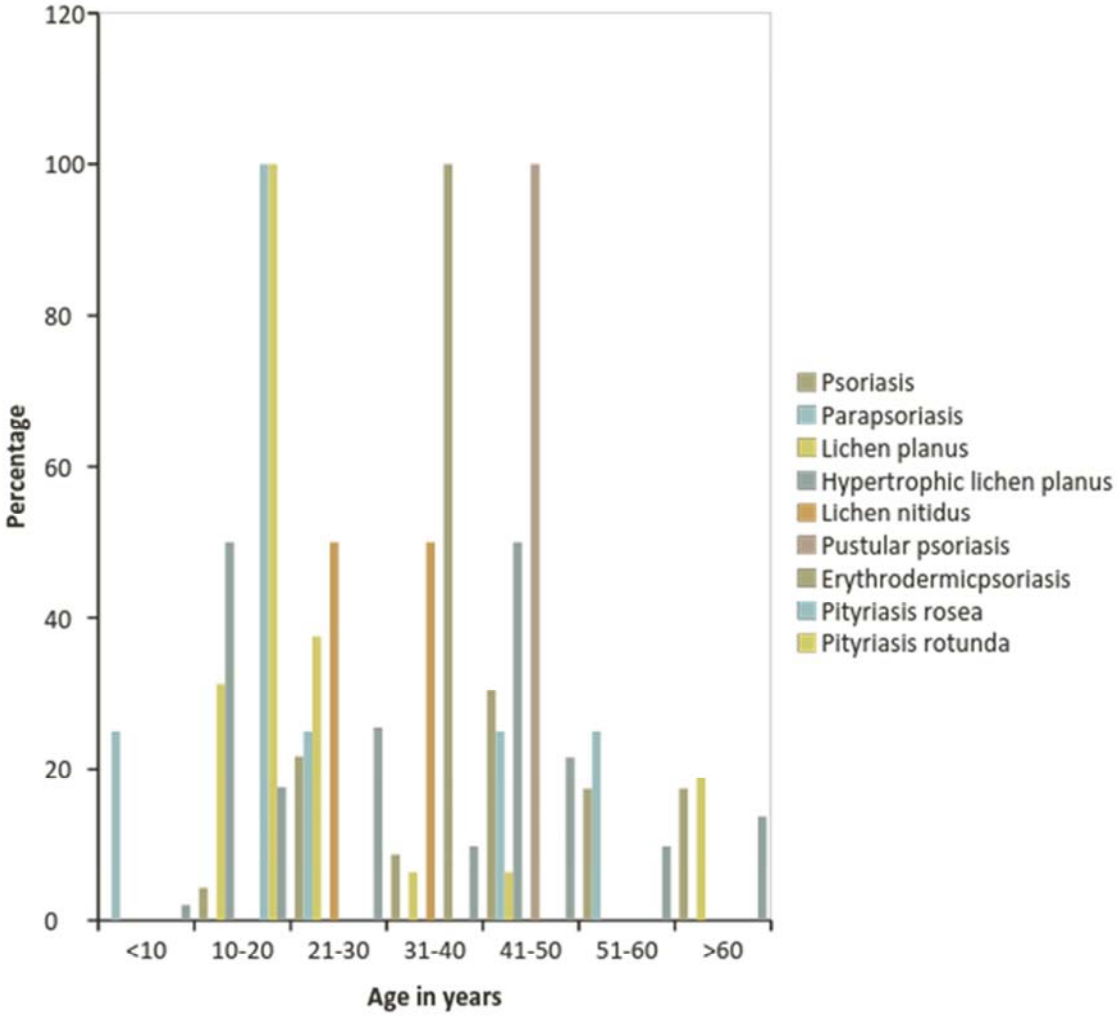


FIG 6: AGE DISTRIBUTION OF PATIENTS STUDIED IN RELATION TO CLINICAL DIAGNOSIS

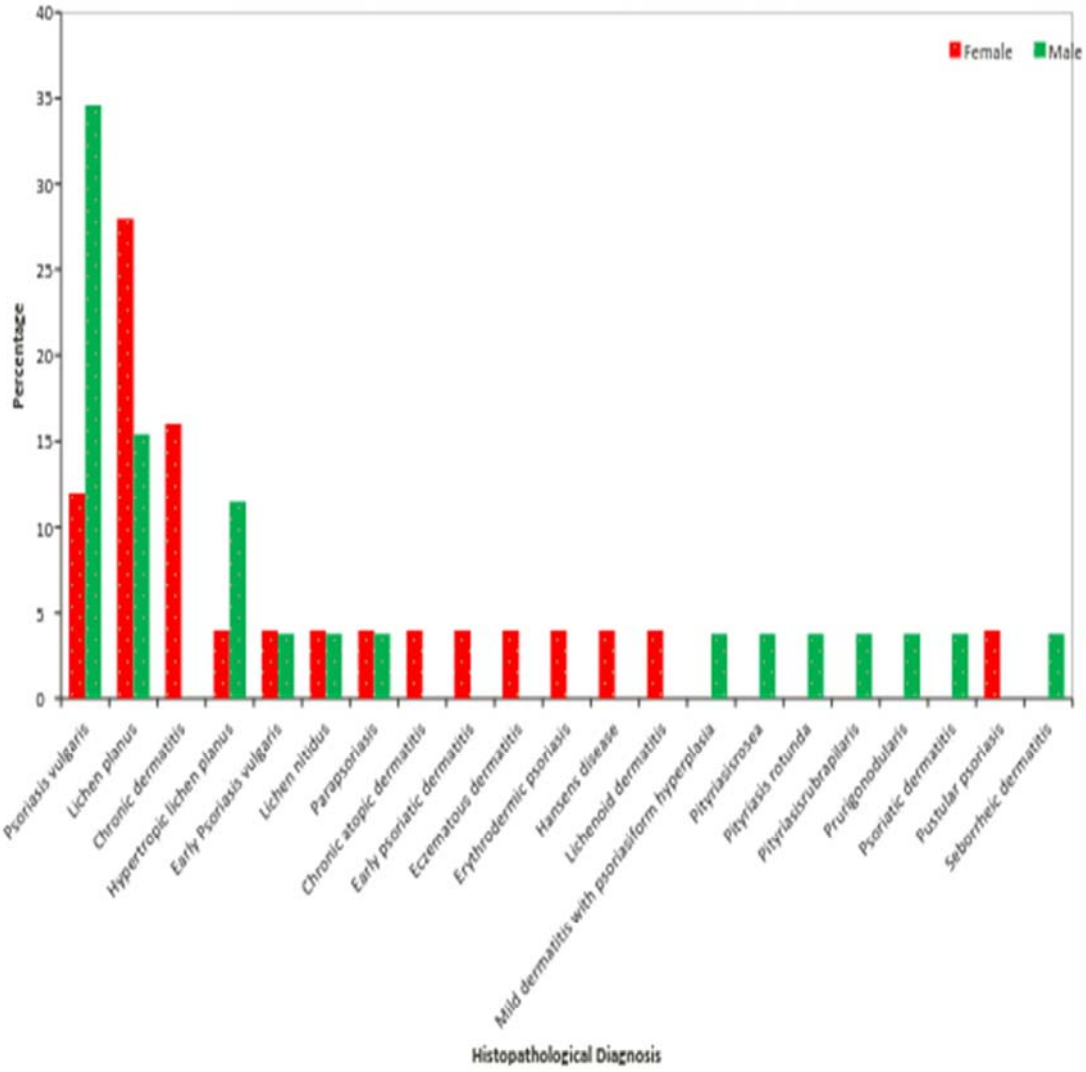
In this study Out of 51 patients, maximum number of psoriasis cases (30.4%) which was diagnosed clinically is seen in the fourth decade (40-50 years). Clinically diagnosed lichen planus are commonly seen in the age group of 20-30 years (37.5%)

