STUDY AND ANALYSIS ON VERRUCOUS SKIN LESIONS OVER THE LOWERLIMBS

Dissertation submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

in partial fulfillment of the requirements for the award of

M.D. DEGREE (BRANCH-XII)

IN

DERMATOLOGY, VENEREOLOGY AND LEPROLOGY

APRIL 2013

CERTIFICATE

This is to certify that the dissertation entitled **"Study and analysis on verrucous skin lesions over the lowerlimbs"** is the bonafide original work of Dr.N.S.Jayanthi in partial fulfillment of the requirements for MD DERMATOLOGY, VENEREOLOGY AND LEPROLOGY BRANCH XII examination of the Tamilnadu DR.M.G.R Medical University, Chennai to be held in April 2013. The period of study was from August 2011 to July 2012.

Date :

Professor & Head of the Department,

Department of Dermatology,

Coimbatore Medical College & Hospital,

Coimbatore.

Date :

Dean,

Coimbatore Medical College & Hospital,

Coimbatore.

DECLARATION

I Dr.N.S.Jayanthi solemnly declare that the dissertation entitled **"Study and analysis on verrucous skin lesions over the lowerlimbs"** is a bonafide work done by me at Coimbatore Medical College Hospital during the year August 2011 to July 2012 under the guidance & supervision of Dr.P.P.Ramasamy M.D.,D.D., Professor & Head of Department, Department of Dermatology, Coimbatore Medical College & Hospital.

The dissertation is submitted to Dr.MGR Medical University towards partial fulfillment of requirement for the award of MD degree branch XII Dermatology, Venereology and Leprology.

PLACE: Dr.N.S.JAYANTHI

DATE:

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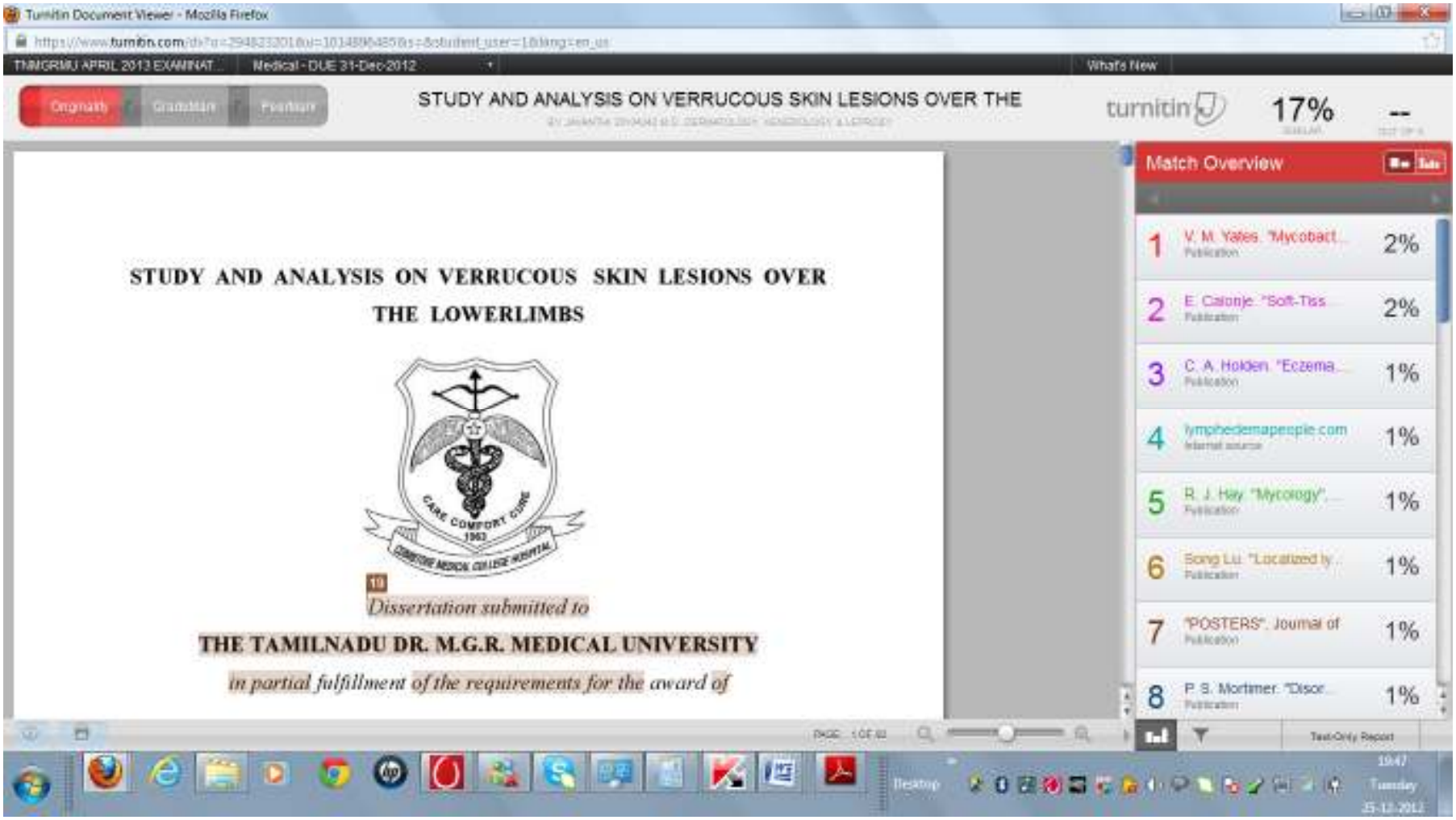
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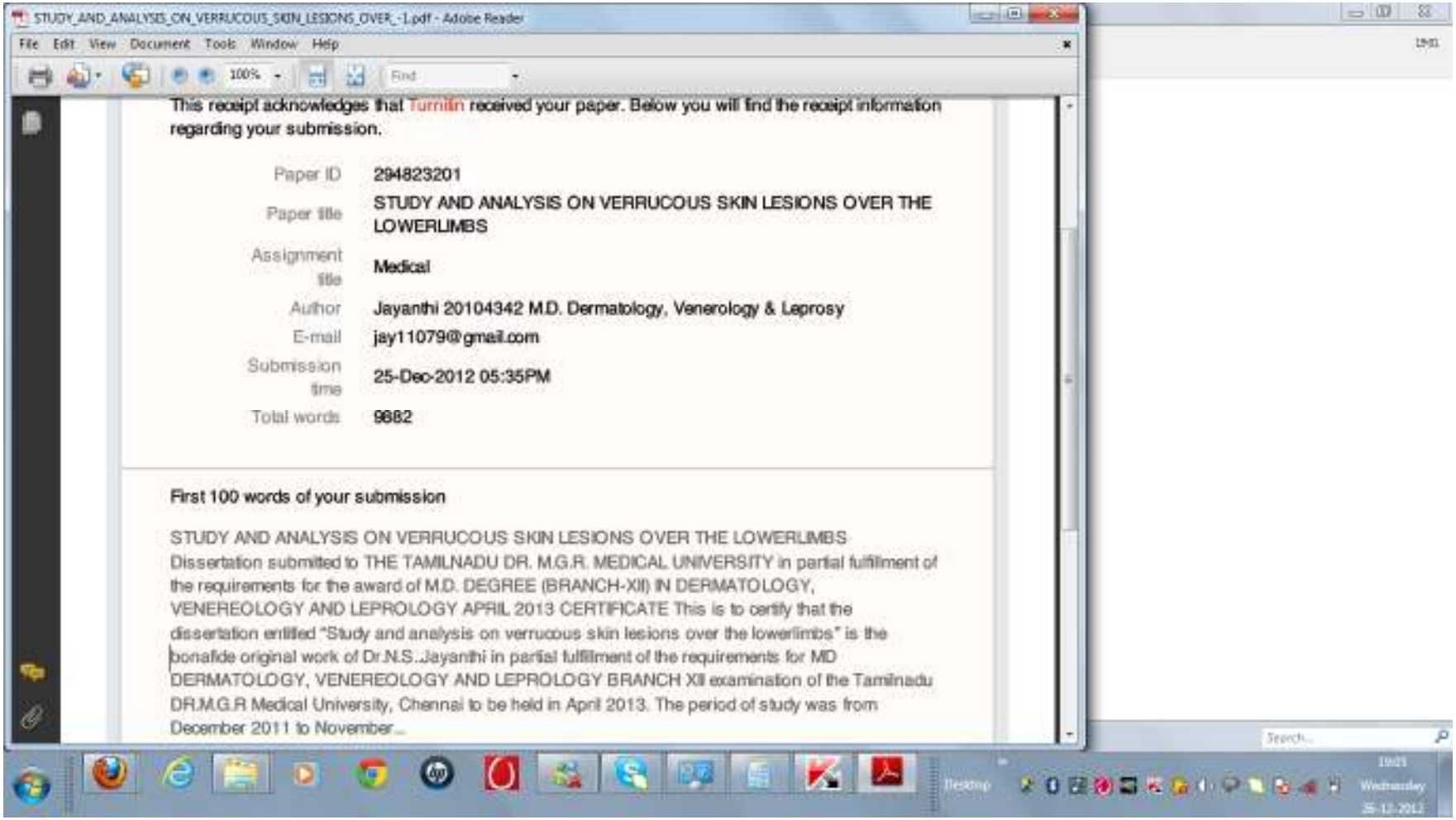
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Coimbatore Medical College

COIMBATORE, TAMILNADU, INDIA - 641 014

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Name of the Candidate : DR. N. S. JAYANTHI

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CONTENTS

ABSTRACT

Title

Study and analysis on verrucous skin lesions over the lowerlimbs.

BACKGROUND

Verrucous skin lesions of lower limbs often brings a diagnostic dilemma to a treating Dermatologist . Much study has not been done on this subject. A proper workup is needed to arrive at a diagnosis. Verrucous skin lesions are more common on lower limbs than upper limbs. Histopathological study is mandatory to differentiate these conditions. Usually we need deep biopsy. At times, serial biopsies should be taken to arrive at the diagnosis.

AIM

To study the clinical and histopathological patterns of various verrucous skin lesions of lower limb and to find out the etiology of these verrucous skin lesions.

Material and methods

During the study period of one year from 1 August 2011 to 31st July 2012 in our outpatient department, department of Dermatology Coimbatore Medical College Hospital. All the patients with verrucous skin lesions of lower limbs of all age groups were included in the study. After clinical examination,

patients underwent routine and certain special investigations to arrive at the diagnosis.

Result

Out of hundred patients, the common causes of verrucous lesions over the lowerlimbs were hypertrophic lichen planus, wart, lichen simplex chronicus, psoriasis and lichen amyloidosis. The less common etiologies were porokeratosis, hypertrophic discoid lupus erythematosus, callosity and Elephantiasis nostros verrucosa cutis.

Conclusion

Hypertrophic lichen planus, Lichen simplex chronicus and wart were the common etiological factors for the occurrence of verrucous skin lesions over the lower limbs. Porokeratosis, hypertrophic discoid lupus erythematosus, elephantiasis nostros verrucosa cutis were the least common cause of verrucous skin lesions of lower limbs. Patients with phlebolymphedema, verrucous carcinoma and verrucous skin lesions of neuropathy, males are mostly affected.

Key words

verrucous skin lesions, hypertrophic lichen planus, wart, lichen simplex chronicus, psoriasis and lichen amyloidosis, phlebolymphedema, verrucous carcinoma and verrucous skin lesions of neuropathy.

INTRODUCTION

Verruca is a Latin word which means a wart like projection, over the back of a toad or on some leaves. The term was first used by Sennertus. He originally used this term for wart because 'they appear on the skin surface like eminence of little hill' .[1] Aulus Cornelius Celsus, who lived during the reign of Tiberius Caesar, in discussing wart-like lesions in his classical work on medicine 'De Medicina', mentioned three types.[2,3,4] Hippocratic writings mentioned about the wart in children.[5]

Verrucous skin lesions of lower limbs often brings a diagnostic dilemma to a treating Dermatologist . Much study has not been done on this subject. A proper workup is needed to arrive at a diagnosis. Verrucous skin lesions are more common on lower limbs than upper limbs. From a clinical standpoint, many verrucous lesions have diverse etiology , but closely resemble each other. Most often they present as hyperpigmented verrucous plaques. Verrucous skin lesions may be due to infectious and non infectious causes. Some verrucous lesions may be the manifestation of different disease process . For that, first step is to identify the underlying pathology. Histopathological study is mandatory to differentiate these conditions. Usually we need deep biopsy. At times, serial biopsies should be taken to arrive at the diagnosis. It is difficult to differentiate verrucous lesions due to lymphedema and phlebolymphedema clinically and also histopathologically. To differentiate these two, we need further workup. Verrucous lesions due to neuropathy also need further investigations to arrive at the diagnosis.

REVIEW OF LITERATURE

Verrucous skin lesions of lower limbs comprise a heterogenous group of disorder. These disorders have been considered diagnostically challenging for the dermatologist.

