"ONE YEAR HOSPITAL BASED CROSS-SECTIONAL STUDY OF DERMOSCOPIC FINDINGS IN VITILIGO CORRELATING WITH LESIONAL ACTIVITY."

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With reference to the above, we wish to inform you that your proposed research project titled **"ONE YEAR HOSPITAL BASED CROSS SECTIONAL STUDY OF DERMOSCOPIC FINDINGS IN VITILIGO CORRELATING WITH DISEASE ACTIVITY ",** is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

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LIST OF ABBREVIATIONS USED

SL. NO.	ABBREVIATION	EXPANSION
1	TRP	Tyrosine related protein
2	HLA	Human leukocyte antigen
3	NBUVB	Narrow band ultra violet B
4	FOXP1	Forkhead boxP1
5	KIT	Receptor tyrosine kinase
6	TYR	Tyrosinase
7	LAMP	Lysosomal associated membrane protein
8	LXR	Liver x receptor
9	TYRP1	Tyrosinase-related protein 1
10	IL -RA	Interleukin 2 receptor
11	CCR6	Chemokine receptor type 6
12	GM-CSF	Granulocyte-macrophage colony-stimulating factor
13	VASI	Vitiligo area and severity index
14	TSH	Thyroid stimulating harmone
15	PUVA	Psoralen and Ultraviolet-A
16	SV	Segmental vitiligo
17	DNA	Deoxyribonucleic acid
19	VEGF	Vascular endothelial growth factor

20	**DEJ**	**Dermo- epidermal junction**
21	**H_2O_2**	**Hydrogen peroxide**
22	**KP**	**Koebner phenomenon**
22	**VIDA**	**Vitiligo disease activity score**
23	**ICAM**	**Intercellular adhesion molecule**
24	**LSEA**	**Lichen sclerosus et atrophicus**
25	**NB-UVB**	**Narrow band-Ultra violet B spectrum**
26	**MBEH**	**Mono benzyl ether of hydroquinone**

ABSTRACT

Background: With a prevalence of 0.5% to 2% and no preference for one race or gender over another, vitiligo is a frequent acquired depigmentary disorder. Clinically, it manifests as white depigmented macules to patches due to melanocyte loss, either from a failure to control cellular stress or through autoimmune destruction. Dermoscopy may help in noninvasive confirmation of the diagnosis by ruling out other clinically imitating hypopigmentary disorders since vitiligo is mostly a clinical diagnosis. Furthermore, dermoscopy is quickly becoming recognized as a crucial auxiliary technique for assessing disease activity. Due to the relative dearth of literature on aspects of correlation between clinical and dermoscopic findings in vitiligo, we undertook this study to determine stability in vitiligo based on dermoscopic findings criteria and to correlate the dermoscopic features with clinical activity of the lesion and eliminate the need for biopsy.

Aims & Objectives:

- **Primary Objective**: To analyze dermoscopic patterns in diagnosed cases of vitiligo.
- **Secondary objective**: To correlate vitiligo clinical activity of lesion with dermatoscopic findings.

Materials and method: This was. a hospital-based cross-sectional study, which was done over a duration of one year. In a total of 100 study subjects, 352 lesions that are clinically diagnosed as vitiligo and screened by woods lamp were included in this study. After obtaining consent, all participants underwent clinical, Wood's lamp and dermoscopic examination making use of ILLUCO IDS -1100 model, under 10X magnification, with findings being noted along with the recording of both clinical and

dermoscopic images. Records were maintained and analyzed statistically. The data was analyzed using statistical software R version 4.0. and Microsoft Excel.

Results: CLINICAL ASSESSMENT ACTIVITY

On clinical examination, out of 100 patients, 74 patients showed unstable activity and 26 patients had stable activity. 10 % of the the study population showed Koebner's phenomenon.

Dermoscopic assessment –

a. **BORDER** on dermoscopic examination out of 352 lesions examined 247 lesions showed ill-defined borders and 105 lesions showed sharp borders.

b. **PIGMENT NETWORK** on examination out of 352 lesions examined 182 showed absent pigmentation and 119 showed reduced pigmentation.

c. **PERIFOLLICULAR HYPERPIGMENTATION** on dermoscopic examination out of 352 lesions examined 11.1 % i.e. 39 showed perifollicular hyperpigmentation.

d. **PERILESIONAL HYPERPIGMENTATION** 9.7% of the study population showed perilesional hyperpigmentation.

e. **SATELLITE LESIONS** of 352 lesions, 9.7 % i.e.., 34 showed satellite lesions.

f. **MICROKOEBNERS** - microkoebners phenomenon was seen in 3.7% of lesions examined.

Using BPleFoSK criteria for dermoscopic assessment of stability out of 352 lesions on dermoscopic examination 100 lesions are scored for stable vitiligo and 252 lesions showed features of unstable vitiligo. A correlation was present between Clinical and Dermoscopic features in 248 lesions (70.5%). However, in the remaining 104 lesions (29.5%) there was no correlation between Clinical and Dermoscopic

assessments. In 45 lesions both assessments i.e., clinical & dermoscopic examination recorded stable activity and in 203 lesions both clinical & dermoscopic assessments recorded unstable activity. But in 49 cases clinical examination recorded stable activity while dermoscopic examination recorded unstable activity. In 55 cases clinica examinationl recorded unstable activity while dermoscopic examination recorded stable activity.

Conclusion: Patients who were diagnosed with unstable vitiligo clinically were diagnosed with unstable vitiligo on dermoscopic evaluation well. However, in 13.9 % of lesions that were clinically diagnosed with stable disease were diagnosed with unstable vitiligo following dermoscopic examination. Henceforth dermoscopy is a better indicator of assessment of stability. It can predict instability in clinically stable lesions and thus aids in management modification, reduces the delay in planning the surgery, and can act as a visual indicator in patient counseling. Altogether, the sensitivity and specificity of dermoscopy are inarguably high, hence making it an important prognostic tool in the assessment of vitiligo.

Limitations: The study's shortcomings were its limited sample size, which could have reduced the power of the study. Additionally, biopsy was not performed and, therefore, tissue diagnosis was not available for confirmation of the clinical and dermoscopic conclusions.

Keywords: Vitiligo, Dermoscopy, Wood’s lamp

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INTRODUCTION

Vitiligo, a widespread depigmentary condition of the skin is also cited as swetakusta in Indian literature. The condition has a 0.5%-2% frequency with no racial or sex preference[1]. On clinical examination, it is characterized by white depigmented macules to patches because of melanocyte depletion which is caused either by defects to manage cellular stress or autoimmune destruction[1].

Conservative medical therapy has constraints and might not cause complete repigmentation in a total subset of patients. However, none of the medical or surgical course of action is yet to guarantee efficacy in all instances. The basis of its management depends on the right categorization of the case into its 2 types-stable vitiligo and unstable vitiligo[2]. Stability is the decisive factor, the cornerstone of vitiligo therapy.[3]

The basis for the clinical categorization of disease activity for stability is issued by prasad Davinder and Somesh Guptha as

i. a patient reporting no new lesions,
ii. No progression of existing lesions
iii. Absence of Koebner phenomenon during past one year[4].

Though vitiligo is of clinical diagnosis, woods lamp will help in differentiating it from other hypopigmented lesions. Woods lamp is a bed side diagnostic tool in which skin, hair, teeth and others are examined. It detects disorders of skin pigment including vitiligo and other skin irregularities. There is reduced or no epidermal melanin in hypopigmented or depigmented lesions of vitiligo. As a result, the light-induced autofluorescence of dermal collagen may be viewed via a glass. The rims of

hypopigmented or depigmented areas look sharper under Wood's light due to the abrupt cut-off in visible emission from lesional skin. Because of autofluorescence, the lesions look vivid blue-white.[5] In practice, diagnosis of vitiligo is clinical, and Dermoscopy may enable noninvasive confirmation by ruling out additional medical disorders that mimic hypopigmentation.[1] More remarkably, dermoscopy is gaining momentum quickly as an indispensable ancillary tool to assess the severity of this condition. Dermoscopic findings observed in vitiligo are perifollicular changes, perilesional changes, disruption in the pigmentary network, and the presence of certain features such as the tapioca sago appearance, comet tail appearance, leukotrichia, telangiectasia, and any new features.[6]

AIMS AND OBJECTIVES

- **Primary Objective**:

  To analyze dermatoscopic patterns in diagnosed cases of vitiligo.

- **Secondary objective**:

  To correlate vitiligo clinical activity with dermatoscopic findings

REVIEW OF LITERATURE

The largest single organ in the human body is the skin, and pigmentation of the skin provides a distinct identity to an individual and provides an identity of race and ethnicity to a population[7]. Like any other organ, the pigmentary system is also prone to disorders that are classified using various parameters.