TABLE 8

HISTOPATHOLOGICAL DIAGNOSIS WITH GENDER CO-RELATION OF THE PATIENTS STUDIED.

Histopathology diagnosis	Gender		Total
	Female	Male	
Psoriasis vulgaris	3 (12%)	9 (34.6%)	12 (23.5%)
Lichen planus	7 (28%)	4 (15.4%)	11 (21.6%)
Chronic dermatitis	4 (16%)	0 (0%)	4 (7.8%)
Hypertropic lichen planus	1 (4%)	3 (11.5%)	4 (7.8%)
Early Psoriasis vulgaris	1 (4%)	1 (3.8%)	2 (3.9%)
Lichen nitidus	1 (4%)	1 (3.8%)	2 (3.9%)
Parapsoriasis	1 (4%)	1 (3.8%)	2 (3.9%)
Chronic atopic dermatitis	1 (4%)	0 (0%)	1 (2%)
Early psoriatic dermatitis	1 (4%)	0 (0%)	1 (2%)
Eczematous dermatitis	1 (4%)	0 (0%)	1 (2%)
Erythrodermic psoriasis	1 (4%)	0 (0%)	1 (2%)
Hansens disease	1 (4%)	0 (0%)	1 (2%)
Lichenoid dermatitis	1 (4%)	0 (0%)	1 (2%)
Mild dermatitis with psoriasiform hyperplasia	0 (0%)	1 (3.8%)	1 (2%)
Pityriasis rosea	0 (0%)	1 (3.8%)	1 (2%)
Pityriasis rotunda	0 (0%)	1 (3.8%)	1 (2%)
Pityriasis rubra pilaris	0 (0%)	1 (3.8%)	1 (2%)
Prurigo nodularis	0 (0%)	1 (3.8%)	1 (2%)
Psoriatic dermatitis	0 (0%)	1 (3.8%)	1 (2%)
Pustular psoriasis	1 (4%)	0 (0%)	1 (2%)
Seborrheic dermatitis	0 (0%)	1 (3.8%)	1 (2%)
Total	25 (100%)	26 (100%)	51 (100%)

FIG 7: HISTOPATHOLOGICAL DIAGNOSIS WITH GENDER CO-RELATION OF THE PATIENTS STUDIED.



Of the 51 biopsies taken from the patients studied, 14 (27.9%) patients are diagnosed with psoriasis vulgaris including early psoriasis vulgaris and 11 (21.6%) patients with lichen planus, 4 (7.8%) of patients with chronic dermatitis and hypertrophic lichen planus, 2 (3.9%) patients with parapsoriasis and lichen nitidus. About 2% of patients are diagnosed

with chronic atopic dermatitis, early psoriatic dermatitis, eczematous dermatitis,erythrodermic psoriasis, hansens disease, lichenoid dermatitis, mild dermatitis with psoriasiform hyperplasia, pityriasis rosea, pityriasis rubra pilaris, pityriasis rotunda, prurigo nodularis, psoriatic dermatitis, pustular psoriasis and seborrheic dermatitis.

From the overall 51 biopsies,those reported as psoriasis or the related psoriasiform dermatitis were subjected to immunohistochemical marker study using CD 34 antibody.The immunostained sections were analysed and the results is as follows:

TABLE 9:

CD34 DISTRIBUTION IN CO-RELATION TO THE GENDER OF THE PATIENTS

CD34	Gender		Total
	Female	Male	
Weak	3 (33.3%)	3 (23.1%)	6 (27.3%)
Moderate	4 (44.4%)	0 (0%)	4 (18.2%)
Strong	2 (22.2%)	10 (76.9%)	12 (54.5%)
Total	9 (100%)	13 (100%)	22 (100%)