Verrucous lesions have jagged, undulating surface [warty] and most often with histopathological findings of papillomatosis.[6]

Verrucous lesions of lower limbs are as follows

Infectious causes :

Viral	–	Wart [7]
Bacterial	–	Tuberculosis verrucosa cutis[6]
		Lupus vulgaris[6]
Fungal	–	Chromoblastomycosis[8]
		Fixed cutaneous sporotricosis [9]
		Cutaneous rhinosporidosis[10]
		Cutaneous blastomycosis[11]
		Coccidioidomycosis [12]
Ectoparasite	–	Crusted scabies[6]

Non infectious causes :

Papulosquamous	–	Hypertrophic lichen planus [6] ,
		Psoriasis [7]

Eczema	_	Lichen simplex chronicus [6]
Prurigo	_	Prurigo nodularis [7]
Collagen vascular disorder	–	Hypertropic variant of Discoid lupus erythematosus[6]
Deposition disorders	–	Lichen amyloidosis[7]
		Lipoid proteinosis. [6]
Keratinization disorders	–	Porokeratosis[6] ,
		Acrokeratosis verruciformis[6] ,
		Darier's disease[7]
Benign tumor	–	Seborrhoeic keratosis[7]
		Stucco keratosis [7]
Pigmentary disorder	–	Incontinentia pigmenti [6]
Nevi	–	Verrucous epidermal nevi[6]
Disorder of lymphatics	–	Lymphangioma circumscriptum[6],
		Elephantiasis nostros verrucosa cutis [16]
Disorders of blood vessel	–	Verrucous hemangioma[13]
		Angiokeratoma circumscriptum[14]
		Phlebolymphedema[15]
Acantholytic disorders	–	Warty dyskeratoma[6]
Malignancy	–	Verrucous carcinoma[6],
		Verrucous variant of malignant melanoma [20],
		Kaposi sarcoma [21].
Neuropathy associated verrucous skin lesions	–	Leprosy[17]
		Diabetes Mellitus[18]
		Other Sensory neuropathies [18].
Drug reactions	–	Iododerma [6]

Bromoderma [6]

HYPERTROPHIC LICHEN PLANUS: [Lichen planus verrucosus] [23]

Lichen planus (Greek leichen, "tree moss" Latin planus, "flat") is a common inflammatory disorder that affects the skin, nails, mucous membranes and hair.[22]

Clinical features:

Hypertrophic lichen planus seen commonly over extremities especially over shin, ankles, interphalangeal joints. Two-thirds of cases occur between the ages of 30 and 60 years. There is no sexual predilection[22]. The lesions are often pruritic, symmetrical, verrucous plaque with central depigmented area surrounded by a hyperpigmented rim. These lesions heal with scarring. Most often it is refractory to treatment. Chronic venous stasis is usually associated with hypertrophic lichen planus[24]. It can rarely transfer into squamous cell carcinoma[25]. Malignant transformation is more common in distal extremities. From the diagnosis of hypertrophic lichen planus to malignancy needs atleast 12 years. [26, 27.]

Histopathology:

Compact orthokeratosis, hypergranulosis, pseudoepitheliomatous hyperplasia, irregular acanthosis, vacuolar degeneration of basal layer. Lymphocytic infiltration at the base of rete ridges. The interface vacuolar

changes are discrete and often to the base of rete ridges. Necrotic keratinocytes are present in lower epidermis and papillary dermis [28].

Lichen simplex chronicus : [Circumscribed neurodermatitis][29]

Definition :

It is an eczematous dermatosis which is characterized by heavily lichenified plaques more often with a single lesion. [29]

Etiopathogenesis :

Patients with LSC are readily conditioned to scratch following an itch stimulus . This may occur as the manifestation of atopic state. Emotional state plays important role in development of LSC.

Clinical features :[30]

Lichen simplex chronicus is uncommon in childhood. Any age group from adolescent age is commonly affected. The most common age group is between 30 and 50 years of age. Women are more commonly affected than men. The common sites are the nape of the neck, lower legs ,ankles, sides of the neck, scalp, upper thighs, vulva, pubis or scrotum, and the extensor forearms. When excoriation continues for many years with lichenification and lax subcutaneous tissues, a solid, tumour-like plaques will be formed. This giant lichenification of Pautrier will be warty and have cribriform surface. This occurs commonly in the genitocrural region.

Histopathology[31]:

Hyperkeratosis, interspersing parakeratosis, acanthosis, papillomatosis with irregular elongation of rete ridges. Broadening of dermal papillae with spongiosis and increased number of fibroblast and vertical orientation of collagen bundles. With chronic rubbing, epidermal hyperplasia and fibrosis become marked.

Psoriasis verrucosa : [32]

It is a rare variant of psoriasis in which two types are recognised. They are dome shaped papules with keratotic plug and crater shaped papule with central depression.

Clinical features:

These lesions occur along with typical psoriatic lesions elsewhere.

Histopathology : [33]

Parakeratosis, epidermal acanthosis with elongation of rete ridges, thin suprapapillary epidermal plates, epidermal hypogranulosis and dilated, tortuous capillaries and a lymphocyte-predominant inflammatory infiltrate, which may contain admixed neutrophils in the papillary dermis. Neutrophil collections in the stratum corneum and stratum spinosum, called 'Munro microabcesses' and 'spongiform pustules of Kogoj', respectively, are the most specific findings for psoriasis. Papillomatosis, epithelial buttressing, and the absence of infection suggest verrucous psoriasis.

VIRAL WART :

HPV is the causative organism of viral wart which infects keratinizing or non - keratinizing stratified squamous epithelium. Cutaneous warts occur at any age. Rarely seen in infancy and early childhood. Peak incidence is seen in school children, adolescence and early adulthood[88,89.] ***Incubation period:*** Variable from few weeks to years[90]. Spreads by direct or indirect contact. Loss of the epithelial barrier function by trauma, maceration or by both leads to inoculation of the virus in the basal epidermal layer. Plantar warts are usually acquired from swimming pool or shower-room floors.

Papillomaviruses are double-stranded DNA viruses. These virus will get integrate into host DNA. The size is 55 nm. More than 200 genotypes of HPV are there which can affect skin and mucous membrane. They are highly species-specific. The viruses infect the basal keratinocyte of the epidermis, through disruptions of the skin or mucosal surface. At this location, the virus remains latent in the cell as a circular episome in low copy numbers. As the epidermal cells differentiate and migrate to the surface, the virus replicates and matures. This results in cutaneous and mucosal wart. Most of the papillomaviruses have different anatomic predilections either skin or genital mucosa. There are many types of viruses which are associated with epidermal

malignancies. They interact with E6 and E7 proteins of host cell function. Clinically, Common warts[91, 92](except plantar warts) are caused mainly due to HPV-2,1,4,27 and 57serotypes. Firm papules with a rough, horny surface, ranging in size from less than 1 mm to over 1 cm in diameter. Common site is on the backs of the hands and fingers. Single wart remains unchanged for months together. It exhibits Köebner-like isomorphic phenomenon at the site of trauma. Compared to plane wart, this phenomenon occurs rarely. Warts in adults heals slower, either with or without treatment. Common warts regresses asymptomatically. It will take several weeks to resolve without any pigmentation. Plantar warts[93] are caused by HPV-1, 2, 4, 27 or 57. The deep 'myrmecia' form is due to HPV-1. Plantar wart starts as 'sago-grain' papule, then becomes a rounded lesion, which is sharply defined with a keratotic, rough surface surrounded by a smooth collar of thickened horn. While on paring, small bleeding points are seen. This helps to differentiate this wart from corn foot. Pain may be a common but variable symptom. Severity may change. Mosaic warts are often painless in children. Spontaneous regression occurs faster in children than in adults. It may be associated with hyperhidrosis or orthopaedic defects. Mosaic warts persist for longer time. Before the lesion separates, there may be blackening from thrombosed blood .[94]

Histopathologically,[95,96] hyperkeratosis, vertical tiers of parakeratosis, acanthosis, papillomatosis, elongated rete ridges ,at the periphery of verruca, bent inward so that it appears to point radially toward the centre [arborisation].

The foci of koilocytes are located at stratum malphigii. Koilocytes posses small ,round deeply basophilic nuclei surrounded by a clear halo and pale staining cytoplasm.

LYMPHOEDEMA AND VENOUS DISEASE (PHLEBOLYMPHOEDEMA):

Phlebolymphedema is a type of secondary lymphedema in which mixed venous and lymphatic insufficiency may present. It cannot be easily recognized and treated. The lymphatic and venous systems are interrelated. When there is venous hypertension, there may be increase in compensatory lymphatic flow.[84.] This increased lymph load leads to failure of local lymphatics which leads to edema. Thrombosis of the major veins and deep vein incompetence affect the small initial and precollecting lymphatics of the skin and subcutaneous tissues of the lower leg which leads to chronic lipodermatosclerosis. Lymphoedema with venous disease leads to the gross swelling and skin changes.[85, 86] Kaposi–Stemmer sign will be positive. Skin creases become enhanced and hyperkeratosis develops. Papillomatosis occurs as the consequence of dilatation of upper dermal lymphatics and fibrosis. As the disease progress this leads to elephantiasis.

Histopathologically [86] the epidermis is acanthotic, with reduplication of the basement membrane. In the dermis, collagen is increased with loss of elastic fibres and anchoring filaments. Overgrowth of the interstitial connective tissue

leads to hard late-stage. Increased protein leads to fibrosis .The number of blood vessels greatly increases. Lymphostatic vasculopathy develop in the blood vessels. In the upper dermis, numerous newly formed vessels can be seen. Highly vascularized dermis develops due to angiogenesis. Complications are hyperpigmentation, stasis dermatitis, lymphostasis, verrucose cutis, lipodermatosclerosis, and ulceration.

Lichen amyloidosis [63]:

Lichen amyloidosis presents as a pruritic eruption of multiple, discrete, scaly, hyperpigmented, hyperkeratotic papules. These papules coalesced to form verrucous plaque which resembles hypertrophic lichen planus or lichen simplex chronicus. It is commonly seen over shin. Other areas of involvement are ankles, calf and dorsum of feet. Abdomen, thigh, chest may be involved. Bullous forms are rarely reported. [64] It is the most common form of cutaneous amyloidosis. [66] This condition is common in Indian subcontinent.[65] Histopathologically, amyloid deposition is restricted to upper dermis. The amyloid deposits will expand the papillae and push the rete ridges laterally.

PRURIGO NODULARIS: [Hyde's prurigo]

Described by Hyde in 1909 [41]. It is a group of skin diseases characterized by intensely pruritic papules or nodules. [42]

Etiopathogenesis :

The cause is unknown. Emotional stress may be a contributory factor. 65–80% of patients are atopic. In 20% of patients, the condition starts after an

insect bite. In most of the cases, the cause could not be found out[43]. Chronic trauma by scratching leads to neural proliferation. Nerve growth factors and receptors are expressed in the skin lesion of prurigo. Increased numbers of calcitonin gene related peptide and substance P immunoreactive neuropeptides may play role in intense pruritus. In 75% of cases, Merkel cells are expressed. These changes do not occur in the lesions of lichen simplex chronicus[44, 45].

Clinical features[43]:

All age group may be affected, but most commonly seen between 20 and 60 years. Male to female ratio is 1:1.

They are small, tiny papules to hard globular nodules, with a raised, warty surface of 1–3 cm in diameter with an irregular ring of hyperpigmentation immediately around the nodules. The nodules will be arranged in groups. Pigmentary changes, crusting and scaling may occur. Xeroderma present over the intervening skin. They usually seen over the distal parts of the limbs, especially over the extensor surfaces. The trunk, face, palms may be affected. Patient's usual complaint is intense pruritus. New nodules develop over time and older nodules remain pruritic for a long time .Some lesions may regress spontaneously leaving a scar.

Histopathology [46,47] :

This is characterised by marked hyperkeratosis, irregular acanthosis, pseudoepitheliomatous hyperplasia and papillomatosis. Papillary dermis shows neural hyperplasia. There will be increased number of Langerhan's cells in

dermis. Dendritic cells and mast cells are enlarged with cytoplasmic granules over the lesional dermis.

TUBERCULOSIS VERRUCOSA CUTIS: (WARTY TUBERCULOSIS) [48]

A warty, indolent, plaque-like form of tuberculosis occurring due to the inoculation of Mycobacterium tuberculosis into the skin of a previously infected patient. This patient will have moderate or high degree of immunity. There are few organisms in these lesion (paucibacillary)[49] .

Pathogenesis:

Organism may be inoculated in three ways.

1. Accidental super infection from exogenous sources: physicians, pathologists and post-mortem attendants are traditionally at risk ('anatomist's warts', 'prosector's warts', 'verruca necrogenica') [50].
2. Autoinoculation with sputum in a patient with active tuberculosis.
3. Already infected children and young adults, who are having some degree of immunity may become infected from sputum by sitting on the ground or walking barefoot. [51,52]

Clinical features:

In Asia knees, ankles and buttocks are mainly involved.[53] In Europe, hands are the common site of involvement. Areas which are exposed to trauma and infected sputum or other tuberculosis material are the common sites. The lesion starts as a symptomless, small, indurated, warty papule with a slight

inflammation. With gradual progression, a verrucose plaque is formed. Irregular extension at the edges leads to a serpiginous outline with finger-like projections to form a massive, infiltrated papillomatous growth. Involution of centre of the lesion may lead to a white atrophic scar. The colour is purplish, red or brown. The consistency is generally firm with few areas of softening. Pus may be expressed from these soft areas or from fissures. Exudation and crusting occur rarely. The lesions may resemble Lupus vulgaris. [54] But the sites are different. Psoriasiform or keloidal appearance may be seen. Sporotrichoid spread and tuberculous lymphadenitis are very rare.[55] Anomalous forms are deeply destructive papillomatous and sclerotic forms, may cause deformity of the limbs[56]. In generalized form, papulonecrotic and lupoid lesions are seen. [57] This form occurs in patients with active disease, which may be due to haematogenous spread with a variable tissue response. An exuberant granulomatous form, Tumour-like forms was described.[52, 53, 58] Without treatment, extension is extremely slow and lesions may remain inactive for months to years together. [56,60 ,61] Spontaneous remission may occur and usually results in atrophic scars. The condition responds to antituberculosis treatment. Bone,joints and lymphnodes should be examined properly[52]. Miliary tuberculosis rarely reported. [59]

Histopathologically[62] it is characterised by hyperkeratosis, acanthosis and pseudoepitheliomatous hyperplasia. Neutrophilic abcess formation in upper dermis. In mid dermis, tuberculoid granulomas with

moderate amount of necrosis are seen. Tubercle bacilli are numerous in this disease when compared to lupus vulgaris.

LEPROSY WITH HYPERKERATOTIC AND VERRUCOUS SKIN LESION ON LOWER EXTREMITIES : [87]

These lesions are characteristically seen on the anterior aspect of ankle joint in Indian leprosy patients. These lesions are seen only in male patients who use stiff plastic shoes with a long tongue to their uppers. Commonly occurs as unilateral lesion but can occur bilateral also. It can occur in any leprosy spectrum. The skin of affected area will be dry or hypoaesthetic. Morphologically, three types are described.

Type1: Hyperkeratotic lesions with thread or finger like projections which resembles filiform warts.

Type 2: Hyperkeratotic lesions had hornlike projections with deep fissures in between.