COLOUR OF THE SKIN

The three factors that play a crucial role in determining the pigment of one's skin are the following[8]:

- Content of hemoglobin in both oxygenated and deoxygenated blood,
- Content of carotenoids[9], and
- Content of the pigment melanin[10].

Among these three main determinants, melanin is the primary pigment that dictates the colour of an individual[11,12]. The differences in the color between races and ethnicities are due to the quantity, dimensions, silhouette, distribution, and degradation of melanosomes, which are melanin-laden organelles that are transferred to the contiguous epidermal keratinocytes.[8,9] Melanin includes a group of polymorphous biopolymers and includes eumelanin, pheomelanin, mixed melanin, and neuromelanin. Mammalian melanocytes create two chemically varied melanin pigment types — eumelanin, a brown-black pigment, and yellowish-red pheomelanin[13–15]. In humans, the following three major classes of integumentary melanin are seen[16,17] (Figure 1)

- Eumelanin: it is manufactured by ellipsoidal melanosomes and is responsible for the brown and black colors of both the skin and hair.[18,19]
- Pheomelanin: It is produced in so-called spherical melanosomes and is accountable for the lighter shades of hair—from yellow to reddish brown.[20,21]
- Trichochromes: Sulphur-containing phaeomelanin pigments with a well-defined structure characterised by a chromophore.[22]

Highly pigmented skin colour contains several large individual melanosomal particles (0.5–0.8 mm in diameter).[15,16,23] The lighter pigmentation contains fewer and smaller melanosomal particles (0.3–0.5 mm in diameter).[15,16] Melanogenesis is a very highly regulated process that results from strong cellular and molecular connections between cell populations in the skin with the key players being fibroblasts, keratinocytes, and melanocytes.[24]

VITILIGO

The most prevalent multifactorial disorder, "**Vitiligo is an acquired, chronic, depigmenting disorder of the skin, in which pigment-producing cells (melanocytes) that determine the colour of skin, hair, and eyes are progressively lost. It appears as milky-white patches of skin and can be cosmetically very disabling, particularly in people with dark skin**".[25] vitiligo is characterised by undyed milky white patches that have different distribution patterns and is caused by the autoimmune destruction of melanocytes. This asymptomatic chronic cosmetic disfiguration has an erratic course and longevity. Destruction of melanocytes by T-cells is what results in depigmentation[25]. Genetic susceptibility, autoimmune disease, and environmental variables all play a role in the etiology. The social stigma associated with vitiligo can have a substantial negative impact on patient's' mental

health. It also has a negative correlation with skin cancer risk but positive correlation with risk of other autoimmune illnesses.

HISTORY

Since ancient times, vitiligo has been recognised . Description of vitiligo has been documented in the period of Aushooryanas as early as 2200 BC by Tarikh-e-Tib-eIran[26]. Diseases with similar description as vitiligo have been reported in ancient Indian historical texts of "Atharva Veda", which dates back to 1400 BC[26] . In an Indian dialect it was known as "Shweta kushtha", which means "white leprosy"[26]. In the Bible, it has been described as "Zoraat".[27]

EPIDEMIOLOGY

Vitiligo is thought to affect fewer than 0.5% of people worldwide on an average.[28] However, another study has reported a prevalence of 0.3–1.1% .[29] In India, several studies have reported rates of 0.46–8.8% in Gujarat [30,31] and 0.5–2% in Chennai. In children, the reported prevalence is 26% in South India[32] and 23.3% in North India[33].

GENETIC BACKGROUND

Associations of vitiligo with other autoimmune or endocrine diseases is rare[34].

Various genes associated with vitiligo and its comorbidities are discussed in [31] (Table 1).

Gene	Protein	Function	Comorbilities
RERE	Atrophin like protein 1	Regulation apoptosis	
PTPN22	Lymphoid specific protein tyrosinase phosphatase nonreceptor 22	Regulation T cell receptor signaling	Type 1 DM, Grave's disease, RA, Addison's disease, psoriasis, IBD
CTLA4B	Cytotoxic T lymphocytes antigen 4	Inhibition of T cells	Type 1 DM, Grave's disease, Hashimoto's thyroiditis, IBD, SLE
FOXP1	Forkhead box P1	Regulation of lymphoid cell development	
TSLP	Thymic stromal lymphoprotein	Regulation of T cell and DC maturation	
CCR6	Chemokine receptor type 6	Regulation of B cell differentiation	IBD, AR, Grave's disease
IL2RA	Interleukin 2 receptor	Regulation of lymphocyte response to bacteria	Type 1 DM, Grave's disease, RA, multiple sclerosis, SLE
GZMB	Granzyme B	Mediator of T cell and NK apoptosis	
FOXP3	Forkhead box P3	Regulation of T-reg	

Table 1: Genes associated with vitiligo classification.[31]

CLASSIFICATION

Vitiligo has been classified using different classifications based on the site, severity, and prognosis.[34]

1. Revised classification as per 2012 global consensus – based on site , broadly classified into non-segmental, segmental, and unclassified.(Table 2)
2. Clinical classification of vitiligo – based on severity – used for evaluating different therapeutics. For example,<10% topical therapy can be preferred.(Table3)
3. Classification based on severity of prognosis : broadly into progressive type of vitiligo, stable type, and improving vitiligo.(Table 3)

Localized		Generalized			Universal
Focal	*Segmental*	*Acrofacial*	*Vulgaris*	*Mixed*	
One or more patches in one area but not in segmental pattern	One or more macules in dermatomal, unilateral distribution.	Affects face and distal extremities	Symmetrical distribution of lesions in typical zones	Segmental along with vulgaris or acrofacial	Involves more than 80% of the body

Table 2: Revised classification as per the 2012 global issue consensus conference based on clinical distribution.[34]

Types	Subtypes
Non-segmental vitiligo	Acrofacial Mucosal (more than one site affected) Generalized or Common Universal Mixed (associated with segmental vitiligo) Rare forms
Segmental	Unisegmental, bisegmental or multisegmental
Unclassified or indeterminate	Focal Mucosal (only one site affected)

Table 3: Revised classification/nomenclature of vitiligo and related issues: the Vitiligo Global Issues Consensus Conference – 2012[34]

Based on severity vitiligo can be divided into 4 stages	
Limited involvement	10%
Moderate	10-25%
Moderately severe	26-50%
Severe disease	>50%

Table 4: Classification based on the severity of disease .[34]

1. Progressive type of vitiligo (V1):
• Occurrence of new lesions
• progressing old lesions
• presence of ill-defined borders
2. Quiescent or stable type of vitiligo (V2):
• Not developing new lesions
• No progression of old lesions
• Well-defined pigmented borders
3. Improving vitiligo type (V3) :
• Disappearing vitiligo lesions

Table 5: Classification based on the severity of prognosis [34]

Aggravating factors: the aggravating causes of vitiligo comprise

- Trauma
- Stress on an emotional level
- Drugs
- Physical strain etc.

PATHOGENESIS

Numerous possible theories have been proposed to explain the pathophysiology of vitiligo.

Genetic Background:

30% of people with vitiligo have a family history, of which 20% of subjects had first-degree relatives. Polygenic traits are those that include more than three di-allelic alleles.[35]

Black people and Moroccan Jews both have HLA-DR4 and HLA-B13 respectively.[26] HLA-A2, HLA-B12 presence co-relates with vitiligo.[36,37]

Autoimmune hypothesis:

Vitiligo is seen as an autoimmune illness due to a related other autoimmune condition.

- Patient's serum contains organ-specific antibodies.
- First-degree relatives of patients with auto-antibodies
- HLA – DR1, HLA-DR4.
- the existence of melanocyte-specific autoantibodies.

Hydrogen peroxide - H_2O_2 theory:

Human tyrosinase is reversibly inhibited by hydrogen peroxide. Lesional and non-lesional epidermis both exhibit reduced catalase activity, which leads to an increase in peroxidase concentration.

Deficiency of Melanocyte growth factor theory

Melanocytes in normal and perilesional skin develop improperly when melanocyte growth factors are present at lower concentrations in vitro.[39]

REDUCED MELANOCYTE LIFESPAN HYPOTHESIS:

Keratinocyte releases interleukin-1. and interferon-gamma, which induced melanocyte apoptosis, where c-KIT plays an important role.[40] People with vitiligo have early apoptosis due to decreased c-KIT receptors in melanocytes.

LIVER X RECEPTOR (LXR ROLE) - the modulation of liver receptors causes

- Reduced permeability barrier performance
- Reduced epidermal proliferation
- Altered differentiation
- Carcinogenesis

When compared to healthy skin, Kumar et al.[41] found that ill people's perilesional skin had higher LXR-/b levels.

