A DESCRIPTIVE STUDY OF LESIONS FOLLOWING THE LINES OF BLASCHKO

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CERTIFICATE

This is to certify that this dissertation entitled **'A DESCRIPTIVE STUDY OF LESIONS FOLLOWING THE LINES OF BLASCHKO'** submitted by **Dr. SHEERJA BALI** to The Tamil Nadu Dr.M.G.R. Medical University, Chennai is in partial fulfillment of the requirement for the award of M.D. [DERMATO VENEREO LEPROLOGY] and is a bonafide research work carried out by her under direct supervision and guidance.

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DECLARATION

I, **Dr. SHEERJA BALI** solemnly declare that I carried out this work on '**A DESCRIPTIVE STUDY OF LESIONS FOLLOWING THE LINES OF BLASCHKO'** at Department of Dermatology, Government Rajaji Hospital during the period of Oct 2009 – Sep 2011.

I also declared this bonafide work or a part of this work was not submitted by me or any other for any award, degree and diploma to any university, board either in India or abroad.

This is submitted to The Tamil Nadu Dr. M.G.R Medical University, Chennai in partial fulfillment of the rules and regulation for M.D., [D.V.L] Degree examination.

Govt. Rajaji Hospital. Dr. SHEERJA BALI

Madurai.

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INTRODUCTION

The concept of Blaschko's lines was first put forth by Alfred Blaschko, a private practioner in dermatology, when he presented his findings on the distribution patterns of linear skin at the German Dermatological Society meeting in Breslau in 1901.[1] In his original description he referred to Blaschko's lines as 'a system of lines on the human skin which the linear nevi and dermatosis follow.'[1] The pattern of Blaschko's lines does not correlate with the distribution of cutaneous nerves, blood vessels, or lymphatics and is distinct from pigmentary demarcation lines . Two mechanisms have been proposed to explain Blaschko's lines: mosaicism and chimerism.

A number of congenital and acquired conditions follow these special lines. Almost all epidermal nevi follow Blaschko's lines. Lesions in various X-linked disorder's like incontinentia pigmenti , Goltz syndrome and chromosomal disorders like hypomelanosis of Ito also respect these lines. This pattern followed by these congenital diseases helps in their diagnosis not only in the newborn and infant, but also in adults.

Other nevoid epidermal disorders like linear lichen planus, nevoid psoriasis, Darier's disease and Hailey- Hailey disease, linear porokeratosis and blaschkitis are some of the acquired diseases that occur along the Blaschko's lines. The

occurrence of these acquired lesions has been known to have special significance with regards to the epidemiology, clinical presentation, treatment and prognosis. There have been many studies with respect to individual diseases following the lines of Blaschko. However, no studies with Blaschko's lines as the central focus could be found in the literature. This study is an attempt in that direction.

AIM OF THE STUDY

1) To study and describe the patterns of Blaschko's lines encountered in various dermatoses.
2) To study the clinico- epidemiological features of various diseases related to the lines of Blaschko.

REVIEW OF LITERATURE

DEFINITION-

The lines of Blaschko represent a pattern assumed by many different nevoid and acquired skin diseases on the human skin and mucosae.[2] These lines are characteristic of mosaic conditions of the epidermis and probably represent the routes of ectodermal cell migration from the neural crest. These were first described by Alfred Blaschko (1858- 1922) , a German Dermatologist in 1901.[1]These are also termed as cutaneous lines of embryogenesis.[3]These lines do not correspond to other patterns such as Langer's lines of cleavage,[4]Voigt's lines (borders between areas of innervations by peripheral cutaneous nerves,[5] embryonic clefts,[6] pigmentary demarcation lines,[7]or the lines of lymphatic drainage or blood supply.[8] These do not correspond to nervous structures.[8]

TYPES- (Figure 1)

Four main types of Blaschko's lines have been described[9]:

TYPE 1a-NARROW BANDS

TYPE 1b- BROAD BANDS

TYPE 2- CHECKER BOARD PATTERN- A flag like arrangement with a strict midline separation[10]

FIGURE 1: TYPES OF BLASCHKO'S LINES

TYPE 1 (a & b) - NARROW AND BROAD BAND

TYPE 2- CHECKERBOARD PATTERN

TYPE 3- PHYLLOID

TYPE 4- PATCHY WITHOUT MIDLINE SEPARATION

TYPE 5- LATERALIZATION

TYPE 3- PHYLLOID PATTERN- Multiple leaf-like or oblong macules reminiscent of the floral ornaments of art nouveau style.[11]Clinical example is novel neuro- cutaneous syndrome in the form of phylloid hypomelanosis.[12]

TYPE 4- PATCHY PATTERN WITHOUT MIDLINE SEPARATION

A fifth type, LATERALIZATION[13] has been described which is classically seen in CHILD syndrome wherein a clear cut midline demarcation is seen.[14]

DISTRIBUTION PATTERN – (Figures 2 & 3)

These assume a V-shape over the upper spine, an S-shape on the abdomen, and an inverted U-shape from the breast area onto the upper arm. There are perpendicular lines down the front and back of the lower extremities.[1]The lines of Blaschko are less well defined on the head and neck.[15]Happle et al added lines to the posterior scalp,[16,17] while Bolognia et al further delineated the lines on the lateral aspect of the face and neck.[15]Brown and Gorlin mentioned vertical striations on the lips, linear midline lesions on the hard and soft palate, and linear unilateral and / or midline bands on the tongue in patients with epidermal nevi.[18]

The anatomic equivalent of Blaschko's lines has been described in the teeth and eyes as well.[19-20] For example, female carriers of X- linked ocular albinism can have a striated pigmentary pattern in the peripheral retina[21] in addition to an alternating spokewheel – like pigmentation of the iris.[22]Sectoral cataracts and radial patterns in the lens have been described in women with X- linked dominant

FIGURE 2: DISTRIBUTION PATTERN OF BLASCHKO'S LINES - TRUNK & EXTREMITIES

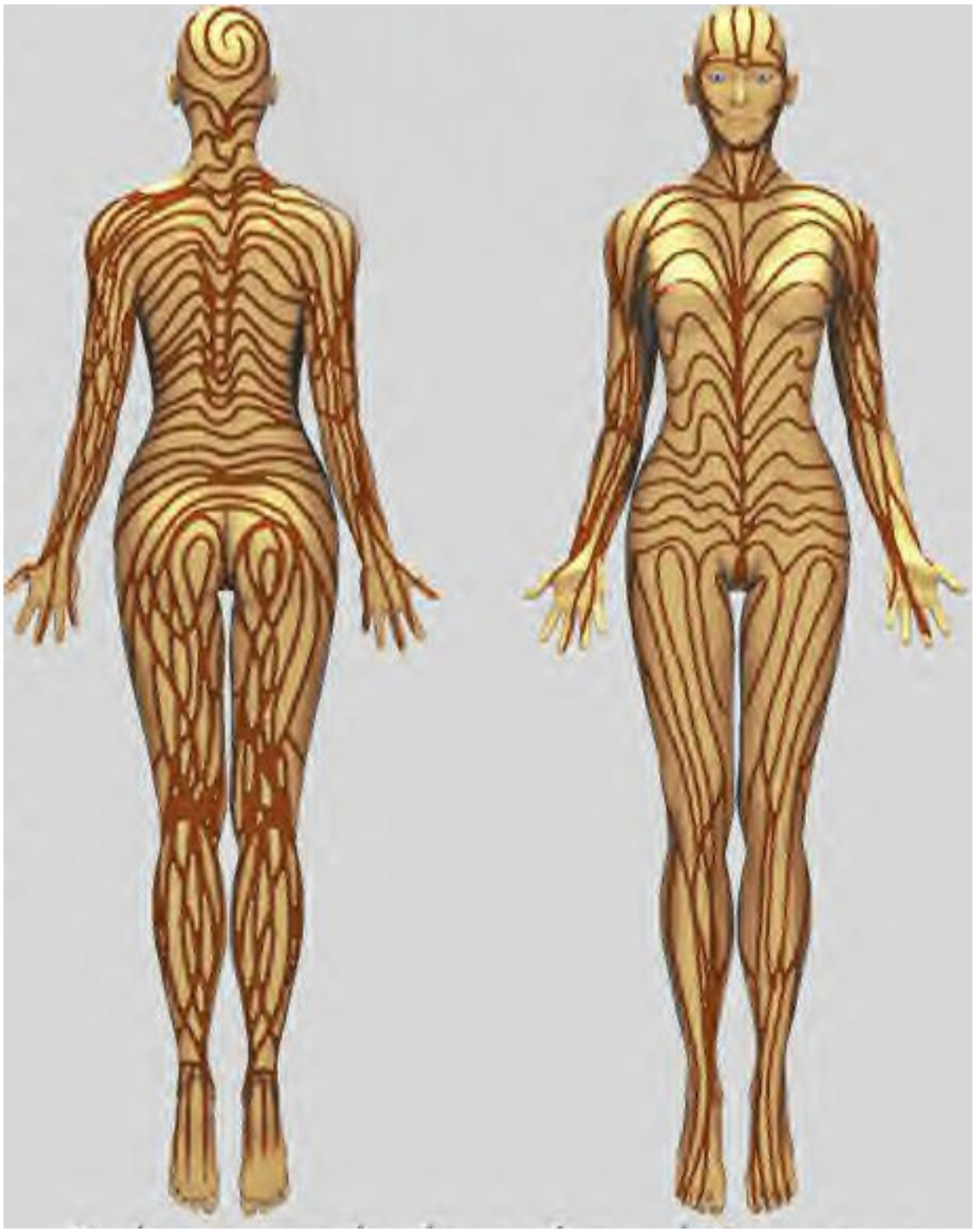
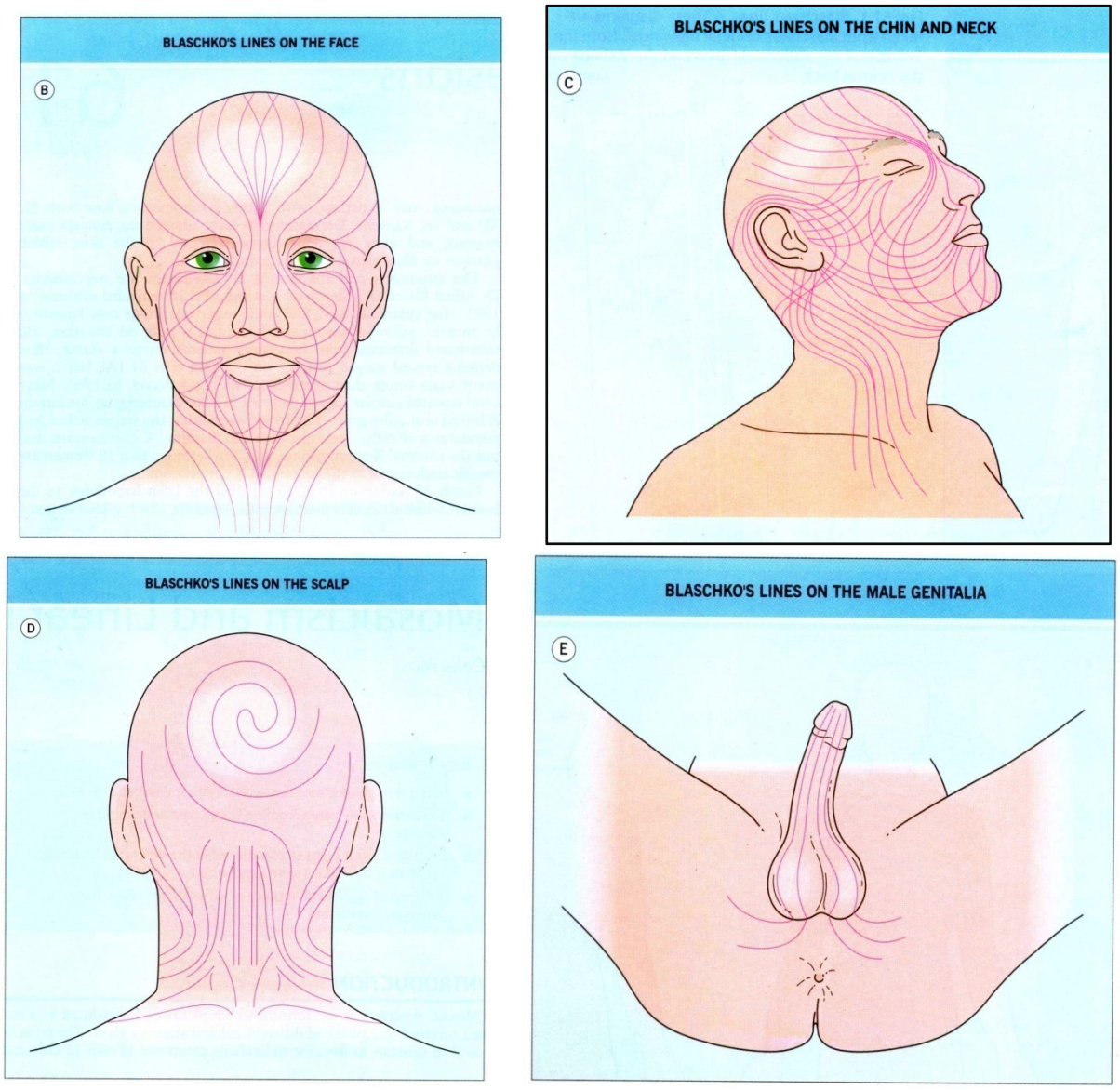


FIGURE 3: DISTRIBUTION PATTERN OF BLASCHKO'S – HEAD, NECK & GENITALS



chondrodysplasia punctata.[23]Witkop also described alternating vertical band of opaque white and translucent (normal- appearing) enamel on the central incisor of women heterozygous for X- linked hypo - maturation amelogenesis imperfecta.[20]

BASIS OF DISTRIBUTION PATTERN OF THE LINES OF BLASCHKO

The embryological basis of distribution pattern of these lines is so far an enigma.[24] Jackson suggested that their localization is partly determined by the dermal tissues.[2]An inborn error of morphogenesis due to a single mutant gene[25] or stretching of the skin[15]during embryogenesis have also been proposed as the underlying mechanisms. Their characteristic distribution pattern points to the presence of two different clones of cells in early embryogenesis. Thus, possibly, these lines represent a form of human 'mosaicism' where two or more genetically distinct cell populations are present in an individual derived from a single zygote. These different clones may be due to lyonization (random inactivation of one of the two X chromosomes in all cells in females), post-zygotic somatic mutations during early embryogenesis or gametid half- chromatid mutation occurring before fertilization, [1, 15]chromosomal non-dysjunction or chimerism. These lines probably represent boundaries between populations of normal and mutant cells and may basically visualize the dorso-ventral outgrowth of a mutant cell clone.[13]

One explanation given for the fountain-like pattern of Blaschko's lines on the back is that transversal proliferation of precursor cells starts from the primitive streak but is interfered with the longitudinal growth and increasing flexion of the embryo.[13] (Figure 4)

Figure 4: Proposed explanation of the fountain-like pattern of Blaschko's lines on the back.

However no single theory as yet clearly elucidates the localization of Blaschko's lines.[24] Interestingly, cutaneous mosaicism does not always follow Blaschko's lines.[8]Assuming that the pattern is determined by cell migration, it will depend on the stage of development at which mosaicism arises. The earlier the mutation, the more widely dispersed and more intimately mixed the mosaic clone will be, and longer the lines of migration.[26]Its timing in relation to the processes of lyonization, lateralization & organogenesis will also influence the pattern.[26]

The pattern of cutaneous mosaicism also varies according to the cell type affected.[26] (Table I)

TABLE I- EXPECTED PATTERN OF CUTANEOUS MOSAICISM ACCORDING TO CELL TYPE

CELL AFFECTED	EMBRYONIC MOVEMENT	EXPECTED PATTERN
MELANOBLAST	Single cell migration	Checker board, bands or phylloid
KERATINOCYTE	Directional proliferation following surface forces	Blaschko - bands

Sometimes, a severe linear lesion is superimposed on a background of milder generalized disease. Happle attributes this to mosaic loss of heterozygosity, and terms it Type 2 mosaicism. Thus, an individual who has inherited an autosomal dominant disorder suffers a postzygotic mutation affecting the other allele ('second hit') resulting in a linear area where the genetic burden is doubled. In fact, many disorders show dense or confluent linear lesions even in the absence of background lesions, for example, Darier's disease, psoriasis and lichen planus. Type 2 mosiacism has now been proven for Hailey- Hailey disease.[27]

The opposite phenomenon - linear absence of skin disease attributed to a back mutation (revertant mosaicism) also occurs as seen in patients with autosomal recessive as well as autosomal dominant forms of epidermolysis bullosa.[28]

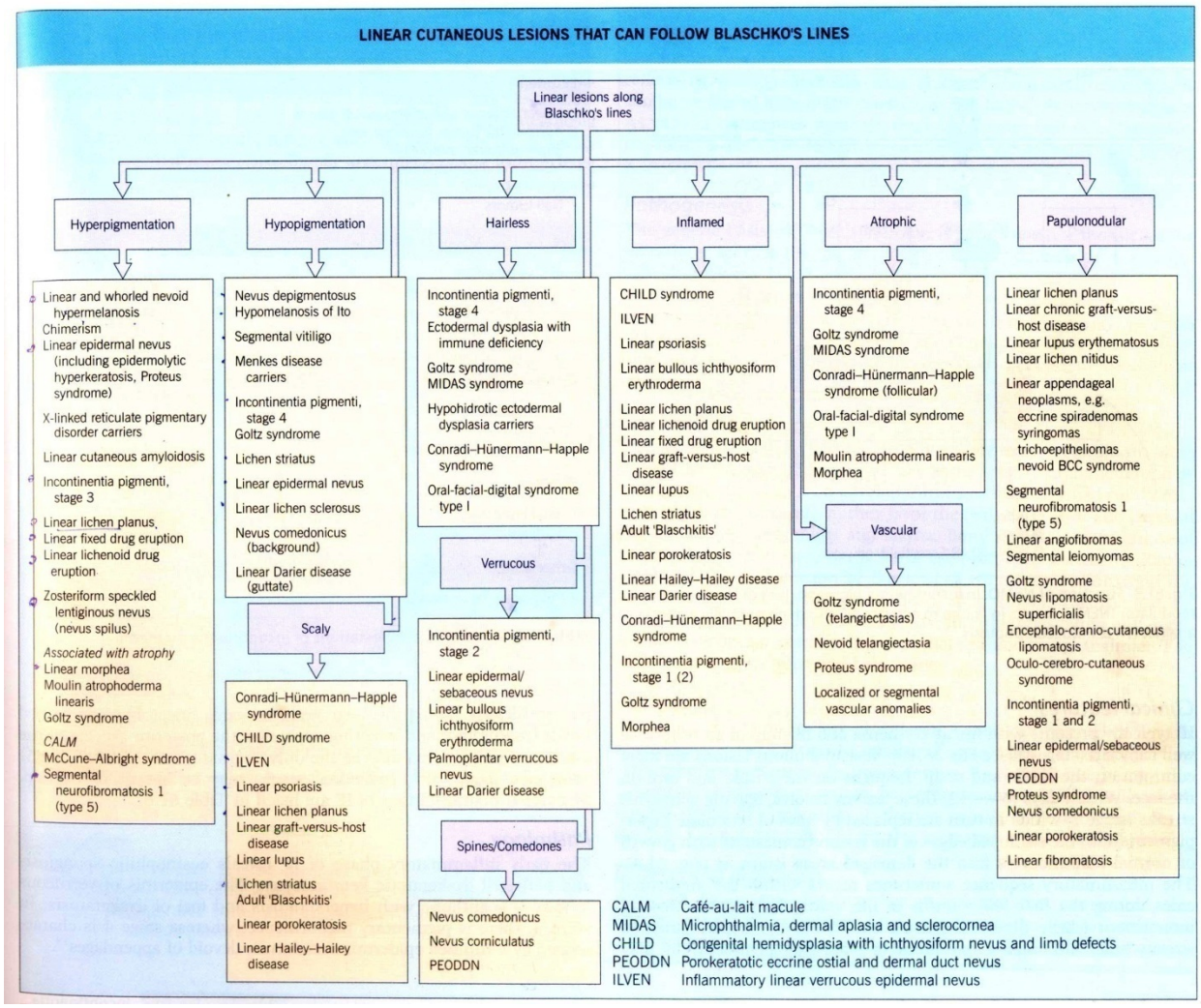
SKIN CONDITIONS FOLLOWING THE LINES OF BLASCHKO- (Table II and Figure 5)

TABLE II- MOSIAC SKIN CONDITIONS CLASSIFIED ACCORDING TO THE NATURE OF THE GENERALIZED CONDITION

X- LINKED DOMINANT LETHAL IN MALES	• Incontinentia pigmenti • Goltz syndrome • Conradi—Hunermann—Happle syndrome (chondrodysplasia punctata) • CHILD syndrome (congenital hemidysplasia with ichthyosiform nevus and limb defects) • MIDAS syndrome • Oral-facial-digital syndrome type I
X-LINKED RECESSIVE	• Hypohidrotic ectodermal dysplasia • Menkes disease (carrier females) • X-Iinked reticulate pigmentary disorder [cutaneous amyloidosis] • Ectodermal dysplasia, hypohidrotic, with immune deficiency (carrier females)
AUTOSOMAL DOMINANT SINGLE GENE DISORDERS	• Linear bullous ichthyosiform erythroderma • Palmoplantar verrucous nevus • Nevus comedonicus • Linear Darier's disease • Linear Halley—Halley disease • Linear porokeratosis • Linear basal cell nevi • Segmental neurofibromatosis type 1 • Linear angiofibromas • Segmental leiomyoma
POSSIBLE AUTOSOMAL DOMINANT SINGLE GENE DISORDERS	• Nevoid telangiectasia • Linear syringomas • Linear trichoepitheliomas • Linear eccrine spiradenomas
MULTIFACTORIAL INFLAMMATORY DISORDERS WITH AUTOSOMAL DOMINANT OR POLYGENIC INHERITANCE	• Linear psoriasis • Linear lichen planus • Linear chronic GVHD • Linear lupus erythematosus • Linear fixed drug eruption

	• Linear lichen nitidus • Lichen striatus • 'Adult blaschkitis' • Linear morphea • Moulin atrophoderma linearis • Segmental vitiligo
PRESUMED AUTOSOMAL DOMINANT LETHAL DISORDER RESCUED BY MOSAICISM (Never Seen In A Generalized Form)	• Linear epidermal /sebaceous nevus • Epidermal nevus syndromes • PEODDN (porokeratotic eccrine ostial and dermal duct nevus) • Proteus syndrome • Nevus lipomatosis superficialis • Encephalocraniocutaneous lipomatosis • Oculocerebrocutaneous syndrome • ILVEN (inflammatory linear verrucous epidermal nevus) • McCune—Albright syndrome • Zosteriform lentiginous nevus • Localized vascular anomalies • Linear fibromatosis
CHROMOSOMAL	• Hypomelanosis of Ito • Nevus depigmentosus • LWNH (linear whorled nevoid hypermelanosis)
CHIMERISM	• Segmental hyperpigmentation

FIGURE 5: LESIONS FOLLOWING THE LINES OF BLASCHKO



A) X-LINKED CONDITIONS FOLLOWING BLASCHKO'S LINES

In women heterozygous for X-linked disorders, mosaicism due to random inactivation (lyonization) may produce skin lesions following Blaschko's lines.