Type 3: Least common type with mild hyperkeratosis and there were no projections.

Factors contributing to these lesions are sensory neuropathy, autonomic neuropathy and prolonged irritation of the skin from the 'tongue' of the stiff plastic shoes during walking. During the movement of ankle joint the tongue of the shoe rub over skin. The patient is not aware of this irritation. Dry and sensory loss further increase the disease process. These lesions are like callosity except for the fissures inbetween the hyperkeratotic plaques.

Histopathologically, compact hyperkeratosis and acanthosis can be made out. In type 1 and type 2 lesions, the acanthosis was massive with pseudoepitheliomatous hyperplasia of epidermis. In type 3 lesions, the hyperkeratosis and acanthosis are mild. The dermis revealed only a mild perivascular mononuclear cell infiltrate without any granuloma.

Verrucous carcinoma[34]:

The term was first coined by Ackerman in 1948. It is a slow-growing neoplasm with a tendency for local recurrence. It rarely metastasizes. It is a form of squamous cell carcinoma which is characterised by slow growing exophytic tumor. Clinically it is characterized by cauliflower like appearance at the site of chronic irritation. Four types has been described according to the anatomical site of involvement (a) oral florid papillomatosis - verrucous carcinoma of the oral cavity (b) giant condyloma of Buschke and Löwenstein - verrucous carcnoma of the genitoanal region (c) epithelioma cuniculatum - verrucous carcinoma of the plantar region (d) cutaneous verrucous carcinoma - verrucous carcinoma occurring in other areas of the skin. [34] The pathogenesis of verrucous carcinoma is not fully understood. Epithelioma cuniculatum is a locally destructive, slow-growing, low-grade tumour. It is typically found on the sole of the foot. It may involve periunguium, mucosa and other locations. It is a warty, soft bulbous mass with discharge of foul-smelling yellow material on the distal part of the sole of the foot. Multiple sinuses open on the surface and greasy, rancid and foul-smelling material may be expressed out[35] of it. The

common site of involvement is anterior weight-bearing area of sole of the foot. When tumour grows, it locally invades and plantar fascia may get affected. When it is advancing toward the dorsal surface of the foot it may destroy metatarsal bones [36].

Histopathology[39,40]:

Large deep biopsy is essential for diagnosing verrucous carcinoma. There will be hyperkeratosis, parakeratosis, acanthosis, well differentiated keratinocyte with a small nucleus. The tumor invades with broad strands and contains keratin filled cyst in their centre, large ,bulbous downward proliferation that compress the collagen bundles and push them aside .Thus the tumor has bulldozing rather than stabling effect. In the deeper portions, nuclear atypia , individual cell keratinisation &horn pearls are absent.

Verrucous skin lesions of neuropathy [68, 69]

Verrucous skin lesions of neuropathy is most commonly associated with diabetes mellitus. In diabetic patients, it may present as verrucous skin lesions on the feet and skin ulcers simultaneously or either ulcer or neuropathies preceed. These associations may be due to neuropathy and diabetic ulcers are interrelated in their aetiology and pathogenesis. Multiple treatment modalities with foot care are necessary for this condition.

ELEPHANTIASIS NOSTROS VERRUCOSA CUTIS :

Nostros means 'of our region'. Synonyms are mossy leg, Lymphangitis recurrens, Elephantogenica, Elephantiasis nostras. It is a complication of

chronic lymphedema. It is due to lymphatic obstruction caused by recurrent bacterial infection. [71]

In 1969, Castellani [70] classified elephantiasis into four subtypes:

1. Elephantiasis tropica - due to filariasis
2. Elephantiasis nostras - due to bacterial infection
3. Elephantiasis symptomatica - due to mycotic, syphilitic, tuberculoid, neoplastic, or traumatic causes of lymphatic obstruction.
4. Elephantiasis congenita - inherited disorders such as Milroy's disease. Bacterial infection, lymphangioma, malignancy, lymphatic fibrosis and prior surgery or trauma, radiation therapy, chronic venous stasis, scleroderma and obesity can lead to lymphatic obstruction and edema. Lymphatic vessels are an important pathway for immune cell trafficking and antigen delivery[72,73]. The protein-rich interstitial fluid of lymphedema leads to inflammation and an accumulation of fibroblasts, adipocytes and keratinocytes that transform the initially soft swollen tissue into a hard fibrotic tissue with stiff, thickened skin.[74,75] Protein rich fluid induces fibroblast proliferation and increases susceptibility to infection and inflammation. Because of this ongoing process, further fibrosis of the dermis and lymph channels can occur. In chronic venous stasis, activated leukocytes may migrate out of the vasculature and release TGF-β1, stimulating collagen production by dermal fibroblasts, which culminates in dermal and lymphatic fibrosis[76, 77, 78, 79]. Complications of lymphedema

include impairment of limb function, recurrent cellulitis which further aggravates the lymphedema and accelerate skin changes. Rarely malignancies such as lymphangiosarcoma (Stewart-Treves syndrome), melanoma , squamous cell carcinoma, Kaposi's sarcoma and lymphoma.[80-82]

Clinically it presents as fatigue and heaviness in the affected limb. The edema increases towards the evening and relieved by bed rest. Early edema is soft and pitting. But later become indurated and non-pitting. The skin over the affected leg becomes thick and hyperkeratotic with pebbled appearance of the skin surface. Squaring of the toes with difficulty in pinching the skin over the second toe known as Kaposi -Stemmer's sign is positive . Investigations like Lymphoscintigraphy (isotope lymphography), Magnetic resonance imaging (MRI), X-ray contrast lymphography are useful.

Histopathologically, pseudoepitheliomatous hyperplasia, dilated lymphatic spaces, and fibrosis are characteristic of ENV. These findings may not be present always during the advanced stage [83].

Hypertrophic Discoid lupus erythematosus [111] :

Hypertrophic Discoid lupus erythematosus is a variant of chronic cutaneous lupus erythematosus. It represents 2% of chronic cutaneous lupus erythematosus. These lesions mainly occur over the face, upper back, limbs, palms and sole. Two types of hypertrophic DLE are papulo nodular and hyperkeratotic type. Clinically, there will be hyperkeratotic scaly

verrucous plaque with adherent scales. These patients usually have classical DLE lesions elsewhere in the body.[112]

Histologically, [113] two patterns are recognised. In the first pattern, hyperplastic papillomatous epidermis with hyperkeratotic scale with dyskeratotic keratinocytes are seen. Thickened basement membrane zone is seen in old lesions. Band like mononuclear infiltration is seen along the dermoepidermal junction which mimicks like lichen planus. Second pattern consists of deep dermal perivascular, periappendageal and interstitial infiltrate with mucin deposition.

POROKERATOSIS:

Porokeratosis was first described by Vittorio Mibelli in 1893. Depending on clinical criteria five clinical variants are recognized: classic porokeratosis of Mibelli, disseminated superficial (actinic and non actinic type) PK, linear PK ,punctuate PK, porokeratosis Palmaris et plantaris disseminata. Rare morphological forms like facial PK, giant PK, hypertrophic verrucous porokeratosis, reticulate PK are also reported. [108] Except the punctate type, all other types are characterized by a keratotic ridge .This feature histologically correlates the presence of cornoid lamella.[109,110] In the disseminated superficial form, the lesions are smaller, more in number, with minimally raised, less hyperkeratotic border is seen.

LUPUS VULGARIS:

Lupus vulgaris is a paucibacillary form of cutaneous tuberculosis which has chronic and progressive course. It is seen in patient with moderate to high degree of immunity. The source of lupus vulgaris is contiguous, haematogenous or lymphatic spread.[98] In European countries face, especially around the nose is commonly involved. In developing countries common sites are lower limbs and buttocks[99], especially in children[100]. It starts as a small, reddish-brown, soft flat plaque with gelatinous consistency. These lesion extends by slow peripheral extension with areas of atrophy. Apple jelly nodules are seen on diascopy. Usually it manifests as a single lesion except in disseminated forms. Sporotrichoid-like spread can occur[102]. Five clinical forms are known depending on the local tissue response to the infection. Those types are tumour-like forms, vegetating form, ulcerative and mutilating form ,plaque form, papular and nodular forms. Histopathologically [97], the epidermis shows atrophy and ulceration. Hyperplastic lesions shows hyperkeratosis, acanthosis, papillomatosis and pseudoepitheliomatous hyperplasia. Tuberculoid granuloma with epithelioid giant cells are seen in the upper dermis. Caseation necrosis within the tubercle is slight or absent. Extensive fibrosis occurs during the healing process. Bacilli are seen very rarely.

Bromodermas and iododermas:

Bromoderma is caused by anticonvulsant therapy with potassium bromide [124]. The Skin lesions are multiple nodules and plaques with verrucous surface seen commonly on limbs and face. Histopathology [123] shows verrucous pseudoepitheliomatous hyperplasia with abscesses containing neutrophils and eosinophils in the epidermis, dense dermal infiltrate initially consists mainly of neutrophils and eosinophils and later lymphocytes, plasma cells and histiocytes. The abundant dilated blood vessels may show endothelial proliferation.

Iododermas are caused by oral intake(cough syrup) ,intravenous (radiographic contrast) and after throidectomy. The skin lesions are urticaria, papulopustules, bulla and vegetating masses. Histopathology shows marked ulceration but there is usually less epithelial hyperplasia. Both conditions must be differentiated from blastomycosis and coccidioidomycosis, and pemphigus vegetans. [124]

Kaposi's sarcoma (Granuloma multiplex haemorrhagicum) [125,126,127]

Kaposi's sarcoma is a multifocal, endothelial proliferative disorder predominantly involving the skin and other organs and associated with formation of vascular channels and proliferation of spindle-shaped cells.

Etiology:

It is causd by Human herpes virus-8 and in HIV positive patients the virus itself causes the disease. It acts by trans-activating protein(Tat-1)

which promotes the development of the lesions by activating cytokines and angiogenic factors.

Types :

1. Classic
2. Endemic
3. Iatrogenic
4. HIV associated

Classic type - It is found mainly in elderly males, particularly in Jews of Eastern European origin. The lesions begin around the ankle and slowly spread up the leg. It begins as blue red macule, evolve into a plaque/nodule later ulcerate. It has a chronic course. Mucosal and visceral involvement is rare.

Endemic type – It is endemic in Africa.It has two types.

1. Benign nodular variety which is similar to classic type.
2. Fulminant lymphadenopathic type which is fatal in 2-3 years.

Iatrogenic type - It is seen in renal transplant patients. The lesions are chronic but aggressive than the classical type.

HIV associated type-It is common in homosexual men, especially in oro-anal intercourse. It may appear at any stage of HIV infection irrespective of CD4 count. The skin lesions occur along the cleavage lines in face and trunk. Mucosal and visceral involvement (GIT/LUNG) is common.

Histopathology:

In the patch stage:

There is a proliferation of jagged, irregular, lymphatic-like vascular channels lined by a single layer of bland endothelial cells. These channels particularly surround the pre-existing blood vessels and adnexal structures. These structures are seem to be floating within the newly formed channels, the so-called "promontory sign". There is prominent mononuclear inflammatory cell infiltrate in which plasma cells are seen.

In the nodular stage:

Well-circumscribed nodules of generally bland, spindle-shaped cells forming cleft like spaces that impart a typical sieve-like appearance. Extravasated red blood cells are plenty. Mitotic figures are common. The periphery of the nodules gives a angiomatous appearance. The lesions of Kaposi's sarcoma that have regressed after chemotherapy or with angiogenesis inhibitors, including Col-3 or HAART, may be very difficult to interpret.

In completely regressed lesions, there is absence of irregular lymphatic-like channels and spindle-shaped cells, increase in the number of capillaries in the superficial dermis and a superficial, perivascular lymphohistiocytic inflamatory cell infiltrate with variable numbers of siderophages. Partially regressed lesions show prominent decrease in the number of lymphatic-like vascular channels and spindle-shaped cells and focal inflammation with siderophages. The residual spindle-shaped cells tend to be arranged around superficial and middermal capillaries.

The Histopathological differential diagnosis are spindle cell haemangioma, tufted angioma, microvenular haemangioma, hobnail haemangioma, progressive lymphangioma, venous dermatitis (acroangiodermatitis). Angiosarcoma is also often considered in the differential diagnosis, but in the latter there is clear evidence of cytological atypia, mitotic activity and multi layering.

CHROMOBLASTOMYCOSIS :

Chromoblastomycosis is the chronic fungal infection which is characterized by slow growing exophytic lesions, seen commonly on the feet and legs[103]. Chromoblastomycosis is caused most commonly by Phialophora verrucosa, F. Compacta, Fonsecaea pedrosoi and Cladophialophora carrionii (carrionii) Rhinocladiellaaquaspersa.[104] Feet, legs, arms, face and neck are sites which are commonly involved. It starts as warty painless papule slowly enlarges to form a verrucous plaque. Secondary ulceration may occur. Lymphatic spread occurs to adjacent areas. Rarely, Haematogenous spread can occur. Squamous cell carcinoma as has been reported in chronic cases[105, 106].

Histology[107] :

Marked pseudoepitheliomatous hyperplasia with transepidermal elimination of fungal cells are seen. Foreign-body granuloma with isolated areas of microabscess formation and muriform or sclerotic cells are the important features of chromoblastomycosis.

Blastomycosis:

Blastomycosis is a chronic suppurative and granulomatous mycosis caused by Blastomyces Dermatitidis. It mainly affects the lungs. During dissemination it affects skin, central nervous system, bones and other sites. Three types of blastomycosis like primary cutaneous, pulmonary and disseminated form are reported. Primary cutaneous form which is very rare. Following trauma, entry of organism leads to an erythematous, indurated plaque appears within 1–2 weeks. This is associated with lymphangitis and lymphadenopathy. Histologically, B.dermatitidis produces broad based budding yeast in tissues. In disseminated forms, pseudoepitheliomatous hyperplasia with intra and subepidermal polymorphonuclear abscesses with granulomatous infiltrate are found in the dermis.