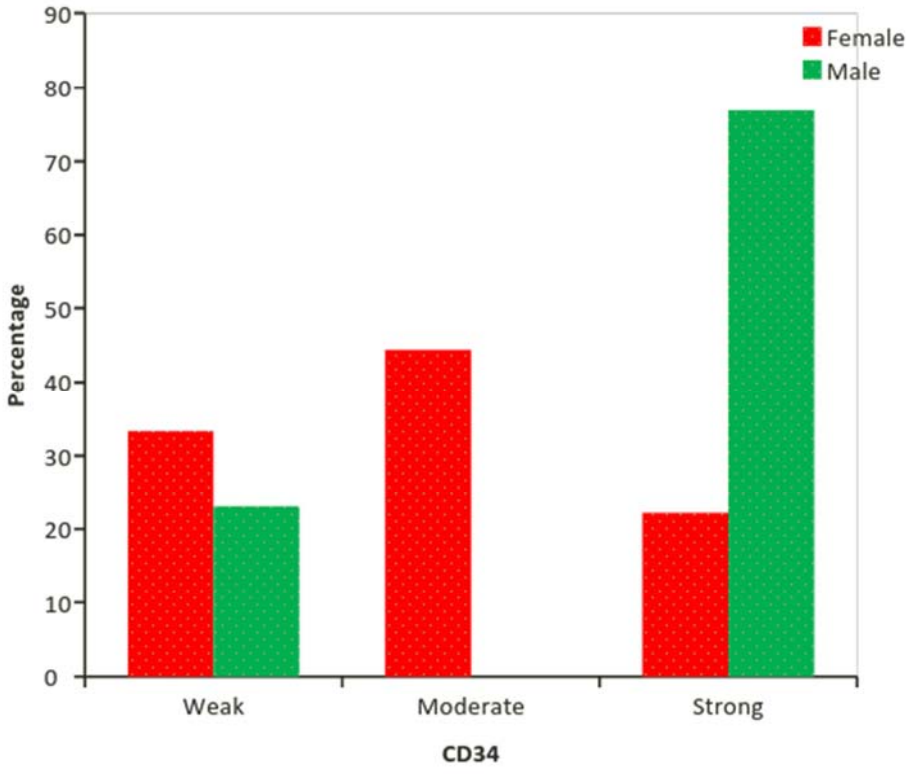


FIG 8: CD34 DISTRIBUTION IN CO-RELATION TO THE GENDER OF THE PATIENTS

Out of 51 patients,biopsy material from 22 cases are subjected to the immunohistochemical marker CD34. Of these 22 cases, 16 patients are diagnosed histopathologically as psoriasis vulgaris, early psoriasis,erythrodermic psoriasis and pustular psoriasis and 6 patients are diagnosed with psoriasiform dermatitis which includes dermatitis with psoriasiform hyperplasia, psoriatic dermatitis and seborrheic dermatitis. Of the 22 cases, 12 cases histopathologically reported as psoriasis show strong positivity for CD34,while 4 other cases of psoriasis including erythrodermic and pustular psoriasis show moderate positivity for CD34. All 6 cases of psoriasiform dermatitis show weak positivity. Ten male patients (76.9%) diagnosed with psoriasis show strong positivity of CD34.

TABLE10:

COMPARISON OF PSORIASIS AND PSORIASIFORM DERMATITIS USING CD34 MARKER

CD34 MARKER	PSORIASIS	PSORIASIFORM DERMATITIS	TOTAL
WEAK	0	6 (27.3%)	6 (27.3%)
MODERATE	4	0	4 (18.2%)
SEVERE	12	0	12 (54.5%)
TOTAL			51 (100%)

TABLE 11:
HISTOPATHOLOGICAL DIAGNOSIS ACCORDING TO CLINICAL DIAGNOSIS

Histopathology	Clinical Diagnosis									Total
	Psoriasis	Parapsoriasis	Lichen planus	Hypertrophic lichen planus	Lichen nitidus	Pustular psoriasis	Erythrodermi cpsoriasis	Pityriasis rosea	Pityriasis rotunda	
Lichen planus, lichen nitidus	1 (4.3%)	0 (0%)	11 (68.8%)	0 (0%)	2 (100%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	14 (27.5%)
Psoriasis vulgaris	13 (56.5%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	13 (25.5%)
Chronic atopic dermatitis	2 (8.7%)	1 (25%)	2 (12.5%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	5 (9.8%)
Hypertropic lichen planus	0 (0%)	0 (0%)	2 (12.5%)	2 (100%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	4 (7.8%)
Early psoriasis vulgaris	3 (13%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	3 (5.9%)
Pityriasisrubrapilaris,pityriasis rotunda	1 (4.3%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (100%)	1 (100%)	3 (5.9%)

Parapsoriasis	0 (0%)	2 (50%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	2 (3.9%)
Eczematous dermatitis	1 (4.3%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (2%)
Erythrodermic psoriasis	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (100%)	0 (0%)	0 (0%)	1 (2%)
Hansens disease	0 (0%)	1 (25%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (2%)
Mild dermatitis with psoriasiform hyperplasia	1 (4.3%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (2%)
Prurigonodularis	0 (0%)	0 (0%)	1 (6.3%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (2%)
Pustular psoriasis	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (100%)	0 (0%)	0 (0%)	0 (0%)	1 (2%)
Seborrheic dermatitis	1 (4.3%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (2%)
Total	23 (100%)	4 (100%)	16 (100%)	2 (100%)	2 (100%)	1 (100%)	1 (100%)	1 (100%)	1 (100%)	51 (100%)

Chi-Square test/Fisher Exact test

In the present study, out of 51 patients,23 cases are clinically diagnosed as psoriasis vulgaris. Out of these 23 cases, 13 (56.5%) cases are diagnosed histopathologically as psoriasis vulgaris, 3 (13%)cases as early psoriasis vulgaris, 2 (8.7%) cases as chronic dermatitis and one case (4.3%) each as that of seborrheic dermatitis, mild dermatitis with psoriasiform hyperplasia, pityriasis rubra pilaris and lichen planus.Of the sixteen clinically suspected cases of lichen planus,11 cases (68.8%) are well correlated histopathologically while 2 (12.5%)cases were reported as chronic atopic dermatitis and hypertrophic lichen planus and 1 case (6.3%) as prurigo nodularis histopathologically.Four cases (100%) which was diagnosed clinically as lichen nitidus, pityriasis rosea and pityriasis rotunda correlated accurately with the histopathological diagnosis. Out of the 4 cases clinically reported as parapsoriasis two cases turned out to be same histopathologically while one case was reported as chronic atopic dermatitis and the other as Hansen's disease.

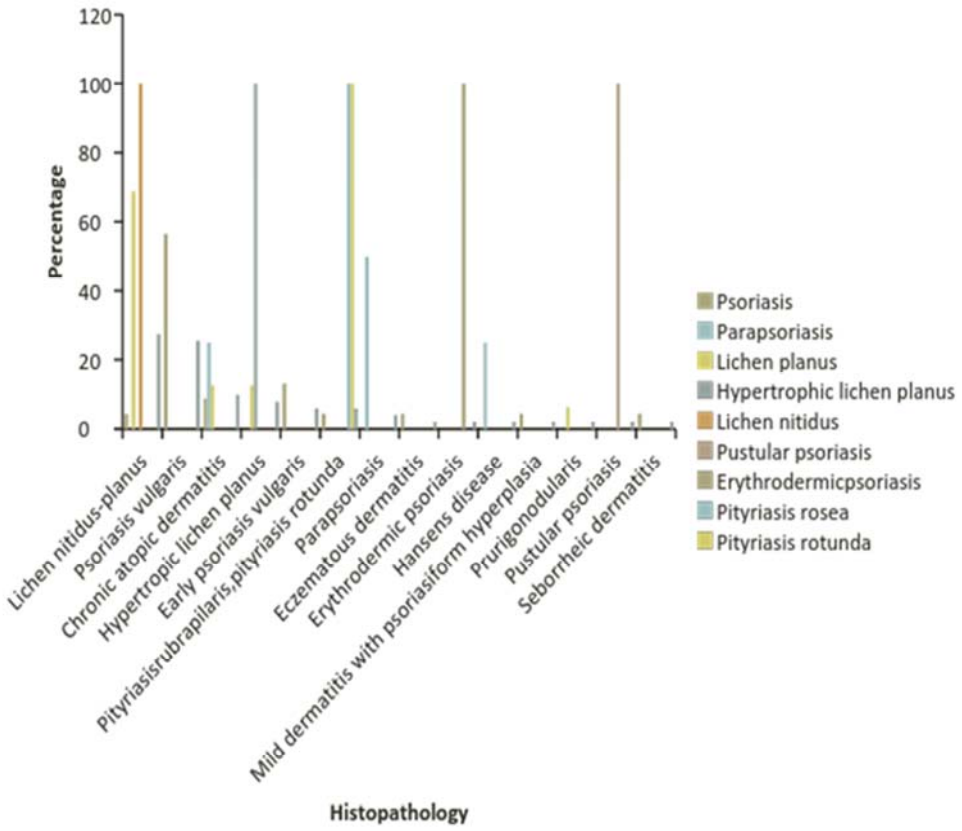


FIG 9: : HISTOPATHOLOGICAL DIAGNOSIS ACCORDING TO CLINICAL DIAGNOSIS

TABLE: 12 HISTOPATHOLOGICAL CORRELATION WITH CLINICAL CORRELATION

HISTOPATHOLOGICAL DIAGNOSIS	CLINICAL DIAGNOSIS
CORRELATED	36 (70.6%)
NOT CORRELATED	15 (29.4%)
TOTAL	51 (100%)