Lyonization occurs synchronously in all cells at about the 1000- cell stage, so the two clones are intimately mixed from the beginning. Therefore, Blaschko's lines in X- linked disorders are typically narrow and numerous. An exception is CHILD syndrome, which characteristically shows large unilateral blocks of abnormal skin; possibly because the mosaicism is due to later somatic mutation on the X chromosome rather than lyonization.

In males, only X-linked recessive conditions are compatible with survival (e.g. hypohidrotic ectodermal dysplasia). X- linked dominant conditions are usually lethal in males in utero itself (e.g. incontinentia pigmenti and Goltz syndrome). This lethal phenotype can only be rescued by mosaicism.

Not all X- linked skin disorders follow Blaschko's lines. For example women who are heterozygous for Fabry's disease, Wiskott – Aldrich syndrome and chronic granulomatous disease, show either no lesions or scattered lesions in a non- linear pattern. A possible explanation is that these abnormalities are systemic, and a local deficiency in parts of the skin is corrected from elsewhere, usually the bone marrow.[26]

Some genes on the X chromosome do not undergo random inactivation.[29] For example, the gene on the short arm of the X chromosome that encodes steroid sulfatase escapes inactivation. This explains why the epidermis of female carriers of X- linked recessive icthyosis lacks a mosaic pattern of scaling.[15]

I) INCONTINENTIA PIGMENTI

(Synonyms-Bloch- Sulzberger syndrome; Bloch- Siemens syndrome)[26]

Incontinentia pigmenti (IP) is a rare X-linked dominant genodermatosis, first described by Bloch and Sulzberger in 1920's.[30]Nearly all the patients (97%) are females. The name refers to the pathologic finding of pigmentary incontinence, especially in the third stage of the disease. The linear skin lesions reflect mosaicism secondary to X inactivation.

In the 1990s, IP1 (the sporadic form) was differentiated from IP2 (inherited form). Patients said to have IP1 have had chromosomal (X/autosome) translocations, often affecting Xp11, in association with pigmentary abnormalities.[26]IP2 represents the X- linked dominant disorder that is caused by mutations in the NEMO gene.[31]

The vesiculo- bullous first stage occurs in approximately 90% of the patients. The lesions are usually present at birth or appear within 2 weeks of life.[32]They occur most commonly on the extremities in a linear distribution. The verrucous second stage usually begins between the second and sixth week of life and is characterized

by linear warty streaks, often overlying the distribution of stage I lesions. This stage occurs in approximately 70% of the patients.[32] The pigmentary or the third stage peaks between the twelfth and the twenty-sixth week of life and is characterized brown or grey streaks and swirls. The pigmentation is characteristic of the syndrome, and the bizarre 'Chinese letter pattern' is diagnostic.[33]

These streaks follow the lines of Blaschko and have a predilection for the trunk. In about 14% of patients, the third stage is said to occur without evidence of prior stages.[32] The hyperpigmentation fades in weeks to years and usually does not persist into adulthood. The fourth stage is characterized by hypopigmented atrophic lesions that occur most commonly on the legs in a linear or reticulated pattern.[34]In an adult, the hypopigmented streaks may be the only manifestation of IP. Additional manifestations of IP include linear absence of hair and sweat glands, nail dystrophy and nail tumors, asymmetric breast development, supernumerary nipples, missing and conical teeth, microphthalmia, retinal vascular anomalies, cataracts, mental retardation, seizures, spastic hemi/ di/ tetraplegia, optic atrophy, skull anomalies, scoliosis and pulmonary hypertension.

In a retrospective study of 40 patients of IP by Smail Razda et al, it was found that during the neonatal period, erythema, vesicles and hyperkeratotic lesions were rarely absent in the patients with IP. Ocular and neurological abnormalities were

frequent (20% and 30%, respectively) but rarely severe (8% and 7.5%, respectively).[35]

II) OTHER DISORDERS

Some other X- linked disorders following Blaschko's lines include Conradi-Hunermann- Happle syndrome, Goltz Syndrome, Congenital Hemidysplasia with Icthyosiform Erythroderma and limb defects (CHILD Syndrome), Menkes' syndrome (female carriers), X- linked hypohidrotic ectodermal dysplasia (HED- female carriers), MIDAS syndrome, Oral- facial – digital syndromes (OFDS), X-Linked Reticulate Pigmentary Disorder- (Partington Cutaneous Amyloidosis), ectodermal dysplasia, hypohidrotic, with immune deficiency.[26]

B) AUTOSOMAL DOMINANT CONDITIONS FOLLOWING BLASCHKO'S LINES

Some classical autosomal dominant skin disorders occasionally occur in a mosaic pattern. While the lesional morphology and histology are similar in the mosaic and the generalized forms, the diagnosis may not be immediately obvious without clues provided by distribution and overall appearance of the generalized disorder. If no generalized disorder exists to match the linear lesions, then it could represent a dominant lethal mutation only survivable in the mosaic form.[26]

Mosaicism for autosomal disorders arises during gametogensis (half- chromatid mutation) or after fertilization (somatic mutation). As expected for a denovo mutation, there is generally no antecedent family history; rare exceptions have been attributed to inheritance of an unstable permutation.[26]

I) VERRUCOUS EPIDERMAL NEVUS

Verrucous epidermal nevi are congenital, non- inflammatory cutaneous hamartomas composed of keratinocytes. Their prevalence in adults is probably 0.1–0.5%, and they occur equally in males and females. [36] They are divided into epidermolytic and non-epidermolytic types based on histopathology and pathogenesis. The epidermolytic verrucous epidermal nevi represents a clone of cells expressing a mutation in one of the BIE genes *KRT1* and *KRT10* involved in the pathogenesis of the autosomal dominant bullous ichthyosiform erythroderma (BIE). Recently, FGFR3 mutations associated with severe achondroplasia has been found in non- epidermolytic verrucous epidermal nevi, hence may represent an autosomal dominant lethal disorder rescued by mosaicism.[26]

These are often present at birth as slightly pigmented velvety or warty streaks or plaques. With age they darken and the surface becomes more warty. They may extend on to an adjacent mucosal surface. Nail ridging, splitting, discoloration or dystrophy may occur where the nail fold is involved. A wide variety of

developmental anomalies may occur in association with verrucous epidermal nevi of the non- epidermolytic type. They include localized anomalies such as megalopinna and aplasia cutis of the scalp, and syndromes such as epidermal nevus syndrome etc.[36]

II) ACNE NEVUS AND NEVUS COMEDONICUS SYNDROME

Nevus comedonicus was first described in 1895.[38]This rare nevus is usually present at birth, but becomes prominent at puberty, when the sebo- follicular element is stimulated by hormones.[26]

Munro observed that the acneiform lesions of nevus comedonicus and the localized skeletal malformations of nevus comedonicus syndrome are generalized in the autosomal dominant disorder Apert Syndrome. This syndrome is due to mutations on FGFR2 and thus comedo nevus is probably due to mosaicism for FGFR2 mutation.[39]This may present as several lesions in a linear, unilateral or, more rarely, bilateral distribution.

III) LINEAR DARIER'S DISEASE

Darier's disease (DD) in a unilateral or localized pattern was first reported at the turn of the last century. This variant often lacks a positive family history and other features that are associated with typical Darier's disease. Otherwise,

morphologically and histologically, linear Darier disease is identical to the generalized form.[40]

Starink and Woerdeman reported seven cases showing unilateral, linear, or zosteriform patterns, without other findings of DD and suggested the name acantholytic dyskeratotic epidermal nevus.[41]

IV) OTHER DISORDERS

Other autosomal dominant conditions following Blaschko's lines include Hailey-Hailey disease, linear porokeratosis, linear basal cell nevus, segmental Neurofibromatosis –type I and linear angiofibromas in Tuberous sclerosis.[26]

V) OTHER LINEAR BENIGN TUMORS

Syringomas, trichoepitheliomas and eccrine spiradenomas are heritable ectodermal tumors and can occasionally occur in a linear distribution.[26]

C) LINEAR INFLAMMATORY DISORDERS

Several inflammatory skin conditions may occasionally be distributed in Blaschko's lines. Most of these disorders, in their generalized form, seem to be multifactorial in origin. They show a familial susceptibility, suggesting the involvement of atleast one gene in their pathogenesis. They usually appear years after birth, implicating environmental contributions. Several display the Koebner's phenomenon as well, implying susceptibility factors localized to the skin. The

linear form of these disorders may therefore reflect mosaicism for a 'susceptibility' mutation.[26] This loss of heterozygosity may occur from a mutation, deletion, or DNA recombination and lead to the formation of a keratinocyte clone that is more susceptible to development of the skin disease. This concept was first introduced in 1991 to describe linear psoriasis and, since then, has been applied to segmental forms of atopic dermatitis, lichen planus, erythema multiforme, pemphigus vulgaris, vitiligo, granuloma annulare and so on.[42,43] Segmental disease may be superimposed on non-segmental lesions, with the segmental lesions usually being more difficult to treat.[44]

I) LICHEN STRIATUS

(Synonyms- Linear Lichenoid Dermatosis)

Lichen striatus is an asymptomatic, uncommon, self- limited, linear dermatosis of unknown etiology that generally affects children. First described by Balzer and Mercier in 1898, and they termed it 'lichenoid trophoneurosis'. Forty years later, Senear & Caro proposed the name 'lichen striatus'.[45]

Lichen striatus is seen primarily in children between the ages of 4 months and 15 years. In 1988, Burton et al. wrote in their Textbook of Dermatology that LS may rarely be seen in adults.[46] The median age of onset is 2 to 3 years and the vast majority of cases occur in preschool - age children.[47]Charles et al. noted that

females are affected two or three times as frequently as males and that the eruption of LS usually involutes within 1year.[48]In certain other studies, reported male: female ratio has varied from 1:1.6 to 1:2.[49]

Although the distribution of lichen striatus along the Blaschko's lines points to somatic mosaicism, neither the genes involved nor the triggering factors are known.

Environmental agents, particularly viruses, have been implicated, given the predominance of the disorder in young children and its seasonal variation (it appears to be more common in summer and spring). However, to date, a viral association has not been proven via serologic testing or cultures.

In theory, during early fetal development, an aberrant clone(s) of epidermal cells produced by somatic mutation migrated out along the lines of Blaschko. Exposure to an infectious agent (e.g. virus, BCG vaccine) or other precipitant could then break previous tolerance to the aberrant clone inducing a novel membrane antigen.[45]

Lichen striatus may represent a manifestation of an atopic diathesis. A retrospective analysis by Patrizi A et al of 115 children with lichen striatus revealed an association with atopic dermatitis in 70 of the cases.[47]In another series, of 26 patients, 80% were atopic.[50]

There are scattered reports of lichen striatus occurring at the sites of injury (e.g. the periphery of a burn scar[49] rather than along the Blaschko's lines. Lastly, axial distribution has been reported in some cases.[51]

The eruption consists of a continuous or interrupted band composed of discrete or clustered pink, skin- colored or tan papules that are flat-topped, smooth or scaly, range in diameter from 2 to 4mm.Infrequently, vesicles may be present. Hypopigmentation may be prominent, especially in dark- skinned persons. Typically, there is a single, unilateral streak on an extremity along the Blaschko's lines; occasionally, there is a bilateral distribution pattern and /or multiple parallel bands.[45]In two-third of patients, an extremity is involved, in particular the arm; in the remainder, the trunk or face is involved.[52, 53] When lesions extend to the ends of the digits, nail involvement may range from fraying, splitting, onycholysis to total nail loss.[54, 55]

The eruption usually appears suddenly, develops fully over days to weeks, and after several months to a year or more, undergoes spontaneous resolution, leaving post-inflammatory hypopigmentation.[45]

II) LINEAR LICHEN PLANUS

Scattered linear lesions often occur in patients with lichen planus (LP) and are a result of scratching and the Koebner phenomenon.[15]Less commonly, unilateral streaks or bands of LP are seen that are longer and wider than the trauma- induced

lesions seen along the Blaschko's lines.[56]Some authors consider linear LP as an intermediate entity between LP and lichen striatus. This form has also been referred as zosteriform but the distribution pattern of LP is not dermatomal (with the rare exception of the koebnerization of LP into the site of a previous herpes zoster infection). Linear LP accounts for less than 0.2 percent of all patients with LP,[44] except in Japan, where up to 10% of reported cases are linear.[57] Because of the polygenic nature of LP, family members may have the condition in its non - segmental form. LP occurs in up to ten percent of first-degree relatives of affected patients.[44]

The linear variant of LP can be persistent,[58] but occasionally may resolve with post inflammatory hyperpigmentation.[59]Usual sites involved are the extremities.[60] Isolated linear lesions are more common in childhood.[61]Linear LP lesions are usually only a few centimeters in length, but long, narrow linear lesions extending along the whole length of a limb may occur. Such cases may overlap with epidermal nevi, and the term lichenoid epidermal nevus has been introduced by Brownstein et al.[61]

If the LP lesions extend to the end of a digit, the nail is often affected. Linear lichen plano- pilaris has been reported, usually on the face.[62]

III) SEGMENTAL (LINEAR) VITILIGO

Vitiligo is a multifactorial disorder that occasionally occurs in a linear distribution. The lesions tend to be broad bands, patches or blocks, corresponding more to dermatomes than Blaschko's lines, perhaps in keeping with a neuronal pathogenesis. This idea is consistent with mosaicism, as the neuronal abnormality could be mosaic, or alternatively, there could be a clonal susceptibility of melanocytes to neuronal or other influences.

Compared with symmetric vitiligo, the linear type is earlier in onset, less likely to spread to other areas of the body, and less frequently associated with other autoimmune diseases.[26]

IV) LINEAR MORPHEA

Linear morphea occurs as a linear band, usually with a single unilateral lesion. The lower extremities are most often involved, followed, in frequency of occurrence, by the upper extremities, frontal area of the head, and anterior thorax.[63]The female-to-male ratio is 4:1. Linear scleroderma tends to affect children and adolescents.

Whether linear morphea follows Blaschko's lines is controversial.[64] Jackson first described it,[2] but later observed that although linear morphea was thought to follow Blaschko's lines it was probably dermatomal.[65] In a detailed review, Bolognia et al could not find a single case of linear morphea following Blaschko's lines.[15]

Subsequently, only few papers described this finding.[66]Three patients with fronto-parietal scleroderma showing multiple lesions have been described. The lesions were in two different lines that seemed to belong to Blaschko's lines.[67]

However, in many cases of linear morphea, it is not clear whether the distribution is segmental, dermatomal or following Blaschko'slines.[15]

V) LINEAR LICHEN SCLEROSUS ET ATROPHICUS

Lichen sclerosus et atrophicus (LSA) is an inflammatory dermatosis of an unclear pathogenesis.LSA was first described by Hallopeau in 1887.[68]Darier described the characteristic histological findings in 1892.[69]It primarily affects the vulvar, perineal and perianal skin of prepubertal, perimenopausal and postmenopausal women.

It is sometimes distributed following Blaschko lines.[70] The first case report of a linear LSA was described in 1995 by Izumi and Tajima.[71] Thereafter, a handful of cases of linear LSA have been reported, among which some developed in a pattern corresponding to the lines of Blaschko. Kim and Lee [72]have summarized 6 cases of linear LSA along the Blaschko's lines, and this occurred on the trunk, limbs or face. Out of the 3 reported cases of linear LSA that appeared on the face, one case showed facial lesion following the Blaschko's line without any oral mucosal lesion, and the other 2 cases showed additional oral mucosal involvement.[72-74]

In the first stages it presents as interfollicular, pearly, polygonal papules, which merge to form atrophic, sclerotic plaques. In more advanced stages, follicular hyperkeratosis and telangiectasias are seen.[75] Histologically, LSA has a characteristic pattern.

VI) LINEAR LICHEN PLANUS PIGMENTOSUS

Lichen planus pigmentosus clinically differs from the classic lichen planus by exhibiting dark brown macules and/or papules, mottled or reticulated hyperpigmentation and a longer clinical course without scalp, nail, or mucosal involvement. It is most common in sun-exposed areas such as the face and neck and the flexural folds, including axillary, inguinal, and submammary regions.[76] Some authors observed a striking predominance of lesions in intertriginous locations, among which axillae are the most common.[77] The most common pattern of pigmentation is diffuse, whereas less common patterns include linear, reticular, and perifollicular patterns.[77]

There are few reports in the literature of unilateral linear LPP. All except one of these cases were females with absence of involvement of mucosa and nails. Their ages varied from 16- 60 years. The sites involved were predominantly the lower extremities and the neck in one patient. One case of bilateral linear LPP in a male in association with hepatitis C infection has also been reported. All the above

patients were asymptomatic and all the lesions in them were along the Blaschko's lines alone.[78]

VII) 'ADULT BLASCHKITIS'-

(Synonyms- Blaschko linear acquired inflammatory skin eruption (BLAISE); Blaschkitis, or, Idiopathic dermatitis along the lines of Blaschko) [79]

It was first described in 1990 by Grosshans and Marot in Bordex, France.[80]

It presents as pruritic papules and vesicles along multiple lines of Blaschko, particularly on the trunk. It follows a relapsing course, with individual episodes resolving spontaneously within days. This disorder may represent an adult counterpart of lichen striatus, but the histology is more eczematous and less lichenoid.[81]

Some authors are trying to find a difference between adult blaschkitis and lichen striatus, but the discussion remains controversial. In trying to unite these two entities, the term 'Blaschko linear acquired inflammatory skin eruption' (BLAISE) has been created.

VIII) NEVOID PSORIASIS

Psoriasis occasionally occurs in a 'nevoid' form,[82] possibly reflecting mosaicism for a gene responsible for psoriasis. Linear psoriasis coexisting with generalized psoriasis may represent loss of heterozygosity for a psoriasis gene. The nevoid

psoriasis might co-exist with or develop as an isomorphic phenomenon [83-86] over a pre-existing inflammatory linear verrucous epidermal nevus (ILVEN). Linear psoriasis is extremely rare with many early cases having been subsequently reclassified as ILVEN.

Onset is usually during childhood, but some otherwise typical cases have presented at birth. In a study of 419 cases of childhood psoriasis from north India by Kumar B et al, infantile psoriasis (below 1 year of age) accounted for 3.5% of patients, the youngest being a four day old child, but none had lesions at birth.[87] The lesions are clinically indistinguishable from ordinary psoriasis other than in their distribution, and are histologically identical. Linear psoriasis can be associated with nail pits and psoriatic arthritis. This condition responds to anti-psoriatic therapy such as ultraviolet radiation or dithranol. Linear psoriasis can distinguished from ILVEN by its minimal pruritus and therapeutic responsiveness.

IX) LINEAR LICHEN NITIDUS

Lichen nitidus (LN) is a disorder of unknown etiology. Most cases occur in children or young adults. Typical LN papules are minute, pinpoint to pinhead sized, and have a flat or dome-shaped, shiny surface. They usually remain discrete, although they may be closely grouped. They are found on any part of the body but

the sites of predilection are the forearms, penis, abdomen, chest and buttocks. The palms or soles can be involved in form of confluent hyperkeratosis.[88]

Mucous membrane lesions occur occasionally. Krook [89] described a generalized case as having mucosal lesions mainly on the hard palate and maxillary alveolar margins, which consisted of fairly closely grouped, greyish yellow, round, sharply demarcated, discrete papules up to 1 mm in diameter. Linear LN has been described by Pringent F et al, but is exceptionally rare.[90]

X) OTHER LINEAR INFLAMMATORY DISORDERS

Several other inflammatory disorders have been reported in a linear distribution, suggestive of mosaicism for a susceptibility mutation. They include lupus erythematosus, fixed eruption, atrophoderma of Pasini and Pierini (linear atrophoderma of Moulin), chronic lichenoid GVHD, mycosis fungoides and mucinosis.[26]

D) LETHAL DISORDERS RESCUED BY MOSAICISM

For some linear disorders no generalized counterparts can be recognized. These cases probably represent mosaicism for a mutation that would be incompatible with life if it involved all cells. Such conditions affect both men and women and can reflect autosomal dominant and X-linked mutations.[91] Alternatively they could

represent clonal loss of heterozygosity in a phenotypically normal carrier of a lethal recessive mutation.