Seborrhoeic keratosis [114]:

Seborrhoeic keratosis is a benign tumour, which is mainly composed of epidermal keratinocytes occurs commonly in elderly individuals. It is usually occurs in fifth decade of life. This lesions are common in tropical countries. It manifests as superficial verrucous plaque which appear to stuck to the epidermis. Histopathologically, hyperkeratosis, acanthosis, marked papillomatosis which gives church spire pattern with melanocyte proliferation in the immature keratinocytes. [115]

Incontinentia pigmenti:

Incontinentia pigmenti is a X-linked dominant hereditary syndrome disorder in which vesicular, verrucous and pigmented cutaneous lesions are

associated with developmental defects of the eye, teeth and central nervous system.[116, 117] Early inflammatory changes can occur even at birth and do not progress after birth. Tense bulla will be arranged in the linear fashion is the most striking feature of incontinentia pigmenti.

Clinical stages are [118]

Stage 1: inflammatory macules, papules, vesicles and pustules

Stage 2: hyperkeratotic and verrucous lesions

Stage 3: grey - brown pigmentation

Stage 4: atrophic, hypopigmented and depigmented bands or streaks that are hairless and anhidrotic and fail to tan on sun exposure

Darier's disease [119]

Darier's disease manifest as firm, brown , rough papule over the Seborrhoeic areas of the trunk and face especially over the the scalp margins, temples, ears and scalp.

In lower legs and arms multiple discrete papules coalesced to form warty plaques with fissures or papillomatous masses. It forms a vegetating and malodorous mass especially in the flexures, anogenital region, the groins and the natal cleft.

Acrokeratosis verruciformis :

Acrokeratosis verruciformis of Hopf [120] is characterized by multiple flesh coloured or lightly pigmented, warty papules on the dorsa of the hands and feet and other sites.

Stucco keratosis

Stucco keratosis is a variant of Seborrhoeic keratosis in which whitish, keratotic plaques that can be removed easily with a fingernail without bleeding. It is seen commonly on extremities, especially around the ankle region.

Verrucous epidermal naevus [122]

Verrucous epidermal naevus is a congenital, non inflammatory cutaneous hamartoma composed of keratinocytes .Two types are recognised.

1. Epidermolytic verrucous epidermal naevus:

   Pigmented linear verrocous papules or plaques seen in young children.

2. Non-epidermolytic verrucous epidermal naevus :

   Usually seen at birth but may appear in later childhood.

AIM OF THE STUDY

To study the clinical and histopathological patterns of various verrucous skin lesions of lower limb and to find out the etiology of these verrucous skin lesions.

MATERIALS AND METHODS

A study was conducted during the period from 1 August 2011 to 31st July 2012 in our outpatient department, department of Dermatology Coimbatore Medical College Hospital, among the patients attending the Dermatology Department as well as those referred from other departments.

Type of study : Cross sectional study.

Inclusion criteria :

1. All the patients with verrucous skin lesions of lower limbs of all age groups were included.

Exclusion criteria:

1. Those patients who are not willing for the study.
2. Pregnant women.
3. Patient with bleeding disorders.

The study was explained to eligible participants. Informed consent was obtained. A detailed history was recorded regarding complaints, duration, evolution of skin lesions, associated local and systemic complaints. Past history of tuberculosis, leprosy, diabetes mellitus, injury, prior drug intake, and history suggestive of malignancy were taken into account.

Clinical examination:

Clinical examination included morphology of the skin lesions, number of lesions and their size, distribution of lesions, symmetry, tenderness, ulceration and secondary infections. Other areas of skin, mucous membrane, palms and soles, hair and nail were examined. A detailed general and systemic examination was done.

Investigations:

1. Routine laboratory investigations included complete haemogram, urine routine, renal function test and liver function test.
2. Slit skin smear for acid fast bacilli was done for the suspected patients of Hansen's disease.
3. Screening for HIV, Hepatitis B, VDRL were done for high risk patients with history of sexual exposure or occupational exposure to blood and blood products.
4. Mantoux test, X ray chest, ultrasonogram of abdomen were done in relevant cases.
5. Lower limb Doppler was done in patients with phlobolymphoedema and Elephantiasis nostros verrucosa cutis.
6. Nerve Conduction Study was done in patients with sensory or motor neuropathy.
7. Biopsy was taken from representative skin lesions. Special stains for AFB

and fungus were done whenever required.

Analysis:

A descriptive analysis of clinical characteristics, laboratory parameters and histopathological features of various verrucous skin lesions was done. These data were analysed and compared with published literature.

OBSERVATION AND RESULTS

Among the one hundred and five patients with verrucous skin lesions over the lower limbs who attended our outpatient department from August 2011 to July 2012, five patients were excluded from the study due to denial for the study(3) and refusal for biopsy(2).

Hundred patients with clinical features of verrucous skin lesions over the lower limbs were included in the study. The common causes of verrucous lesions over the lowerlimbs were hypertrophic lichen planus (29), wart (10),lichen simplex chronicus(10), psoriasis (7) and lichen amyloidosis(7). The less common etiologies were porokeratosis(2), hypertrophic discoid lupus erythematosus (2), callosity (2) and Elephantiasis nostros verrucosa cutis(1).

Table 1

Clinical spectrum of verrucous skin lesions of lower limbs

Disease	No.of patients	Percentage
Hypertrophic lichen planus	29	29
Lichen simplex chronicus	10	10
Wart	**10**	**10**
Psoriasis	**7**	**7**
Lichen amyloidosis	**7**	**7**
Phlebolymphoedema with verrucous skin lesions	7	**7**
Leprosy with hyperkeratotic and verrucous skin lesion on lower extremities	**6**	**6**
Prurigo nodularis	**5**	**5**
Tuberculosis verrucosa cutis	**5**	**5**
Verrucous carcinoma	**4**	**4**
verrucous skin lesions of neuropathy	**3**	**3**
Others	**7**	**7**
Total	**100**	**100**

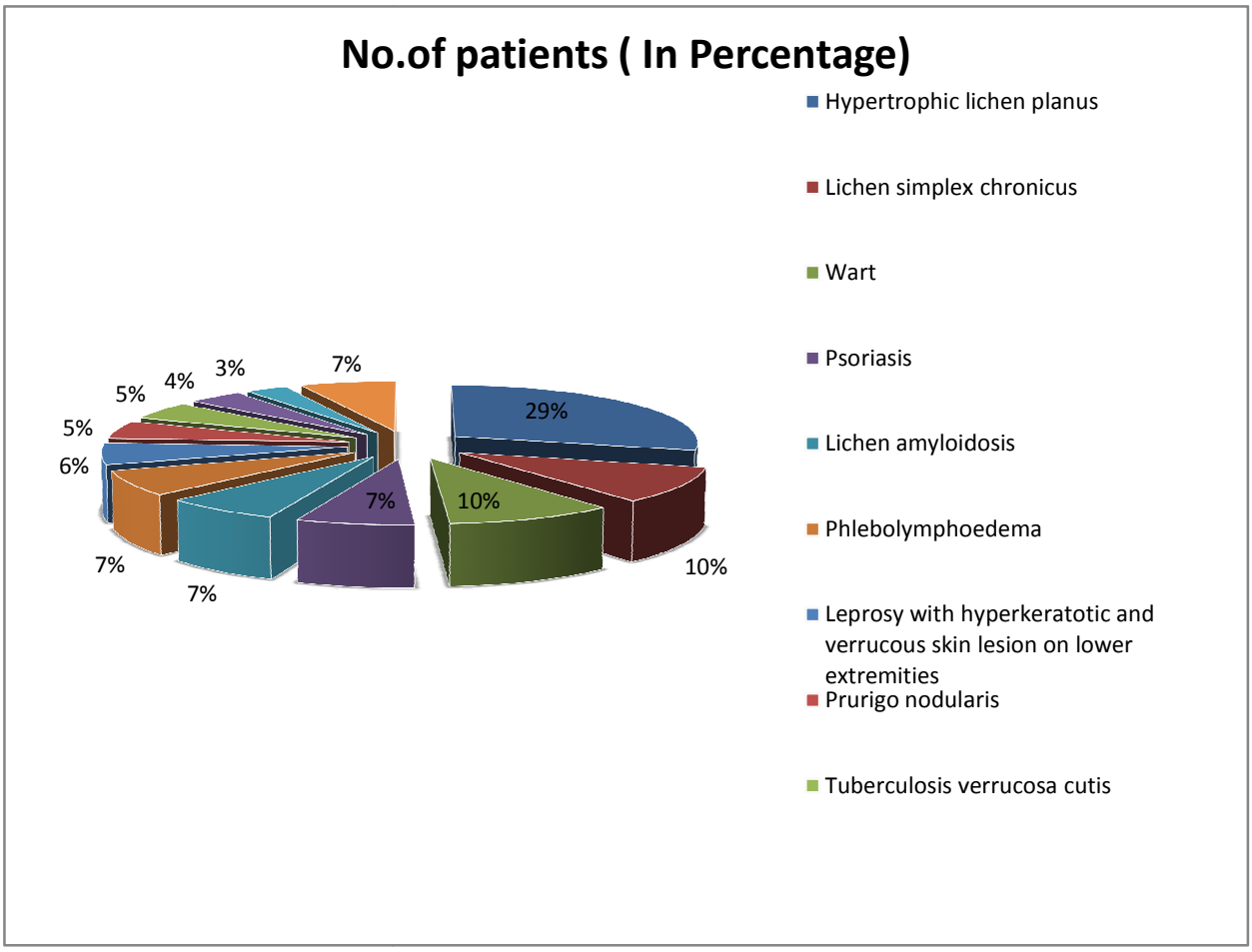
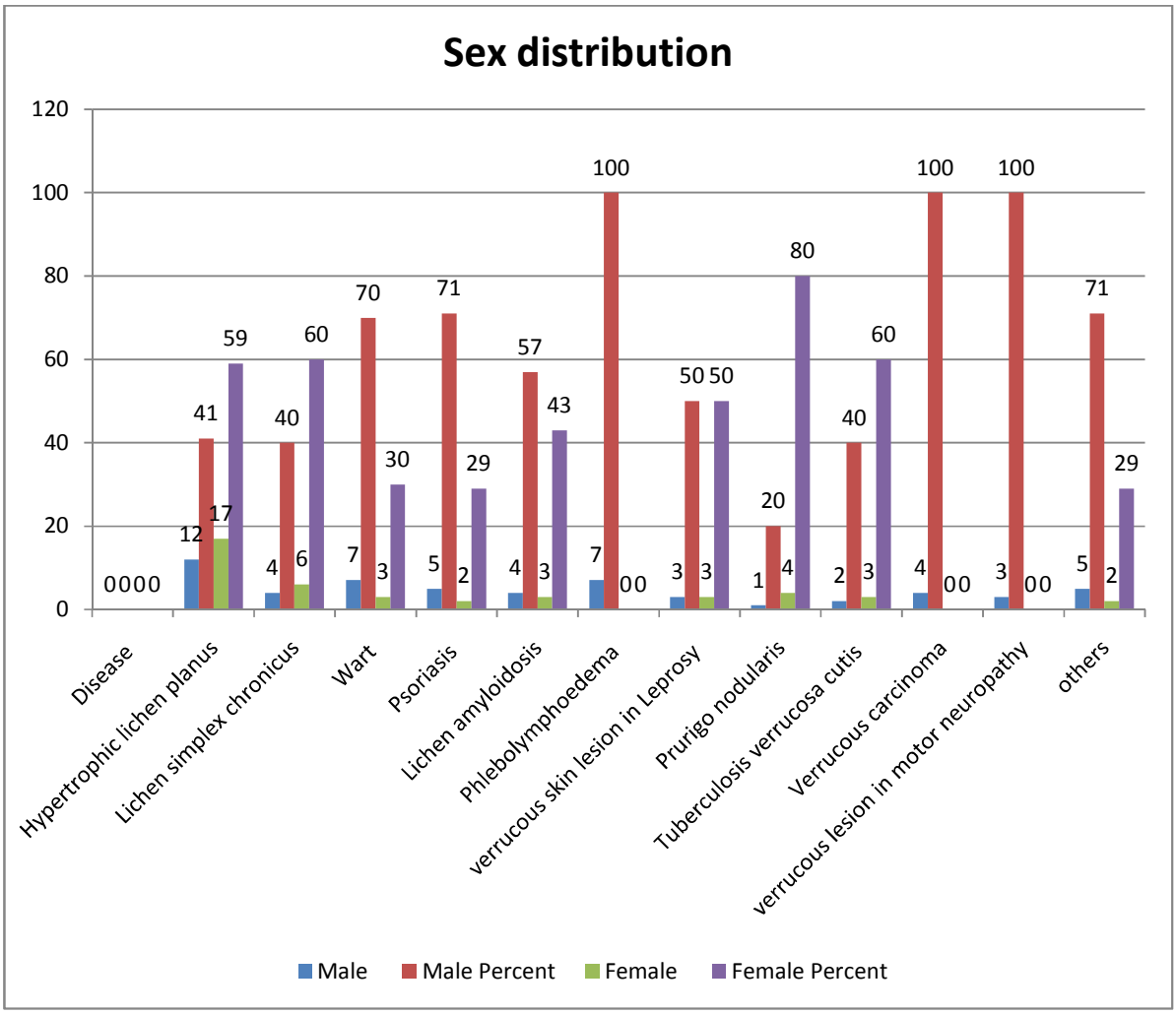

No.of patients (In Percentage)
29%
10%
10%
7%
7%
7%
6%
5%
5%
4%
3%
7%
Hypertrophic lichen planus
Lichen simplex chronicus
Wart
Psoriasis
Lichen amyloidosis
Phlebolymphoedema
Leprosy with hyperkeratotic and verrucous skin lesion on lower extremities
Prurigo nodularis
Tuberculosis verrucosa cutis

Table 2

Sex distribution

	Male		Female	
Disease	No.	%	No.	%
Hypertrophic lichen planus	12	41	17	59
Lichen simplex chronicus	4	40	6	60
Wart	7	70	3	30
Psoriasis	5	71	2	29
Lichen amyloidosis	4	57	3	43
Phlebolymphoedema with verrucous skin lesions	7	100	-	-
Verrucous skin lesion in Leprosy	3	50	3	50
Prurigo nodularis	1	20	4	80
Tuberculosis verrucosa cutis	2	40	3	60
Verrucous carcinoma	4	100	-	-
verrucous skin lesions of neuropathy	3	100	-	-
Others	5	71	2	29
TOTAL	57	57	43	43

In our study, 57 patients were male while 43 were female. Male to Female ratio was 1.3:1(57% &43%).