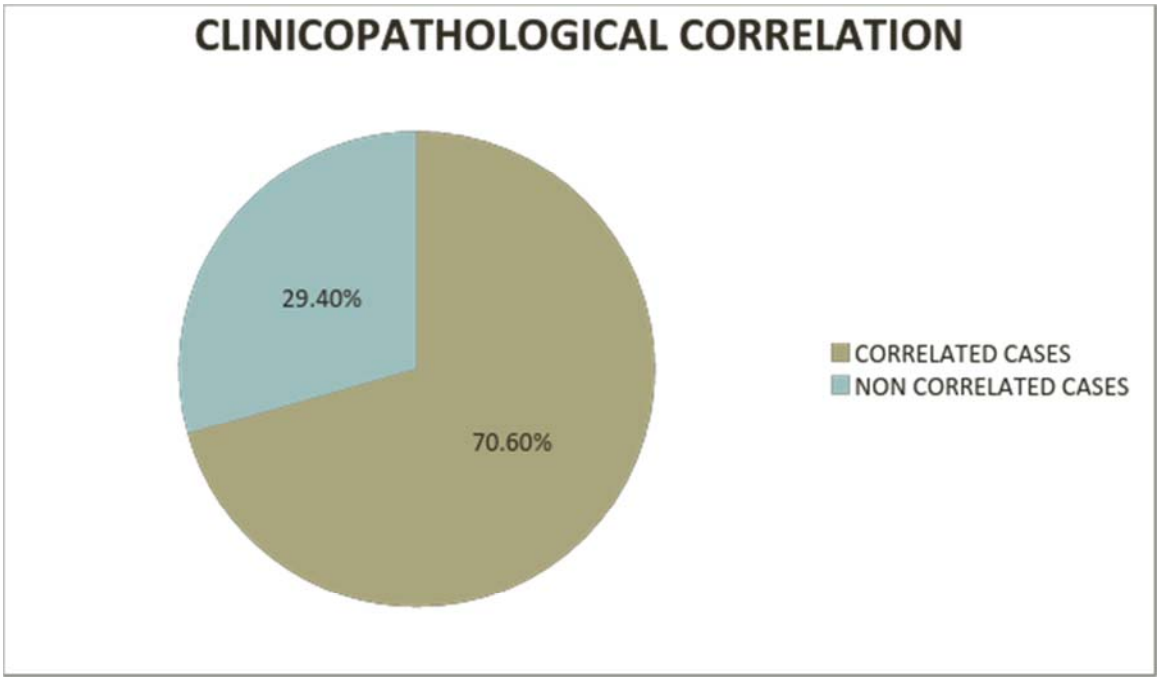


FIG 10: CLINICOPATHOLOGICAL CORRELATION

Out of 51 patients, histopathological diagnosis of 36 patients (70.60%) correlates with the clinical diagnosis. Histopathological diagnosis of 15 patients (29.40%) does not correlate with the clinical diagnosis

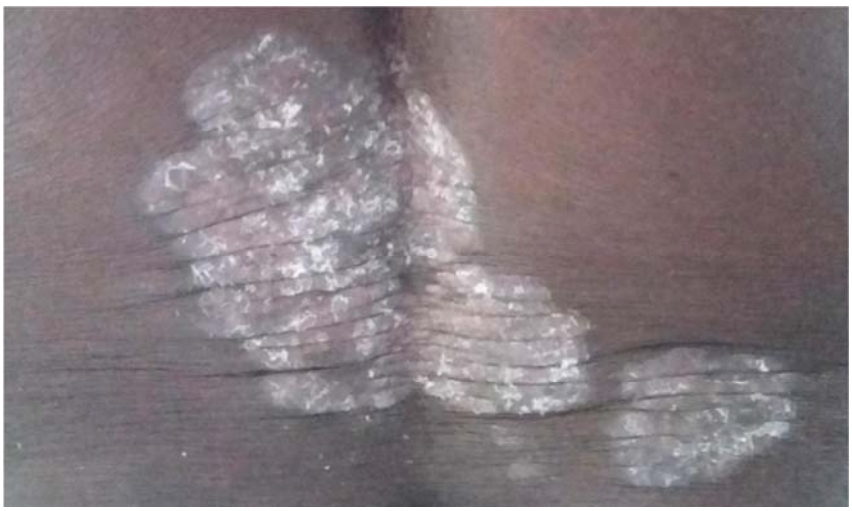


FIG 1: Hypopigmeted scaly lesion in the back

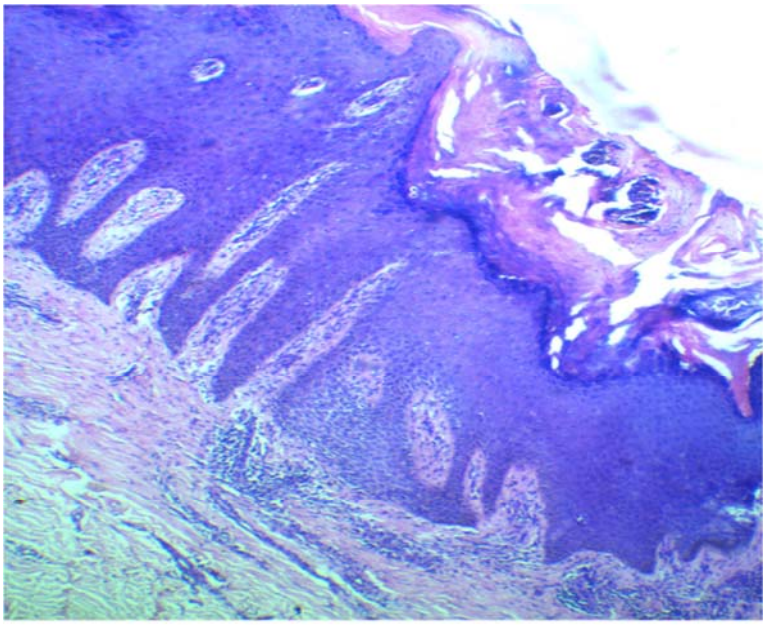


FIG2: Histological section show parakeratosis, acanthosis and elongation of rete ridges-PSORIASIS VULGARIS(H &E 40X)