I) NON- EPIDERMOLYTIC VERRUCOUS EPIDERMAL NEVUS- (see verrucous epidermal nevus)

II) EPIDERMAL NEVUS SYNDROME

(Synonyms- Sebaceous nevus syndrome, Schimmelpenning's syndrome, Feuerstein–Mims syndrome, Organoid nevus syndrome, Jadassohn's nevus phakomatosis)

Epidermal nevus syndrome (ENS) describes the association of sebaceous and/or verrucous nevi with other developmental defects, particularly of the central nervous system (CNS), eye and skeleton. This was first reported by Feuerstein and Mims[92] and Schimmelpenning [93] and named by Solomon in 1975.[94] Happle in 1995 suggested that no one epidermal nevus syndrome exists, but rather includes at least six separate disorders, namely: Schimmelpenning's syndrome, Proteus syndrome, comedo nevus syndrome, CHILD syndrome, Becker's nevus syndrome and phakomatosis pigmentokeratotica.[95] The term 'epidermal nevus syndrome' as used here does not include syndromes where the nevus is not a sebaceous or verrucous epidermal nevus.

ENS occurs in a sporadic fashion, probably reflecting genetic mosaicism for an autosomal dominant mutation which would be lethal if not 'rescued' by mosaicism.

Other skin manifestations include hypo – or hyperpigmentation, café-au- lait macules, hamangiomas, aplasia cutis congenita and melanocytic nevi.[26]

Significant developmental anomalies occur in approximately1.7% of all neonates [96] and 10% of children with epidermal nevi, the risk correlating poorly with number and extent of lesions. The most common systemic abnormalities are neurological seen in 50% of these patients.[97] Neurological abnormalities are much more frequent in patients who have sebaceous nevi on the head and neck,[97] but the location of the skin lesions does not provide a reliable prediction of the laterality of intracranial brain anomalies [98] which include hemi- megaloencephaly, seizures, hemiparesis and retardation.

Some 35–70% of patients have ocular abnormalities, the commonest of which is involvement of the eyelid or conjunctiva by the epidermal nevus, sometimes causing trichiasis or interfering with lid closure. Other ocular problems have included colobomas of the eyelid, iris and retina, choristomas, optic nerve hypoplasia, retinal dysplasia, cortical blindness, microphthalmia, macrophthalmia, anophthalmia, corneal opacities and cataracts.[99]

Skeleton abnormalities include asymmetry, abnormal skull shape, kyphoscoliosis and limb hypertrophy.[26]

Other reported associations include cardiac and genitourinary anomalies.[26] Endocrine disease, including inappropriate antidiuretic hormone (ADH) secretion and precocious puberty.[100]

Benign and malignant transformation may occur in these patients, exactly as it may when such nevi exist without associated abnormalities. A relatively high incidence of systemic malignancies has been found in these patients, often at a very early age.[101]

III) INFLAMMATORY LINEAR VERRUCOUS EPIDERMAL NEVUS (ILVEN) (Synonym- Dermatitic epidermal nevus)

Altman and Mehregan first coined the phrase "inflammatory linear verrucous epidermal nevus" to describe a subset of epidermal nevi that were erythematous , inflamed, and pruritic. These nevi follow Blaschko's lines.[15]

ILVEN is probably due to mosaicism for a dominant mutation, as yet unidentified, which would be lethal if it affected all cells and is 'rescued' by mosaicism.[102] ILVEN is usually sporadic, but there have been reports of familial cases.[103]

Although the lesions may be present at birth, the majority of ILVEN appear during infancy and childhood. Fifty percent are evident by 6 months of life and 75% by 5

years of age.[104]About 25% extend beyond their initial presentation, usually over a few months, but in one case extended over 9 years.[105]

They are characterized by pruritus, which may be intense. The lesions are linear, most commonly on a limb, and comprise eczematous or psoriasiform papules. There is a slight preference for the left side.[104] Occasionally, ILVEN is bilateral and widespread. Nail dystrophy may occur when the nail fold is affected.[106]

Unlike the non- inflammatory epidermal nevi, the ILVEN is not associated with the neurologic defects. Rarely, there are ipsilateral skeletal abnormalities, usually reduction deformities, suggesting that possibly ILVEN is a forme-fruste of CHILD syndrome. An alternative acronym is PEN/ PENCIL (psoriasiform epidermal nevus + congenital ipsilateral limb defects).[107]

ILVEN can be distinguished from true nevoid psoriasis by pruritus and lack of response to anti-psoriatic treatments.[108]

IV) NEVUS SEBACEOUS

Nevus Sebaceous was first described by Jadasshon in 1895.These are present in approximately 0.3% of newborns and appear as a waxy to verrucous plaque.[109] Although usually congenital, they are occasionally first reported later in life.[110] The sex incidence is equal.[36] Typically, there is a yellow to orange hue that reflects hyperplasia of sebaceous glands. Although the most common location is the head

and neck, [36] they can occur on the extremities as well as the trunk. The distribution of sebaceous nevi is along the lines of Blaschko, but this may be difficult to appreciate on the scalp, face or neck.[15]They can extend on to the oral mucosa.[111]

V) OTHER DISORDERS

Some other probably lethal diseases that can involve the Blaschko's lines and are rescued by mosaicism include porokeratotic eccrine ostial and dermal duct nevus (PEODDN), encephalocraniocutaneous lipomatosis, oculocerebro- cutaneous syndrome, Mc-Cune – Albright syndrome, zosteriform speckled lentiginous nevus, linear fibromatosis and localized vascular malformations.[26]

E) CHROMOSOMAL MOSAICISM

I) HYPOMELANOSIS OF ITO

(Synonyms- Linear Nevoid Hypopigmentation, Pigmentary Mosaicism, Incontinentia pigmenti achromians -historical)

The term hypomelanosis of Ito (HI) is a description rather than a diagnosis. The phenotype of this multisystem disorder is highly variable, except in the skin, where it always presents as hypopigmentation following Blaschko's lines.[26]

HI was first described by Japanese dermatologist Minor Ito in 1952. [112] HI is the third most common neurocutaneous disorder, after neurofibromatosis and tuberous

sclerosis.[113] It is diagnosed in 1 per 8000-10000 unselected patients in general paediatric outpatient clinic and 1 out of every 790 in a paediatric dermatology clinic.[114] Unlike incontinentia pigmenti, HI affects both sexes. It probably occurs in all races, but is easier to see in darkly pigmented skin.[26]

There have been a few reports of familial HI, but the majority of cases are sporadic.[115] Chromosomal mosaicism can be identified in the blood in about a third of patients. The mosaic karyotype anomalies reported include a variety of defects of chromosome structure and number and can affect autosomes or X chromosomes.[26] However, no consensus exists about the identity of the HI gene.

In contrast to the variable systemic manifestations, the consistency of the skin appearance is remarkable. Clinically, lesions appear at birth or infancy as asymmetric, whorled or streaked lesions in a "marble cake pattern" along the lines of Blaschko, occurring on any part of the body.[116] The hypopigmented streaks can be unilateral or bilateral. Less often the distribution is patchy sparing the palms and soles.[117] Lesions usually appear by 1 year of age in 77% of patients and an initial increase in extent of involvement can be followed by a gradual repigmentation. [118] A Wood's lamp enhances the pattern. When the scalp is involved, streaks of hypopigmented hairs are observed. [119] There is also evidence for somatic mosaicism in the eye in which striated and mottled hypopigmentation of the fundus and iris can occur in a pattern similar to that in X- linked ocular albinism.[120]

Congenital abnormalities, mental retardation, and seizures are the most commonly associated conditions, as reported in the medical literature. Cerebral malformations may occur and visual impairment may be cortical in nature.[121] Glomerulocystic kidney disease has been reported.[122] Other anomalies include cleft palate, hemi hypertrophy of limbs, hand and/or foot abnormalities, nail abnormalities, hypotonia, teeth abnormalities, hair anomalies, face and/or skull anomalies. The associated anomaly rate is approximately 30%.[26]

II) NEVUS DEPIGMENTOSUS

(Synonym- Nevus achromicus)

This is a localized area of depigmented skin, first described by Lesser in 1884. [123] It occurs in 1 in 50 – 75 individuals.[26]The name is a bit of a misnomer as the areas of leukoderma are actually hypomelanotic not amelanotic.

This circumscribed area of hypopigmentation is congenital, but may not be apparent at birth. There are three clinical variants- isolated, segmental and systematized.[124]The commonest variant is the single, isolated circumscribed, rounded lesion. Segmental and systematized forms are very rare, and may resemble hypomelanosis of Ito. The systematized form often consist of broad whorls and streaks that follow Blaschko's lines.[125,126] Block-like areas of hypopigmentation that respect the midline can also be seen.[127] Most lesions measure a few

centimeters in diameter and have irregular but well- defined borders . Hairs within the hypopigmented macules are usually depigmented.

The cutaneous findings in nevus depigmentosus are identical to those in HI, but are fixed and usually more limited in distribution; and more importantly extracutaneous associations are lacking.[26] However, neurological abnormalities such as seizures have been rarely reported.[129]

III) PHYLLOID HYPOMELANOSIS [26]

This is a newly recognized syndrome occurring in patients with mosaic trisomy 13. The hypomelanosis consists of: (1) round or oval lesions; (2) large asymmetric areas reminiscent of the leaves of a begonia; (3) and pear shaped areas or oblong macules. These patients also have associated CNS defects.

IV) LINEAR AND WHORLED NAEVOID HYPERMELANOSIS (LWNH)

This disorder was first described by different groups of authors in 1970s as a unique pigmentary condition. The features and nomenclature were clarified by Kalter and colleagues in 1988.[130]They characterized LWNH as follows: (1) onset within a few weeks of birth and then progressing for 1-2 years before stabilizing ; (2) linear and whorled nevoid hyperpigmentation following Blaschko's lines without preceding bullae or verrucae; (3) hyperpigmented areas with increased pigmentation of the basal layer and prominence of melanocytes without

incontinence of pigment; (4) sporadic male and female incidence; (5) sparing of mucous membranes, eyes, palms and soles; and (6) possible associations with congenital anomalies.

The exact pathogenesis is not known .Somatic mosaicism that develops during embryogenesis appears to be the underlying etiology.[26]

Patients can have swirls and streaks that follow Blaschko's lines as well as reticulated pigmentation. The trunk, extremities, neck, face and genitalia are the typical sites affected.[131,132,133]The pigmentation tends to persist indefinitely.[26] Associated systemic abnormalities include atrial septal defect, dextrocardia, deafness and neurological and musculoskeletal defects.[15]

Moulin et al [134] described five patients with linear atrophic streaks that were unilateral and followed Blaschko's lines. Onset occurred between 6 and 20 years and the pigmentation was caused by increased epidermal melanosis as in LNWH. Whether this disorder represents a tardive form of LNWH or is a separate entity awaits clarification.

MATERIALS AND METHODS

This study was conducted in the Out Patient Department of Government Rajaji Hospital, Madurai during the period October 2009 to September 2011(24 months).

INCLUSION CRITERIA

All consenting patients with -

1) Lesions related to Blaschko's lines whose diagnosis could be confirmed on clinical basis alone.

2) Lesions related to Blaschko's lines that were confirmed by histopathology where clinically there was a diagnostic dilemma.

EXCLUSION CRITERIA

1) Patients not willing to give consent.

2) Patients with lesions related to Blaschko's lines, but where the diagnosis could not be confirmed on clinical basis and histological and other relevant investigations could not be performed due to various reasons.

After applying the criteria mentioned above, cases were enrolled. After their informed consent thorough dermatological and systemic examination was conducted.

The parameters studied were the age of onset, sex, side of involvement, site, type of Blaschko pattern followed, symptoms, extension/ regression of the lesions and direction of extension of the lesions, associations, family history and birth history. No literature clearly defining the width of narrow and broad – band types of Blaschko's lines could be found. Hence, in this study we have arbitrarily categorized Blaschko's lines in the type 1 pattern with width less than 3 centimeters to be narrow – band and those with a width more than 3 centimeters to be broad- band .

Skin biopsy was done in cases where there was doubt in diagnosis. Relevant investigations like Chest X Ray, EEG, CT -Brain were carried out wherever necessary.

OBSERVATIONS AND RESULTS

In this study, 218 cases with lesions following Blaschko's lines were encountered in the outpatient department of Dermatology, Government Rajaji hospital, Madurai during the period of October 2009 – September 2011. The following observations were made.

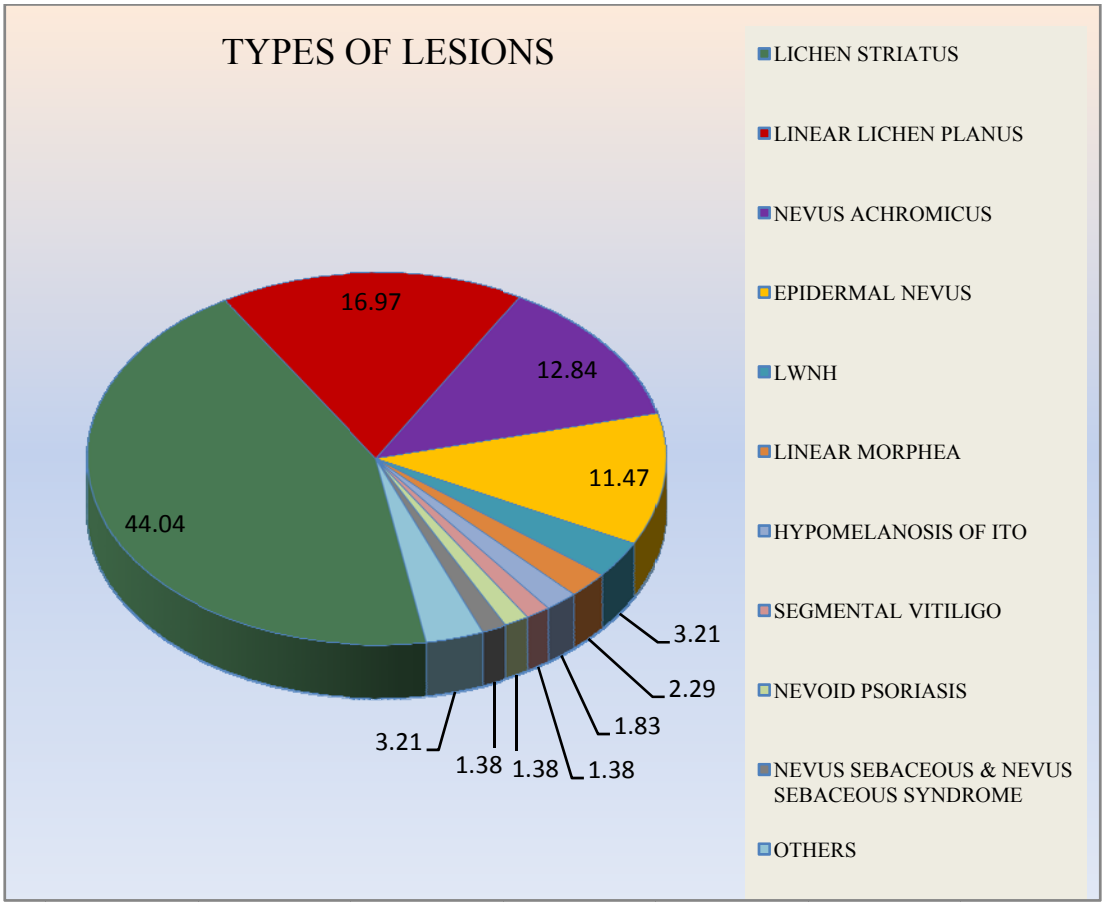
I) INCIDENCE:

The overall incidence of lesions following the lines of Blaschko was found to be 2.1 per 1000 cases with dermatological complaints. The incidence in males was 2 per 1000 and in females was 3 per 1000 dermatology patients.

II) TYPE OF LESIONS:

Of the 218 patients, 96 (44.04%) cases had lichen striatus followed by linear LP in 37 (16.97%) cases, nevus achromicus with 28 (12.84%) cases, epidermal nevus with 25 (11.4%) cases. There were 7 (3.21%) cases of LWNH, 5 (2.29%) cases of linear morphea, 4 (1.83%)cases of HI, 3 (1.38%)cases each of linear vitiligo and nevoid psoriasis; and 2 (0.92%) cases of nevus sebaceous and one(0.46%) case of nevus sebaceous syndrome. Other diseases included Darier's disease, IP, LSA, lichen planus pigmentosus, lichen nitidus, dyschromatosis universalis & soft fibroma in one (0.46%) case each. (Figure 6)

FIGURE 6: DISTRIBUTION OF TYPES OF LESIONS



Others- Incontinentia Pigmenti, Darier's Disease, Dyschromatosis Universalis Hereditaria, LSA, Lichen Nitidus, Lichen Planus Pigmentosus, Soft Fibroma

III) SEX RATIO

In our study 93 cases were males and 125 cases were male, the overall male: female ratio being 0.75: 1. (Table III)

TABLE III- SEX DISTRIBUTION

DISEASE	MALE	FEMALE	M:F	TOTAL
Lichen Striatus	39	57	0.7:1	96
Linear Lichen Planus	9	28	0.3:1	37
Nevus Achromicus	17	11	1.5:1	28
Epidermal Nevus	15	10	1.5:1	25
LWNH	4	3	1.3:1	7
Linear Morphea	2	3	0.7:1	5
Hypomelanosis Of Ito	2	2	1:1	4
Segmental Vitiligo	1	2	1:2	3
Nevoid Psoriasis	1	2	1:2	3
Nevus Sebaceous & Nevus Sebaceous Syndrome	3	0	-	3
Others	0	7	-	7
Total	93	125	0.75:1	218

OTHERS- Darier's Disease, Incontinentia pigmenti, Dyschromatosis universalis hereditaria, Lichen nitidus, LSA, Lichen Planus Pigmentosus, Soft fibroma

On considering the individual diseases, male predominance was seen in nevus achromicus, epidermal nevus and LWNH. Equal sex ratio was found in hypomelanosis of Ito. Female predominance was noted in lichen striatus, linear LP, linear vitiligo and nevoid psoriasis. All the cases of nevus sebaceous were males.

The patients in the other diseases like Darier's disease, IP, LSA, Lichen planus pigmentosus, lichen nitidus, dyschromatosis universalis and soft fibroma were all females. (Figure 7)

IV) AGE AT ONSET – DISTRIBUTION :

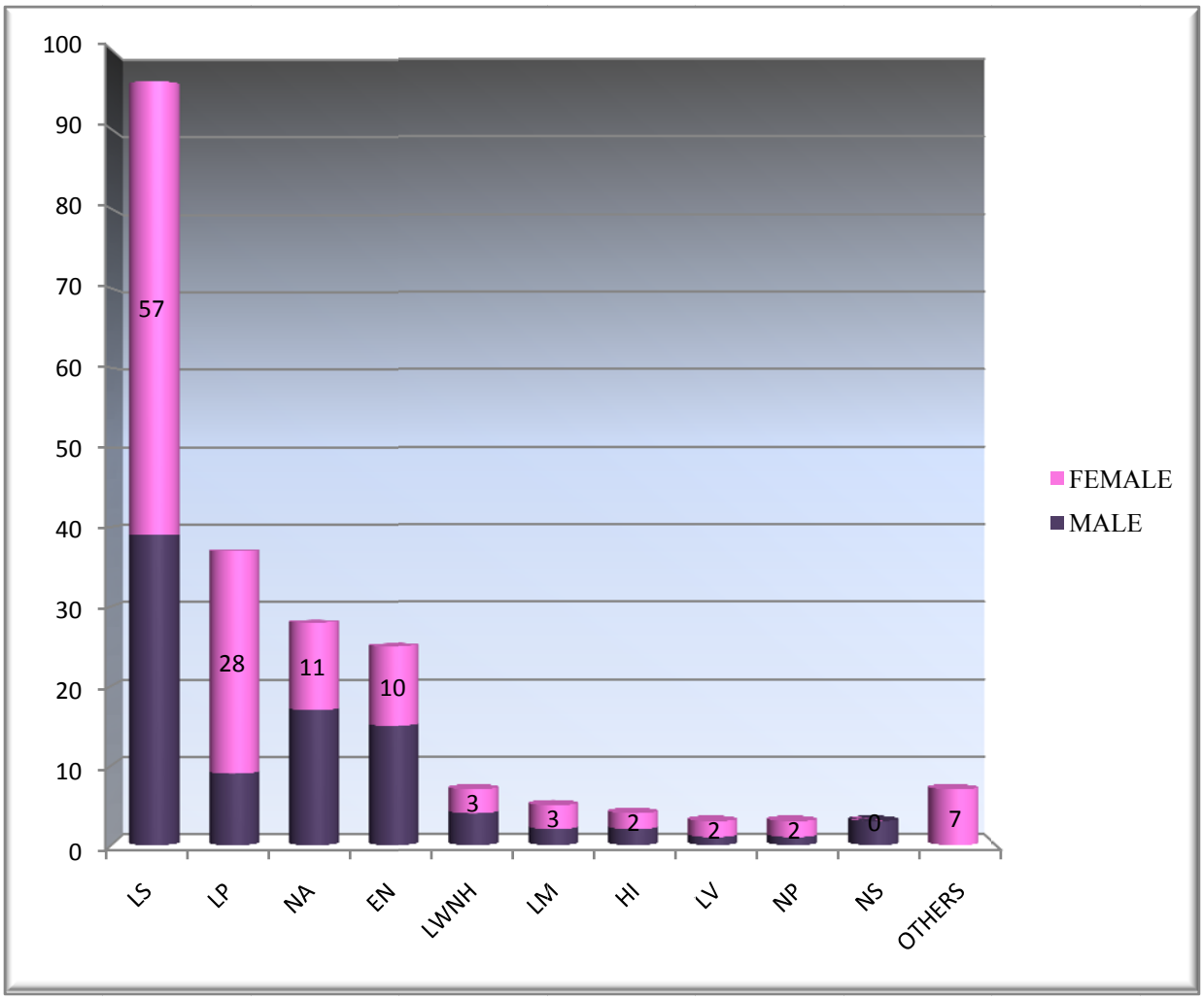
The combined (male + female) age at onset in various diseases is shown.