Sex distribution
120
100
80
60
40
20
0
0000
12
41
17
59
4
40
6
60
7
70
3
30
5
71
2
29
4
57
3
43
7
100
00
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50
3
50
1
20
4
80
2
40
3
60
4
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3
100
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5
71
2
29
Disease
Hypertrophic lichen planus
Lichen simplex chronicus
Wart
Psoriasis
Lichen amyloidosis
Phlebolymphoedema
verrucous skin lesion in Leprosy
Prurigo nodularis
Tuberculosis verrucosa cutis
Verrucous carcinoma
verrucous lesion in motor neuropathy
others
Male
Male Percent
Female
Female Percent

Table -3

Age distribution

Age in years	<10	10-20	21-30	31-40	41-50	51-60	>60	Total
Hypertrophic lichen planus	-	3	4	3	6	7	6	**29**
Lichen simplex chronicus					3	2	5	**10**
Wart	-	5	1	1	2	-	1	**10**
Psoriasis	-	-	-	2	2	2	1	**7**
Lichen amyloidosis			1	3	1	1	1	**7**
Phlebolymphoedema with verrucous skin lesion				1	2	2	2	**7**
Leprosy with hyperkeratotic and verrucous skin lesion on lower extremities					3	2	1	**6**
Prurigo nodularis			2	1	-	1	1	**5**
TBVC		2	1	-	1	1	-	**5**
Verrucous carcinoma					1	2	1	**4**
Verrucous skin lesions of neuropathy					1		2	**3**
others				2	2	1	2	**7**
Total		**10**	**9**	**13**	**24**	**21**	**23**	**100**

Majority of the patients were between the age of 41-80 yrs.

Mean age of presentation was 48.95 years.

Out of hundred cases, 86 patients had non infective etiology of verrucous skin lesions. 14 patients had infectious etiology of verrucous lesions.

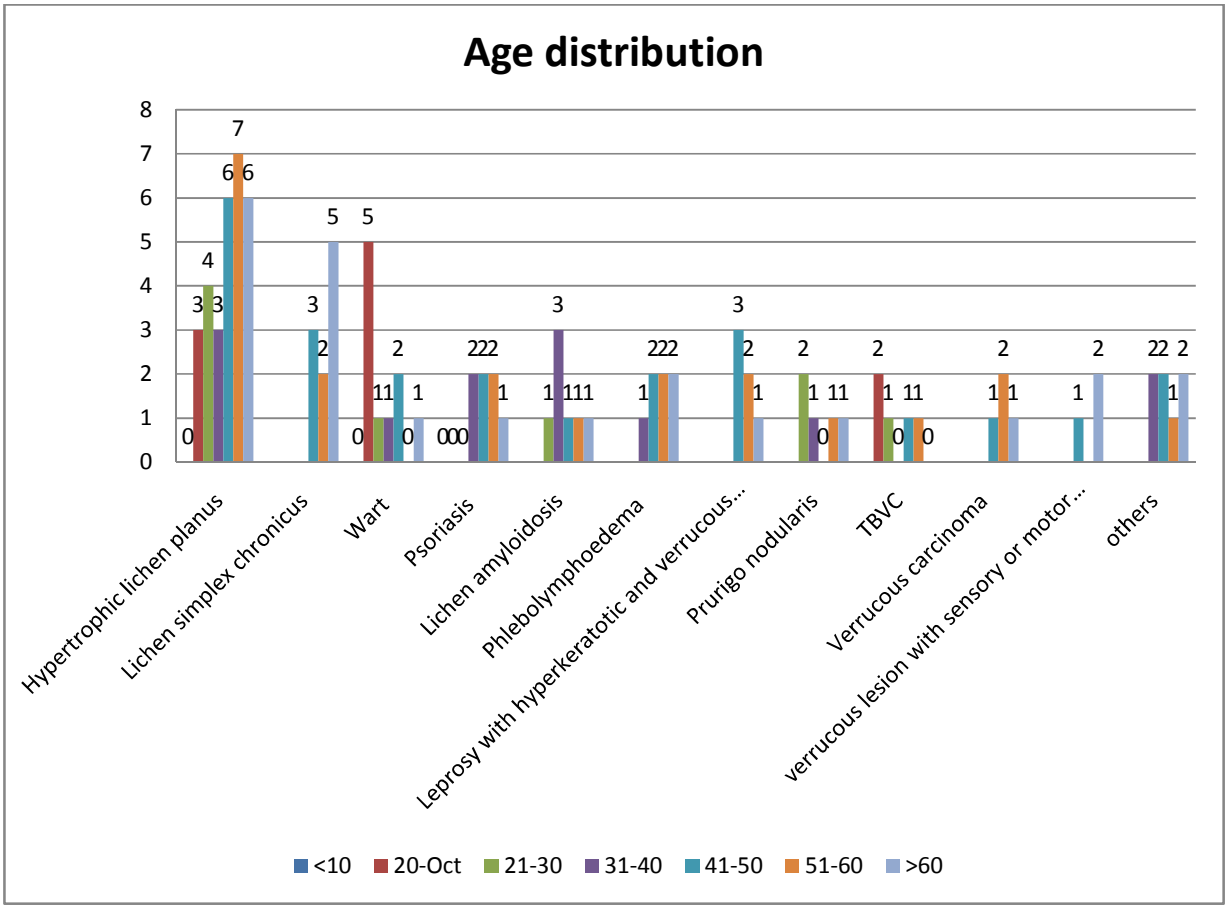

Table -4

Distribution of cases - Verrucous skin lesions of infective etiology

Infective etiology	No.of cases	Percentage
Wart	10	71
TBVC	4	29

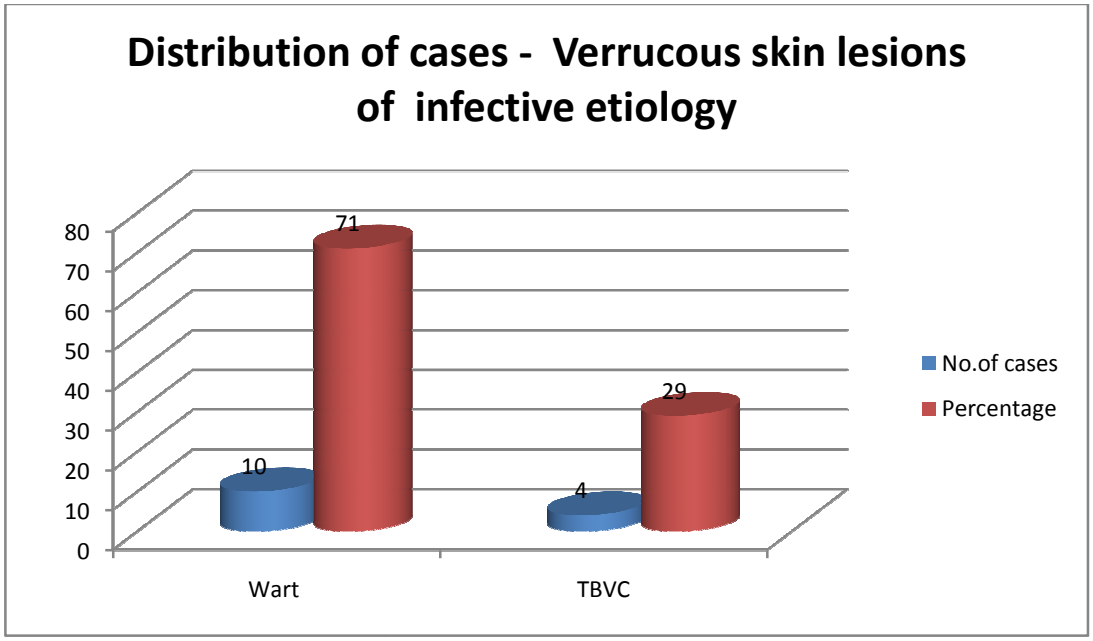

Table -5

Distribution of cases- Verrucous skin lesions of non infective etiology

Non infective etiology	**No.of cases**	**Percentage**
Hypertrophic lichen planus	29	34
Lichen simplex chronicus	10	11.6
Psoriasis	7	8.1
Lichen amyloidosis	7	8.1
Phlebolymphoedema with verrucous skin lesions	7	8.1
Leprosy with hyperkeratotic and verrucous skin lesion on lower extremities	6	6.9
Prurigo nodularis	5	5.8
Verrucous carcinoma	4	4.7
verrucous skin lesions of neuropathy	3	3.5
others	7	8.1

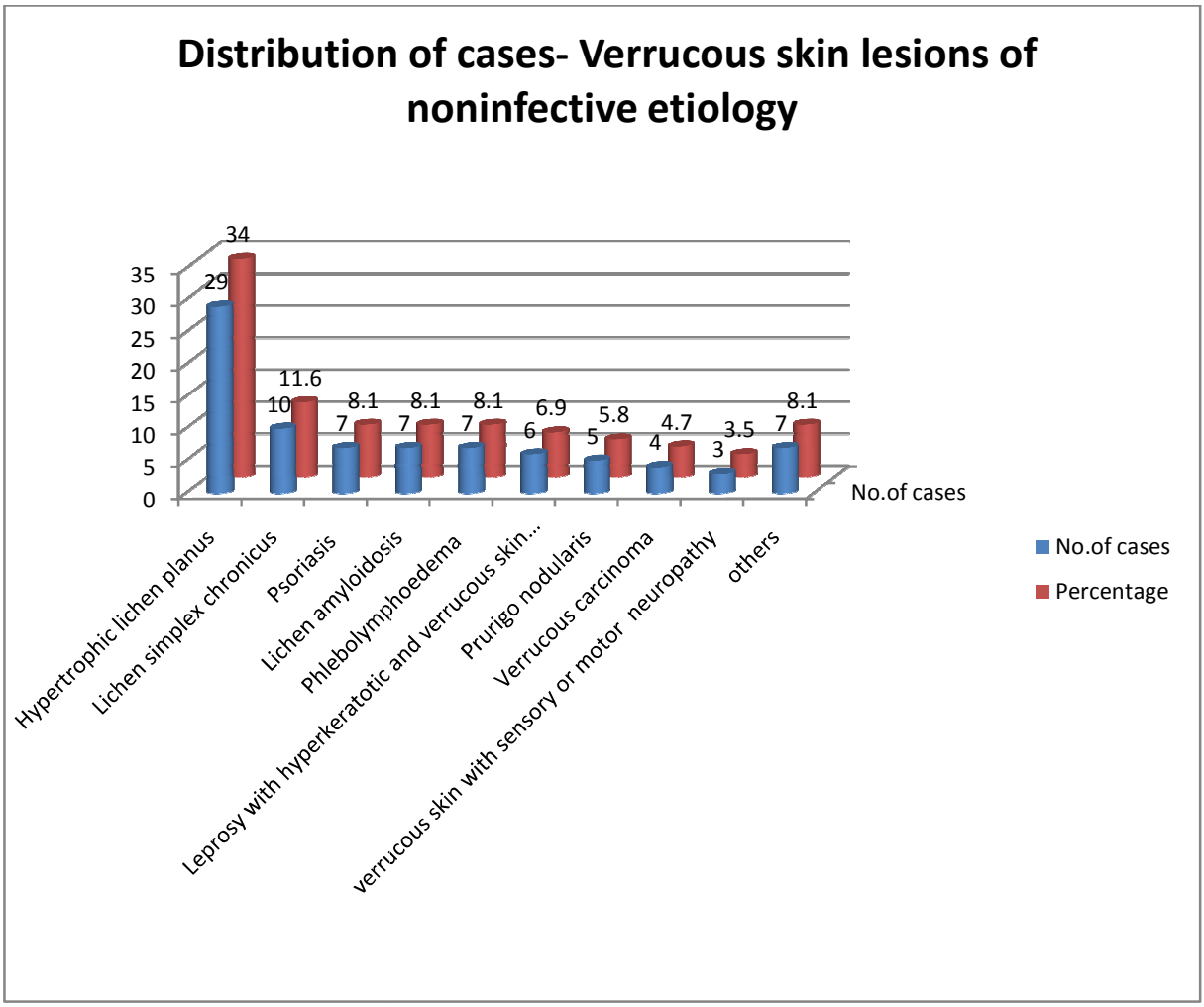
Distribution of cases- Verrucous skin lesions of noninfective etiology
35
30
25
20
15
10
5
0
34
29
11.6
10
8.1
7
8.1
7
8.1
7
6.9
6
5.8
5
4.7
4
3.5
3
8.1
7
No.of cases
Hypertrophic lichen planus
Lichen simplex chronicus
Psoriasis
Lichen amyloidosis
Phlebolymphoedema
Leprosy with hyperkeratotic and verrucous skin...
Prurigo nodularis
Verrucous carcinoma
verrucous skin with sensory or motor neuropathy
others
No.of cases
Percentage

Table- 6

Papulosquamous disorders with verrucous skin lesions

Distribution of cases

Papulosquamous disorder	**No.of cases**	**Percentage**
Hypertrophic LP	29	81
Psoriasis	7	19

Table -7

Rare verrucous skin lesions –distribution of cases

Rare verrucous skin lesions	**No.of cases**	**Percentage**
Porokeratosis	2	28.57
Callosity	2	28.57
Hypertrophic DLE	2	28.57
ENVC	1	14.28

DESCRIPTION OF CASES AND FINDINGS:

Hypertrophic Lichen planus

There were 29 cases of hypertrophic lichen planus.

Clinical profile:

The age group of patients presented with features of hypertrophic lichen planus was between 16 and 85 years. The male to female ratio was 1:1.4. The mean duration of the skin lesions was 1.9years. All the patients had hyperpigmented verrucous lesions over the lower limbs. Twenty five (86%) patients had hyperpigmented lesions with depigmentation in the centre of the lesions. Three (10%) patients had associated nail involvement. Five (17%) patients had associated mucous membrane involvement. Two (6.8%) patients had secondary infection. One (3.4%) patient had ulceration of the lesion. Two (6.8%) Patients had associated venous stasis.