NEURAL HYPOTHESIS

Leners theory stated that Melanocytes are poisoned by the neurochemical compounds produced by nerve terminals.[42] this can be justified by the " vitiligo are spared in limbs which are paralyzed, peripheral nerve injury followed by the appearance of vitiligo."[43] The histopathological examination of vitiligo shows thickness of basement membrane of Schwann cells, the striking finding observed.[44]

Auto Cytotoxic Hypothesis[45]

Quinine, an intermediate product of melanin synthesis, is toxic to melanocytes. Tyrosinase (TRP-1&2, Calnexin, and LAMP-1) helps in melanin repigmentation. Mutation in TRP-1 protein results in the degeneration of cells, finally leading to melanocyte apoptosis. Destruction of melanocytes is protected by the Compartmentalization of melanosomes in normal individuals.

ASSOCIATIONS

There are connections between vitiligo and other autoimmune diseases, such as thyroid abnormality, pernicious anemia, Addison's.Disease., alopecia areata, diabetes mellitus, pemphigus vulgaris, and polyglandular autoimmune syndrome. An association rate of 1.3% was observed in an Indian study.[46] The most common associations are observed in non-segmental vitiligo [47]. Mazereeuw–Hautieret al.[48]reported a rate of association of 11.3% with thyroid function abnormalities without frank clinical disease; additionally, the Only non-segmental vitiligo showed a connection; segmental vitiligo did not show any association.

In adults with vitiligo, autoimmune thyroiditis leading to hypothyroidism is seen in approximately 30% [49] of patients when compared to 10% in the common population [50]. Lacovelli et al. [47] evaluated paediatric cases of vitiligo and noted that 16% of children with non-segmental Vitiligo showed impaired thyroid function metrics but none of the children with. segmental .vitiligo patients had the same defects. Additionally, in girls, abnormal thyroid values were more frequently detected., and hypothyroidism status was more commonly seen in these children than in adults with vitiligo. While associated endocrinopathies are rare in children, asymptomatic presence of autoantibodies is seen sometimes [51]. Antinuclear antibodies are less common in children [52]. Vitiligo-associated halo nevi are more common in children with vitiligo, especially non-segmental vitiligo .[46,51-53]

FUTURE COURSE OF THE DISEASE

Only a few people develop a progressive or recurring pattern of the disease; the course is mostly stable or regressive in most of the patients.[34] Absolute spontaneous re-pigmentation in non-segmental vitiligo is not common. However,

spontaneous re-pigmentation is common in a few .[5] Following onset of the disease, Segmental vitiligo spreads rapidly just along the dermatome that is affected and does not alter for the course of the patient's life.[55]

VITILIGO AS A PART OF HERIDITARY SYNDROMES

Vitiligo may be a component of several of the following disorders in older kids and teenagers.[54]

1. **Vogt–Koyanagi–Harada syndrome**: Its distinctive symptoms include uveitis, aseptic meningitis, dysacusia, alopecia, poliosis, and vitiligo. Lesions from vitiligo often affect the head, neck, and trunk and are symmetrical. A typical location for vitiligo is the sacral area.

2. **Alezzandrini syndrome**: Defining features of this syndrome are poliosis, ipsilateral uveitis with impaired visual acuity, segmental vitiligo (cheek), and ipsilateral partial loss of hearing. The symptoms often appear throughout adolescence. The predominant clinical hallmarks of each of these illnesses are uveitis and accompanying ocular symptoms.

In these conditions, vitiligo develops later and is typically treatment-resistant.[52]

CLINICAL FEATURES:

- **Segmental vitiligo:** Early onset, unilateral distribution, and quick stabilization of vitiligo are characteristics of this variant of vitiligo. This is less frequently associated with autoimmune disorders and family history than other forms of vitiligo. The face is the most frequent area to develop segmental vitiligo, followed in by the scalp, neck, limbs, and trunk.in order of decreasing frequency.

Autoimmune destruction of melanocytes due to[27]

(i) somatic mosaicism,

(ii) neuronal mechanism

(iii) microvascular skin homing

A dermatomal pattern may or may not be present in segmental vitiligo. In segmental vitiligo, surgical techniques gives a good repigmentation.

Non-segmental vitiligo: This is a type of vitiligo which has an indolent course, which progresses all throughout life. In few patients, advancement may be halted sometimes due to various factors.[56]

Focal vitiligo - Most cases of vitiligo commence as focal vitiligo. It is recognized by one or a few macules that primarily follow the distribution of the trigeminal nerve. It may also appear in the neck or the trunk.

Acrofacial vitiligo- this variant of vitiligo which normally involves ocular and oral orifices peripherally with involvement of extremities mostly, distal parts of the extremities. (upper limb involvement is more commonly seen than lower limb)

Lip-tip vitiligo – This kind of vitiligo affects the lips, nipples, and penis in addition to the upper and lower limbs**.**

Mucosal vitiligo-depigmented patches on the mucous membrane of the oral cavity & genitalia are features of this variant.

Generalized vitiligo -- The most typical kind of vitiligo is generalized vitiligo. Depigmented macules and patches are the hallmarks of vitiligo vulgaris. The distribution of this variant is symmetrical.

Universal vitiligo -- A kind of vitiligo known as universal vitiligo involves whole or almost complete body depigmentation.

A surgical procedure is considered as the first option of management if in case vitiligo is stable.[57] The vitiligo is said to be active in state if there is :

- appearance of new vitiligo lesions,
- progression of old vitiligo lesions, and
- presence of Koebnerisation.

Koebner's phenomenon:

"The Koebner phenomenon (KP), first described in 1876 by Heinrich Koebner, is the appearance of new skin lesions on previously unaffected skin secondary to trauma. This phenomenon is also termed the isomorphic (from Greek, "equal shape") response, given the fact that the new lesions that appear are clinically and histologically identical to the patient's underlying cutaneous disease".[58]

When lesions in vitiligo patients stay stationary and undergo remission, and the Koebner phenomenon is nonexistent, this is referred to as stable vitiligo.[58]

VITILIGO VARIANTS:

- Trichrome vitiligo**:** In between the depigmented region and the periphery of normal skin, there is a zone of tan hue color. this pattern is referred as trichrome vitiligo which is an unstable type of vitiligo.
- Quadri-chrome vitiligo: Perifollicular pigmentation is seen with trichrome vitiligo in a lesion that is repigmenting or healing.

- Penta-chrome vitiligo: "The inner white depigmented zone which extends to the margin of normal healthy skin comprises of 5 zoned pigmented patterns."[59]
- Inflammatory vitiligo - The vitiligo patch shows erythematous raised borders. Patient may have itching and burning sensation. This type appears after vigorous treatment.
- Blue vitiligo - It appears as a bluish grey area at the point of post-inflammatory hypermelanosis.[60] In Histopathological examination, dermis displays melanophages whereas the basal layer lacks melanocytes.[61]

Stability of the disease:

Clinical, serological, biochemical, and ultrastructure are the methods are used to evaluate stability or disease activity. The clinical classification of disease activity was based on the definition for stability issued by the Standard guidelines of care for Vitiligo surgery by Prasad Davinder and Somesh Guptha Indian Association of Dermatology, Venereology and Leprology as "**a patient reporting no new lesions, no progression of existing lesions, and absence of Koebner phenomenon during the past one year**" .[4]

According to Falabella,[56]

- a lesion with lack of progression or no new lesions for 2 years,
- absence of recent Koebner phenomenon
- positive mini grafting test
- absence of Koebner phenomenon at the donor site, and
- evidence of repigmentation of existing patches either spontaneously or with medical therapy is considered stable.

It may be difficult to accurately determine stability based on a patient's past, and it is not practical to do minigraft in every case. [4] Furthermore, the same patient may have both stable and unstable lesions in line with the concept of lesional stability, making it nearly difficult to biopsy every lesion.