TABLE IV- AGE AT ONSET DISTRIBUTION

AGE / Δ	0-12m	1-5y	6-10y	11-20y	21-30y	31-40y	41-50y	>50y	NR	TOTAL
LS	4	52	23	16	1	-	-	-	-	96
LP	-	3	2	9	5	7	5	6	-	37
NA	26	-	-	1	1	-	-	-	-	28
EN	21	-	1	1	-	-	-	-	1	25
LWNH	5	-	1	-	-	-	-	-	1	7
LM	-	-	1	3	1	-	-	-	-	5
HI	2	-	2	-	-	-	-	-	-	4
LV	-	2	1	-	-	-	-	-	-	3
NP	3	-	-	-	-	-	-	-	-	3
NS	2	-	-	-	1	-	-	-	-	3
DD	-	-	-	-	-	-	-	1	-	1
IP	1	-	-	-	-	-	-	-	-	1
LSA	-	-	-	1	-	-	-	-	-	1
LPP	-	-	-	1	-	-	-	-	-	1
LN	-	-	1	-	-	-	-	-	-	1
S Fibroma	-	-	-	-	-	1	-	-	-	1
DUH	-	1	-	-	-	-	-	-	-	1
TOTAL	65	58	32	32	9	8	5	7	2	218

LS- lichen striatus, LP- lichen planus, NA- nevus achromicus, EN- epidermal nevus, LM- linear morphea, HI- hypomelanosis of Ito, LV- linear vitiligo, NP- nevoid psoriasis, NS- nevus sebaceous & nevus sebaceous syndrome, LPP- Lichen Planus Pigmentosus, LN- Lichen nitidus, S Fibroma- soft fibroma, DUH- dyschromatosis universalis hereditaria, DD – Darier's disease.

FIGURE 7: SEX- WISE DISTRIBUTION



LS- Lichen striatus, LP- linear lichen planus, NA- nevus achromicus, EN- Epidermal Nevus,LM- Linear Morphea,HI- Hypomelanosis Of Ito, LV-Linear Vitiligo, NP-Nevoid Psoriasis, NS-Nevus Sebaceous & Nevus Sebaceous Syndrome, Others- Incontinentia Pigmenti, Darier's Disease, Dyschromatosis Universalis Hereditaria, LSA, Lichen Nitidus, Lichen Planus Pigmentosus,Soft Fibroma

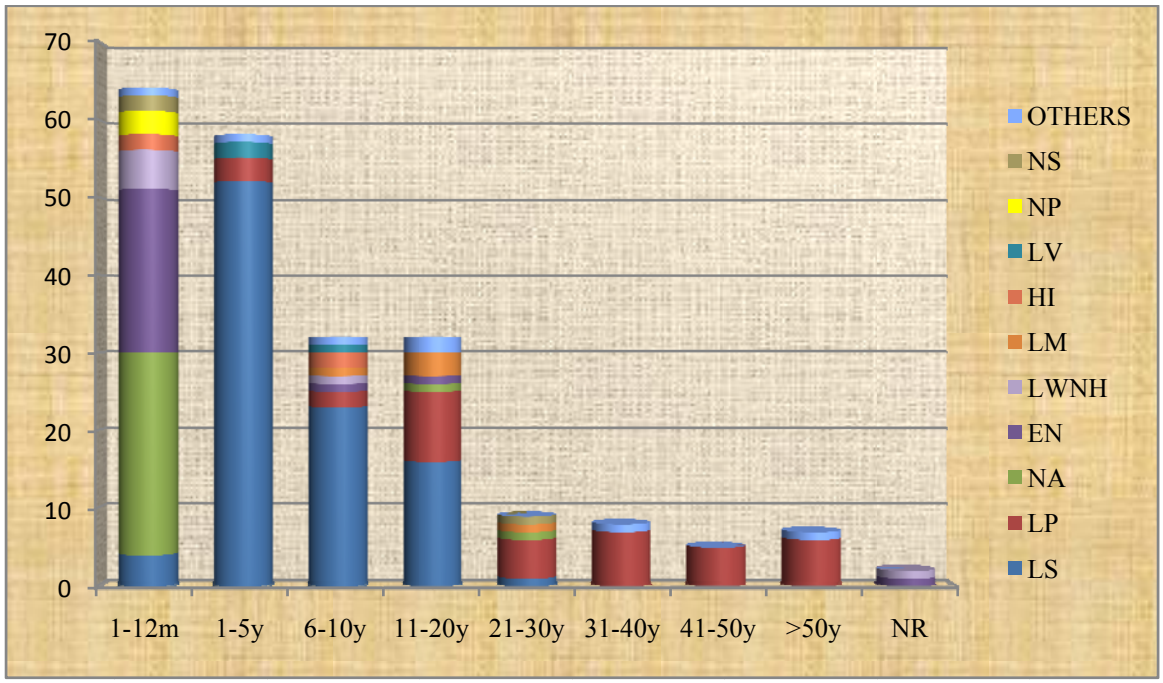
The maximum number of cases {65 (29%)} occurred in the first year, more so in the first 1month with 56 (25.7%) cases , representative of epidermal nevi and pigmentary disorders which included nevus achromicus, LWNH and Hypomelanosis of Ito. The second most common age group was 1-5 years with 58 (25.8%) cases predominantly constituted by lichen striatus. (Figure 8)

AGE AT ONSET DISTRIBUTION- MALES AND FEMALES

The age at onset distribution in males and females separately is similar to that of the total distribution. However in males, the maximum number of cases occurred in the age group 0-12 months with 36 (38.7%) cases, followed by 25(26.9%) cases in 1-5 years, 13(14%) cases in the ages 11-20 years and 10 (10.7%) cases in the ages 6-10 years. The number of cases with age at onset above 20 years was 9(9.7%).

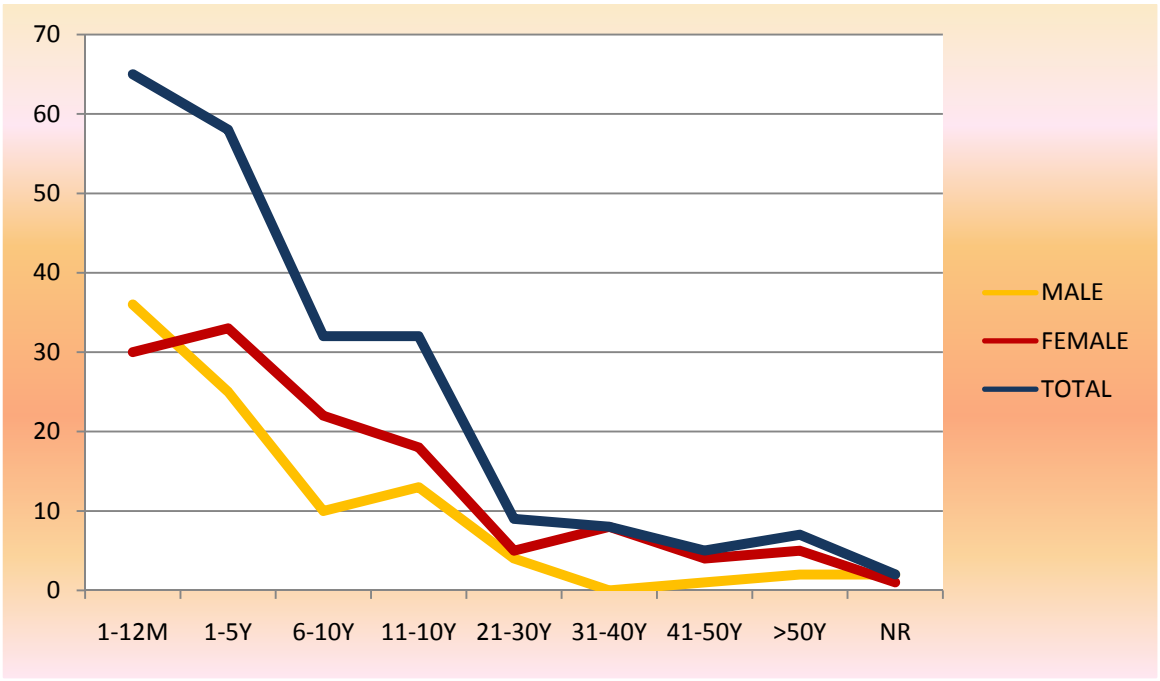
In females, the maximum number of cases {33 (26.4%)} occurred in the age-group of 1-5 years, closely followed by 30(24%) cases in the first year. However, as compared to the age distribution in males, the number of cases above 21 years was 22 (17-6%). Twenty – two (17.6%) cases developed lesions between 6- 10 years and 18 (14.4%) cases developed lesions in the ages 11-20 years. (Figure 9)

FIGURE 8: DISTRIBUTION OF AGE AT ONSET- TOTAL



LS- Lichen striatus, LP- linear lichen planus, NA- nevus achromicus, EN- Epidermal Nevus,LM- Linear Morphea,HI- Hypomelanosis Of Ito, LV-Linear Vitiligo, NP-Nevoid Psoriasis, NS-Nevus Sebaceous & Nevus Sebaceous Syndrome, Others- Incontinentia Pigmenti, Darier's Disease, Dyschromatosis Universalis Hereditaria, LSA, Lichen Nitidus, Lichen Planus Pigmentosus, Soft Fibroma , NR- not recalled

FIGURE 9 -COMPARATIVE DISTRIBUTION OF AGE OF ONSET IN MALES, FEMALES AND COMBINED



V) SIDE- WISE DISTRIBUTION

Overall, the right side and left sides each were involved in approximately 45% of the patients and both the sides were simultaneously involved in the remaining 10 % cases. On scrutinizing the individual diseases, significant right side predominance was observed in linear LP and left side predominance in nevus achromicus. Lichen striatus, linear vitiligo and nevoid psoriasis involved the right side preferentially while epidermal nevus, LWNH and nevus sebaceous preferentially involved the left side. Linear morphea showed equal occurrence on left and right side. (Table V)

TABLE V- SIDE OF INVOLVEMENT

DISEASE	RIGHT	LEFT	BOTH	TOTAL
Lichen Striatus	49	45	2	96
Linear Lichen Planus	21	13	3	37
Nevus Achromicus	5	16	7	28
Epidermal Nevus	10	13	2	25
LWNH	1	3	3	7
Linear Morphea	2	2	1	5
Hypomelanosis Of Ito	2	-	2	4
Linear Vitiligo	2	1	-	3
Nevoid Psoriasis	2	1	-	3
Nevus Sebaceous & Nevus Sebaceous Syndrome	-	3	-	3
Others	4	2	1	7
TOTAL	98	99	21	218

OTHERS- Darier's Disease, Incontinentia pigmenti, Dyschromatosis universalis hereditaria, Lichen nitidus, LSA, Lichen Planus Pigmentosus, Soft fibroma

VI) DISTRIBUTION OF TYPE OF BLASCHKO'S LINES

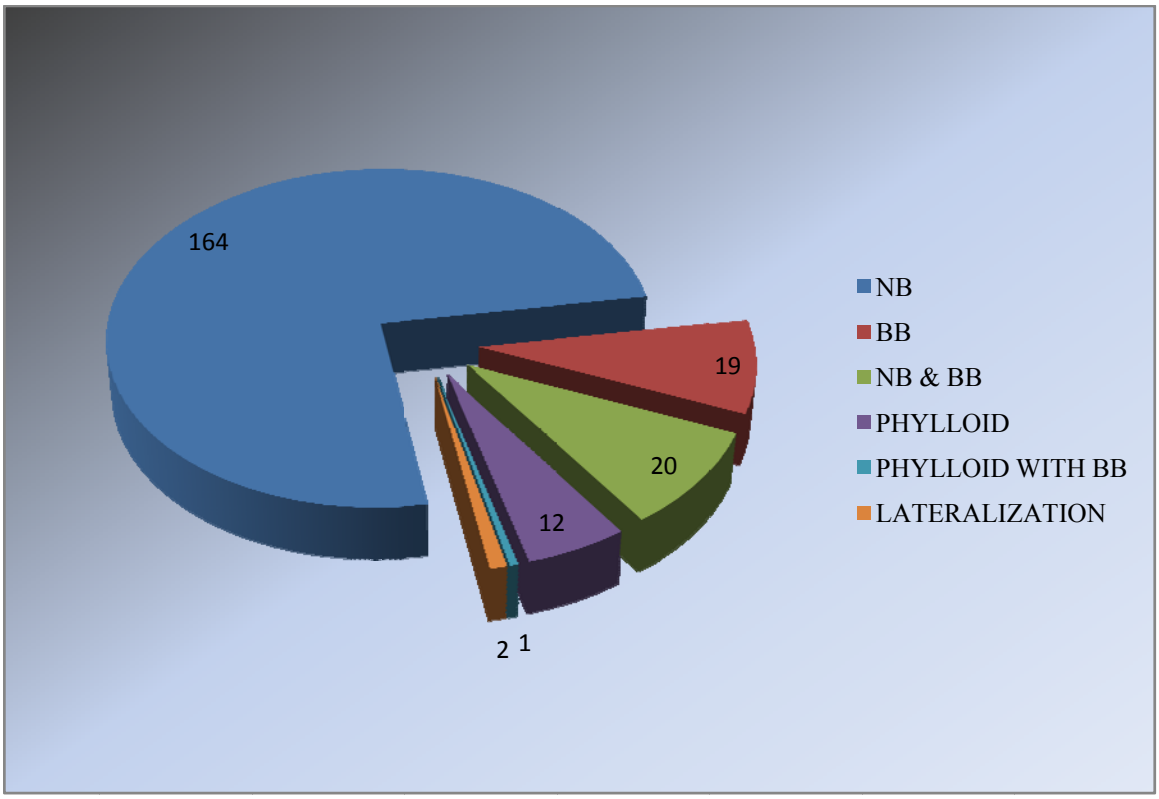
Overall, the most common type of Blaschko pattern observed was the narrow band type, followed by a combination of the narrow and broad- band types of Blaschko's lines, broad –band type alone and the phylloid pattern. Lateralization was observed in two patients with nevus achromicus and a combination of broad-band and phylloid in pattern in one patient. (Table VI and Figure 10)

TABLE VI- TYPE OF BLASCHKO'S LINES FOLLOWED

DISEASE	NB	BB	NB & BB	PHYLLOID	PHYLLOID WITH BB	LATERAL-IZATION
Lichen Striatus	87	2	7	-	-	
Linear Lichen Planus	33	2	2	-	-	
Nevus Achromicus	9	3	5	8	1	2
Epidermal Nevus	19	2	4	-	-	
LWNH	2	2	2	1	-	
Linear Morphea	-	5	-	-	-	
Hypomelanosis Of Ito	3	1	-	-	-	
Segmental Vitiligo	-	-	-	3	-	
Nevoid Psoriasis	3	-	-	-	-	
Nevus Sebaceous & Nevus Sebaceous Syndrome	2	1	-	-	-	
Others	6	1	-	-	-	
Total	164	19	20	12	1	2

OTHERS- Darier's Disease, Incontinentia pigmenti, Dyschromatosis universalis hereditaria, Lichen nitidus, LSA, Lichen Planus Pigmentosus, Soft fibroma

FIGURE 10: DISTRIBUTION OF TYPES OF BLASCHKO'S LINES



Narrow Band, BB- Broad Band

VII) **SITE DISTRIBUTION-** This is represented and discussed according to the individual diseases.

a) SITES OF INVOLVEMENT - LICHEN STRIATUS

Multiple sites were involved in 64.6 % patients. The single most common site involved was the thigh in this study. (Table VII and Figure 11)

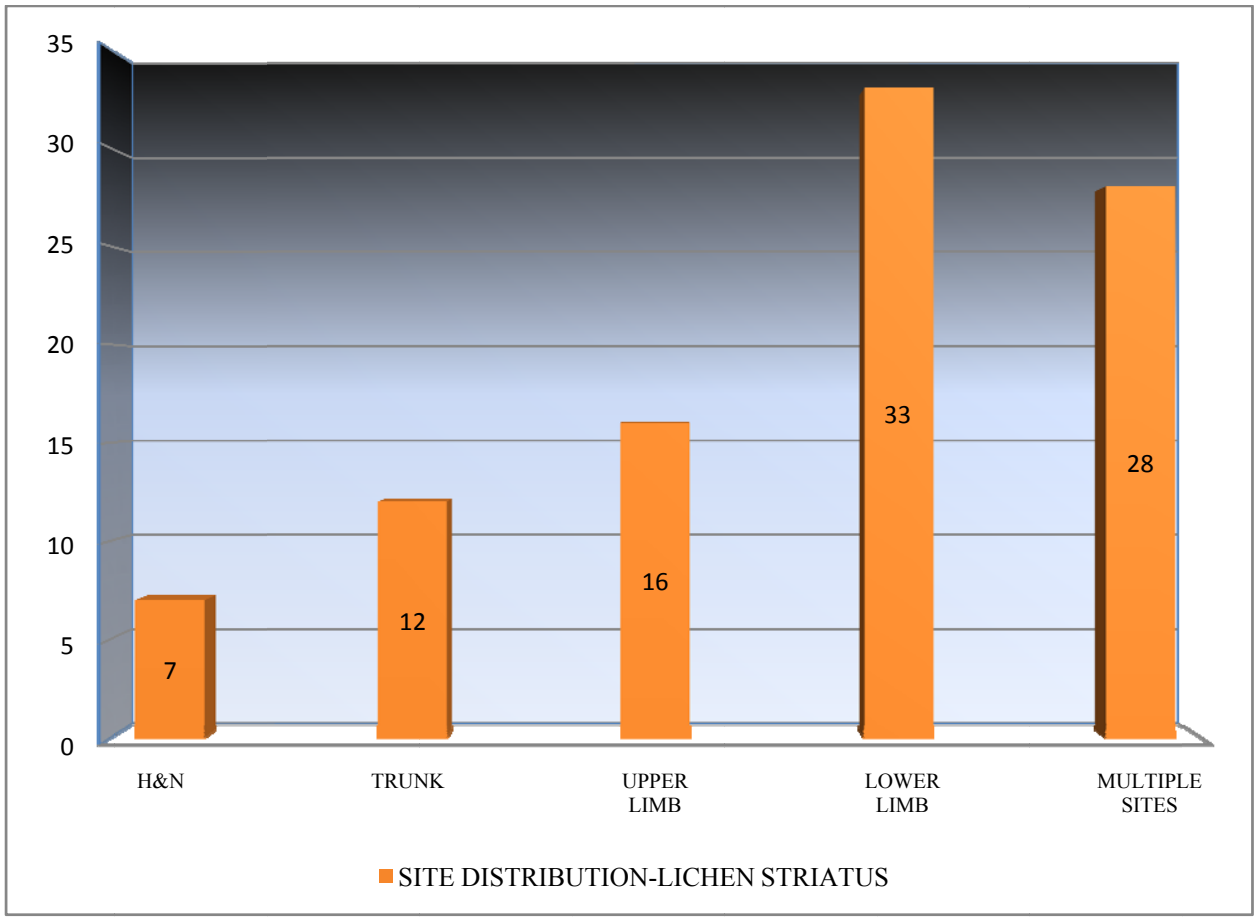
TABLE VII- SITE – WISE DISTRIBUTION- LICHEN STRIATUS