Histopathology:

Among the 29 patients, twenty six (90%) showed the features of hyperkeratosis, pseudoepitheliomatous hyperplasia with irregular acanthosis, wedge shaped hypergranulosis, discrete interface vacuolar changes and pigmentary incontinence were seen. Dermal fibrosis was seen in two patients.

Table -8

Hypertrophic lichen planus clinical features

Laboratory investigations and histopathology

Age	No.of patients	DSL yrs	Asso.nail invol	Asso.mucous mem invol	Asso.inf	Asso. ulcer	Asso.venous stasis	H/E PEH	H/Edermal fibrosis
<10	-								
10-20	3	1.5						2	
21-30	4	2	1	1				4	
31-40	3	1.2		2			1	2	
41-50	6	2.1	1	1				6	
51-60	7	2.4	1	1	1	1	1	8	1
>60	6	2.2			1			8	1

Key-DSL=duration of skin lesions, Asso.inf –associated infection, Asso.ulcer- Associated ulcer, PEH-pseudoepitheliomatous hyperplasia, Asso.nail invol- associated nail involvement, H/E –hematoxylin and eosin stain.

Lichen simplex chronicus (LSC)

There were 10 patients of LSC with verrucous morphology.

Clinical profile:

The age group of patients with LSC was between 41 and 70 years the male to female ratio is 1:1.5.The mean duration of illness was 2.5 years.Six (60%) patients had verrucous hyperpigmented plaques around the ankle. Four (40%) patients had lesions over the lateral aspect of legs. Two (20%) patients had secondary infections. One (10%) patient had associated depression.

Histopathology :

Among the ten patients of lichen simplex chronicus, eight (80%) patients showed hyperplasia of all the components of epidermis. Vertically oriented collagen bundles and increased fibroblasts was seen in seven (70%) patients. Two (20%) patients showed mild spongiosis.

Table -9

Lichen simplex chronicus-clinical features

Laboratory investigations and histopathology

Age	**No.of patients**	**DSL years**	**Asso.disease**	**Asso.inf**	**H/E EH**	**Vertically oriented collagen**
<10						
10-20						
21-30						
31-40						
41-50	3	1.5		1	3	2
51-60	2	2.6			2	2
>60	5	3.4	1		3	3

Key-DSL=duration of skin lesions, asso.inf –associated infection, asso. Disease-associated disease, EH-epidermal hyperplasia, H/E-Hematoxylin and eosin stain.

Viral Wart:

There was 10 patients with viral wart .

Clinical profile:

The age group of the patients with features of wart was between 12 and 61 years. The mean duration of illness was 9 months. Seven (70%) patients had plantar warts. Two (20%) patients had common wart over the legs and knees. One (10%) patients had single hyperpigmented verrucous nodule over lateral aspect of right leg. This lesion was excised and confirmed with biopsy. Two (20%) patients had associated lesions over the upper limbs and face.

Table- 11
Viral wart – Clinical features

Age	No.of patients	DSL months	plantar	Common wart
<10				
10-20	5	12	4	2
21-30	1	8		1
31-40	1	9	1	1
41-50	2	7	1	1
51-60				
>60	1	9	1	

Key-DSL=duration of skin lesions

Histopathology:

All the patients (100%) showed the features of hyperkeratosis, vertical tiers of parakeratosis, papillomatosis, incurving of rete ridges, the foci of koilocytes are located at Stratum Malphigii.

Psoriasis:

7 patients had psoriasis with verrucous morphology.

Clinical profile:

The age group of patients presented with the features suggestive of Psoriasis with verrucous morphology was between 33 years and 71 years. The male to female ratio was 2.5:1. The mean duration of illness was 2.5 years. Five (71%) patients had associated lesions over the trunk and upper limbs. Two (28%) patients had palmoplantar psoriasis. These patients had hyper pigmented verrucous plaque over the lower limbs, which mimicked hypertrophic lichen planus. We confirmed the diagnosis with biopsy.

Histopathological findings:

Specimens of all the patients showed hyperkeratosis, parakeratosis, acanthosis, regular elongation of rete ridges, papillomatosis, suprapappilary thinning, dilated capillaries. 1(14%) specimen showed Munro micro abcess.

Lichen amyloidosis :

There were 7(7%) patient with lichen amyloidosis showing verrucous morphology.

Clinical profile:

The mean age of presentation was 57.5 years. Male to female ratio was 1.3:1. The mean duration of illness was 3.2 years. Hyperpigmented, bilaterally symmetrical discrete, verrucous papules coalesced to form verrucous plaques were seen over both anterior aspect of legs in six (85%) patients. One (14%) patient had unilateral lesion over the right leg.

Histopathology :

All (100%) specimens showed the features of hyperkeratotic, hyperplastic epidermis, rounded dermal papillae and homogenous deposits of amyloid filled up the entire dermal papillae with pigmentary incontinence seen in all patients.

Phlebolymphoedema:

There were 7 cases of phlebolymphedema.

Clinical profile:

The age group of patients presented with features suggestive Phlebolymphoedema was between 40 and 70 years. The mean duration of illness was 5 years. All patients were males. Four (57%) patients had bilateral leg oedema and limb hypertrophy. Two (28%) patients had bilateral verrucous plaques over the second toe and third toe. Two (28%) patients had ulcer over the medial malleolus with verrucous plaque surrounding the ulcer. Venous Doppler was done for all patients.

Findings in venous Doppler:

Five (71%) patients showed incompetence of saphenofemoral system with extensive lymphedema. One (14%) patient had saphenopopliteal incompetence. One (14%) patient had isolated perforator incompetence.

Prurigo nodularis :

Five (5%) patients had prurigo nodularis.

Clinical profile:

The age group of the patients showing prurigo nodularis was between 20 and 72 years. The mean duration of skin lesions was 1.4 years. The male to female ratio was 1:4. Four patients (80%) had hyperpigmented verrucous nodules over legs and dorsum of foot. One (20%) patient had depigmented

nodules of right leg with depigmented plaque around the ankle. One (20%) patient had secondary infection.

Histopathology :

All the patients (100%) showed the features of hyperkeratosis, acanthosis, pseudoepitheliomatous hyperplasia, vertically oriented collagen bundles.

Tuberculosis verrucosa cutis (TBVC):

There were 5 patients presented with features suggestive of TBVC.

Clinical profile:

The age group of the patients showing TBVC was between 10 and 62 years. The mean duration of skin lesions was 1.5 years. The male to female ratio was 1:1.5. One (20%) patient had verrucous plaque over the left foot extending from dorsum of foot to plantar aspect of the foot laterally. One (20%) patient had hyperpigmented verrucous plaque over the dorsum of foot. Three (60%) patients had verrucous plaque around the ankle joints. Out of these, One (20%) patient had pus discharge from the lesion.

Histopathology:

Hyperkeratosis, acanthosis, pseudoepitheliomatous hyperplasia, neutrophil microabcess in upper dermis, epitheloid granuloma in lower dermis.

Investigatory findings:

All the patients were positive for Mantoux. One (20%) patient had associated pulmonary tuberculosis.

Verrucous carcinoma:

In this study, 4 patients showed the features of verrucous carcinoma.

Clinical profile :

The age group of the patients showing verrucous carcinoma was between 51 and 65 years. The mean duration of skin lesions was 4.2years. All patients were males. One (25%) patient had cauliflower flower growth over the left foot over the dorsum and plantar aspect. One (25%) patient had verrucous growth over the right lateral malleolus and hyperkeratotic verrucous plaque over the left lateral malleolus. One (25%) patient had pedunculated verrucous growth just above the right knee. One (25%) patient had verrucous growth over left thigh. Out of these, two (50%) patients were old case of Hansen's disease and had chronic ulcer. Two (50%) patients needed multiple deep biopsy to arrive at the diagnosis. One (25%) patient had destruction of metatarsal bones.

Xray findings:

One patient had destruction of metatarsal bone.

Histopathology :

On histopathological examination hyperkeratosis, parakeratosis, acanthosis with keratin filled cyst. Well differentiated keratinocyte with a small nucleus with nuclear atypia with downward proliferation which compress the collagen bundles seen in all specimens.

Leprosy with hyperkeratotic and verrucous skin lesion on lower extremities:

Six (60%) patients had verrucous skin lesions associated with leprosy.

Clinical profile:

The age group of patients with hyperkeratotic and verrucous skin lesion on lower extremities was between 43 and 65 years. The mean duration of disease was 4 years. Three (50%) patients had verrucous and hyperkeratotic lesion over anterior aspect of the ankle joints. Two (33%) patients had verrucous plaque around the trophic ulcer. One (16%) patient had verrucous plaque over the toes. One (16%) patient had associated foot drop. All patients completed their MDT.

Histopathology :

Compact hyperkeratosis, acanthosis with pseudoepitheliomatous hyperplasia and dermal infiltration were seen in all patients.

verrucous skin lesions with sensory or motor neuropathy:

Three (30%) patients had verrucous skin lesions with sensory or motor neuropathy.

Clinical profile:

The age group of patients with features of verrucous skin lesions with sensory or motor neuropathy was between 50 to 72 years. Mean duration of illness was 3.5 years. All patients were male. Two (66%) patients had depigmented verrucous plaque over right ankle joint. One (33%) patient had depigmented verrucous plaque over the anterior aspect of left leg. Out of these, one (33%) patient had diabetic neuropathy.

Nerve conduction study:

All the patient underwent nerve conduction study. Two (66%) had severe demyelinating sensory and motor neuropathy. One (33%) patient had pure sensory diabetic neuropathy.

Porokeratosis :

There were two (20%) patients with features suggestive of porokeratosis.

Clinical profile:

A 50 year old male patient presented with hyperkeratotic verrucous plaques with central crater over both upper and lower limbs of size varying

from 2x1cm to 3x2cm for 3 years. Another patient, 45year old female with hyperkeratotic depigmented verrucous plaque over the great toe extended to the anterior aspect of foot with peeling of skin. On histopathological examination, both the patients showed keratin filled invagination of epidermis with coronoid lamella in the center and absent granular layer beneath the coronoid lamella.

Hypertrophic Discoid lupus erythematosus:

There were two patients with features suggestive of Hypertrophic Discoid lupus erythematosus.

Clinical profile:

Two patients presented with the complaints of photosensitivity. A 39 year old male patient presented with hyperpigmented verrucous plaques both thighs and legs. Depigmented plaques with sorrounding hyperpigmentation. Another patient, 45 year old male patient with hyperpigmented verrucous plaque over the right leg with SCLE lesions over the face, upper trunk.

Histopathology:

Hyperkeratosis, acanthosis, papillomatous hyperplasia, basement membrane degeneration and deep dermal infiltration of lymphocytes perivascular, periappendageal infiltration with mucin deposition seen on histopathological examination.

Callosity:

There were two (2%) cases of callosity.

Clinical profile:

The mean age of presentation was 63 years. The duration of skin lesions was 5 years. Both the patients were males. Both of them had hyperpigmented verrucous plaque over both knees.

Histopathology:

Hyperkeratosis, acanthosis, papillomatosis with increased dermal collagen and fibrosis around the neurovascular bundles seen in both patients.

ELEPHANTIASIS NOSTROS VERRUCOSA CUTIS (ENVC)

One (10%) patient had the features suggestive of ENVC.

A 37 year old female patient had a hyperpigmented verrucous plaque involving all the toes of right foot of size 12x7cm for the past 3 years. Hyperkeratotic verrucous plaque present over the dorsum of right foot. Little toe was completely encircled by verrucous plaque**.**

Investigations

Biopsy showed the pappillomatosis, non specific chronic inflammation with fibrosis . Doppler showed the features chronic of lymphedema with normal saphenofemoral and saphenopopliteal valve.

DISCUSSION

The verrucous skin lesions of lower limbs are diverse group of skin disorders with different aetiology. These lesions are morphologically similar, characterised by jagged, undulating surface with papillomatosis on histopathology. Detailed history, histopathological examination and certain specific investigations are necessary to arrive at the diagnosis.

There are limited studies on verrucous skin lesions of lower limbs. We discussed and compared with the available resources.

In our study, the age group of patients presented with features of hypertrophic lichen planus was between 16 and 85 years. The male to female ratio was 1:1.4. The mean duration of the skin lesions was 1.9years. Two (6.8%) Patients had associated venous stasis. In a study by Dilip Kachhawa et al, a clinicopathological study on lichen planus [128], the most common age group with hypertrophic lichen planus was between 40 and 60 years of age. We also observed the same incidence in our study group. Another study by Bhattacharya M et al on Lichen planus, associated mucosal involvement was 16.8% and nail involvement was 15.1%. In our study 10% patients had associated nail involvement. 17% patients had associated mucous membrane involvement.

In our study, the age group of patients with LSC was between 41 and 70 years. The male to female ratio is 1:1.5.The mean duration of illness was 2.5 years. Six (60%) patients had verrucous hyperpigmented plaques around the ankle. Four (40%) patients had lesions over the lateral aspect of legs. One(10%) had associated depression. In a study by Julius L et al [130] incidence of Lichen Simplex Chronicus in Orientals and Caucasians was 3%. In a study by Thappa et al[131] on patterns of lower leg and foot eczema in south India, LSC was common between 30-50 years of age. The age group affected by LSC in our study was between 40-70 years.

The age group of the patients with features of wart in our study were between 12 and 61 years. The mean duration of illness was 9 months. Berth Jones and Hutchinson [133] in their study on 400 patients of warts, found 54% patients in the age group of 11–25 years. In our study, 10-20 years old patients were commonly affected with wart. These findings were comparable with our study. A study by Sudhakar Rao et al on clinical study of wart[132], plantar wart were commonest presentation of wart on lower limbs when compared to common wart. This is comparable with our study in which 70% patients had plantar warts and 20% patients had common wart over the legs and knees.

In our study, the age group of patients presented with the features suggestive of psoriasis was between 33 and 71 years. The male to female ratio was 2.5:1.On histopathological examination, we observed psoriasiform hyperplasia, acanthosis, papillomatosis with epidermal buttressing. In a study of verrucous psoriasis by Khalil FK et al[134] 50% patients had

verrucous lesions over the lower limbs with male to female ratio of 1.4:1 . The age group of the patients affected in his study was between 38 and 93 years with histopathological features comparable with our study.