Clinical parameters of unstability or active lesion is defined by presence of following parameters

- Progression of old lesion
- Development of new lesion
- Koebner phenomenon
- presence of trichrome pattern features , inflammatory like lesions
- Mini grafting-- positive
- VIDA score

Biochemical parameters:

- Morrone et al**.** claims that higher quantities of homovanillin acid and vanillyl mandelic acid can be found in urine in those with active vitiligo.[42]
- Cucchi et. al. suggests that norepinephrine, normetanephrine, homovanillin acid, and 3-methoxy-4-hydroxyphenylglycol levels rise in the presence of active vitiligo.[63]
- Ines et.al. studies declare that stable patches of vitiligo had shown reduced levels of glutathione peroxidase and elevated malondialdehyde, super oxide dismutase.[64] There are further factors. Plasma neuropeptide Y levels[65] are greater, homocysteine levels[66] are higher, GM-CSF levels[67] are lower, and IL-6 levels are lower in progressive vitiligo.[68]

Serological parameters:

- Serological findings such as Anti-tyrosine hydroxylase antibodies are absent in stable condition of lesion but are present in 27% of active state of the diseases.[69]
- Hann et.al said through his studies that on treatment following systemic immune suppressive agents like Steroids , melanocyte destruction has been decreased. The reduction in melanocyte cytotoxicity is an indication of therapy success.[70]

Ultrastructural parameter:

Unstable vitiligo on histopathology exhibits dermal lymphocytes and basal cell vacuolization& melanophages in perilesional skin.[71]

- According to Al badri AM, e.t. al.,[72] ICAM-1 is six times more abundant in the perilesional skin in active vitiligo.
- As per Kumar et. al. Perilesional melanocytes in individuals with active illness have demonstrated on HPE retracting dendrites.[73]
- Benzenkri et. by his studies observed that a lesion with poorly defined margins and hypomelanosis.[74]

As a necessity, a noninvasive approach is required to evaluate all lesions. Dermatoscopy allows for the evaluation of all lesions as well as the assessment of subtle characteristics that may not be visible to the naked eye. Vitiligo dermatoscopy is underappreciated, and there are very few studies supporting it.

DIFFERENTIAL DIAGNOSIS

Various nevoid (figure 2) and hereditary disorders with depigmentation may be confused with vitiligo. Differentiating these disorders from vitiligo is important as

these hereditary disorders may have multisystem associations. Additionally, the treatment options are standardized in vitiligo.

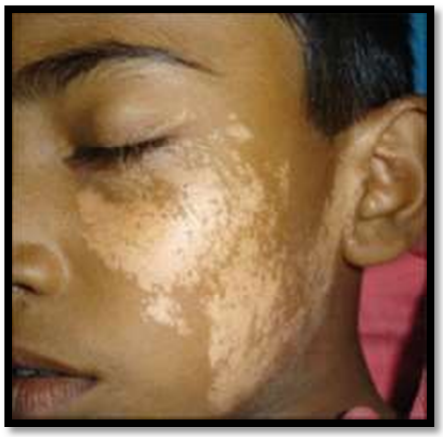


Figure 2 : Nevus depigmentosus [75]

DIFFERENTIAL DIAGNOSIS OF VITILIGO [76]

Congenital

1. Naevoid pigmentary disorders
   - Nevus depigmentosus : serrated margins of the lesion
   - Nevus anaemicus: pale lesion, which on diascopy becomes unnoticeable
2. Ash-leaf spots (tuberous sclerosis): Lanceolate shape more commonly seen over the trunk
3. Piebaldism: Patterned depigmented patches, white forelock seen in 85% of cases
4. Waardenburg syndrome: Since birth depigmented patches seen, with a white forelock. Most common associated features are heterochromia of irides, dystopia canthorum, and congenital sensorineural deafness
5. Linear lesions

- Hypomelanosis of Ito: Hypopigmented, linear streaks and whorls are seen along Blaschko's lines.
- Incontinentia pigmenti: (fourth stage) history of previous lesions will be present, Linear, atrophic, hypopigmented streaks are seen with loss of hair and sweat pores over it.

6. Oculo-cutaneous albinism depigmentation mostly observed in skin, hair, and eyes with leukotrichia seen

- Acquired

1.Inflammatory

- Pityriasis alba: Hypopigmented macules/patches with fine scaling, mostly seen over face.
- Lichen striatus: linear hypopigmented shiny papules.
- Post-inflammatory hypopigmentation history pertaining to the past lesion will be present.[76]

2.Infection

- Pityriasis versicolor : hypopigmented macules with furfaraceous scales, most commonly seen in seborrheic distribution.
- Leprosy: hypopigmented patches with loss of sensation and peripheral nerve involvement seen.
- Post-Kala azar dermal leishmaniasis symmetric hypopigmented macules which are widespread. associated nodules may be seen.
- Pinta: mostly seen in endemic areas and family members are also affected Widespread slate-blue hyperpigmentation initially which is replaced by depigmented macules are seen.[76]

3.Miscellaneous

- Polymorphous light eruption: Pruritic, scaly, hypopigmented, papule/macule/patch seen in photo exposed areas with a history of photo exacerbation seen.
- Contact depigmentation: only over the contact areas patterned depigmentation seen (footwear, diaper).
- Lichen sclerosus et atrophicus (LSEA): Porcelain-white atrophic macules. In case of genital lesions, atrophy, and resorption of genital structure are seen in girls and phimosis is seen in boys.
- Topical steroid abuse history of pre-existing dermatoses and chronic abuse of steroid.[76]

Woods lamp

Robert W. Wood developed the Wood's lamp in 1903 which is named after him. It works by emitting long-wave ultraviolet radiation, also known as black light, which is produced by mercury arc under high pressure fitted with nickel oxide at 9% and barium silicate filter, otherwise known as the "Wood's filter." It filters out all light rays except light belonging to wavelength between 320-400 nm.[77] When Wood's (UV) light is absorbed, tissues fluoresce and it emits radiation with a larger wavelength, notably visible light. Typically, Wood's lamp produces little light. (< 1 mw /cm2). Normal skin has very little to no fluorescence and is mostly as a result of elastin components, aromatic amino acids, and melanin precursors or products.[77]

EXAMINATION TECHNIQUE FOR WOOD'S LAMP - A Wood's light may be operated without much expertise. However, to prevent incorrect interpretation of results, various practical considerations should be borne in mind.[78-80]

- The light should preferably be let approximately a minute to warm up..
- The examining space should be completely dark, ideally without any windows or with black occlusive blinds.
- For the examiner to clearly see the contrast, they should become dark-adapted.
- The distance from the light source to the lesion should be 4 to 5 inches.
- Avoid cleaning the area before exposing it to a black light evaluation since due to the pigment's dilution, it could provide falsely negative findings. Lint, soap residues, and topical medications can also produce misleading negative findings.
- Less or no epidermal melanin can be seen in lesions that are depigmented or hypopigmented. Subsequently, a window exists through which dermal collagen's light-induced autofluorescence may be seen.

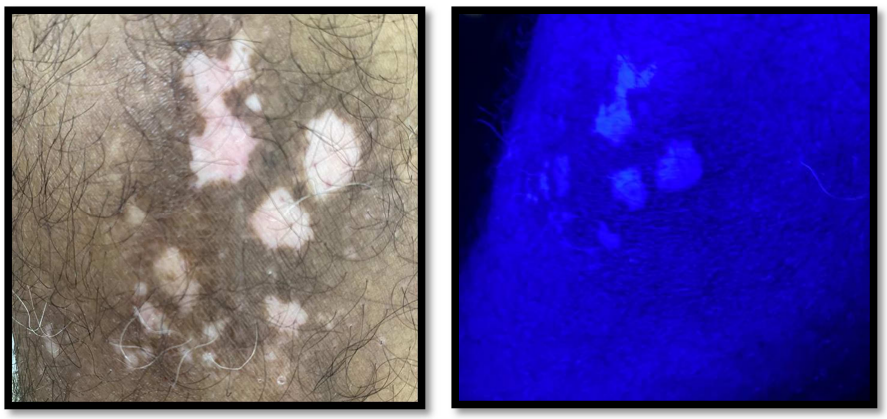


Figure 3 : clinical lesions on woods lamp examinatinon

The use of a basic bed side tool, woods lamp light examination can rule out other hypopigmentary conditions. The borders of hypopigmented or depigmented patches look sharper under Wood's light owing to the abrupt cut-off in the visible emission from vitiligo skin. Because of autofluorescence, the lesions show vivid blue-white.[80]

HISTORY OF DERMATOSCOPY

The practice of using skin surface microscopy was prevalent even during the mid- 17th century when Peter Borelus and Johan used examination of nail-fold capillaries to evaluate dermatological disorders .[81] It was during the beginning of the 20thcentury that Otfried Müller first assembled portable monocular and binocular microscopes so that they could be easily used in daily practice[82]. The term "dermoscopy" was coined by Goldman in 1920, and it was officially introduced in 1921 by Joe Saphier[83,84] .

DERMOSCOPY

Dermoscopy is also called dermatoscopy and incident light microscopy[83-85] . Dermoscopy is a non-invasive test that helps in visualising cutaneous and subcutaneous structures, which in turn aids in establishing an accurate diagnosis[86]. This test is simple and quick; therefore, it can help in diagnosing faster. It was initially used for evaluation of changes in suspicious nevi, in differentiating inflammatory skin conditions; however, over the course of time, its use has expanded to diagnose infectious and pigmented skin conditions[83-85]. When dermoscopy is used in diagnosing conditions associated with inflammation, the term "inflammascopy" is used[83].