SITE	TOTAL
Head & neck	7
Trunk	12
Upper limb	16
Lower limb	33
Multiple sites	28
Total	96

b) SITES OF INVOLVEMENT- LINEAR LICHEN PLANUS

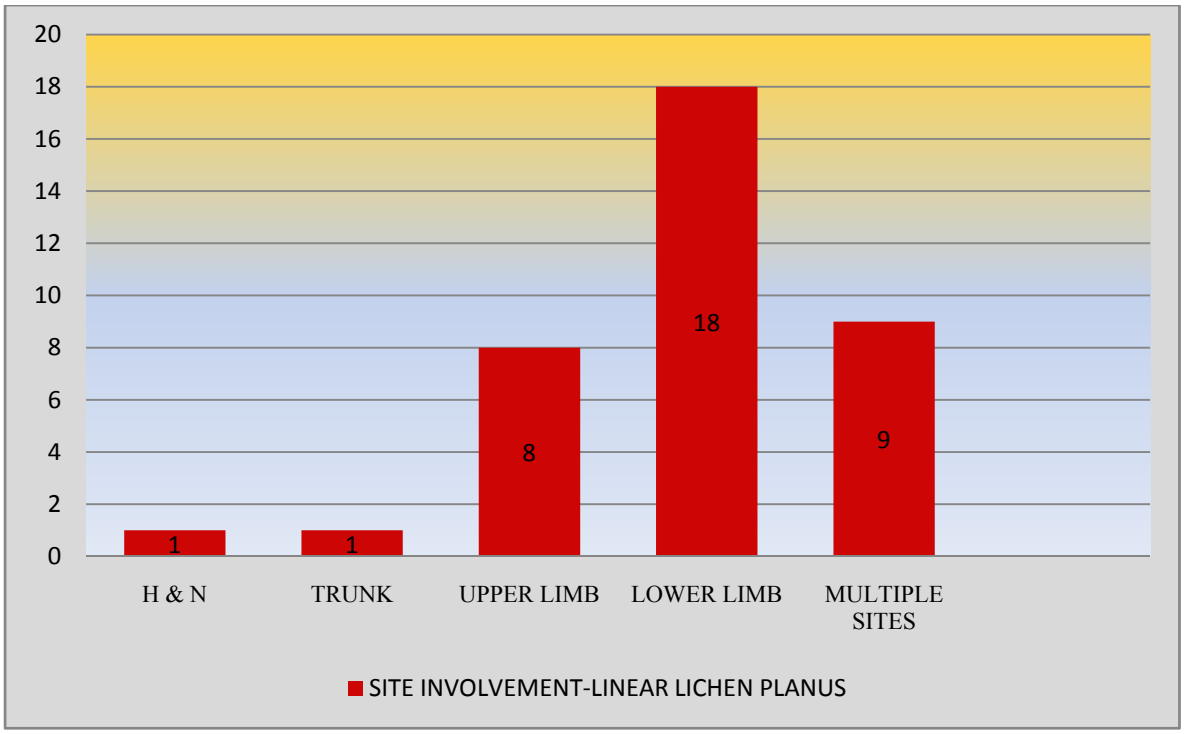
Multiple sites were involved in 78 % of the patients (including cases with coexisting oral lesions), the predominant site being the leg. Coexisting oral mucosal involvement was seen in 7 (19%) patients of which one was male and six were female patients. (Table VIII and Figure 12)

FIGURE 11: SITE- WISE DISTRIBUTION – LICHEN STRIATUS



H & N - Head & Neck

FIGURE 12: SITE – WISE DISTRIBUTION IN LINEAR LICHEN PLANUS



H & N - Head & Neck

TABLE VIII- SITE- WISE DISTRIBUTION- LINEAR LICHEN PLANUS

SITE	TOTAL
Head & neck	1
Trunk	1
Upper limb	8
Lower limb	18
Multiple sites	9
Total	37

c) SITES OF INVOLVMENT- NEVUS ACHROMICUS

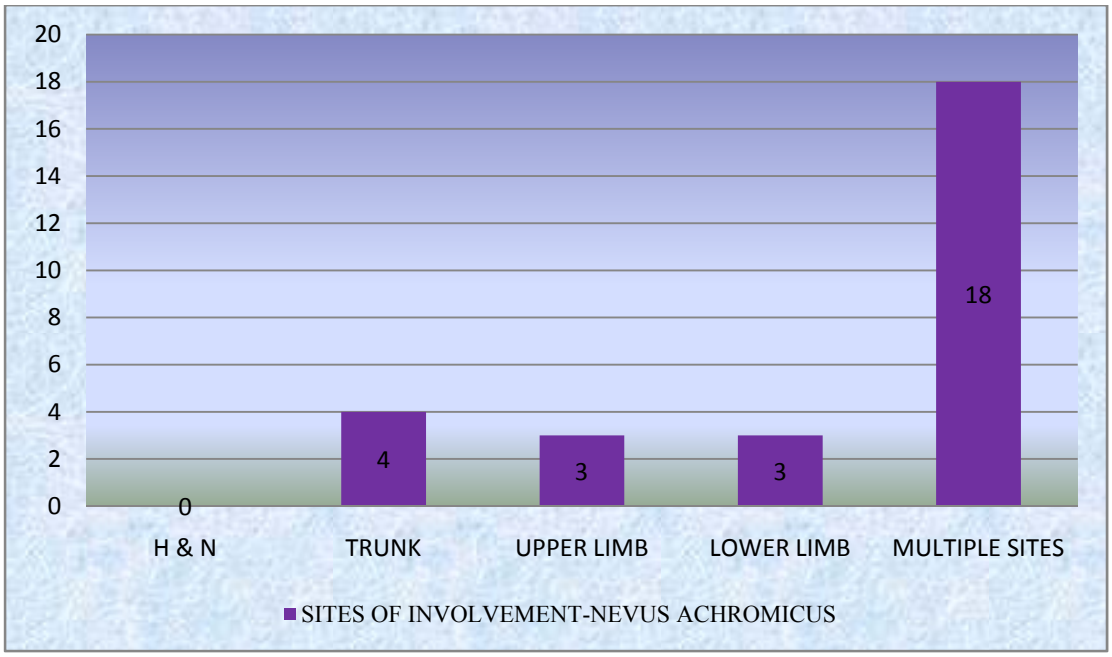
In this study, 24 (86%) out of 28 of patients had involvement of multiple sites, the most common site being the trunk. (Table IX and Figure 13)

TABLE IX- SITE - WISE DISTRIBUTION - NEVUS ACHROMICUS

SITE	TOTAL
Head & neck	-
Trunk	4
Upper limb	3
Lower limb	3
Multiple sites	18
Total	28

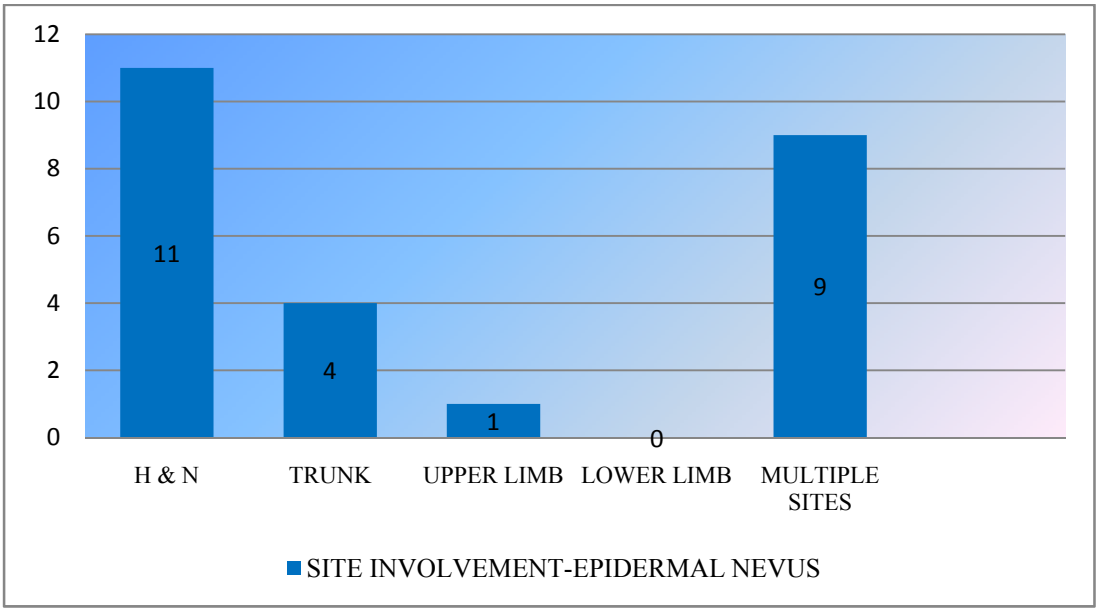
d) SITES OF INVOLVEMENT- EPIDERMAL NEVUS

In our study, the most common site involved in epidermal nevi patients was the head and neck region, followed by the involvement of multiple sites. One patient had systematized form of the disease. (Table X and Figure 14)



H & N - Head & Neck

FIGURE 14: SITE- WISE DISTRIBUTION IN EPIDERMAL NEVUS



H & N - Head & Neck

TABLE X- SITE - WISE DISTRIBUTION - EPIDERMAL NEVUS

SITE	TOTAL
Head & neck	11
Trunk	4
Upper limb	1
Lower limb	-
Multiple sites	9
Total	25

e) SITES OF INVOLVEMENT- OTHER DISEASES

The sites involved in the other diseases are shown in Table XI. In general, in most patients multiple sites were involved. LWNH preferentially involved the lower limbs and linear morphea preferentially involved the upper limbs.

TABLE XI- SITE - WISE DISTRIBUTION - OTHER DISEASES

SITE / Δ	HEAD & NECK	TRUNK	UPPER LIMB	LOWER LIMB	MULTIPLE SITES	TOTAL
LWNH	-	-	1	4	2	7
LM	-	-	3	2	-	5
HI	1	-	-	-	3	4
LV	-	-	-	1	2	3
NP	-	-	2	1	-	3
NS	3	-	-	-	-	3
DD	-	-	-	-	1	1
IP	-	-	-	-	1	1
LSA	-	-	-	1	-	1
LPP	-	-	1	-	-	1
LN	-	-	1	-	-	1
S FIB	1	-	-	-	-	1
DUH	-	-	-	-	1	1
TOTAL	5	-	8	9	10	32

LM- linear morphea, HI- hypomelanosis of Ito, LV- linear vitiligo, NP- Naevoid psoriasis, DD- Darier's disease, LPP- lichen planus pigmentosus, S fib- soft fibroma, DUH- dyschromatosis universalis hereditaria, NS – Nevus Sebaceous and nevus sebaceous syndrom

VIII) SYMPTOM - WISE DISTRIBUTION

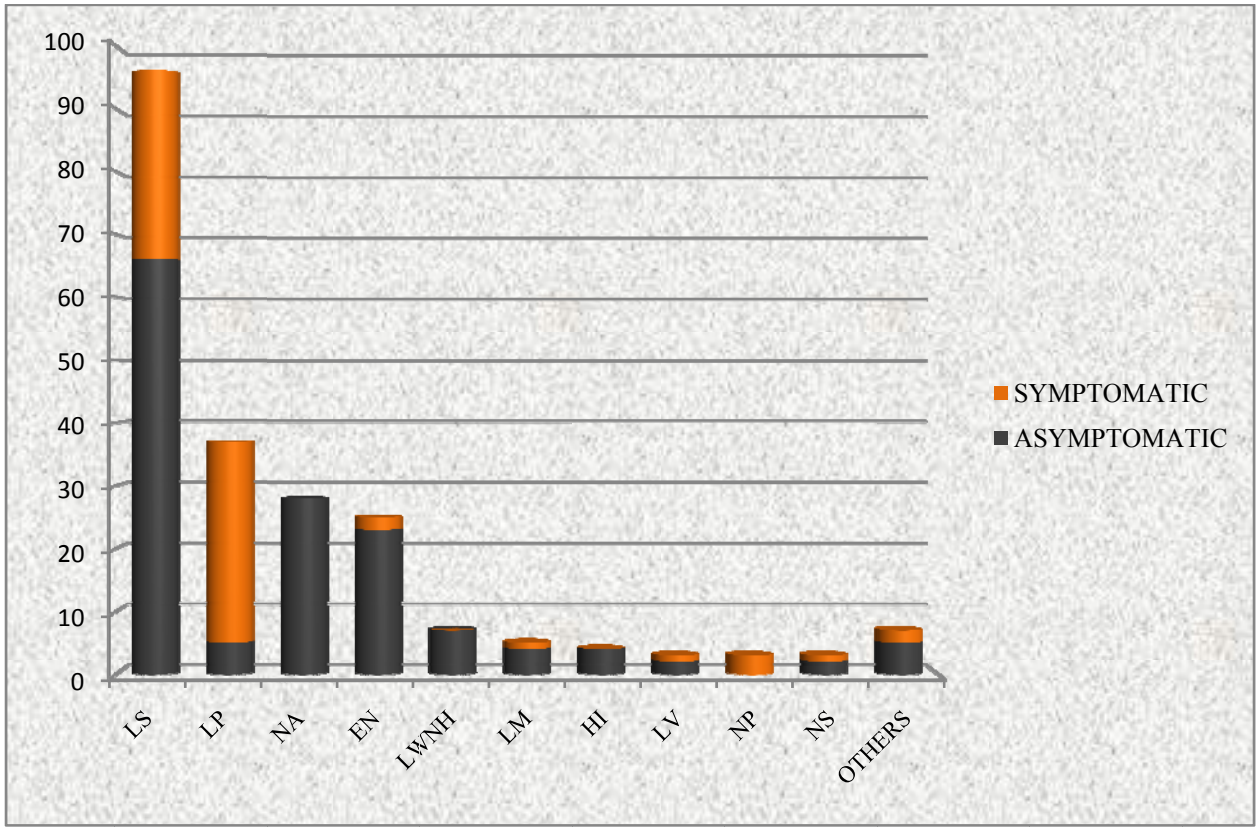
Overall, most of the patients in this study were asymptomatic. However, linear lichen planus was symptomatic in 86%. Thirty percent of patients with lichen striatus experienced pruritus. (Table XII and Figure 15)

TABLE XII- SYMPTOM- WISE DISTRIBUTION

DISEASE	ASYMPTOMATIC		SYMPTOMATIC	
	M	F	M	F
Lichen Striatus	30	36	9	20
Linear Lichen Planus	1	4	8	24
Nevus Achromicus	17	11	-	-
Epidermal Nevus	14	9	-	2
LWNH	4	3	-	-
Linear Morphea	1	3	1	-
Hypomelanosis Of Ito	2	2	-	-
Linear Vitiligo	-	2	1	-
Nevoid Psoriasis	-	-	1	2
Nevus Sebaceous & Nevus Sebaceous Syndrome	2	-	1	-
Others	-	5	-	2
Total	71	75	21	49

OTHERS- Darier's disease, lichen planus pigmentosus, Soft fibroma, Dyschromatosis universalis hereditaria, Incontinentia pigmenti, Lichen nitidus, LSA

FIGURE 15: SYMPTOM- WISE DISTRIBUTION



LS- Lichen striatus, LP- linear lichen planus, NA- nevus achromicus, EN- Epidermal Nevus,LM- Linear Morphea,HI- Hypomelanosis of Ito, LV-Linear Vitiligo, NP-Nevoid Psoriasis, NS-Nevus Sebaceous & Nevus Sebaceous Syndrome, Others- Incontinentia pigmenti, Darier's Disease, Dyschromatosis Universalis Hereditaria, LSA, Lichen Nitidus, Lichen Planus Pigmentosus, Soft Fibroma

IX) EVOLUTION OF LESIONS- (Table XIII)

TABLE XIII- EVOLUTION OF LESIONS

Disease	Extending	Static	Not recalled	Initial extension, now static	Total
Lichen striatus	73	1	18	4	96
Linear lichen planus	30	1	4	2	37
Nevus achromicus	7	17	1	3	28
Epidermal nevus	6	13	3	3*	25
LWNH	2	4	-	1*	7
Linear morphea	3	-	1	1	5
Hypomelanosis of ito	1	2	1	-	4
Linear vitiligo	3	-	-	-	3
Nevoid psoriasis	3	-	-	-	3
Nevus sebaceous & nevus sebaceous syndrome	-	2	-	1	3
Others	3	4	-	-	7
Total	131	44	13	7	218

Others- Incontinentia pigmenti, Darier's disease, Lichen planus pigmentosus, soft fibroma, dyschromatosis universalis hereditaria, lichen nitidus, LSA

(*) regression of lesions

In this study, most acquired lesions were still extending at the time of presentation,these included lichen striatus, linear lichen planus, linear morphea, linear vitiligo. The congenital disorders like epidermal nevus and nevus achromicus were static in nature, exception being nevoid psoriasis which was spreading beyond the original site of involvement in all the three patients.

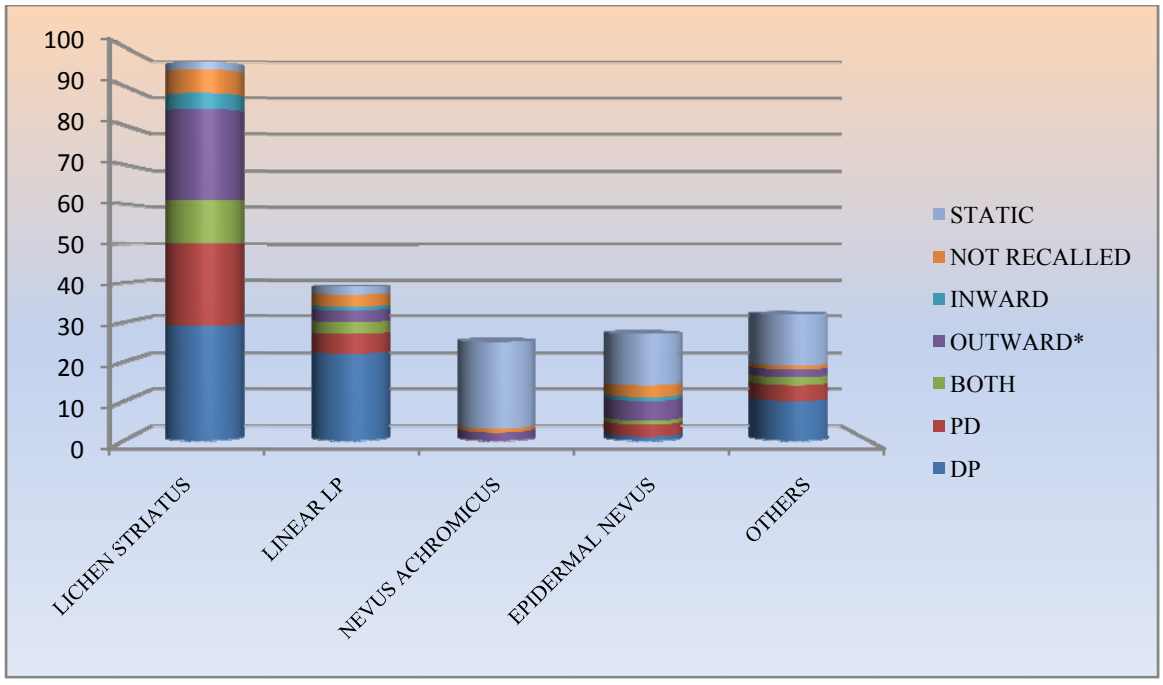
X) DIRECTION OF EXTENSION

In lichen striatus, the predominant direction of extension was distal to proximal over the limbs and outward over the trunk and head and neck region. In Linear LP, most lesions extended in the distal to proximal direction over the limbs and outward direction over the trunk and head & neck region. In nevus achromicus, the predominant direction of extension was outward over the trunk. Predominant direction of extension in epidermal nevus was proximal to distal over the limbs & outward over the trunk and head & neck regions. (Figure 16)

XI) ASSOCIATIONS

Diabetes mellitus was recorded in 3 patients and hypertension in one patient with linear lichen planus. Our patient with Darier's disease was both diabetic and hypertensive. One patient with lichen striatus had coexistent pityriasis rosea. One patient with linear LP had coexistent lesions of morphea not following the Blaschko's lines and another patient with linear LP had epilepsy with Parkinson's disease. Three patients with nevus achromicus had CNS abnormalities while one patient had congenital atrial septal heart defect. One patient of linear morphea gave

FIGURE 16: DIRECTION OF EXTENSION OF LESIONS



Others- LWNH, Linear Morphea, Hypomelanosis Of Ito, Linear Vitiligo, Nevoid Psoriasis, Nevus Sebaceous & Nevus Sebaceous Syndrome, Incontinentia Pigmenti, Darier's Disease, Dyschromatosis Universalis Hereditaria, LSA, Lichen Nitidus, Lichen Planus Pigmentosus,Soft Fibroma

(*)- Lesions On Trunk With/ Without Limb Lesions, PD- proximal to distal, DP- distal to proximal

history of temporal association with tetanus vaccination and pregnancy. One of the three patients with nevoid psoriasis had congenital valvular heart disease. Our patient with soft fibroma was a known case of pemphigus vulgaris.

XII) BIRTH HISTORY

In this study, 170 (78%) patients were born out of non- consanguineous marriage while 30 (14%) patients were born out of third degree consanguineous marriage and 18 (8%) patients were born out of second degree consanguineous marriage.

XIII) FAMILY HISTORY

Only 2 patients reported with medical disorders in the family. The older female sibling of the patient with incontinentia pigmenti died on 5^{th} postnatal day due to unknown causes. She did not have lesions of IP according to the history. The daughter of one patient with epidermal nevus was diagnosed to have epidermodysplasia verruciformis.

DISCUSSION

The lines of Blaschko represent a pattern assumed by many different nevoid and acquired skin diseases on the human skin and mucosae. Various congenital and acquired dermatoses follow the Blaschko's lines. There are five patterns of Blaschko's lines which include the narrow and broad – band pattern, checker board pattern, phylloid pattern, patchy pattern without midline separation and the lateralization pattern.

DISCUSSION ON EPIDEMIOLOGY OF BLASCHKO'S LINES

A) INCIDENCE

Out of a total 1, 03, 536 cases visiting the Dermatology outpatients during the study period, the cases with lesions following Blaschko's lines were 218 in total. Thus the incidence rate of lesions following the lines of Blaschko was 2.1 per 1000 cases.