In our study, age group of patients presented with features of Phlebolymphoedema with verrucous plaques over the lower limbs was between 40 and 70 years . According to Song Lu et al,[135] etiology of lower limbs lymphedema are cardiac , renal failure, protein losing conditions, lipedema, deep vein thrombosis, myxedema and chronic venous insufficiency.

T Salim et al [136] who made a study on lichen amyloidosis, shin was the common site of involvement in lichen amyloidosis with the history of using scrubs for more than 2 years. 63.3% were males and 36.7% females with a mean age of 43.13 years. We observed that male to female ratio was 1.3:1. The mean duration of illness was 3.2 years and site of involvement was anterior aspect of both legs. The mean age of the patients affected by this illness was 49.5

A study on prurigo nodularis by Nils Weigelt et al [137] showed the male to female ratio was 1:1, mean age was 58.38. In our study, the mean age of the patients showing prurigo nodularis was 36.4 years. The mean duration of skin lesions was 1.4 years. The male to female ratio was 1:4. Nils Weigelt et al [137], showed on histopathological examination there was thick compact orthohyperkeratosis and hairy palm sign pseudoepitheliomatous hyperplasia, focal parakeratosis, hypergranulosis, fibrosis of the papillary dermis with

vertically arranged collagen fibers. We observed the same histopathological features in our study.

According to Padmavathy et al on clinical, pathological and epidemiological study of 71 cases[138] tuberculosis verrucosa cutis, the mean duration of illness was four years and ten months. According to Panthi et al [139], 97.1% of patients showed mantoux positive and AFB positive on cytology was 10.3%. In our study of five patients with TBVC, Mantoux was reactive in all patients with characteristic histopathological features. The age group of the patients with TBVC was between 10 and 62 years. The mean duration of skin lesions was 1.5 years.

According to A.H.Patki[87], hyperkeratotic and verrucous skin lesion on lower extremities in leprosy patients was observed only in male patients. The age group was between 26 and 52 years. The duration of illness was between two to six years. We observed the age group was between 43years and 65 years. The mean duration of disease was 4 years. All the patients were males who completed their MDT regimen.

According to Durox H et al [141] who made a study on verrucous carcinoma of lower limb**,** the mean age of patients was 78 years. The male to female ratio was 1:2. These patients had previous etiology of varicose ulcer (5 cases), mixed ulcer (3 cases), burn (2 cases) or traumatic lesion (1 case). We observed the mean age of patients was 59 years. We observed all patients were

male. Two patients had history of Hansen's disease with chronic ulcer. We could not find out the etiology in another two patients of verrucous carcinoma of lower limb which was proved histopathologically.

Sueki et al [68]on association of verrucous skin lesions and skin ulcers on the feet in patients with diabetic neuropathy, he reported three diabetic neuropathy patients with verrucous skin lesions and ulcers on feet .In our study one patient had diabetic neuropathy with verrucous skin lesions of lower limbs. Another two patients had skin lesions with verrucous morphology with sensory and motor neuropathy confirmed by nerve conduction study. There are only limited studies on this entity.

Two patients had porokeratosis with verrucous morphology.On histopathological examination, we found classical coronoid lamella with dyskeratosis and absent granular layer just beneath the coronoid lamella. These features were consistent with features of porokeratosis described in Lever's, Histopathology of the skin[142].

A study by Daldon PE [143] a clinicopathological study of hypertrophic DLE revealed the features of discoid lesions with verrucous plaque, commonly involving upper limb and face but rarely seen on lower limbs. Two cases of hypertrophic DLE lesions seen over the lower limbs.

Fang-Yih Liaw et al [144] reported a case of elephantiasis nostros verrucosa cutis of a 70 year old male with congestive heart failure and diabetes mellitus. Doppler sonography showed deep vein thrombi bilaterally in the lower

limbs revealed partial thrombi bilaterally in the common femoral vein and in the right deep femoral vein. Magnetic resonance imaging revealed generalized swelling in the subcutaneous layer and lymphedema of the bilateral lower legs. We had a case of ENVC following trauma and doppler showed the features of lymphedema with competent saphenofemoral and saphenopopliteal valve.

Habib Ur Rehman [145] who reported prayer nodules over the dorsum of feet ,knees and lateral malleolus .We found two patients with callosity over knees due to chronic friction .This was due to their religious prayer.

SUMMARY

A cross sectional study of clinical and histological features of verrucous skin lesions of lower limbs was done for a period of 12 months from August 2011 to July 2012.

100 patients with verrucous skin lesions of lower limbs were included in the study. The average age of presentation was the male to female ratio was 1.3:1(57% &43%).

The common etiology of verrucous skin lesion over the lower limbs were hypertrophic lichen planus (29), wart (10) and lichen simplex chronicus (10).

The less common etiology were porokeratosis(2), hypertrophic discoid lupus erythematosus (2), callosity (2) and ENVC(1). Clinically most of the lesions were presented with hyperpigmented verrucous plaque and most often located over the lower legs, ankles, dorsum of foot.

Hypertrophic lichen planus with pseudoepitheliomatous hyperplasia with lymphocytic infiltration at the base of rete ridges and the interface vacuolar changes was the most common histopathological finding observed. The clinical and histopathological profile of psoriasis and wart were similar to other studies. All the cases of phlebolymphedema, verrucous carcinoma, verrucous skin lesions of neuropathy occurred in men in contrast to other studies.

CONCLUSION

1. Hypertrophic lichen planus, wart and lichen simplex chronicus were the common etiological factors for the occurrence of verrucous skin lesions over the lower limbs.
2. Noninfective causes of verrucous skin lesions were seen 86 % of patients and verrucous skin lesions of infectious etiology were seen in 14 % of patients.
3. The mean age of presentation was 48 yrs with the male to female ratio was 1.3:1. In phlebolymphedema, verrucous carcinoma and verrucous skin lesions of neuropathy, males are mostly affected.
4. The unusual causes of verrucous skin lesions are Porokeratosis, verucous skin lesions of neuropathy and hypertrophic discoid lupus erythematosus .
5. Clinicopathological correlations with appropriate investigations are necessary for the proper diagnosis of verrucous skin lesions of lower limbs.

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PROFORMA

Name : Age : Sex:

Hosp.no : S.I.No :

Presenting Complaints :

Duration of Skin Lesions :

Type of skin lesion : Papule

Plaque

Nodule

Number of lesions :

Site of involvement :

Tenderness :

Ulceration :

Secondary infection :

Description of lesions :

Scarring :

Others :

Systemic symptoms : YES/NO

Evening rise of temperature :

H/O injury :

General examination :

Weight :

Pallor :

Temperature :

LNE :

Description :

Varicose veins :

Systemic examination :

CVS :

RS :

CNS :

GIT :

Investigations:

Complete Haemogram :

LFT :

S.Bilirubin :

SGOT :

SGPT :

A/G Ratio :

Sr.creatinine :

VDRL :

HBsAg :

HIV :

HCV :

Skin smear for AFB :

ANA :

MANTOUX TEST :

U/S DOPPLER :

NERVE CONDUCTION STUDY :

BIOPSY FINDINGS :

SPECIAL STAIN :

Final Diagnosis: clinical Final Diagnosis: pathological

CONSENT FORM

Yourself Mr./Mrs./Ms…….. are being asked to be a participant in the research study titled ""CLINICAL STUDY OF VERRUCOUS SKIN LESIONS OF LOWER LIMB""in CMC Hospital, Coimbatore, conducted by Dr.N.S.JAYANTHI, Post Graduate Student, Department of Dermatology, Coimbatore Medical College. You are eligible after looking into the inclusion criteria. You can ask any question you may have before agreeing to participate.

Research Being Done

CLINICAL STUDY OF VERRUCOUS SKIN LESIONS OF LOWER LIMBS

Purpose of Research : To study the prevalence and etiology of various verrucous skin lesions of Lowerlimbs

To evaluate the various pattern of verrucous lesions of lower limbs

Timely diagnosis and guiding treatments accordingly

Decline from Participation

You have the option to decline from participation in the study existing protocol for your condition.

Privacy and Confidentiality

Privacy of individuals will be respected and any information about you or provided by you during the study will be kept strictly confidential.

Authorization to publish Results

Results of the study may be published for scientific purposes and/or presented to scientific groups, however you will not be identified.

Statement of Consent

I volunteer and consent to participate in this study. I have read the consent or it has been read to me. The study has been fully explained to me, and I may ask questions at any time.

---------- ----------

Signature /Left thumb impression Date

(volunteer)

---------- ----------

Signature of witness Date

ABBREVIATION

HPV	–	Human papilloma virus
DNA	–	Deoxyribo Nucleic Acid
TGF –β1	–	Transformation Growth Factor
MRI	–	Magnetic Resonance Imaging
ENV	–	Elephantiasis Nostros Verrucosa Cutis
CCLE	–	Chronic Cutaneous Lupus Erythematosus
DLE	–	Discoid Lupus Erythematosus
PK	–	Porokeratosis
HIV	–	Human ImmunoDeficiency Virus
VDRL	–	Venereal Disease Research Laboratory
AFB	–	Acid Fast Bacilli
DSL	–	Duration of Skin Lesions
PEH	–	Pseudoepithliomatous Hyperplasia
LSC	–	Lichen Simplex Chronicus
EH	–	Epidermal Hyperplasia
TBVC	–	Tuberculosis verrucosa cutis
SCLE	–	Subacute cutaneous Lupus Erythematosus
TAT-1	–	Trans-Activating Protein-1
GIT	–	GastroIntestinalTract

s.no	op.no	name	age	sex	tos	si	rec	knee	ankle	legs	dof	oai	hiv	cxr	sss	doppler	ncs	bf	cd	pd
1	412501/11	karuppaiya	20	M	VP	NIL	NIL	P	A	A	P	NO	NR	NAD	NEG	NORMAL	NORMAL	hlp	hlp	hlp
2	61264	Thangavelu	58	M	VP	NIL	NIL	A	A	A	p	NO	NR	NAD	NEG	NORMAL	NORMAL	hlp	hlp	hlp
3	386170	Sekar	46	M	VP	NIL	NIL	A	A	A	P	NO	NR	NAD	NEG	NORMAL	NORMAL	ns	hdv	hdv
4	344862	Krishnasamy	70	M	VP	NIL	NIL	A	P	P	A	NO	NR	NAD	NEG	NORMAL	sn/mn	hlp	hlp	hlp
5	332731	Maragatham	37	F	VP	NIL	NIL	A	A	P	P	NO	NR	NAD	NEG	le	NORMAL	pn	pn	pn
6	243526	Rakshnadevi	10	F	VP	NIL	NIL	A	A	A	P	NO	NR	NAD	NEG	NORMAL	NORMAL	hlp	hlp	hlp
7	492780	Muthusamy	78	M	VP	NIL	NIL	A	P	P	A	NO	NR	NAD	NEG	sfi	NORMAL	pl	pl	pl
8	678453	Kathiresan	24	M	VP	NIL	NIL	A	P	A	A	NO	NR	NAD	NEG	NORMAL	NORMAL	hlp	hlp	hlp
9	78916	Manickam	46	M	VP	NIL	NIL	A	P	P	A	NO	NR	NAD	NEG	NORMAL	NORMAL	la	la	la
10	1003	Sankaran	40	M	VP	NIL	NIL	A	A	A	P	NO	NR	NAD	NEG	sfi	NORMAL	wart	wart	wart
11	796047	Govindaraj	27	M	VP	NIL	NIL	A	A	A	P	NO	NR	NAD	NEG	NORMAL	NORMAL	tbvc	tbvc	tbvc
12	80748	Balu	46	M	VP	NIL	NIL	A	A	P	A	NO	NR	NAD	NEG	NORMAL	NORMAL	lsc	lsc	lsc
13	79082	Anandha kumar	27	M	VP	NIL	NIL	A	P	P	P	NO	NR	NAD	NEG	NORMAL	NORMAL	pn	pn	pn
14	637629	Shanmugam	26	M	VP	NIL	NIL	A	A	P	A	NO	NR	NAD	NEG	NORMAL	NORMAL	wart	wart	wart
15	546372	Thangamani	60	F	VP	NIL	NIL	A	P	A	P	NO	NR	NAD	NEG	NORMAL	NORMAL	pn	pn	pn
16	154135	Thulasi	60	F	VP	NIL	NIL	A	A	P	A	NO	NR	NAD	NEG	NORMAL	NORMAL	wart	wart	wart
17	381431	Krishnaveni	55	F	VP	NIL	NIL	A	A	A	P	NO	NR	NAD	NEG	NORMAL	NORMAL	hlp	hlp	hlp
18	478591	Janakiyammal	58	F	VP	NIL	NIL	A	P	P	A	NO	NR	NAD	NEG	NORMAL	NORMAL	lsc	lsc	lsc
19	544065	Maragathamani	29	F	VP	NIL	NIL	A	P	P	A	NO	NR	NAD	NEG	NORMAL	NORMAL	hlp	hlp	hlp
20	117606	Rajeshwari	60	F	VP	NIL	NIL	A	A	P	A	NO	NR	NAD	NEG	NORMAL	NORMAL	hlp	hlp	hlp
21	91600	Krishnavani	38	F	VP	NIL	NIL	A	A	P	A	NO	NR	NAD	NEG	NORMAL	NORMAL	ps	ps	ps
22	156939	Rukmani	62	F	VP	NIL	NIL	A	A	P	A	NO	NR	NAD	NEG	NORMAL	NORMAL	la	la	la
23	119881	Manickam	56	M	VP	NIL	NIL	A	P	A	A	NO	NR	NAD	NEG	NORMAL	NORMAL	vc	vc	vc
24	60144	Madhuvandhi	16	F	VP	NIL	NIL	A	A	P	A	NO	NR	NAD	NEG	NORMAL	NORMAL	hlp	hlp	hlp
25	35341	Rajammal	60	F	VP	NIL	NIL	A	A	P	P	NO	NR	NAD	NEG	NORMAL	NORMAL	ps	ps	ps
26	82377	Kandasamy	15	M	VP	NIL	NIL	A	P	P	A	NO	NR	NAD	NEG	NORMAL	NORMAL	wart	wart	wart
27	16084	Nagaraj	51	M	VP	NIL	NIL	A	P	A	P	NO	NR	NAD	NEG	NORMAL	NORMAL	hlp	hlp	hlp
28	153834	Vignesh	38	M	VP	NIL	NIL	A	A	A	P	NO	NR	NAD	NEG	NORMAL	NORMAL	hdle	hdle	hdle
29	477134	Nabisa	45	F	VP	NIL	NIL	A	A	P	A	NO	NR	NAD	NEG	NORMAL	NORMAL	wart	wart	wart
30	642813	Mylathal	64	F	VP	NIL	NIL	A	P	A	A	NO	NR	NAD	NEG	NORMAL	NORMAL	hdv	hdv	hdv
31	733818	Mohammed sultan	67	M	VP	NIL	NIL	P	A	A	A	NO	NR	NAD	NEG	NORMAL	NORMAL	hlp	hlp	hlp
32	161072	Shajar	16	M	VP	NIL	NIL	A	A	A	P	NO	NR	NAD	NEG	NORMAL	NORMAL	tbvc	tbvc	tbvc
33	170653	Selvaraj	43	M	VP	NIL	NIL	A	A	P	A	NO	NR	NAD	NEG	NORMAL	sn/mn	ns	vsn	vsn
34	171320	Muthusamy	64	M	VP	NIL	NIL	A	A	P	A	NO	NR	NAD	NEG	NORMAL	NORMAL	ns	pl	pl