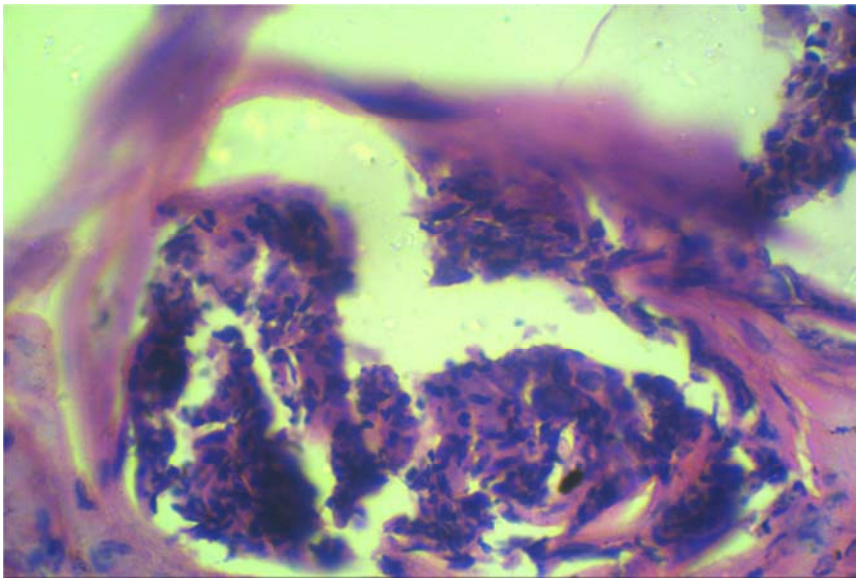


FIG 3: Histological section shows Munro's microabscess-PSORIASIS VULGARIS(H &E 100X)

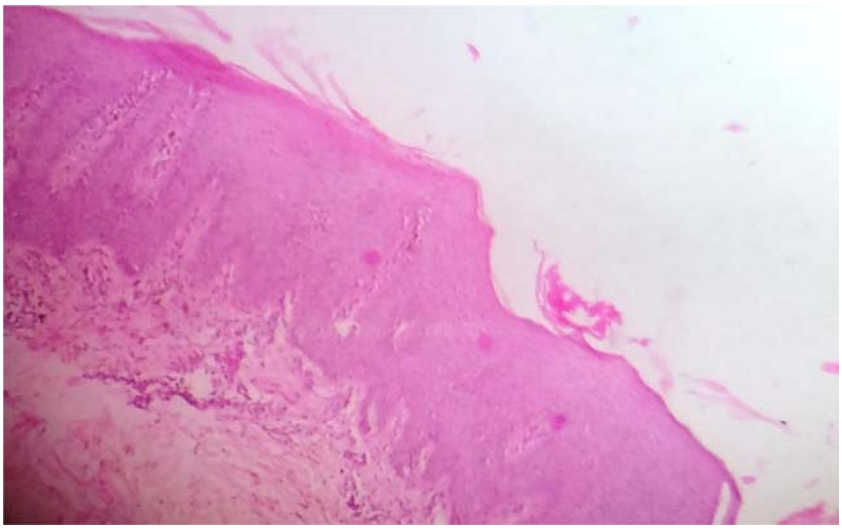


FIG 4: Histological section reveals epidermal hyperplasia and spongiosis-PSORIASIFORM DERMATITIS (H & E 40X)

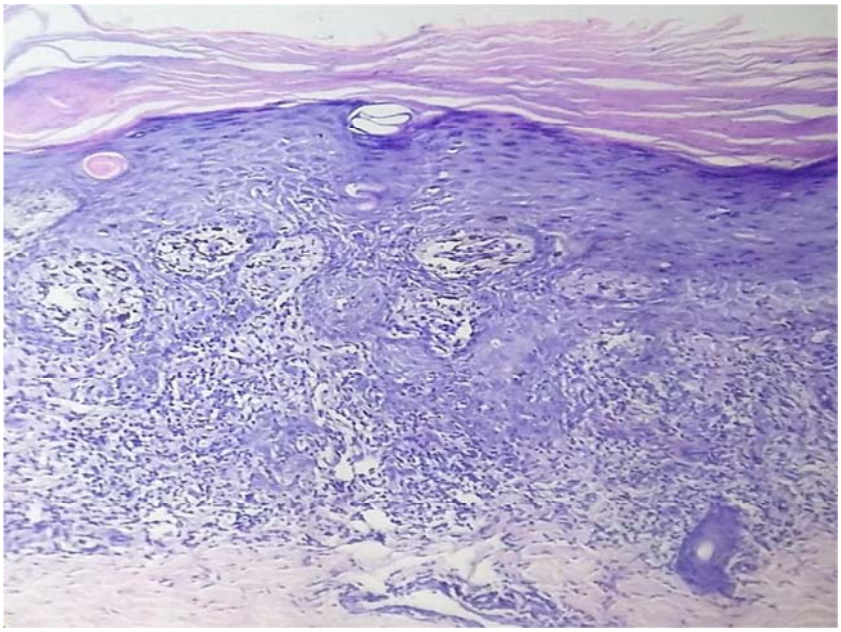


FIG5 : Histological section showing orthokeratosis, wedge shaped hypergranulosis and band like inflammatory infiltrate in the upper dermis-LICHEN PLANUS(H &E 40X)

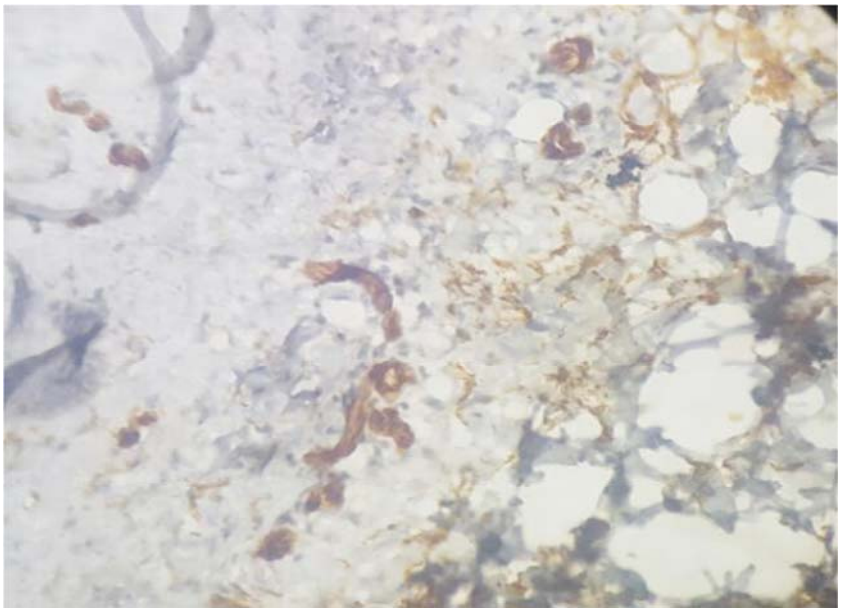


FIG 6 : CD34 Immunostaining showing strong positivity(400X)