B) CASE DISTRIBUTION

The most common disease with lesions along the Blaschko's lines was lichen striatus, followed by linear LP, nevus achromicus and epidermal nevus. Other diseases with few cases included LWNH, linear morphea, hypomelanosis of Ito, linear vitiligo, nevoid psoriasis and nevus sebaceous nevus & sebaceous syndrome.

Few rare conditions in which lesions are known to exist along the Blaschko's lines encountered were Darier's disease, incontinentia pigmenti, LSA, linear lichen nitidus and lichen planus pigmentosus . One case of soft fibroma following Blaschko's lines and one patient in whom universal dyschromatosis sparing the skin along Blaschko's lines was recorded.

C) SEX DISTRIBUTION

The overall sex distribution in the study of all the lesions following Blaschko's lines with a male to female ratio of 0.75:1 indicating a slight female predominance.

D) AGE OF ONSET – DISTRIBUTION

Two peaks were seen in the age distribution pattern. The first peak occurred in the age group of 0-1month reflecting the occurrence of nevi and nevoid conditions at birth and soon after birth. The second peak occurred at 1-5 years the bulk of which was formed by cases of lichen striatus. The next most common age groups involved were 6-11years again representative of lichen striatus; and 11-20 years representing both lichen striatus and linear LP.

The overall age distribution in males also showed a similar pattern having two peaks at 0-1month and again at 1-5 years.

In females, most of the cases belonged to the ages between 1-5 years and 6-10 years constituted mainly by lichen striatus.

E) SIDE DISTRIBUTION

The overall distribution of the side involved in all the diseases was found to be equal with 45% cases having right or left side involvement each. In 10 percent of the patients both right and left sides were involved.

The left side was involved found to be involved more frequently in males (49%) as compared to females (42%).

However, on individual analysis of the various diseases, left side involvement was more common in nevus achromicus, epidermal nevus and LWNH.

F) DISTRIBUTION ACCORDING TO THE TYPE OF BLASCHKO'S LINES FOLLOWED

Overall, the most common type of Blaschko's lines followed was Type 1A or the narrow – band pattern. The next most common pattern encountered was the broad-band and a combination of narrow and broad – band patterns. Lichen striatus, linear LP, epidermal nevi, linear morphea, hypomelanosis of Ito, nevoid psoriasis, nevus sebaceous, incontinentia pigmenti, Darier's disease, LSA, lichen nitidus, lichen planus pigmentosus and soft fibroma all followed only the type 1 pattern of

Blaschko's lines. Phylloid type alone was seen only in 12 patients all of whom had either nevus achromicus or linear vitiligo and in one patient of LWNH. A combination of phylloid and type 1 pattern was seen in one patient of nevus achromicus. Lateralization pattern was found in two patients both of whom were diagnosed with nevus achromicus.

No similar study of all the lesions following the Blaschko's lines could be found on reviewing the literature. Hence we have presented our findings with regards to Blaschko's lines.

G) OTHER PARAMETERS STUDIED-

- Data collected regarding the direction of extension showed no relevance to the severity/ course of the disease.
- Information about the birth history with regards to consanguinity was collected from all the patients. However, consanguinity did not show any effect on the incidence or presentation of the disease following Blaschko's lines.
- Family history was not contributory to the the clinical- epidemiological aspects in the diseases in this study

DISCUSSION ON INDIVIDUAL DISEASES

1) LICHEN STRIATUS

Lichen striatus formed the bulk of the cases in this study contributing 96 out of the total 218 cases. The lesions in 90.6% of the the patients followed the narrow- band pattern and 7.3 % a combination of narrow and broad- band types. Only in 2.1% cases the lesions followed the broad- band pattern alone. No other pattern of Blaschko's lines was followed. No reference for the type of Blaschko's line's followed in lichen striatus could be found.

The male: female distribution was 1:1.46 which is in concurrence with the study by Zhang Y & Mcnutt NS and where the ratio was 1: 1.6.[49] However the female predominance is not as high as two to three times as found in the study by Charles et al. [48] The maximum patients in our study were in the age group of 1-5 years followed by the age group 6-10 years in both males and females, the median age of onset being 4 years which concurs with the study by Patrizi et al where the median age was 4 years and 5 months.[47] The youngest patient was 1month in our study and the oldest patient was 21 years of age. The right side was preferentially involved in our study. Only two patients had involvement of both the sides. We could not find any data on the side involvement in the disease in the literature. Multiple sites were involved in 64.6 % patients. Overall, the thigh was the most common site of involvement in our study as compared to the arm as found in the study by Taieb et

al and Sittart et al. [52, 53] Nail involvement was seen in only two patients. The lesions were asymptomatic in 69 % of our patients and pruritic in 30%. Only one patient experienced pain. In our study more number of females (35%) experienced symptoms as compared to males (23%). In our study, the direction of progression of lesions was predominantly distal to proximal over the extremities and outwards over the trunk. No previous study with regards to these aforesaid parameters could be found in the literature. None of our patients were atopic in contrast to the study by Patrizi et al and Toda et al. [47, 50] This could perhaps reflect the lower incidence of atopic dermatitis in Indian children. One patient had coexistent pityriasis rosea which may throw some light to the ongoing studies on etio-pathogenesis of lichen striatus which is still not fully known.

II) LINEAR LICHEN PLANUS

Linear LP was the second most common disease following the Blaschko's lines in this study constituting 16.97% of the total number of cases (excluding the one case of linear lichen planus pigmentosus discussed later). The predominant type of Baschko's lines followed was the narrow- band pattern in nearly 90% patients. The broad- band and a combination of both narrow and broad- bands was found in 5% each. No other pattern of Blaschko's lines was observed. In one patient, the lesions occurred along multiple Blaschko's lines in a symmetrical fashion over the legs.

One case of linear LP occurring as multiple streaks affecting several different areas of the body has been reported by Long and Finlay. [135] The predominant direction of extension of the linear lesions on the limbs was distal to proximal followed by proximal to distal direction.

The male: female ratio was 0.3:1, similar to generalized lichen planus where predilection for females is found. [136] The average age of onset in our study was 31years which is in accordance with the age of onset (33years) in the cases reviewed by Bolognia et al. [15]Our youngest patient was 2 years old. The predominant side involved was the right side in 57% patients followed by the left side in 35%. Only 3 patients had bilateral involvement. Multiple sites were involved in 78 % of the patients, the predominant site being the leg which is in concurrence with the literature reviewed[60] Coexisting oral mucosal involvement was seen in 7 (19%) patients of which one was male and six were female patients as compared to generalized LP in which mucosal involvement occurs in 60- 70%. [137] Both sides of the oral mucosa were involved and in only one patient the mucosal lesions were limited to the side of skin involvement. In three of our patients nail involvement in the form of longitudinal ridging was seen when the linear LP lesions extended upto the proximal nail fold. Only 8 (22%) of our patients had coexisting LP lesions which did not follow the Blaschko's lines over other parts of the body. One patient had patchy scarring alopecia over the scalp and

morphea lesions over the face, back and lower limbs. One patient had palmo-plantar keratoderma. In all 70% of our patients had involvement of other sites including mucosa, nails, scalp and hair as compared to 30 % in the cases reviewed by Bolognia et al. [15] Nearly 86% of our patients experienced pruritus which correlates with the literature studied. Of the 37 patients, only three were diabetic, one patient was hypertensive, and one patient had epilepsy with Parkinson's disease.

III) NEVUS ACHROMICUS

Nevus achromicus was the third most common lesion occurring along the Blaschko's lines in this study accounting for 12.8% of the total cases. The most common pattern of Blaschko's lines found was the narrow – band (32%) , followed by the phylloid pattern in 8 (29%) patients , broad- pattern in 3 (10%) and lateralization in 2 (7%) of our patients. A combination of narrow and broad- band pattern was found in 5 (18%) patients and a combination of phylloid and broad-band pattern in one patient. Nevus achromicus formed the majority of cases in which the lesions followed the phylloid type of Blaschko's lines. The lesions extended for the first two months in one patient and for the first 1 year in another; the lesions showing extension in the outward direction.

The male: female ratio was 1.5:1. No reference for sex incidence in nevus achromicus could be found. In our study, the lesions were present at birth in 22 (79 %) patients and appeared within 2 months of birth in another 3 (7%) patients in accordance with the study by Lee HS, Chun YS,and Hann SK. [128] Two of our patients had late onset of lesions at the ages of 18 years and 21 years. The lesions were static in nearly 61% of the patients while the lesions extended after birth in 7 (28%) patients in contrast to the fixed nature of the disease mentioned in the literature. [26] The predominant side involved was the left side in 57% followed by both side involvement in 25% and right side involvement in 18%. Multiple sites were involved in 86% of our patients. In this study, the most common site involved was the chest as compared to the back and thighs in the study by Lee HS et al. [128] In one of our patients there was longitudinal ridging of one half of the nail which was continuous with the linear patch of nevus achromicus on the forearm. No reference with regards to nail involvement in nevus achromicus could be found. Two (7%) cases had coexistent seizure disorder of which one patient also had mild mental retardation. Another patient had microcephaly with craniostosis. These cases add to the few case reports of association of nevus achromicus with neurological defects. [129] One of our patients had atrial septal defect. However, no reference could be found for congenital heart disease in nevus achromicus.

IV) EPIDERMAL NEVUS

Epidermal nevus constituted the fourth most common condition following the Blaschko's lines, including one patient with systematized epidermal nevus. The most common pattern followed was the Type 1, narrow band pattern in 19 (76%) patients, broad band in 2 (8%) patients and a combination of narrow and band – bands in 4(16%) patients. No other pattern was observed. In 13 (52%) patients the lesions were static from birth, only becoming prominent with age. Spread beyond the site was seen in 36% of our patients in concurrence with the study by Maureen et al where extension of lesions was noted in 37% cases. [37] Our patient with systematized nevus noted regression of lesions over the extremities after 13 years of age.

The male: female ratio in our study was 1.5:1 as compared to the equal incidence in males and females reported in the literature. [36] In 17 (68%) of the patients, the lesions were present at birth and in a further 2 (8%) patients the nevi appeared within 2 weeks of birth. The lesions developed in the first 6months of age in 3(12%) of the patients. The highest age of onset in our study was 11 years in accordance with the study by Maureen et al where the age of onset ranged from birth to 14 years. [37] The right side was involved in 10 (40%) and the left side in 13 (52%) of the patients. Both the sides were involved in 2 (8%) of the cases. No literature on the side of involvement could be found. Head and neck involvement

was the most common constituting 11 (44%) cases, in concurrence with the study by Maureen et al. [37] This was followed by multiple sites of involvement seen in 10 (40%) of our patients. Only one(4%) patient had systematized epidermal nevus with bilateral involvement of the head, neck, entire trunk, both extremities and external genitalia, sparing only the palms, soles and mucosa as compared to widespread involvement in 13% in the study by Maureen et al. [37] In another patient, the sole was involved in continuity with the nevus on the leg. No mucosal or nail involvement was noted. None of our patients had any other coexisting cutaneous or systemic findings. This is in contrast to 38% of the patients who had other abnormalities as per the study by Maureen et al. [37]

V) DARIER'S DISEASE

One case of Darier's disease following Blaschko's lines was encountered. Our patient was a female aged 55years with skin lesions limited to the left half of the trunk, external genitalia and extremities since 3 years. On examination multiple hypopigmented macules of varying sizes over the arms and legs, and multiple hyperkeratotic pigmented non-scaly papules of varying sizes over the left submammary region, groin and vulva along the Blaschko's lines were noted. Other features associated with classical Darier's disease like cobble-stone appearance of oral mucosa and V- nicking of nails were absent. She experienced no

photosensitivity or seasonal exacerbation. There was no similar family history. Biopsy was confirmatory. The lack of family history and other signs of the disease suggest nevoid origin. [40, 41]

VI) SOFT FIBROMA

One case of soft fibroma following Blaschko's lines was recorded in a 42 years old female who was known case of Pemphigus vulgaris on pulse therapy. Multiple soft pigmented verrucous papules over the left side of neck along the Blaschko's lines were present for a duration of 3 years. Biopsy confirmed the diagnosis of soft fibroma. On reviewing the literature, a single case of a 17 years old male who presented with large pedunculated soft fibromas restricted to a circumscribed area of the right abdomen along the Blaschko's lines has been reported by Worm et al[138] and it has been suggested that these nevoid bag-like soft fibromas represent a new malformation in the heterogenous group of nevoid tumors.

VII) DYSCHROMATOSIS UNIVERSALIS HEREDITARIA

We came across a 20 years old female patient with dyschromatosis universalis hereditaria (DUH) in whom sparing of the skin along the lines of Blaschko along broad bands were noted. The lesions were asymptomatic and static in nature, and were noticed at the age of 5 years. Family history was lacking.

A variable autosomal mode of inheritance of DUH has been described. In DUH, skin lesions are usually present in the first years of life. The trunk and extremities are the dominant sites. [139] No reference with DUH sparing the skin along Blaschko's lines could be found. Our case probably represents a case of revertant mosaicism in which there is linear absence of skin disease attributed to a back mutation. [28]

VIII) INCONTINENTIA PIGMENTI

We recorded one case of incontinentia pigmenti in a 3 days old female baby born out of a non-consanguineous marriage. She had the classical vesicular lesions of IP extending from the right lower limb and right side of chest along the Blaschko's lines (narrow- band pattern).The baby was otherwise normal. Family history was lacking. Examination of the mother was normal. Tzank smear from the vesicle showed plenty of eosinophils. Neuro-sonogram and ophthalmological examination were normal. Biopsy could not be carried out due to the neonatal age group.

IX) LICHEN SCLEROSUS ET ATROPHICUS

In this study, an 11 years old female patient was diagnosed to have the classical lesions of LSA in the narrow band pattern over the right thigh. Classical lesions of LSA, not following the Blaschko's lines, were also present over other areas and the

genitals. Oral mucosa was normal. Histopathology was consistent with LSA. This case adds to the not so uncommon cases reports of extragenital linear LSA,[140] and the uncommon case reports of LSA following Blaschko's lines.[71-74]

X) LINEAR LICHEN NITIDUS

One case of linear lichen nitidus (LN) was encountered in a 12 years old female who presented with the classical lesions of lichen nitidus along the Blaschko's lines (narrow- band pattern) over the right upper limb. Similar lesions were present over the neck, trunk and both forearms and hands not following the Blaschko's lines. Tiny, shiny papules were present over the palate. Nails were normal. Biopsy was consistent with LN. Linear LN is very rare with only one reference available.[90] In addition, our patient had oral mucosal involvement which is also very rare. [89]

XI) LINEAR LICHEN PLANUS PIGMENTOSUS

A single case of LPP following Blaschko's lines was recorded in a 19 years old female who presented with asymptomatic pigmented skin lesions over the back and right upper limb since two years. On examination, multiple pigmented macules and patches in reticulate and scattered pattern were seen over the back and both shoulders. On the right side, linear pigmented streaky patches were seen over the axilla, chest and upper limb. These linear lesions followed the narrow band pattern

of Blaschko's lines. Oral mucosa and nails were normal. Biopsy was consistent with LPP. These findings are consistent with other case reports in the literature. [78] However, our patient also had LPP lesions in reticulate and scattered pattern in other areas.

XII) LINEAR MORPHEA

We came across 5 cases of linear morphea of which 3 patients were females and 2 male patients, following the Blaschko's lines (broad – band pattern). The sex ratio (F:M) was 3:2, as compared to 4:1 in the literature.[141] Predominant age of onset was adolescence, except in one patient where the age of onset was 28 years. However, this female patient developed the lesions during pregnancy and a few weeks after tetanus immununization both of which are implicated in the pathogenesis of morphea. [142] In 4 of our patients, unilateral involvement and in one patient bilateral involvement, along the Blaschko's lines was seen. Three of these patients had morphea lesions elsewhere which did not follow the Blaschko's lines. In contrast to the literature reviewed where it is stated that the lower limbs are more commonly affected, [64] the linear lesions in our study were present over the upper limbs in 3 patients; lower limb being involved in two patients. The biopsy in all our patients was consistent with morphea.

XIII) LINEAR VITILIGO

Three patients in this study presented with vitiligo lesions along Blaschko's lines, two of whom were females and one male. In all the patients the lesions followed the phylloid pattern of Blaschko's lines. Our patients were young children, the early age of onset in concurrence with the literature reviewed. [26] However, in contrast to the literature reviewed where vitiligo lesions over other sites are usually absent, [26] two of our patients also had vitiligo not along the Blaschko's lines at other sites also. No association with other autoimmune disease similar to the literature studied. [26] However, our sample of 3 cases is too small to compare the data correctly.

XIV) NEVUS SEBACEOUS & NEVUS SEBACEOUS SYNDROME

We came across two patients of nevus sebaceous and one patient diagnosed with Nevus sebaceous syndrome. The left side was involved in all. The narrow- band pattern was followed in two and the broad- band pattern in one patient. The sex distribution as per the literature is equal.[36]However all the three patients in this study were males. The face alone was involved in them in concurrence with the literature reviewed.[36] In two of the patients the lesions appeared at birth also in accordance with the study by Connor et al. [110]

In the patient diagnosed with Nevus sebaceous syndrome, the lesions were noticed only at the age of 23 years and the lesion increased in extent for the first 2 years.

The biopsy was consistent with nevus sebaceous. He also had facial asymmetry with jaw deviation to the right side. He had history of seizures and CNS shunt surgery following an episode of meningitis at the age of 14 years; thus we could not attribute the CNS symptoms entirely to the syndrome.

XV) NEVOID PSORIASIS

Three cases of nevoid psoriasis following the Blaschko's lines were recorded, of which one was male and two were females. Onset of lesions occurred at birth in two cases and at 6 months of age. Two cases had upper limb involvement and in one case the lower limb was involved. In all the three cases, the lesions followed the narrow- band pattern of Blaschko's lines. All the three patients complained of extension of the lesions and occasional pruritus. In one patient classical psoriatic lesions, not following the Blaschko's lines, were present in other areas including the scalp. No nail involvement was noted. Biopsy was confirmatory in all cases. Our male patient had congenital valvular heart disease. On reviewing the literature, no case reports of nevoid psoriasis with coexisting such developmental defects could be found. These patients add to the handful reported cases of nevoid psoriasis .[82, 143]

XVI) HYPOMELANOSIS OF ITO

A total of four cases of hypomelanosis of Ito were encountered of which two patients were male and two were female. The lesions followed the narrow- band pattern of Blaschko's lines in three patients and the broad- band pattern in one patient. Two patients had bilateral involvement and two had unilateral involvement. In three of the patients the skin lesions were present at birth, but in one female patient the lesions were first noticed at the age of 8 years. One patient had lesions confined to the head and neck while the trunk and extremities were involved in the other three. None of our patients had any associated systemic involvement which is found in 30- 50 % cases as per the literature reviewed. [26]

XVII) LWNH

In this study 7 cases of linear and whorled nevoid hypermelanosis were recorded, of which 4 were male and 3 were female patients. The predominant pattern of Blaschko's lines followed was Type 1, with the broad- band and narrow – band pattern seen in two patients each and a combination of both patterns seen in two patients. The phylloid pattern was seen in one patient. One patient also had reticulate pigmentation which is also a common finding in LWNH. [131, 132, 133] Bilateral predominantly asymmetrical involvement was seen in 3 patients and unilateral involvement in 4 patients. The lesions were present at birth in 4 patients

and noticed at 1m of age in one patient in concurrence with the study by Kalter et al. [130] However in one patient, the lesions were noticed at the age of 7 years, probably representing the tardive form of the disease as described by Moulin et al.[134]The lesions were static in 4 patients, spread beyond the initial site in two and regressed in one patient.

RELEVENCE OF HISTOPATHOLOGICAL ANALYSIS IN THIS STUDY

In 33 (15%) out of the 218 cases histopathological studies were conducted. These cases included -

1. Two out of 96 cases of lichen striatus – to differentiate from lichen planus and from lichen nitidus in one.
2. Sixteen out of 37 patients of lichen planus- to confirm the diagnosis.
3. In all the 5 cases of morphea- to confirm the diagnosis.
4. In all three cases of nevoid psoriasis – to differentiate from ILVEN
5. In one out of the 3 cases of nevus sebaceous – to distinguish it from epidermal nevus.
6. In the cases of Darier's disease, dyschromatosis universalis, soft fibroma and lichen planus pigmentosus , lichen sclerosus et atrophicus and lichen nitidus– to confirm the diagnosis.

SUMMARY

1. A total of 218 cases of lesions following Blaschko's lines were encountered in the two year study period.
2. Lesions following the lines of Blaschko constituted about 2 in 1000 cases visiting the Dermatology OPD.
3. Various dermatoses occurred along the lines of Blaschko, of which the most common was lichen striatus.
4. Overall, the lesions following Blaschko's lines were more common in females.
5. The most common dermatoses following Blaschko's lines in infancy were nevus achromicus and epidermal nevi; in toddlers and young children was lichen striatus; and in adults was linear lichen planus.
6. The overall side distribution of these lesions in various dermatoses was equal.
7. The most common pattern of Blaschko's lines occurring in various dermatoses was the narrow- band pattern followed by the broad- band pattern and a combination of the two. Phylloid pattern and lateralization were most commonly encountered in nevus achromicus.
8. Lichen striatus was more common in females, and the most common age group affected was that of 1-5 years. Narrow- band was the predominant

type of Blaschko's lines followed by lichen striatus. The most common site involved was the thigh.