It was in 1878 that the first use of immersion oil in light microscope was described by Abbe [84]. It was in 1893 that a similar principle was used to evaluate conditions related to the cutaneous surface under the microscope by a German physician Unna[87]. He coined the term "Diaskopie" which refers to the use of the technique of immersion oil and a glass spatula in order to interpret lesions associated with lichen planus and infiltrations associated with lupus erythematosus[87]. The first ever series on dermoscopic data in the evaluation of skin disorders related to pigmentation was initially published by Goldman in 1950 in USA[87].

It was in 1971 that Rona Mac Kie highlighted that there was a significant improvement in the preoperative evaluation of patients with disorders of skin pigmentation using surface microscopy; this technique could also be advantageous in differentiating benign conditions from malignant ones[83,84,88]. The first ever consensus conference on the cutaneous surface under microscopy was held in 1989 in Hamburg, and the next consensus meeting on dermoscopy was held in 2001 in Rome, which was the first international conference of this nature[84].

In the present era, the field of dermoscopy has progressed so much that it is no longer a setback tool; rather, it is a routinely used method in the evaluation of dermatological conditions and is gaining global acceptance quickly[87].

DERMOSCOPIC CRITERIA USED TO INTERPRET SKIN LESIONS

Dermoscopy is a very useful tool as it helps in identifying several structures and colours that cannot be visualised by the naked eye. The components that are visualised to characterise the dermoscopic findings include the colour, network of pigments, and vascular pattern.

TYPES OF DERMOSCOPY

Based on the type of light used, there are two types of dermoscopy: non-polarised and polarised dermoscopy. Non-polarised dermoscopy entails coming into contact with the skin's surface and a fluid interface between the skin's surface and the glass. In contrast, polarised light does not require contact fluids and penetrates deeper than the non-polarised light [89]. The visuals obtained from a dermoscope can be digitally photographed or recorded for future reference.

	Non-Polarised	**Polarized**
Factor	Classic contact dermoscopy	Contact and noncontact dermoscopy
technique	Requires a liquid interface and direct contact between the scope and the skin	Although it can be used in contact or noncontact mode, and can be used with or without a liquid interface, direct contact and liquid interface provide superior image clarity
Skin layers	Superficial layers are better visualized	Deep layers of epidermis and papillary dermis (depth of polarized. light approximately 60 to 100 um) are better visualized
Colour and structures	• Blue white veil due to orthokeratosis is more conspicuous • Milla-like cysts and comedo -like openings are more conspicuous • Steel blue colour in blue nevi appears more homogenous • Regression structures (peppering, blue white areas, and gray colour) are more conspicuous • Ability to visualize vascular structures depends on the amount of pressure applied to the skin • White shiny structures cannot be visualized adequately	• Pink and red colours are more conspicuous • Milia-like cysts and comedo-like openings are less conspicuous • Blue colour in blue nevi will appear darker, with differing hues • White scar-like areas are more conspicuous • Vascular structures and collagen are more conspicuous. • White shiny streaks, also known as crystalline structures, are more conspicuous

Table 6: Polarised and non-polarised type of dermoscopy[85]

COLOUR

The most important criterion that cannot be visualised by the naked eye but can be clearly identified on dermoscopy is the colour of the skin, which is a critical observation on dermoscopy. The most common skin colours are white, black, red, blue, light brown, blue green, dark brown, and yellow[88-91] (Figure 3). The colour observed on dermoscopy based upon a variety of variables. The two primary chromophores found are melanin, which is visible as black, brown, and bluish, and or greyish based on how deeply it penetrates the skin, and haemoglobin, which appears reddish, blue, purple colour, depending on the level of depth, degree of oxidation, and the presence of thrombosis[91]. The most important chromophore responsible for the colour in neoplasms associated with melanocytes is the pigment melanin and the colour of this pigment is dependent on its location within the skin[88-91]. When melanin is perched in the superficial epidermis and horny layer the colour appears black. When melanin is located within the epidermis the colour of the skin is light-to-dark brown. When melanin is located within the papillary dermis the colour of the skin is grey-to-grey blue. When melanin is located within the reticular dermis the colour of the skin appears steel blue. Therefore, when the skin appears blue, melanin is most often localized in the deeper skin layers as a result of shorter wavelength components of visible light —the blue -violet side of the spectrum of visible light—is more dispersed compared with the part of the spectrum of visible light with longer wavelengths[92]. Commonly, the colour red is linked to an increase in blood vessels. or as a consequence of dilatation of the blood vessels, trauma, or neovascularization[92].The dermoscopic structures as proposed by the consensus meeting[36].

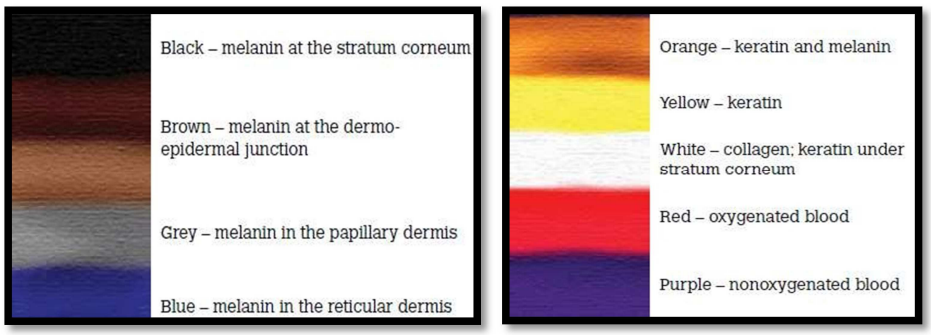


Figure 4: Colours observed in dermoscopy, which determines the colour and type of lesion[94].

PIGMENT NETWORK

One of the most important parameters evaluated on dermoscopy is the network of pigments. A grid of tiny brown lines that represents the pigment network that are scattered over a backdrop that has a soft brown hue with a structure similar to that of a honeycomb[94]. When we evaluate the anatomy of the pigment network, it is believed that this type of network is a result of the arrangement of the melanin pigment within the melanocytes or the arrangement of melanocytes along the DEJ. Within the epidermis, reticulation is represented in the form of a pattern of rete ridges[22,90,91,95]. There are relatively hypomelanotic holes that exist within the reticulation, and correspond to the tips of the dermal papilla. The cutaneous suprapapillary plates also contribute to the hypomelanotic holes in the reticulation[96] (Figure 4).

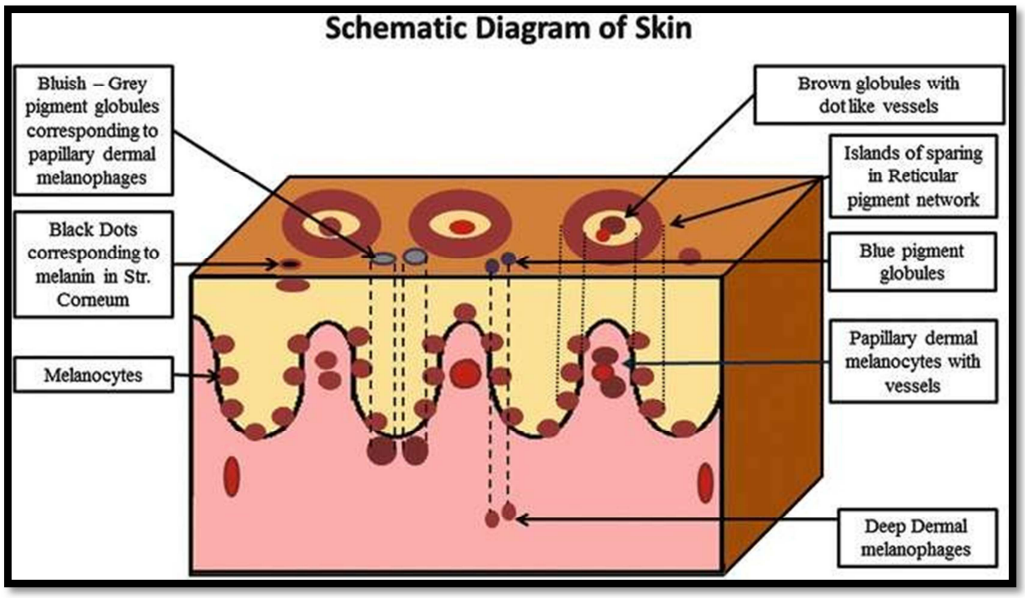


Figure 4 : Level of the pigment and the appearance in dermoscopy [42].