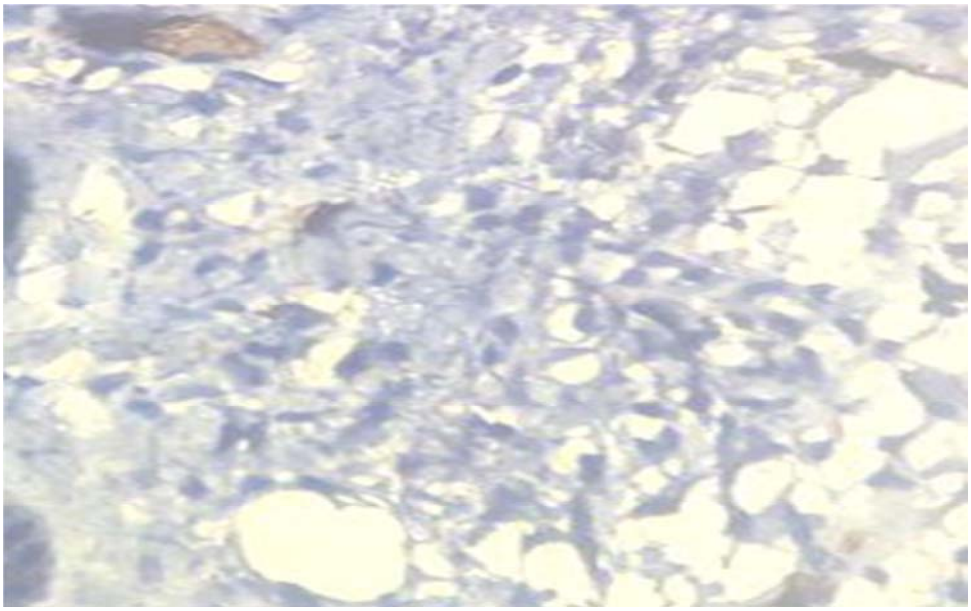


FIG 7 : CD 34 Immunostaining showing weak positivity (400X)

Discussion

DISCUSSION

The accurate diagnosis of any non-infectious scaly skin lesion is important for its effective treatment and evaluation of its prognostic significance. Most of these scaly skin lesions have a similiar clinical presentation, hence the histopathological study is considered as the gold standard for the evaluation of these lesions.

The present study was conducted to determine the age and sex incidence of scaly non-infectious skin lesions and its clinical correlation.Fifty one patients who came with complaints of scaly skin lesions and clinically thought to arise from an non-infectious etiology were subjected to biopsy and evaluated histopathologically.

TABLE: 13

COMPARISON OF AGE IN DIFFERENT STUDIES

STUDY	AGE RANGE (YEARS)
Yonus et al[88]	21-30 years
Rajasekhar et al[1]	31-40 years
Vijay et al[89]	21-30 years
Grace et al[90]	31-40 years
Present study	21-30 years

Yonus et al and Vijay et al showed that maximum number of cases are seen in the age group of 21-30 years. In the present study maximum number of cases are detected in the same age group.

TABLE :14

COMPARISON OF SEX DISTRIBUTION IN DIFFERENT STUDIES

STUDY	MALE	FEMALE
Grace et al	60.25%	39.75%
Rajasekhar et al	77.5%	22.5%
Present study	51%	49%

Studies by Grace et al and Rajasekhar et al study show that the incidence of noninfectious scaly skin lesions is higher in males as in concordance with our present study which shows a slightly higher incidence (51%) in males.

TABLE:15

COMPARISON OF CLINICAL COMPLAINTS IN VARIOUS STUDIES

CLINICAL COMPLAINTS	PRESENT STUDY	VELDHURTHY VS et al
HYPERPIGMENTED LESION	49%	36.9%
HYPOPIGMENTED LESION	49%	31.5%

In the study by Veldhurthy vs et al, hyperpigmented scaly skin lesions was the common presentation accounting for 36.9% followed by hypopigmented lesions (31.5%).However in this study, both hypo and hyperpigmented lesions share equal incidence accounting for about 49% each.

TABLE:16

HISTOPATHOLOGICAL DIAGNOSIS-COMPARISON OF PSORIASIS IN VARIOUS STUDIES

STUDY	PSORIASIS
YONAS ET AL	Psoriasis (36.8%)
RAJASEKHAR ET AL	Psoriasis (42.5%)
VELDHURTHY VS ET AL	Psoriasis (11.95%)
PRESENT STUDY	Psoriasis (27.9%)

Psoriasis is the most common disease accounting for 36.8% and 42.5% in the studies done by Yonus et al and Rajasekhar et al respectively while it accounts for about 11.95% of cases in Veldhurthy et al study. The present study though having a percentage in betweeen the earlier said studies also reported Psoriasis (27.9%) as the most common histopathological diagnosis.

TABLE: 17

HISTOPATHOLOGICAL DIAGNOSIS- COMPARISON OF LICHEN PLANUS IN DIFFERENT STUDIES

STUDY	LICHEN PLANUS
YOUNAS et al	31.5%
RAJASEKHAR et al	30%
VELDHURTHY et al	26%
PRESENT STUDY	21.6%

In the present study, lichen planus accounts for 21.6%, which is the second most common lesion after psoriasis. In the study by Younas et al, lichen planus account for about 31.5% while Rajasekhar et al reported lichen planus in 30% of their cases. In the study of Veldhurthy et al,lichen planus accounted for 26% of the total study population.

TABLE : 18

CLINICAL AND HISTOPATHOLOGICAL CORRELATION IN VARIOUS STUDIES

STUDY	CORRELATION
GRACE et al	97.52%
YOUNAS et al	76.30%
PRESENT STUDY	70.60%

In Grace et al study 97.52% of clinical diagnosis was compatible with that of the histopathological diagnosis. Younas et al study showed 76.30% compatibilty while our study has 70.60% clinico-histopathological diagnosis compatibility.

In this study, histopathological diagnosis of lichen nitidus, parapsoriasis, pityriasis rosea and pityriasis rotunda completely correlated with the clinical diagnosis (100%) while there was an observed clinico-histopathologic diagnostic incompatibilty in 15 cases presenting with the other diagnosis. Out of 15 cases, 4 cases which was clinically diagnosed as lichen planus were found to be chronic atopic dermatitis, prurigo nodularis and chronic dermatitis with pigment alteration.The rest (9 cases) which were clinically diagnosed as psoriasis were found to be eczematous dermatitis, lichen planus, pityriasis rubra pilaris and psoriasiform dermatitis. 2 cases of clinically diagnosed parapsoriasis was found to be psoriasiform dermatitis and Hansen’s disease.