9. Linear lichen planus was more common in females, the average age of onset being 31 years. The predominant type of Blaschko's lines followed was the narrow- band pattern. The most common site of involvement was the legs. Oral mucosal and nail involvement were less common in linear LP.
10. Nevus achromicus was more common in males, the lesions being present at birth in most cases. The predominant side involved was the left side. The most common type of Blaschko's lines followed was the narrow- band type, followed by the phylloid pattern. The most common site involved was the chest. One patient had nail involvement.
11. Most epidermal nevi presented within the first 2 weeks of life and were more common in males. The left side was more frequently affected. Most epidermal nevi occurred along the narrow band pattern, the head and neck being the most common site of involvement. Epidermal nevi were not associated with developmental defects in this study.
12. The less common disorders of pigmentation that followed Blaschko's lines included linear whorled nevoid hypermelanosis, hypomelanosis of Ito and linear vitiligo.

13. Developmental abnormalities in hypomelanosis of Ito and LWNH were not found to be as common as given in the literature.
14. Linear morphea followed the lines of Blaschko, despite the controversies stated earlier.
15. Sebaceous nevi occurred along Blaschko's lines, and were also noted to develop in older age.
16. Certain diseases like Darier's disease, lichen nitidus, lichen planus pigmentosus, LSA infrequently occurred along the Blaschko's lines.
17. Interesting findings included were three cases of nevoid psoriasis, reflecting that this condition is probably not as rare as thought to be.
18. A unique observation made was the presence of revertant mosaicism in dyschromatosesis universalis hereditaria wherein sparing of areas of normal skin along the lines of Blaschko occurred.
19. A rare finding encountered of was soft fibroma occurring along the Blaschko's lines.
20. Conditions like nevoid psoriasis and nevus achromicus were found to be associated with developmental and neurological abnormalities. Hence, it may be prudent to evaluate and follow such cases regularly.
21. Only a few cases required biopsy for the confirmation of diagnosis, which was done in the study.

CONCLUSION

The incidence of lesions following the lines of Blaschko is 2 per 1000 dermatological cases, the most common disease being lichen striatus followed by linear lichen planus, nevus achromicus and epidermal nevus. The infrequently encountered diseases are linear whorled nevoid hypermelanosis, hypomelanosis of Ito, nevoid psoriasis, sebaceous nevi, linear morphea, linear vitiligo, Darier's disease, lichen nitidus, lichen sclerosus, lichen planus pigmentosus and incontinentia pigmenti. Lesions following Blaschko's lines are more common in females. The most common type of Blaschko's lines followed is the narrow- band. The age of onset distribution shows two peaks constituted by nevus achromicus and epidermal nevi in the first 1 year of life and by lichen striatus in the toddler age group. Disorders like nevus achromicus and nevoid psoriasis can also be associated with congenital defects. Rarely, soft fibroma can follow the lines of Blaschko and sparing of the skin along the Blaschko's lines can occur in dyschromatosis universalis hereditaria.

To the best of our knowledge, this is the first study of its kind. More similar studies are needed for further comparisons and validation. The obstacle faced in this study was the lack of a clear distinction between narrow and broad – band patterns of Blaschko's lines. Thus there is a need to reach a clear and universal definition of the various types of Blaschko's lines. The differences in the course,

prognosis and treatment aspects of various dermatoses following the Blaschko's lines as compared to the generalized forms of the diseases are not yet completely elucidated. Hence, further studies in this direction will be of profound help in the management of such cases.

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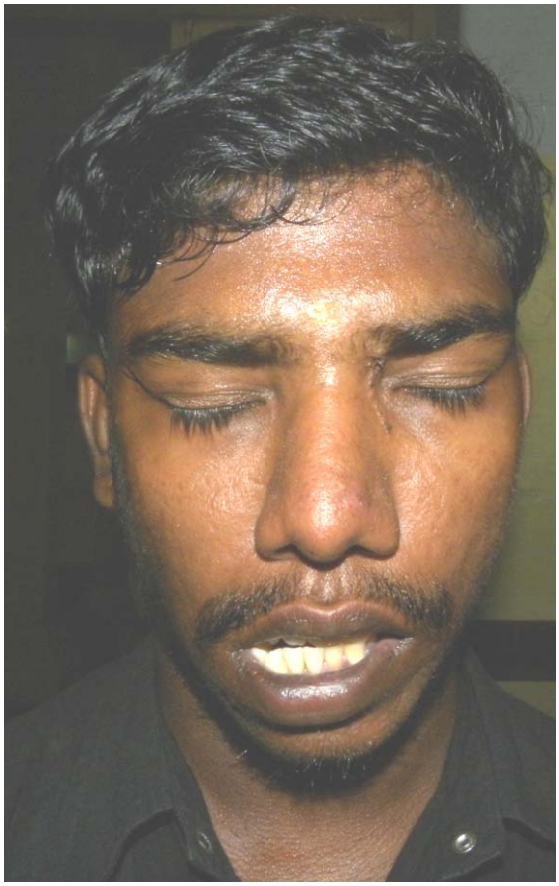
LESIONS ALONG NARROW- BAND PATTERN OF BLASCHKO'S LINES

A) LICHEN STRIATUS

B) LINEAR LICHEN PLANUS

LESIONS ALONG NARROW- BAND PATTERN OF BLASCHKO'S LINES



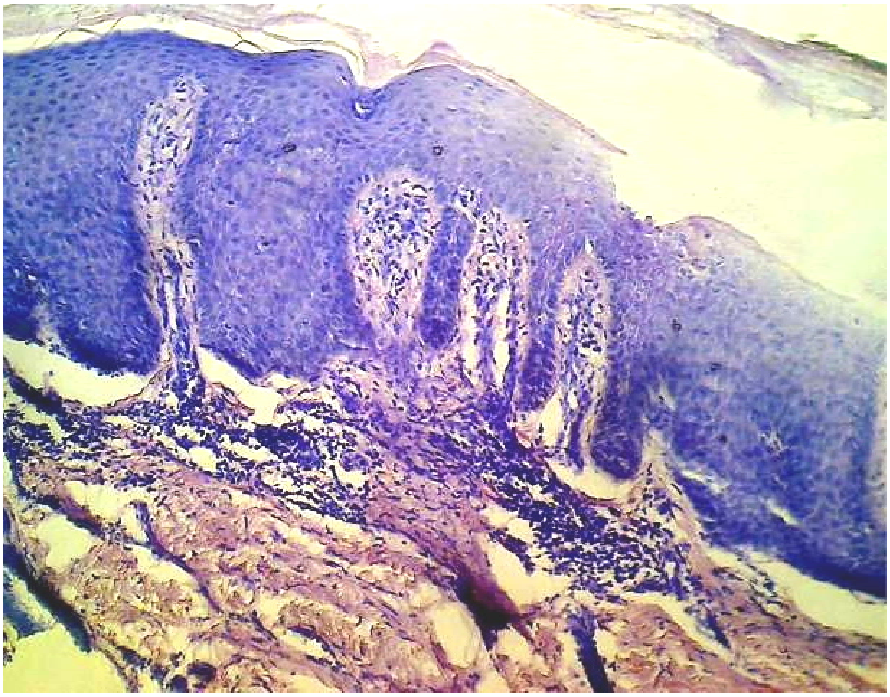
C) NEVUS SEBACEOUS SYNDROME (WITH FACIAL ASYMMETRY)

D) SOFT FIBROMA

LESIONS ALONG NARROW- BAND PATTERN OF BLASCHKO'S LINES

E) NEVOID PSORIASIS

i) Clinical picture



H & E, 10x ii) Histopathological picture

LESIONS ALONG NARROW- BAND PATTERN OF BLASCHKO'S LINES

F) LINEAR LICHEN PLANUS PIGMENTOSUS

G) LINEAR LICHEN SCLEROSUS ET ATROPHICUS

LESIONS ALONG NARROW- BAND PATTERN OF BLASCHKO'S LINES

H) INCONTINENTIA PIGMENTI

I) HYPOMELANOSIS OF ITO

LESIONS ALONG NARROW- BAND PATTERN OF BLASCHKO'S LINES

J) DARIER'S DISEASE

i) Hyperkeratotic pigmented papules along Blaschko's Lines

ii) Hypopigmented macules along Blaschko's Lines

LESIONS ALONG BROAD - BAND PATTERN

OF BLASCHKO'S LINES

A) LICHEN STRIATUS

B) NEVUS ACHROMICUS

C) LINEAR LICHEN PLANUS

LESIONS ALONG BROAD - BAND PATTERN

OF BLASCHKO'S LINES

D) LINEAR MORPHEA

E) DYSCHROMATOSIS UNIVERSALIS HEREDITARIA – SPARING OF SKIN ALONG BLASCHKO'S LINES

LESIONS ALONG BOTH NARROW - BAND AND BROAD - BAND PATTERN OF BLASCHKO'S LINES

A) SYSTEMATIZED EPIDERMAL NEVUS

B) LINEAR WHORLED AND NEVOID HYPERMELANOSIS

LESIONS ALONG PHYLLOID - PATTERN

OF BLASCHKO'S LINES

A) NEVUS ACHROMICUS

B) LINEAR WHORLED AND NEVOID HYPERMELANOSIS

LESIONS ALONG PHYLLOID - PATTERN OF BLASCHKO'S LINES

C) LINEAR VITILIGO

LESION ALONG LATERALIZATION PATTERN – OF BLASCHKO'S LINES

NEVUS ACHROMICUS

PROFORMA

- Sl no
- DATE
- Name
- Age/sex
- Occupation
- Address
- Phone no
- Chief complaints
- Skin lesions
- Age at onset
- Duration
- Site
- Side of involvement
- Symptoms-
    - Asymptomatic
    - Pruritus
    - Pain
    - Others
- Progression
    - Extending
    - Static
    - Regressing
- Direction of extension-

        - LIMBS- Distal to proximal/ Proximal to distal
        - TRUNK & FACE - Outward/ inward
- Similar lesions elsewhere
- Other skin lesions
- Associations-other diseases
        - CNS
        - Eyes
        - Bones/Joints
- Family history
            - Similar lesions
            - Other skin lesions
            - Other diseases
- Birth history
- Examination
        - General
        - Systemic
        - Skin-
            - site
            - surface
            - distribution/pattern
            - size
            - shape
            - colour
            - palpation
            - induration
            - tenderness
            - mucosa
            - scalp & hair
            - nails
            - palms & soles
            - Genitals

- PATTERN OF BLASCHKO'S LINES FOLLOWED-
    - Narrow- band/ Broad – band
    - Checkerboard
    - Phylloid
    - Patchy without midline separation
    - Lateralization

- Probable diagnosis/final diagnosis

- Investigations
    - Biopsy
    - Routine
    - Special

KEY TO MASTER CHART

ASD	-	Atrial Septal Defect
B	-	Both
BB	-	Broad- band
CHD	-	Congenital heart disease
Cm-2	-	Second degree consanguineous
Cm-3	-	Third degree consanguineous
D	-	Days
DD	-	Darier's disease
DM	-	Diabetes mellitus
DP	-	Distal to proximal
DUH	-	Dyschromatosis universalis hereditaria
E	-	Extending
EDV	-	Epidermodysplasia verruciformis
F	-	Female
HI	-	Hypomelanosis of Ito
HTN	-	Hypertension
IE	-	Initial extension
I	-	Inward
IP	-	Incontinentia pigmenti
L	-	Left
LM	-	Linear morphea
LN	-	Lichen nitidus

LP	-	Lichen planus
LPP	-	Lichen planus pigmentosus
LSA	-	Lichen sclerosus et atrophicus
LV	-	Linear vitiligo
LWNH	-	Linear and whorled nevoid hypermelanosis
M	-	Male
mo	-	Months
MR	-	Mental retardation
n	-	None
NA	-	Not applicable
NB	-	Narrow- band
Ncm	-	Non- consanguineous
NP	-	Nevoid psoriasis
NR	-	Not Recalled
NS	-	Nevus sebaceous
NSS	-	Nevus sebaceous syndrome
O	-	Outward
p	-	Present
p-i	-	Present - itching
PD	-	Proximal to distal
R	-	Right
S	-	Static
Yr	-	Years
y	-	Yes

ABSTRACT

BACKGROUND: Blaschko's lines have been described as 'a system of lines on the human skin which the linear nevi and dermatoses follow'. These lines probably represent the routes of ectodermal cell migration from the neural crest. The pattern of Blaschko's lines does not correlate with the distribution of cutaneous nerves, blood vessels, or lymphatics.

AIM: To study and describe the patterns of Blaschko's lines followed by various dermatoses and the clinico-epidemiological features of these dermatoses.

MATERIALS AND METHODS: The study was conducted in the outpatient department of Government Rajaji Hospital, Madurai during the period October 2009 to September 2011. All consenting patients with dermatoses related to Blaschko's lines in whom the diagnosis could be confirmed on clinical and/or histopathological basis were enrolled.

RESULTS AND CONCLUSION: In this study 218 patients with lesions following the Blaschko's lines were encountered. The incidence of lesions following the Blaschko lines was 2 per 1000 cases. The various dermatoses that followed in descending order were lichen striatus (44.04%), linear lichen planus (16.97%), nevus achromicus (12.84%), epidermal nevus (11.4%), linear and whorled nevoid

hypermelanosis (3.21%), linear morphea (2.29%), hypomelanosis of Ito (1.83%), linear vitiligo (1.38%), nevoid psoriasis (1.38%), nevus sebaceous and nevus sebaceous syndrome (1.38%). One (0.46%) case each of incontinentia pigmenti, lichen sclerosus et atrophicus, lichen nitidus, lichen planus pigmentosus were noted. Rare cases encountered were soft fibroma following Blaschko's lines and one case of dyschromatosis universalis hereditaria sparing the skin along the lines of Blaschko. Overall, the lesions following Blaschko's lines were found to be more common in females. The most common dermatoses following Blaschko's lines in infancy were the nevus achromicus and epidermal nevi; in toddlers and young children was lichen striatus; and in adults was linear lichen planus. The most common pattern of Blaschko's lines occurring in various dermatoses was the narrow- band pattern. The overall side distribution of these lesions in various dermatoses was equal. Nevoid psoriasis and nevus achromicus were found to be associated with developmental heart and neurological abnormalities.

KEY WORDS- Blaschko's lines, nevoid.

EPIDERMAL NEVUS																									
S.No	NAME	TYPE	AGE OF ONSET	SEX	SIDE	HEAD & NECK	CHEST	ABDOMEN & PELVIS	BACK	THIGH	LEG	ARM	FOREARM	HANDS	FEET	PALMS & SOLES	GENITALS	SYMPTOMS	EVOLUTION	DIRECTION OF EXTENSION	ASSOCIATIONS	BIRTH HISTORY	FAMILY HISTORY	BIOPSY	COMPATIBLE
1	Amudavalli	NB	8 Yr	F	R				p			p						n	NR	NR	n	Ncm	Daughter-EDV	n	NA
2	Ganesh	NB	NR	M	L		p	p										n	NR	NR	n	Ncm	n	n	NA
3	Prashanth	NB	birth	M	L	p												n	S	S	n	Ncm	n	n	NA
4	Pandiyan	NB	birth	M	R		p		p			p	p					n	E	PD	n	Ncm	n	n	NA
5	Gokulkrishnan	NB	5mo	M	L								p					n	E	DP	n	Cm-3	n	n	NA
6	D/o Jayabarashakti	BB	birth	F	L		p											n	S	S	n	Ncm	n	n	NA
7	Vishwa	NB	3D	M	L			p		p	p				p			n	NR	B	n	Ncm	n	n	NA
8	Karupaiah	NB	6mo	M	R	p	p					p	p	p				n	E	I/PD	n	Ncm	n	n	NA
9	Mohsan	NB	birth	M	L	p												n	IE	O	n	Ncm	n	n	NA
10	Rajimeena	NB	birth	F	L	p												n	S	S	n	Ncm	n	n	NA
11	Satyamurthy	NB	1 Yr	M	R	p												n	S	S	n	Ncm	n	n	NA
12	Praveen	NB	birth	M	R		p	p		p							p	n	S	S	n	Cm-3	n	n	NA
13	C. Subramaniyan	NB	birth	M	L				p									n	S	S	n	Ncm	n	n	NA
14	Senthil Kumar	NB	4mo	M	R			p	p	p	p				p	p		n	S	S	n	Ncm	n	n	NA
15	Naveen	NB	birth	M	L		p											n	S	S	n	Cm-2	n	n	NA
16	Nandini	NB & BB	birth	F	R	p												n	IE	O	n	Ncm	n	n	NA
17	Sabya	BB	birth	F	L	p												n	S	S	n	Ncm	n	n	NA
18	Prasanth	NB & BB	birth	M	B	p												n	S	S	n	Ncm	n	n	NA
19	Mariammal	NB & BB	birth	F	L	p												p-i	IE	O	n	Cm-3	n	n	NA
20	Sumati	NB & BB	birth	F	B	p	p	p	p	p	p	p	p	p	p		p	n	IE	NR	n	Cm-2	n	n	NA
21	Raja	NB	birth	M	L	p												n	S	S	n	Ncm	n	n	NA
22	Kamaraj	NB	birth	M	R		p					p						n	IE	PD	n	Cm-2	n	n	NA
23	Vanita	NB	15D	F	R	p						p	p	p				n	S	S	n	Ncm	n	n	NA
24	Mugashree	NB	birth	F	L	p												n	IE	O	n	Cm-2	n	n	NA
25	Laxmi	NB	birth	F	R	p												n	S	S	n	Ncm	n	n	NA

	LINEAR LICHEN PLANUS																								
S.No	NAME	TYPE	AGE OF ONSET	SEX	SIDE	HEAD & NECK	CHEST	ABDOMEN & PELVIS	BACK	THIGH	LEG	ARM	FOREARM	HANDS	FEET	PALMS & SOLES	GENITALS	MUCOSA	SYSMPTOMS	EVOLUTION OF LESION	DIRECTION OF EXTENSION	ASSOCIATIONS	BIRTH HISTORY	FAMILY HISTORY	BIOPSY	COMPATIBLE
1	Taufida	NB	2 Yr	F	R						p								p-i	E	DP	n	Ncm	n	Y	Y
2	Thangam	NB	45 Yr	F	R					p	p								p-i	E	B	n	Ncm	n	Y	Y
3	Ramayee	NB	55 Yr	F	L					p	p								p-i	E	DP	n	Ncm	n	Y	Y
4	Uma	NB	38 Yr	F	L	p													p-i	E	O	n	Ncm	n	n	NA
5	Shakuntala	NB	39 Yr	F	R					p	p								p-i	E	PD	n	Ncm	n	n	NA
6	Gurulakshmi	NB	16 Yr	F	L						p								p-i	E	S	n	Ncm	n	n	NA
7	Kaviya	NB	4 Yr	F	R					p									p-i	E	NR	Scarring alopecia/morphea	Ncm	n	Y	Y
8	Shivakumar	BB	11 Yr	M	R				p			p	p	p					p-i	E	DP	3rd R finger nail dystrophy	Ncm	n	Y	Y
9	Panjavarnam	NB	33 Yr	F	L					p	p								p-i	IE	DP	n	Ncm	n	Y	Y
10	Anagam	NB	25 Yr	F	L							p	p						p-i	E	DP	n	Ncm	n	Y	Y
11	Kala	NB	15 Yr	F	R					p	p								n	IE	NR	n	Ncm	n	n	NA
12	Sarathi	NB	26 Yr	M	R						p				p				p-i	E	B	LP other sites	Ncm	n	n	NA
13	Revathi	NB	19 Yr	F	L							p	p						p-i	E	DP	LP other sites	Ncm	n	n	NA
14	Muniyammal	NB	60 Yr	F	B					p	p								p-i	E	DP	LP other sites	Ncm	n	n	NA
15	Parameshwari	NB	48 Yr	F	L					p								p	p-i	E	DP	DM	Ncm	n	n	NA
16	Rubini	NB	21 Yr	F	R						p				p				p-i	NR	NR	n	Ncm	n	n	NA
17	Atmaram	NB	64 Yr	M	R				p			p	p	p				p	p-i	E	O,PD	HTN	Ncm	n	n	NA
18	Abdul Naseer	NB	46 Yr	M	R						p								p-i	NR	DP	LP other sites	Ncm	n	n	NA
19	Sudha	NB	28 Yr	F	R		p		p			p	p	p					p-i	E	B	n	Ncm	n	Y	Y
20	Saroja	NB	35 Yr	F	R							p							p-i	E	PD	n	Ncm	n	Y	Y
21	Mahalaxmi	NB	38 Yr	F	R					p	p							p	p-i	E	DP	n	Ncm	n	Y	Y
22	Sahana	NB	4 Yr	F	R				p			p		p					n	E	DP	n	Cm-3	n	Y	Y
23	Devasena	NB	38 Yr	F	R				p			p	p						p-i	E	DP	n	Cm-3	n	n	NA
24	Muthukrishnan	NB & BB	61 Yr	M	L							p	p						n	S	S	n	Cm-3	n	Y	Y
25	Saravanan	NB	18 Yr	M	L								p						p-i	E	DP	n	Cm-3	n	Y	Y