35	142664	Ramasamy	72	M	VP	NIL	NIL	A	P	A	P	NO	NR	NAD	NEG	NORMAL	NORMAL	hlp	hlp	hlp
36	140920	Jaya	66	F	VP	NIL	NIL	A	P	A	A	NO	NR	NAD	NEG	NORMAL	NORMAL	pn	pn	pn
37	456372	Balasubramaniam	54	M	VP	NIL	NIL	A	A	P	A	NO	NR	NAD	NEG	NORMAL	NORMAL	la	la	la
38	10836	Maragatham	48	F	VP	NIL	NIL	A	A	P	P	NO	NR	NAD	NEG	NORMAL	NORMAL	envc	envc	envc
39	146075	Raseed	52	M	VP	NIL	NIL	A	P	P	P	NO	NR	NAD	NEG	spi	NORMAL	hlp	hlp	hlp
40	142664	Ramasamy	72	M	VP	NIL	NIL	A	P	A	P	NO	NR	NAD	NEG	NORMAL	sn	ns	vsn	vsn
41	653426	Anitha	19	F	VP	NIL	NIL	A	A	P	A	NO	NR	NAD	NEG	NORMAL	NORMAL	tbvc	tbvc	tbvc
42	216929	Mubeena	25	F	VP	NIL	NIL	A	A	A	P	NO	NR	NAD	NEG	NORMAL	NORMAL	hlp	hlp	hlp
43	243273	Ramesh	33	M	VP	NIL	NIL	A	P	P	A	NO	NR	NAD	NEG	NORMAL	NORMAL	ps	ps	ps
44	243156	Mano	24	F	VP	NIL	NIL	A	A	P	A	NO	NR	NAD	NEG	NORMAL	NORMAL	pn	pn	pn
45	9987	Murugeshan	55	M	VP	NIL	NIL	A	P	P	A	NO	NR	NAD	NEG	NORMAL	NORMAL	vc	vc	vc
46	543242	Kamatchiamma	66	F	VP	NIL	NIL	A	A	A	P	NO	NR	NAD	NEG	NORMAL	NORMAL	hlp	hlp	hlp
47	226826	Janakiammal	55	F	VP	NIL	NIL	A	A	P	A	NO	NR	NAD	NEG	NORMAL	NORMAL	hlp	hlp	hlp
48	263874	Ganesan	44	M	VP	NIL	NIL	A	P	P	A	NO	NR	NAD	NEG	NORMAL	NORMAL	ps	ps	ps
49	263880	Thulasiammal	60	F	VP	NIL	NIL	A	P	A	A	NO	NR	NAD	NEG	NORMAL	NORMAL	tbvc	tbvc	tbvc
50	264315	Pappathy	38	F	VP	NIL	NIL	A	A	A	P	NO	NR	NAD	NEG	NORMAL	NORMAL	la	la	la
51	33875	Palanivel	60	M	VP	NIL	NIL	A	P	A	P	NO	NR	NAD	NEG	NORMAL	NORMAL	hdle	hdle	hdle
52	266830	Palanammal	50	F	VP	NIL	NIL	A	A	A	A	NO	NR	NAD	NEG	NORMAL	NORMAL	hlp	hlp	hlp
53	4931	saran	42	M	VP	NIL	NIL	A	P	A	A	NO	NR	NAD	NEG	NORMAL	NORMAL	ps	ps	ps
54	269913	Mahesh	56	M	VP	NIL	NIL	A	P	A	A	NO	NR	NAD	NEG	spi	NORMAL	ns	pl	pl
55	263801	Lakshmi	16	F	VP	NIL	NIL	A	A	A	P	NO	NR	NAD	NEG	NORMAL	NORMAL	wart	wart	wart
56	269177	Padmanaban	63	M	VP	NIL	NIL	A	P	A	A	NO	NR	NAD	NEG	NORMAL	NORMAL	ps	ps	ps
57	268731	Lakshmi	67	F	VP	NIL	NIL	A	A	A	P	NO	NR	NAD	NEG	NORMAL	NORMAL	hlp	hlp	hlp
58	27227	Suseela	45	F	VP	NIL	NIL	A	A	P	A	NO	NR	NAD	NEG	NORMAL	NORMAL	pk	pk	pk
59	253672	Albert	50	M	VP	NIL	NIL	A	A	P	P	NO	NR	NAD	NEG	NORMAL	NORMAL	ns	hdv	hdv
60	268201	kuppammal	67	F	VP	NIL	NIL	A	P	A	A	NO	NR	NAD	NEG	NORMAL	NORMAL	lsc	lsc	lsc
61	297805	Loganathan	52	M	VP	NIL	NIL	A	A	A	P	NO	NR	NAD	NEG	NORMAL	NORMAL	hlp	hlp	hlp
62	283031	Sadhasivam	26	M	VP	NIL	NIL	A	A	P	A	NO	NR	NAD	NEG	NORMAL	NORMAL	hlp	hlp	hlp
63	55441	Kuppusamy	65	M	VP	NIL	NIL	A	A	A	P	NO	NR	NAD	NEG	NORMAL	NORMAL	ns	cs	cs
64	299198	Rajendran	57	M	VP	NIL	NIL	A	A	P	A	NO	NR	NAD	NEG	sfi	NORMAL	ns	pl	pl
65	309343	Ramasamy	55	M	VP	NIL	NIL	A	P	A	A	NO	NR	NAD	NEG	NORMAL	NORMAL	ns	hdv	hdv
66	297560	Thangaraj	65	M	VP	NIL	NIL	A	A	P	A	NO	NR	NAD	NEG	NORMAL	NORMAL	hlp	hlp	hlp
67	317682	Rathinavel	39	M	VP	NIL	NIL	A	A	P	A	NO	NR	NAD	NEG	NORMAL	NORMAL	la	la	la
68	373620	Madiyalagan	35	M	VP	NIL	NIL	A	A	A	P	NO	NR	NAD	NEG	NORMAL	NORMAL	wart	wart	wart
69	315834	Serinammal	48	F	VP	NIL	NIL	A	P	A	A	NO	NR	NAD	NEG	NORMAL	NORMAL	hlp	hlp	hlp

70	308827	Kannamal	60	F	VP	NIL	NIL	A	A	P	A	NO	NR	NAD	NEG	NORMAL	NORMAL	ns	hdv	hdv
71	324395	Chandrasekar	37	M	VP	NIL	NIL	A	A	A	P	NO	NR	NAD	NEG	NORMAL	NORMAL	hlp	hlp	hlp
72	234820	Mahalingam	69	M	VP	NIL	NIL	a	p	p	p	NO	NR	NAD	NEG	NORMAL	sn/mn	ns	vsn	vsn
73	311450	Alagirisamy	64	M	VP	NIL	NIL	A	A	A	P	NO	NR	NAD	NEG	NORMAL	NORMAL	ns	cs	cs
74	328203	Balasubramani	77	M	VP	NIL	NIL	A	A	P	A	NO	NR	NAD	NEG	NORMAL	NORMAL	lsc	lsc	lsc
75	9345	Arunachalam	44	M	VP	NIL	NIL	A	A	P	P	NO	NR	NAD	NEG	sfi	NORMAL	ns	pl	pl
76	329011	Kavitha	45	F	VP	NIL	NIL	P	A	P	A	NO	NR	NAD	NEG	NORMAL	NORMAL	ns	hdv	hdv
77	88234	Rajasekar	11	M	VP	NIL	NIL	A	A	P	A	NO	NR	NAD	NEG	NORMAL	NORMAL	wart	wart	wart
78	738136	Anantha kumar	56	M	VP	NIL	NIL	A	A	A	P	NO	NR	NAD	NEG	NORMAL	NORMAL	lsc	lsc	lsc
79	483080	krishnammal	40	F	VP	NIL	NIL	A	A	A	P	NO	NR	NAD	NEG	NORMAL	NORMAL	hlp	hlp	hlp
80	432523	Subaiyan	67	M	VP	NIL	NIL	A	P	A	P	NO	NR	NAD	NEG	NORMAL	NORMAL	lsc	lsc	lsc
81	543231	Tajudin	44	M	VP	NIL	NIL	A	A	A	P	NO	NR	NAD	NEG	NORMAL	NORMAL	ns	pl	pl
82	151316	Vijay	57	M	VP	NIL	NIL	P	A	A	A	NO	NR	NAD	NEG	NORMAL	NORMAL	ps	ps	ps
83	345214	Rani	45	F	VP	NIL	NIL	A	A	P	A	NO	NR	NAD	NEG	pi	NORMAL	lsc	lsc	lsc
84	347045	Bagyalakshmi	45	F	VP	NIL	NIL	P	A	A	P	NO	NR	NAD	NEG	NORMAL	NORMAL	hlp	hlp	hlp
85	2449	Mariappan	65	M	VP	NIL	NIL	A	A	A	F	NO	NR	NAD	NEG	NORMAL	NORMAL	vc	vc	vc
86	324511	yamuna	32	F	VP	NIL	NIL	P	P	A	A	NO	NR	NAD	NEG	NORMAL	NORMAL	hlp	hlp	hlp
87	740161	Arogyasamy	18	M	VP	NIL	NIL	p	A	A	P	NO	NR	NAD	NEG	NORMAL	NORMAL	wart	wart	wart
88	357556	Usha	42	F	VP	NIL	NIL	A	A	A	P	NO	NR	NAD	NEG	NORMAL	NORMAL	hlp	hlp	hlp
89	351233	Lakshmi	46	F	VP	NIL	NIL	P	A	A	P	NO	NR	NAD	NEG	NORMAL	NORMAL	tbvc	tbvc	tbvc
90	202780	Maruthamuthu	37	M	VP	NIL	NIL	A	A	A	P	NO	NR	NAD	NEG	NORMAL	NORMAL	la	la	la
91	355272	Sudha	58	F	VP	NIL	NIL	P	A	A	P	NO	NR	NAD	NEG	NORMAL	NORMAL	lsc	lsc	lsc
92	201734	Rukmani	50	F	VP	NIL	NIL	P	A	A	P	NO	NR	NAD	NEG	NORMAL	NORMAL	hlp	hlp	hlp
93	773057	Manickam	48	M	VP	NIL	NIL	P	A	P	A	NO	NR	NAD	NEG	NORMAL	NORMAL	vc	vc	vc
94	267496	Ramakrishnan	16	M	VP	NIL	NIL	P	P	A	A	NO	NR	NAD	NEG	NORMAL	NORMAL	wart	wart	wart
95	361275	Ravi	50	M	VP	NIL	NIL	P	A	A	P	NO	NR	NAD	NEG	NORMAL	NORMAL	pk	pk	pk
96	366375	Maragadam	68	F	VP	NIL	NIL	A	A	P	P	NO	NR	NAD	NEG	NORMAL	NORMAL	lsc	lsc	lsc
97	403126	Sundara pandian	37	M	VP	NIL	NIL	P	A	A	A	NO	NR	NAD	NEG	sfi	NORMAL	ns	pl	pl
98	524177	Yasodha	21	F	VP	NIL	NIL	A	A	P	A	NO	NR	NAD	NEG	NORMAL	NORMAL	la	la	la
99	543628	Aboorvam	65	F	VP	NIL	NIL	A	P	A	A	NO	NR	NAD	NEG	NORMAL	NORMAL	lsc	lsc	lsc
100	523345	Akila	42	F	VP	NIL	NIL	P	A	A	P	NO	NR	NAD	NEG	NORMAL	NORMAL	hlp	hlp	hlp

CODE FOR MASTER CHART

TOS	-	Type of skin lesion
SI	-	Secondary infection
REC	-	Recurrence
DOF	-	dorsum of foot
HIV	-	Human Immunodeficiency Virus
CXR	-	Chest XRay
SSS	-	Slit Skin Smear
NCS	-	Nerve conduction study
Bf	-	Biopsy findings
CD	-	Clinical diagnosis
PD	-	Provisional diagnosis
HLP	-	Hypertrophic lichen planus
HDV	-	verrucous skin lesion in leprosy
PN	-	Prurigo nodularis
PL	-	Phelebolymphedema
LA	-	lichen planus
TBVC	-	Tuberculosis verrucosa Cutis
LSC	-	Lichen simplex chronicus
PS	-	Psoriasis
VC	-	Verrucous Carcinoma

HDLE	-	hypertrophic discoid lupus erythematosus
VSN	-	Verrucous skin lesions of neuropathy
ENVC	-	Elephantiasis nostros verrucosa cutis
PL	-	Phelebolymphedema
PK	-	Porokeratosis
CS	-	Callosity
VSN	-	verrucous skin lesions of neuropathy