In this study it is noted that majority of histologically diagnosed cases of psoriasiform dermatitis clinically presented with features mimicking Psoriasis. This emphasis the need for histopathological evaluation of all clinically suspected cases of psoriasis to rule out psoriasiform dermatitis since the treatment for both varies. Clinical management was definitely benefited in non-correlated cases by histopathological examination.

In addition to that dermal vascular changes was studied to compare between psoriasis and psoriasiform dermatitis, using an immunohistochemical marker CD34 in this study. It shows that CD34 was strongly and moderately expressed in psoriasis and weakly expressed in psoriasiform dermatitis.

Summary

SUMMARY

A study of the histological spectrum in patients presenting to the dermatology department with noninfectious scaly skin lesions was undertaken as a prospective observational study. The period of study was for 2 years between July 2014 to July 2016.

Analysis of the skin biopsies from the study population (51 cases) show a wide histopathological spectrum with Psoriasis being the most common lesion accounting for about 27.9% followed by Lichen planus. The other histologic diagnosis given were chronic nonspecific dermatitis, Parapsoriasis, Lichen nitidus, chronic atopic dermatitis, eczematous dermatitis, erythrodermic psoriasis, lichenoid dermatitis, Psoriasiform dermatitis, Pityriasis rosea, pityriasis rubra pilaris, pityriasis rotunda, prurigo nodularis,and seborrheic dermatitis.

Maximum number of patients with scaly skin lesions were in their second decade. Male preponderance was noted, with 51% of the individuals with scaly skin lesions being males and 49% females.

High percentage (70.60%) of clinico-histopathological correlation was noted in this study with incompatibility observed in only 29.40% of the cases studied.

The Vascular proliferation validated by CD34 expression was strong to moderate in all cases of psoriasis and weak in psoriasiform dermatitis.

Conclusion

CONCLUSION

Papulosquamous lesions are the most common skin disease encountered. These present clinically as a scaly skin lesion with pigment alteration. The most common of this group being Psoriasis vulgaris followed by Lichen planus. This study reiterates that these lesions show a definite male preponderance with maximum patients in their second decade (20-30 years). Histopathological confirmation is mandatory for the treatment protocol, since most of the papulosquamous lesion have similar clinical presentation.

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36. No •. Vii •., Morphologia •. Fig. 1. A. Early psoriatic changes in the dermal vessels, reaction with CD34 in the endothelium of tortuous and dilated vessels lined with high columnar endothelium, IHC method (×1000). Б. Membrane reaction of multiple T-lymphocytes

with CD3 marker, IHC method (×1000). B. Mixed reaction of S100 marker in the Langerhans cells (elongated cells with branched processes), IHC method (×1000). Γ. Cytoplasmic reaction of single CD68 macrophages, IHC method (×1000).

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Annexures

PROFORMA

PATIENT DETAILS:

Name: Age: Gender:

Hospital Number: Histopathology Number :

Type of biopsy:

Site of biopsy:

CLINICAL DETAILS:

Site:

Type of lesion:

Duration:

Single/ multiple:

Associated symptoms/ signs/ lesions:

1. Itching
2. Scaling
3. Erythema
4. Pigmentation
5. Plaque
6. Papule
7. Others

CLINICAL DIAGNOSIS

HISTOPATHOLOGICAL DIAGNOSIS

INFORMED CONSENT FORM FOR PARTICIPATION IN A RESEARCH STUDY

HISTOPATHOLOGICAL ANALYSIS OF SCALY SKIN LESIONS OF NONINFECTIOUS ETIOLOGY

CONSENT FORM 1

INTRODUCTION

You are invited to take part in a research study conducted by **MANIMEGALAI .S** at **CHENNAI MEDICAL COLLEGE HOSPITALAND RESEARCH CENTRE**. You are invited to participate because of presence of "**SCALY SKIN LESION**". If you take part in this study, you will be one of people expected to participate in study. This study is conducted under the supervision of the course instructor, **DR.S. PRIYA BANTHAVI**. No funding has been received for this study.

PROCEDURES

If you agree to participate, you will be interviewed about your "SKIN LESION". The interview will last about 20 – 30 MINUTES followed by **CLINICAL EXAMINATION AND BIOPSY.**

RISKS

To the best of our knowledge, participation in this study has no risk or harm to you. It is possible that some of the questions will make you uncomfortable, but you are free to refuse to answer any question, or to stop at any time.

BENEFITS AND COSTS

You do not have to pay to participate in this study. You will not receive any direct benefit from participating in this study, but we hope to gather information that will help us to evaluate the "Histopathological analysis of scaly skin lesions

of noninfectious etiology**"**. You will not be compensated for participating in this study.

WITHDRAWAL FROM THE STUDY

Participation in this study is voluntary. You have the right to refuse to take part in this study. If you choose to participate, you have the right to withdraw at any time without penalty or loss of benefits to which you are otherwise entitled. If there are any new findings during the study that may affect whether you want to continue to take part, you will be told about them as soon as possible. The student researchers may decide to discontinue your participation without your permission because they may decide that staying in the study will be bad for you or for any other reason.

CONFIDENTIALITY

Protecting participants confidentiality is of the utmost importance throughout this research study. All information obtained in the interview will remain confidential. Only the student researcher **MANIMEGALAI.S** will have access to the link between participant name and Study ID, which will be stored separately AFTER the interview.

The information from the interviews will be used for research papers only. Participants identity will not be revealed in any paper resulting from this project.

QUESTIONS OR PROBLEMS

You are encouraged to ask questions now, and at anytime during the study. You can reach Dr.**MANIMEGALAI. S** at **CHENNAI MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE** for any queries . You could be contacted by the Student Researcher if she has any questions about your participation in this study.

CONSENT FORM II

PARTICIPANT CONSENT FORM

CERTIFICATION

I have read the Informed Consent Form (or the form has been read to me). I believe that I understand this Consent Form. I understand the purpose of the research study and what I will be asked to do. I have been given the chance to ask questions and they have been answered to my satisfaction.