26	Rani	NB	51Yr	F	R								p	p				p	p-i	E	DP	LP other sites/Epileptic/ Parkinson's	Cm-2	n	n	NA
27	Gurulakshmi	NB	16Yr	F	L						p								p-i	E	DP	n	Ncm	n	Y	Y
28	Vinodini	NB	10Yr	F	R								p	p					n	NR	PD	R thumb nail-dystrophy	Cm-3	n	Y	Y
29	Shalini	NB & BB	7Yr	F	R						p				p				p-i	E	PD	LP other sites	Ncm	n	n	NA
30	Ayesha Beewi	NB	39Yr	F	B						p				p				n	NR	DP	DM/HTN	Ncm	n	Y	Y
31	Sethammal	NB	60Yr	F	R					p	p								p-i	E	DP	LP other sites	Ncm	n	n	NA
32	Gautam Sundaram	NB	18Yr	M	R						p								p-i	E	DP	n	Cm-3	n	n	NA
33	Palanimurugan	NB	13Yr	M	R					p	p								p-i	E	DP	n	Ncm	n	n	NA
34	Shantaram	NB	17Yr	M	L			p											p-i	E	O	LP other sites	Ncm	n	n	NA
35	Panjavaram	NB	48Yr	F	L								p					p	p-i	E	DP	DM	Ncm	n	n	NA
36	Kamakshi	BB	48Yr	F	B						p							p	p-i	E	DP	LP other sites	Ncm	n	n	NA
37	Meeran Beewi	NB	25Yr	F	L				p			p	p	p				p	p-i	E	I,DP	L thumb nail dystrophy	Ncm	n	n	NA

	LICHEN STRIATUS																							
S.No	NAME	TYPE	AGE OF ONSET	SEX	SIDE	HEAD & NECK	CHEST	ABDOMEN & PELVIS	BACK	THIGH	LEG	ARM	FOREARM	HANDS	FEET	PALMS & SOLES	GENITALS	SYMPTOMS	EVOLUTION OF LESIONS	DIRECTION OF LESIONS	ASSOCIATIONS	BIRTH HISTORY	FAMILY HISTORY	BIOPSY	COMPATIBLE
1	Santosh	NB	2Yr	M	L		p	p										n	E	O	n	Cm-2	n	n	NA
2	Sangeet ram	NB & BB	2Yr	M	R					p								n	NR	DP	n	Ncm	n	n	NA
3	Karthika	NB	9Yr	F	R					p								n	NR	DP	n	Ncm	n	n	NA
4	Swetha	NB	5Yr	F	L					p	p							p-pain	NR	DP	n	Ncm	n	Y	Y
5	Vineet kumar	NB	1.5Yr	M	R		p					p						n	E	O	n	Ncm	n	n	NA
6	Durga devi	NB	1Yr	F	R							p	p					p-i	NR	DP	n	Ncm	n	n	NA
7	Ashwati	NB	7Yr	F	R							p	p					n	E	DP	n	Cm-3	n	n	NA
8	Thayamani	NB	9Yr	F	L					p								n	E	DP	n	Ncm	n	n	NA
9	Vanumati	NB	3Yr	F	R				p									n	E	O	n	Ncm	n	n	NA
10	Santosh	NB	1.5Yr	M	R				p									n	E	O	n	Ncm	n	n	NA
11	Shridarshini	NB	2Yr	F	R					p								n	E	PD	n	Cm-3	n	n	NA
12	Vaishnavi	NB	2Yr	F	L					p								p-i	E	PD	n	Ncm	n	n	NA
13	Mutish kumar	NB	1.25Yr	M	L							p	p	p				n	E	DP	n	Ncm	n	n	NA
14	Krithika	NB	2Yr	F	R					p								n	E	B	n	Ncm	n	n	NA
15	Sarvana Kumar	NB	8Yr	M	L		p		p	p	p							n	NR	O	n	Cm-3	n	n	NA
16	Panju	NB	1Yr	F	L		p					p						n	NR	B	n	Ncm	n	n	NA
17	Jeevarajan	NB	11Yr	M	R	p												n	E	O	n	Ncm	n	n	NA
18	Jeevadarshini	BB	1Yr	F	R			p	p									p-i	E	I	n	Ncm	n	n	NA
19	Kirupa	NB	6mo	F	R	p												n	E	I	n	Ncm	n	n	NA
20	Wajid	NB	2Yr	M	R			p		p								p-i	E	B	n	Ncm	n	n	NA
21	K prabhakar	NB	21Yr	M	R		p		p	p		p	p					n	NR	NR	n	Ncm	n	n	NA
22	Rajapriya	NB	10Yr	F	R		p	p										n	E	NR	n	Ncm	n	n	NA
23	Chandralekha	NB	15Yr	F	L							p	p	p				p-i	IE	B	n	Ncm	n	n	NA
24	Bhuvaneshwari	NB	4Yr	F	R						p							p-i	E	DP	n	Ncm	n	n	NA
25	Manikandan	NB	4Yr	M	L		p		p	p		p	p					p-i	E	B	n	Ncm	n	n	NA
26	Kalejashree	NB	2Yr	F	L			p	p									n	IE	O	n	Ncm	n	n	NA
27	Pooja	NB	4Yr	F	R	p												n	E	O	n	Ncm	n	n	NA
28	Nirmal Kumar	BB	11Yr	M	L					p								n	E	DP	n	Ncm	n	n	NA
29	Sabish	NB	2Yr	M	R		p					p	p					n	E	DP	n	Ncm	n	n	NA
30	Aruneshwaran	NB	1.5Yr	M	R					p								n	E	DP	n	Ncm	n	n	NA
31	S. Siddharth	NB	7Yr	M	L		p											n	E	O	n	Ncm	n	n	NA
32	Deepadarshini	NB	6Yr	F	L					p	p							n	E	PD	n	Ncm	n	n	NA

33	Shanmugasundari	NB	6Yr	F	R	p											n	E	O	n	Ncm	n	n	NA
34	Nandini	NB	3Yr	F	L			p									n	NR	S	n	Ncm	n	n	NA
35	Peth Raj	NB	7Yr	M	L				p			p					n	E	O	n	Ncm	n	n	NA
36	Kanija	NB	3Yr	F	L		p		p								n	E	I	n	Cm-3	n	n	NA
37	Kaleeshwari	NB	10Yr	F	R					p	p						p-i	E	B	n	Ncm	n	n	NA
38	Sethupriya	NB & BB	10Yr	F	R					p	p				p		n	E	DP	n	Ncm	n	n	NA
39	Asharuddin	NB	14Yr	M	R	p											n	E	O	n	Ncm	n	n	NA
40	Satyra	NB	6Yr	M	R	p	p										n	S	S	n	Cm-3	n	n	NA
41	Tirupati Selvi	NB	1.5Yr	F	R				p			p	p	p			n	E	O	n	Cm-3	n	n	NA
42	Jagan	NB	11Yr	M	L					p	p						p-i	E	B	n	Ncm	n	n	NA
43	Vineetha	NB	8Yr	F	R					p	p						p-i	NR	PD	n	Cm-3	n	n	NA
44	Ramachandran	NB	6Yr	M	R				p			p					p-i	IE	O	n	Ncm	n	n	NA
45	Selvam Shabari	NB & BB	3Yr	M	L					p	p						n	E	DP	n	Ncm	n	n	NA
46	Priyadarshini	NB	3Yr	F	L						p						n	E	PD	n	Ncm	n	n	NA
47	B. Rishikesh	NB	2Yr	M	L								p	p			n	E	PD	n	Ncm	n	n	NA
48	Sahana	NB	1.75Yr	F	L		p		p								n	E	I	n	Ncm	n	n	NA
49	Balakrishnan	NB	2.75Yr	M	R									p			n	E	DP	R 3rd finger nail dystrophic	Ncm	n	n	NA
50	Bhaskaran	NB & BB	9Yr	M	R						p				p		p-i	E	DP	R 4th toe nail dystrophic	Ncm	n	n	NA
51	Sushmita	NB	8Yr	F	L				p			p	p				p-i	E	PD	n	Cm-3	n	n	NA
52	Malavender	NB	12Yr	M	R					p	p						n	E	PD	n	Ncm	n	n	NA
53	Yasid Kumar	NB	5Yr	M	R				p			p					p-i	E	O	n	Ncm	n	n	NA
54	Sumati	NB	19Yr	F	L								p				n	E	O	n	Cm-3	n	n	NA
55	Selvalakshmi	NB	3Yr	F	R		p		p			p					n	NR	O	n	Cm-3	n	n	NA
56	Pandichelvi	NB	1mo	F	R				p			p					n	NR	O	n	Ncm	n	n	NA
57	Dharanya	NB	3Yr	F	L					p	p						p-i	E	B	n	Ncm	n	n	NA
58	Kartika	NB	6Yr	F	L		p					p	p				n	E	PD	n	Ncm	n	n	NA
59	Pavitra	NB	2Yr	F	L							p	p				n	E	DP	n	Ncm	n	n	NA
60	Vidya	NB	4Yr	F	L							p	p				p-i	E	PD	n	Ncm	n	n	NA
61	Shiva Abhimanyu	NB	1Yr	M	L					p							n	NR	DP	n	Ncm	n	n	NA
62	Aarti	NB & BB	7Yr	F	R					p	p						p-i	E	DP	n	Cm-3	n	n	NA
63	Manikandan	NB	2Yr	M	B				p-L	p-R		p-L					n	E	PD	n	Ncm	n	n	NA
64	Nitin Raj	NB	2Yr	M	R				p			p					n	NR	NR	n	Ncm	n	n	NA
65	Anjali	NB	NR	F	L						p						n	NR	NR	n	Cm-3	n	Y	Y
66	Muneera	NB & BB	9Yr	F	R				p			p					p-i	E	DP	n	Ncm	n	n	NA
67	Suryaprakash	NB	3Yr	M	L					p							n	E	DP	n	Ncm	n	n	NA
68	Kartikapriya	NB	11Yr	F	L							p					n	E	PD	n	Ncm	n	n	NA
69	Logesh Kumar	NB	4Yr	M	B					p-R	p-R	p-L					n	E	DP	n	Ncm	n	n	NA
70	Kalpana Devi	NB	5Yr	F	R					p							p-i	E	PD	n	Ncm	n	n	NA
71	Satya	NB	20Yr	F	L							p	p				p-i	E	PD	n	Ncm	n	n	NA

72	Aishwarya	NB & BB	9Yr	F	R						p							n	NR	DP	n	Ncm	n	n	NA
73	Yashika	NB	14Yr	F	R							p	p					p-i	E	B	n	Ncm	n	n	NA
74	Darshiya	NB	5Yr	F	L							p	p	p				p-i	E	B	n	Ncm	n	n	NA
75	Divya	NB	11Yr	F	R					p								n	E	DP	n	Cm-3	n	n	NA
76	Jayasurya	NB	1.5Yr	M	R					p								p-i	E	PD	n	Ncm	n	n	NA
77	Bharati	NB	11Yr	F	L							p						n	E	PD	n	Ncm	n	n	NA
78	Priya	NB	9Yr	F	R		p					p	p					n	E	O	n	Cm-2	n	n	NA
79	Anushree	NB	1.5Yr	F	R		p											n	E	O	n	Cm-2	n	n	NA
80	Roshini	NB	7Yr	F	L						p				p			p-i	E	DP	n	Ncm	n	n	NA
81	Harish Babu	NB	5Yr	M	L	p												n	NR	NR	n	Ncm	n	n	NA
82	Mareeshwari	NB	3Yr	F	R		p		p			p	p					p-i	E	O	n	Ncm	n	n	NA
83	Ajay	NB	3Yr	M	R					p	p							n	E	DP	n	Ncm	n	n	NA
84	Preeti	NB	4Yr	F	R					p	p							p-i	E	PD	n	Cm-3	n	n	NA
85	Gokila	NB	19Yr	F	L				p			p	p					n	NR	DP	n	Ncm	n	n	NA
86	V. Kumaran	NB	3Yr	M	L							p	p	p				n	NR	DP	n	Cm-2	n	n	NA
87	Pooja	NB	8mo	F	L					p	p							n	E	B	n	Ncm	n	n	NA
88	Edwin Raj	NB	11Yr	M	L	p												p-i	E	O	Pityriasis rosea	Ncm	n	n	NA
89	Manimeghalai	NB	14Yr	F	L						p							n	E	PD	n	Cm-3	n	n	NA
90	Revathy	NB	3Yr	F	R		p		p	p		p	p					n	E	PD	n	Cm-2	n	n	NA
91	Surya	NB	8Yr	M	R		p		p			p						n	E	PD	n	Ncm	n	n	NA
92	Aparna	NB	3mo	F	L				p			p	p					n	IE	O	n	Ncm	n	n	NA
93	Ulageshwaran	NB	1.5Yr	M	L								p	p				p-i	E	PD	n	Ncm	n	n	NA
94	Kanira	NB	2Yr	F	L		p					p	p					p-i	E	NR	n	Cm-2	n	n	NA
95	Madesh	NB	2Yr	M	R					p								n	E	DP	n	Ncm	n	n	NA
96	Aramugam	NB	1.75Yr	M	L				p			p	p	p				n	E	DP	n	Cm-3	n	n	NA

	NEVUS ACHROMICUS																								
S.No	NAME	TYPE	AGE OF ONSET	SEX	SIDE	HEAD & NECK	CHEST	ABDOMEN & PELVIS	BACK	THIGH	LEG	ARM	FOREARM	HANDS	FEET	PALMS & SOLES	GENITALS	SYMPTOMS	EVOLUTION OF LESIONS	DIRECTION OF EXTENSION	ASSOCIATIONS	BIRTH HISTORY	FAMILY HISTORY	BIOPSY	COMPATIBLE
1	Bharati	BB	birth	F	R			p										n	S	S	ASD	Cm-2	n	n	NA
2	Devibala	NB	birth	F	L								p					n	S	S	seizure disorder	Ncm	n	n	NA
3	B/O Muthumari	BB & Phylloid	birth	M	R	p	p					p						n	S	S	n	Ncm	n	n	NA
4	B/O Sundari	Phylloid	birth	F	B		p-L	p-R		p-R		p-L						n	S	S	n	Ncm	n	n	NA
5	Ajay kumar	BB	birth	M	L		p					p						n	IE	O	n	Ncm	n	n	NA
6	Sanjay Pandi	Phylloid	birth	M	L		p		p									n	NR	NR	Mild MR with seizure disorder	Cm-3	n	n	NA
7	Punishwara	BB	birth	M	B		p-L	p-R		p-L							p	n	S	S	n	Ncm	n	n	NA
8	Yogeshwaran	BB	birth	M	B	p	p-B	p										n	S	S	n	Ncm	n	n	NA
9	Karthikeyan	Phylloid	20 D	M	L		p	p				p						n	S	S	n	Ncm	n	n	NA
10	Subhash	Phylloid	birth	M	L			p	p									n	S	S	n	Ncm	n	n	NA
11	Kamesh	Phylloid	1.5mo	M	R		p	p		p								n	S	S	n	Ncm	n	n	NA
12	Nandini Mahalaxmi	NB & BB	15D	F	L	p	p		p			p						n	S	S	n	Ncm	n	n	NA
13	K Pandi	BB	18Yr	M	L		p		p									n	S	S	n	Cm-3	n	n	NA
14	Sundari	Lateralization	birth	F	L		p		p			p	p					n	S	S	n	Cm-3	n	n	NA
15	Anees Fatima	NB & BB	birth	F	L					p	p							n	S	S	n	Ncm	n	n	NA
16	Neha	NB	birth	F	R					p	p							n	S	S	n	Ncm	n	n	NA
17	Tirupati	NB & BB	21Yr	M	L							p						n	S	S	n	Ncm	n	n	NA
18	A Daneeshwari	BB	birth	F	B	p			p									n	S	S	n	Ncm	n	n	NA
19	Priyadarshini	BB	8D	F	B	p-R	p-L					p-L					p	n	S	S	n	Cm-2	n	n	NA
20	Harish kumar	NB & BB	birth	M	L					p	p						p	n	S	S	n	Ncm	n	n	NA
21	Hemant	Phylloid	birth	M	L					p	p							n	S	S	n	Ncm	n	n	NA
22	Hariharan	Phylloid	birth	M	B		p	p		p		p-R	p-R					n	S	S	n	Ncm	n	n	NA
23	Srimati	NB	birth	F	L								p					n	S	S	Craniostosis/microcephaly	Ncm	n	n	NA
24	Madesh Bharati	Phylloid	birth	M	R		p	p				p					p	n	S	S	n	Cm-2	n	n	NA
25	Gabilan	BB	birth	M	B	p		p-L		p-B	p-B						p	n	S	S	n	Ncm	n	n	NA
26	Sinchana	NB & BB	birth	F	L		p	p	p			p	p	p				n	S	S	L thumb nail dystrophy	Cm-2	n	n	NA
27	T J Udaykumar	Lateralization	birth	M	L		p	p	p			p						n	S	S	n	Ncm	n	n	NA
28	Yogeshwaran	BB	birth	M	L		p		p			p						n	IE	O	n	Ncm	n	n	NA

OTHER DERMATOSES

S.No	NAME	TYPE	DIAGNOSIS	AGE OF ONSET	SEX	SIDE	HEAD & NECK	CHEST	ABDOMEN & PELVIS	BACK	THIGH	LEG	ARM	FOREARM	HANDS	FEET	PALMS & SOLES	GENITALS	SYMPTOMS	EVOLUTION OF LESIONS	DIRECTION OF EXTENSION	ASSOCIATIONS	BIRTH HISTORY	FAMILY HISTORY	BIOPSY	COMPATIBLE
1	Pandiammal	NB	DD	52Yr	F	L		p	p		p	p	p	p				p	p-i	S	S	DM &HTN	Ncm	n	Y	Y
2	Poongavana	BB	DUH	5Yr	F	B		p	p	p	p	p	p	p					n	S	S	n	Ncm	n	Y	Y
3	Gowri	NB	HI	8Yr	F	B	p												n	S	S	n	Ncm	n	n	NA
4	Renuka Devi	NB	HI	birth	F	R			p		p								n	IE	O,PD	n	Ncm	n	n	NA
5	Vijaybharati	NB	HI	birth	M	R				p			p						n	NR	NR	n	Ncm	n	n	NA
6	Nagapandiyan	BB	HI	birth	M	B		p	p	p	p	p	p	p					n	S	S	n	Ncm	n	n	NA
7	B/O Punita	NB	IP	1D	F	R		p											n	S	S	n	Ncm	F sibling died 5 D after birth	n	NA
8	Devadarshini	NB	LN	10Yr	F	R							p	p					p-i	E	S	LN other sites	Ncm	n	Y	Y
9	Semmala	NB	LPP	17Yr	F	R		p					p	p	p		p		n	E	O,PD	LPP other sites	Ncm	n	n	NA
10	Praveen kumar	BB	LM	10Yr	M	R					p	p				p			p-i	IE	DP	n	Ncm	n	Y	Y
11	Shyamaladevi	BB	LM	13Yr	F	B					p	p							n	NR	DP	n	Ncm	n	Y	Y
12	Ponni	BB	LM	12Yr	F	L							p	p					n	E	DP	morphea other sites	Ncm	n	Y	Y
13	Thangaselvi	BB	LM	28Yr	F	L							p	p	p				n	E	PD	morphea other sites, TT vaccine/ pregnancy	Ncm	n	Y	Y
14	Seetharaman	BB	LM	14Yr	M	R							p	p	p				n	E	DP	morphea other sites	Ncm	n	Y	Y
15	Vasanthi	Phylloid	LV	4Yr	F	R				p			p						n	I	B	n	Ncm	n	n	NA
16	Kalidas	Phylloid	LV	10Yr	M	L					p								p-i	IE	DP	n	Ncm	n	n	NA
17	Rajeshwari	Phylloid	LV	5Yr	F	R					p	p						p	n	IE	PD	vitiligo other sites	Cm-3	n	n	NA
18	Saranya	NB	LSA	11Yr	F	R					p								p-i	E	DP	vitiligo other sites	Ncm	n	Y	Y
19	Abi	BB	LWNH	birth	F	B				p	p							p	n	E	DP	n	Ncm	n	n	NA
20	Darshadevi	NB	LWNH	birth	F	B					p- R & L	P-L							n	E	S	n	Ncm	n	n	NA
21	Sreekanth	BB	LWNH	7Yr	M	R								p	p				n	IE	B	n	Cm-2	n	n	NA
22	S/O Chitrakala	Phylloid	LWNH	birth	M	L					p	p							n	S	S	n	Ncm	n	n	NA
23	Manikandan	NB & BB	LWNH	1mo	M	L					p								n	R	NR	n	Ncm	n	n	NA

24	Kalidas	NB	LWNH	NR	M	L		p	p				p						n	S	S	reticular pattern back	Cm-2	n	n	NA
25	Shivashankari	NB & BB	LWNH	birth	F	B					p	p							n	S	S	n	Ncm	n	n	NA
26	Karthik	NB	NS	birth	M	L	p												n	S	S	n	Ncm	n	Y	Y
27	S/O Srirangam	BB	NS	birth	M	L	p												n	S	S	n	Ncm	n	n	NA
28	Pandiyan	NB	NSS	23Yr	M	L	p												p-i	IE	PD	facial asymmetry/ seizures	Ncm	n	Y	Y
29	Sudarshan	NB	NP	birth	M	R								p	p				p-i	E	DP	valvular CHD	Cm-3	n	Y	Y
30	Raji	NB	NP	birth	F	L	p						p	p	p				p-i	E	PD	psoriasis other sites	Ncm	n	Y	Y
31	Saranya	NB	NP	6mo	F	R					p	p							p-i	E	DP	n	Ncm	n	Y	Y
32	Panju	NB	SF	39Yr	F	L	p												n	S	static	pemphigus vulgaris	Ncm	n	Y	Y