The pigment network based on its pattern can be classified into i.typical pigment network and ii.atypical pigment network. Typical pigment network refers to a relatively uniformly arranged homogeneous colour with a regular meshed network and this network is most often toward the periphery. In an atypical network, in contrast to the typical network, there is a non-uniform staining with darker or broader lines and heterogeneous colour and shape. These broadened hyperpigmented lines abruptly stop as they reach the periphery[96,97]. Another point to be noted about the reticular network is that in cases of small or less pigmented areas, it may be very difficult to visualise the pigment network[93]. If the network does not have any signs of regression and there is no structural network, it is referred to as "structureless area".[98]

DOTS

The term "dots" refers to rounded small structures with a diameter smaller than 0.1 mm. The colour of these dots may vary from black-brown to grey-or-blue grey. The colour of the dots depends on the location of pigment accumulation as described earlier. The grey-blue granules are also known as "peppering" and are a result of tiny

melanin pigments in structures that lie within the papillary dermis[85]. These grey-blue or blue granules are often a result of loose lying pigment melanin, fine particles of melanin, merely the dust of melanin within the melanophages, or free lying melanin situated in the deep papillary or reticular dermis.[85,94,99,100]

GLOBULES

The term "globules" refers to symmetrical round-to-oval structures that can vary in colour from red-black or brown (Figure 5). Most often these are larger than dots and are wider in diameter more than 0.1 mm. Globules corresponds to colonies of melanocytes, which may be either benign or malignant. They can also be a result of melanin clumps or melanophages that are located in the deep parts of the epidermis, DEJ, or capillary dermis[85,99,101].

Dots and globules both can occur in malignant melanocytic proliferation and benign melanocytic proliferation, but the difference is that, in benign lesions they are often standard in size and shape with an even dispensing of colour commonly toward the center of the legion. In malignancy, however, they tend to have a variable size and shape and are most often located at the periphery of lesional skin[102].

BRANCHED STREAK

Branched streaks refer to altered pigment networks and implies that the pigment network is either broken or disrupted. Pathologically, branch streaks imply remnants of either the pigment or rete ridge with bridging colonies of melanocytes that lie within the papillary dermis[90].

RADIAL STREAMING

These are perceived as radial parallel linear expansions that are organized asymmetrically that lie at the periphery of the lesion (Figure 5)[90,94].

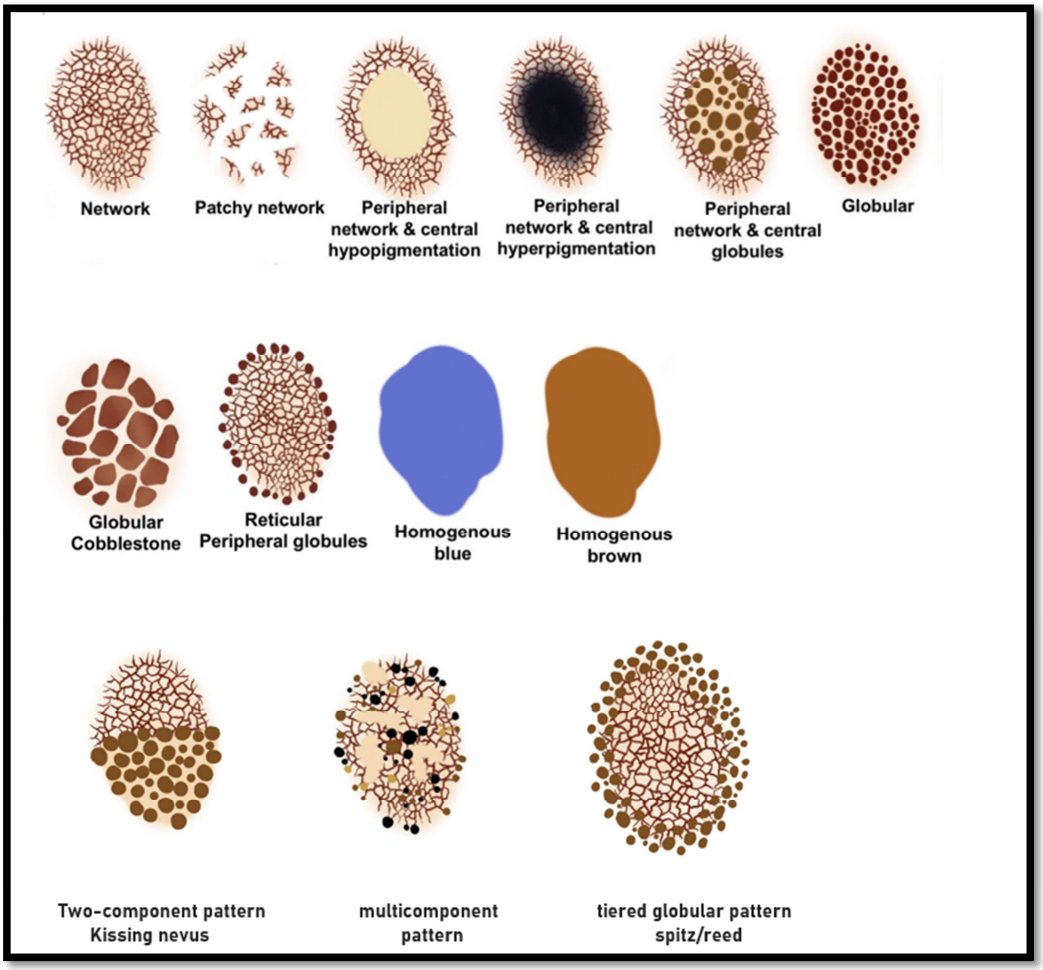


Figure 5: Dermoscopic findings of pigment[103]

PATTERNS OF LINES

Overall, five patterns are recognised (Figure 6)[100]:

- Reticular (figure 6F)
- Branched (figure 6G)
- Parallel (figure 6H)
- Radial (figure 6I)
- Curved (figure 6J)

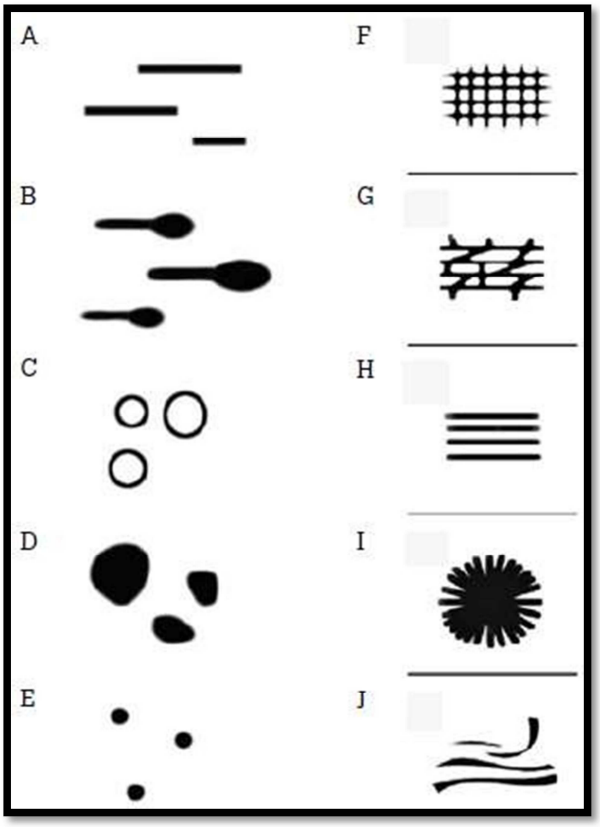


Figure 6 : Patterns of lines on dermoscopy[100].

SKIN CHANGES IN VITILIGO.

The usual reticulate pattern of healthy skin matches to pigmentation. along rete ridges having patches of pallor that correlate to the papillary dermis. In vitiligo lesions, the reticulate pigmentary pattern varies. Therefore, a dermoscopic study is useful in the early vitiligo identification. The normal reticulate pattern is reversed in early vitiligo lesions [48]. Dermoscopy, an *in vivo* non-invasive technique used to observe pigmented and hypomelanotic skin lesions.[54]

DERMOSCOPIC PATTERNS

Various patterns on dermoscopy are [29]:

- Marginal hyperpigmentation
- Perifollicular pigmentation
- Reticulate pigmentation
- Perifollicular depigmentation
- Altered pigment network
- Leukotrichia
- Intra/perilesional erythema with telangiectasia
- Trichrome
- Starburst appearance
- Comet tail appearance
- Polka dot appearance
- Diffuse white glow is the characteristic dermoscopic feature of vitiligo in which the lesion has an appearance similar to the glow of a full moon. This is because of complete reflection of light from the upper dermal collagen fibers, which is normally absorbed by the melanin in the basal layer[104].