- I understand that participation in this study is voluntary and I may refuse to participate or may discontinue participation at any time.
- I understand that the researchers will work to keep the information I give them confidential.
- I hereby give my informed and free consent to be a participant of this study.
- I permit the use of stored sample (tissue/blood) for future research.
  Yes [ ] No [ ]

SIGNATURES: **DATE :**

1. _____

CONSENT SIGNATURE OF PARTICIPANT

2. _____

NAME OF PARTICIPANT

3. _____

NAME AND SIGNATURE OF THE
PERSON CONDUCTING INTERVIEW

Master Chart

Sl. No	NAME	AGE	SEX	COMPLAINTS	CLINICAL DIAGNOSIS	H.NO	HISTOPATHOLOGY	CD34
1	Prabavathy	25	F	Hyperpigmented scaly lesion	lichen planus	H1248/15	Chronic atopic dermatitis	
2	Barakath nisha	19	F	hyperpigmented Scaly lesion	lichen planus	H2417/16	Lichen planus	
3	Rajesh	23	M	Hypopigmented scaly lesion	Lichen nitidus	H2419/16	Lichen nitidus	
4	Joe clinton	11	M	Hyperpigmented scaly lesion	Hypertrophic lichen planus	H2235/16	Hypertropic lichen planus	
5	Narayanasamy	49	M	hyperpigmentedScaly lesion	Lichen planus	H2874/16	Hypertropic lichen planus	
6	Vennila	45	F	Hyperpigmented scaly lesion	hypertrophic lichen planus	H2640/16	Hypertropic lichen planus	
7	Deepika	21	F	Erythematous scaly lesion	parapsoriasis	H819/15	Parapsoriasis	
8	Bhuvaneshwari	30	F	Hyperpigmented scaly lesion	psoriasis	H644/15	Eczematous dermatitis	
9	Uma maheshwari	28	F	Hyperpigmented scaly lesion	Lichen planus	H1865/15	Lichen planus	
10	Pradeepa	25	F	hyperpigmented Scaly lesion	Lichen planus	H86/15	Lichen planus	
11	Vikash	12	M	Hypopigmented scaly lesion	pityriasis rotunda	H436/15	Pityriasis rotunda	
12	Arun	32	M	Erythematous,scaly lesion	Lichen planus	H2563/15	Lichen planus	
13	Raja	21	M	Hyperpigmented scaly lesion	Lichen planus	H2486/15	Prurigo nodularis	
14	Manivel	17	M	Hyperpigmented scaly lesion	Psoriasis	H1411/15	Lichen planus	
15	Prema	40	F	Hypopigmented scaly lesion	Lichen nitidus	H1541/15	Lichen nitidus	
16	Anandh	24	M	Hyperpigmented scaly lesion	Lichen planus	H5387/15	Lichen planus	
17	Sundari	42	F	Hypopigmented scaly lesion	Parapsoriasis	H5184/15	Hansens disease	
18	Tamilselvi	42	F	hyperpigmented, scaly lesion	Psoriasis	H5591/15	Chronic dermatitis	
19	Murugaiyan	66	M	hyperpigmented, scaly lesion	Lichen planus	H1443/16	Lichen planus	
20	Manasa	10	F	Erythematous scaly lesion	Lichen planus	H1927/14	Lichenoid dermatitis	
21	Muthulakshmi	65	F	Hyperpigmented scaly lesion	lichen planus	H2009/14	Lichen planus	
22	Mohandoss	51	M	hypopigmentedScaly skin lesion	Parapsoriasis	H4228/14	Parapsoriasis	
23	Sarojini	18	F	Hypopigmented scaly lesion	Lichen planus	H4347/14	Chronic dermatitis with pigment alteration	
24	Kalamani	65	F	hyperpigmented scaly lesion	Lichen planus	H4483/14	Lichen planus	
25	Rasheeda begam	30	F	Hyperpigmented scaly lesion	Lichen planus	H4431/14	Lichen planus	
26	Imaran	17	M	Hyperpigmented scaly lesion	lichen planus	H4516/14	Hypertropic lichen planus	
27	Adarsh	18	M	Hyperpigmented scaly lesion	pityriasis rosea	H1587/15	Pityriasis rosea	
28	Chinnathambi	57	M	hyperpigmented Scaly lesion	Psoriasis	H1300/14	Pityriasis rubra pilaris	
29	Geetha	17	F	hyperpigmented Scaly lesion	Lichen planus	H3851/14	Lichen planus	
30	Balkis beevi	75	F	Hyperpigmented scaly lesion	psoriasis	H3899/14	Psoriasis vulgaris	moderate
31	Nazar mohamed	60	M	hypopigmented Scaly lesion	Psoriasis	H1936/15	Psoriasis vulgaris	strong
32	Shankar	52	M	Hyperpigmented scaly lesion	Psoriasis	H1447/15	Psoriasis vulgaris	strong
33	Mohan	30	M	hypopigmented Scaly lesion	psoriasis vulgaris	H2091/15	Psoriasis vulgaris	strong
34	Nandha kumar	29	M	Erythematous scaly lesion	Psoriasis vulgaris	H2496/14	Psoriasis vulgaris	strong
35	Gurunathan	45	M	Erythematous scaly lesion	Psoriasis vulgaris	H1808/14	Psoriasis vulgaris	strong
36	Palaniyammal	78	F	Hyperpigmented scaly lesion	Psoriasis	H1900/14	Psoriasis vulgaris	moderate
37	Anbazhagan	34	M	Scaly lesion	Psoriasis vulgaris	H2154/14	Early Psoriasis vulgaris	strong
38	Alwar	65	M	Erythematous scaly lesion	Psoriasis vulgaris	H3449/14	Psoriasis vulgaris	strong
39	Ashok kumar	45	M	erythematous scaly lesion	psoriasis	H3465/14	Psoriasis vulgaris	strong
40	Govindharaj	30	M	Erythematous scaly lesion	Psoriasis vulgaris	H3750/14	Psoriasis vulgaris	strong
41	Shanthi	29	F	hypopigmented Scaly lesion	Psoriasis vulgaris	H4215/14	Psoriasis vulgaris	strong
42	Mohamed ibrahim	58	M	hypopigmented Scaly lesion	Psoriasis	H1292/15	Psoriasis vulgaris	strong
43	Nabila banu	4	F	Hypopigmented scaly lesion	Parapsoriasis	H3312/14	Chronic dermatitis with psoriasiform hyperplasia	weak
44	Vanisree	36	F	Erythematous scaly lesion	Psoriasis vulgaris	H3934/14	Chronic dermatitis with psoriasiform hyperplasia	weak
45	Vanitha	45	F	Erythematous scaly lesion	Psoriasis vulgaris	H3388/14	Early psoriasis vulgaris	strong
46	Ladha	35	F	Erythematous scaly lesion	Erythrodermic psoriasis	H3548/14	Erythrodermic psoriasis	moderate
47	Rajalingam	65	M	Hyperpigmented scaly lesion	Psoriasis	H2480/14	Mild dermatitis with psoriasiform hyperplasia	weak
48	Chandrasekar	50	M	Scaly lesion	Psoriasis vulgaris	H2204/14	Psoriatic dermatitis	weak
49	Charles	43	M	Erythematous scaly lesion	Psoriasis	H3272/14	Seborrheic dermatitis	weak
50	Meera	45	F	scaly erythemayous lesion	Pustular psoriasis	H2316/14	Pustular psoriasis	moderate
51	Sumathy	46	F	scaly hyperpigmented lesion	psoriasis	H3456/15	Early psoriatic dermatitis	weak