Dermoscopic patterns may overlap in multiple lesions on the same subject.

DERMOSCOPIC INDICATORS OF DISEASE ACTIVITY IN VITILIGO

A few dermoscopic features are associated with disease activity and are linked to progressive vitiligo. These include ill-defined borders with decreased pigment network (trichrome lesion), micro-Koebnerisation, perilesional depigmented globules (Polka dot, tapioca sago), and starburst also described as feathery pattern. Micro-

Koebner appearance {also known as comet tail appearance} is one of the well-researched dermoscopic characteristics of vitiligo and presents as linearly arranged streaks of depigmentation. Typically, a naked eye examination does not reveal anything about them. The other marker of active vitiligo is the emergence of several white globules surrounding a central depigmented macule. It has been described metaphorically as having a polka dot/tapioca sago pattern. The starburst Pattern is a metaphorical description for depigmented lines radiating outward from center of primary lesion[99].Dermoscopic patterns of marginal hyperpigmentation and perifollicular pigmentation are most commonly observed in stable vitiligo and patients on treatment. Trichrome is most commonly observed in patients with unstable vitiligo.[85]

DERMOSCOPIC FINDINGS

1. **Dermoscopic pattern in subjects with stable vitiligo**:

i. marginal hyperpigmentation : perilesional hyperpigmentation seen in a vitiligo patch

ii. perifollicular hyperpigmentation: Another common dermoscopic pattern is perifollicular pigmentation. It is important to recognise that perifollicular depigmentation or perifollicular re-pigmentation takes into consideration the background skin colour. A loss of perifollicular pigment with a retained interfollicular pigment network is referred to as perifollicular depigmentation.; it is considered as a marker of disease activity [8]. Re-occurrence of a pigmentary network around follicle that had previously been entirely erased is known as perifollicular re-pigmentation.; it occurs around the hair follicle in a lesion and is a feature of re-pigmenting vitiligo. Perifollicular re-pigmentation, which can happen naturally or as a result of therapy,

resembles residual pigmentation. On examination both cannot be distinguished on dermoscopy, and only longitudinal examination may be used to evaluate them[105]. As an outcome, dermoscopy can be used to track the progression of the disease and its response to therapy.

iii. Repigmentation in vitiligo When exposed to sunlight, spontaneous repigmentation may take place. After treatment, repigmentation might either happen in follicular, marginal areas, or widespread pattern. It can also happen in mixed pattern. Possible causes of repigmentation include melanocyte migration followed by melanocyte proliferative growth. Multiple immunopathological study shows mature melanocytes in the upper epidermis.

1. Additional dermoscopic symptoms in individuals receiving therapy[29] :

- atrophy
- erythema
- telangiectasia

2. Dermoscopic findings in unstable vitiligo

- Trichrome: An area with normal pigmentation encircles a rim of hypopigmentation which in turn is around an depigmented patch[50].
- Comet tail: unstable lesion showing depigmented patch with Koebnerisation in a comet tail pattern[50].
- Starburst: multiple white lines radiating from primary lesion.
- Polka dot: these are described as spilled depigmented macules in a polka dot appearance around a central depigmented patch (unstable lesion). Few macules show pigmentation around perifollicular lesions as well[29] .

- Distorted Pigment Network -Due to the lack of melanocytes in the basal layer, the typical reticular pigmentary network seen on smooth non-facial skin is not present in vitiligo. The boundaries of evolving vitiligo demonstrate areas of broken pigmentary network with ill-defined margins.

3. Other dermoscopic findings in vitiligo

a. Amoeboid pattern: is described as relatively well-defined edges of the depigmented patch extending in pseudopod-like fashion [50].

b. Leucotrichia: a fairly well-defined amoeboid appearance of vitiligo patch demonstrating leucotrichia—white hairs[50].

c. Reduced pigment network [34]

d. Absent pigment network [34]

e. Reversed pigment network: A white or depigmented net-like pattern with normal skin colouMr in between. This finding is very well seen on dermoscopy in melanocytic nevus and also in melanoma where it is described as reticular light areas with dark holes in between in a net like pattern[106]. A similar picture is seen in vitiligo. Based the findings reported by Thatte et al.[91], it is safe to say that in dubious situations, dermoscopy may prevent the requirement for a skin biopsy.

EVOLVING VITILIGO

Early vitiligo also called as previtiligo. Clinically, it might be challenging to identify newly developing vitiligo lesions from other hypopigmentation-causing conditions. Dermoscopy is very handy in such cases as it leads to early diagnosis of vitiligo based on the aforementioned patterns. Thatte et al.[34] observed many changes in pigment network of vitiligo patients such as reverse pigment network, perifollicular

hyperpigmentation & perilesional hyperpigmentation in new lesions of vitiligo. In this study, cases of stable and re- pigmenting vitiligo were excluded.

Investigations:

Although the diagnosis of vitiligo is dependent on clinical evidence, a biopsy can assist distinguish vitiligo from hypopigmentary diseases.

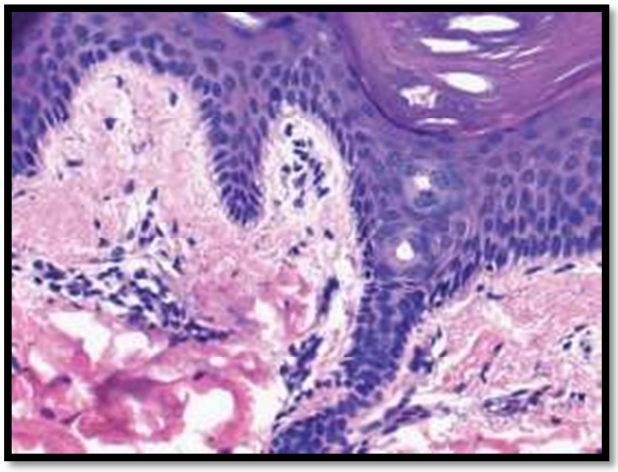


Figure 29: Histopathology in evolving lesions of vitiligo shows reduced melanocytes with sparse perivascular lymphocytic infiltrate.[34]

- Epidermis : shows absence of melanocytes & melanin in the s.basale.
- Lymphocyte and histiocyte infiltration on histopathology with an clinically erythematous border can be seen in inflammatory vitiligo.
- Immune-histochemical studies of lesional skin had shown melanocytes only occasionally.[107]

Thyroid disorders, pernicious anaemia, diabetes mellitus, Addison disease, and alopecia areata may all be linked to vitiligo.

So, laboratory work includes:

- Thyroid panel T3,T4,TSH
- Antinuclear antibody
- Anti-thyroid peroxidase antibody
- CBC count
- Differential count

Treatment Options: Way to approach to treat the condition:

- The disease in the first place is to be stabilized.
- Once the condition is stabilized, treatment for repigmentation is planned.

Various treatment modalities for vitiligo include the following: [4]

1. Medical
    a. Topical
        - Corticosteroids
        - Tacrolimus/pimecrolimus
        - Calcipotriol
        - Combination
    b. Systemic
        - Corticosteroids (oral methylprednisolone with betamethasone/ methyl prednisolone)
        - Steroid sparing drugs for follow maintenance
2. Phototherapy
    - Topical PUVA
    - NB- UVB
    - Systemic PUVA (> 12 years)

- Phenylalanine + PUVA
- Excimer laser (308nm)- targeted NB-UVB phototherapy

3. Surgical therapy options include
    - Conventional methods
        i. Mini punch graft
        ii. Epidermal suction blister
        iii. Thin Thiersch graft
    - Newer cellular transplantation techniques
        i. Epidermal cell suspension
        ii. Cultured melanocyte suspension
        iii. Cultured epidermis
4. Cosmetic camouflage to cover blemishes.
5. Total depigmentation using MBEH—mono benzyl ether of hydroquinone.

MATERIALS AND METHODS

- **Study source:** This study was conducted in the Department of Dermatology, Venereology and Leprosy, in tertiary care hospital, Belgaum as a part of the MD academic curriculum.
- **Study duration:** The study was conducted between 1st January 2021 to 31st December 2021
- **Ethical clearance:** Clearance was taken from the Ethical Committee of the institute.
- **Study design:** Hospital based cross sectional study.
- **Sample size:** Formula used for sample size calculation is

$$n = \frac{\widehat{S_p}(100 - \widehat{S_p})Z^2}{d^2 p}$$

n is the sample size required, $\mathbf{S_p}$ is the specificity observed, **p** is the percentage occurrence of a state or condition (proportion or prevalence), **d** is the precision of estimate, **Z** is the value corresponding to level of confidence required.

We assume that 15% of the subjects will have stable lesions and observed specificity of 96.8% in predicting stable vitiligo by network within the vitiligo [1]. With precision of 10% at 95% confidence level sample size is given by,

$$n = \frac{96.8 \times (100 - 96.8) \times (1.96)^2}{10^2 \times 15}$$

$$n = 79.33 \approx 79$$

Hence minimum sample size required is 79. Larger the sample size, better the precision.

- **Sample selection criteria:** Random sampling method
- **Inclusion criteria:**
  - All patients regardless of age and sex, with clinically diagnosed vitiligo screened under woods lamp and attending dermatology department were included in study.
- **Exclusion criteria:**
  - Non consenting patients.
  - Mucosal/ lip tip vitiligo.
  - Syndromes associated with vitiligo.
- **Data collection:**
  - Informed consent was taken from all the study patients.
  - All the study subjects were made to go through a detailed history taking, general physical, systemic and dermatological examination.
  - Data was collected by a single examiner and recorded in case record proforma.
  - All participants underwent clinical, Wood's lamp and dermoscopic examination (ILLUCO IDS -1100 model, under 10X magnification) with findings being noted along with the recording of both clinical and dermoscopic images.
  - Records were maintained and analysed statistically.

- **Details of criteria used for clinical examination findings**
  - Clinical assessment of disease activity according to standard guidelines of care for vitiligo surgery by Devinder Prasad and Somesh Gupta as

a. History of progression: Absence of new lesion

b. Extension of old lesions: No extension of old lesions

c. Koebner phenomenon: Absence of Koebner phenomenon either based on history or by checking for experimentally induced vitiligo.

- **Details of criteria used for to describe stability of disease**

DERMOSCOPY PARAMETERS	**SCORE**
1. Border sharp(sharp/ill-defined/trichrome)	+1
2. Pigment network (absent / reticulate/reduced/reverse)	+1
3. Perilesional hyperpigmentation	+1
4. Perifollicular hyperpigmentation	+1
5. Satellite lesions/tapioca sago/polka dot	-1.5
6. Micro Koebner phenomenon/comet tail	-2

According to **BPleFoSK** criteria, predictors of stability are sharp border, absent / reticulate pigment network, perilesional, perifollicular hyperpigmentation are markers of stability. A cut of score of ≥1.5 is marked as stable vitiligo.

- **Statistical Method for Data Analysis**

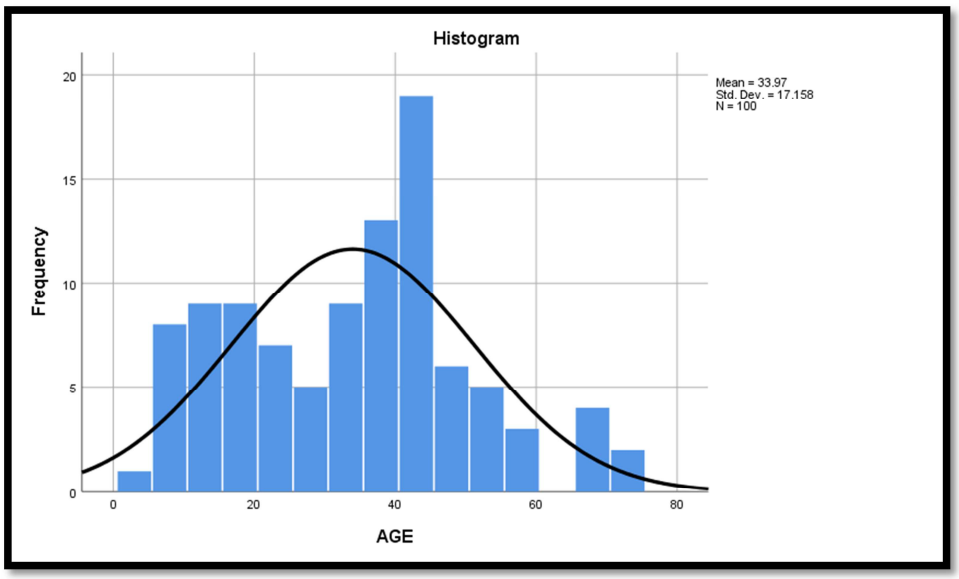
➢ Data was collected and stored in Microsoft Excel.

➢ Data was analyzed using statistical software R and Microsoft Excel.

- Continuous variables were given in mean± SD/median (range).
- Categorical variables were represented by frequency.
- To check the dependency between attributes Chi-square test was used.
- P-value less than or equal to 0.05 shows statistical significance.

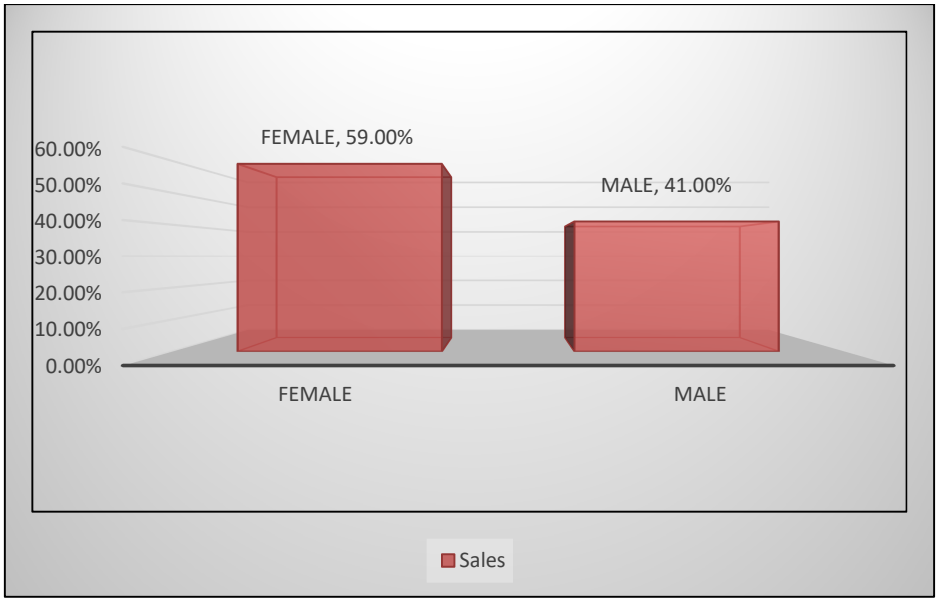
RESULTS

1. **AGE GROUP** - In our study, a total of 100 subjects were enrolled. Out of them, 41 were males and 59 were females with a female-to-male ratio of 2:3. The minimum age observed in the sample was 3 years and the maximum observed was 73 years. The average age of presentation with standard deviation was 33.97±17.158 years.



Graph 1. Distribution of subjects by age

2. **Gender distribution:** There was a female predominance seen i.e., 59% (n=59) of the study population were females and the remaining 41% (n=41) were males.

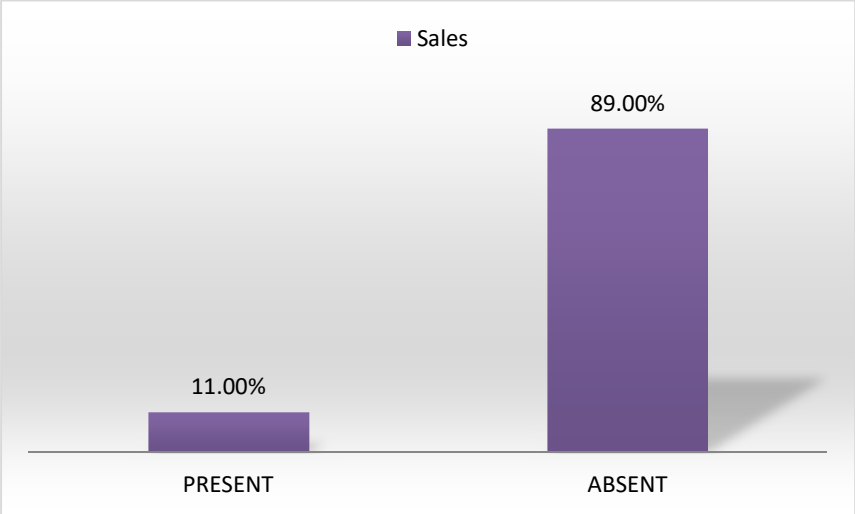


Graph 2: Distribution of the sexes in the study population

	Frequency	***Percent***
FEMALE	59	59.0
MALE	41	41.0
Total	100	100.0

Table 1: Distribution of the sexes in the study population

3. **FAMILY HISTORY OF VITILIGO --** At this time, the general view is that family incidence ranges from 20% to 30%. the reported findings on this score in India and abroad vary between 7.5% and 41% respectively by Dutta AK. Vitiligo: Neural and immunologic linkages[13], which is in support of this study with a family history of 11%.

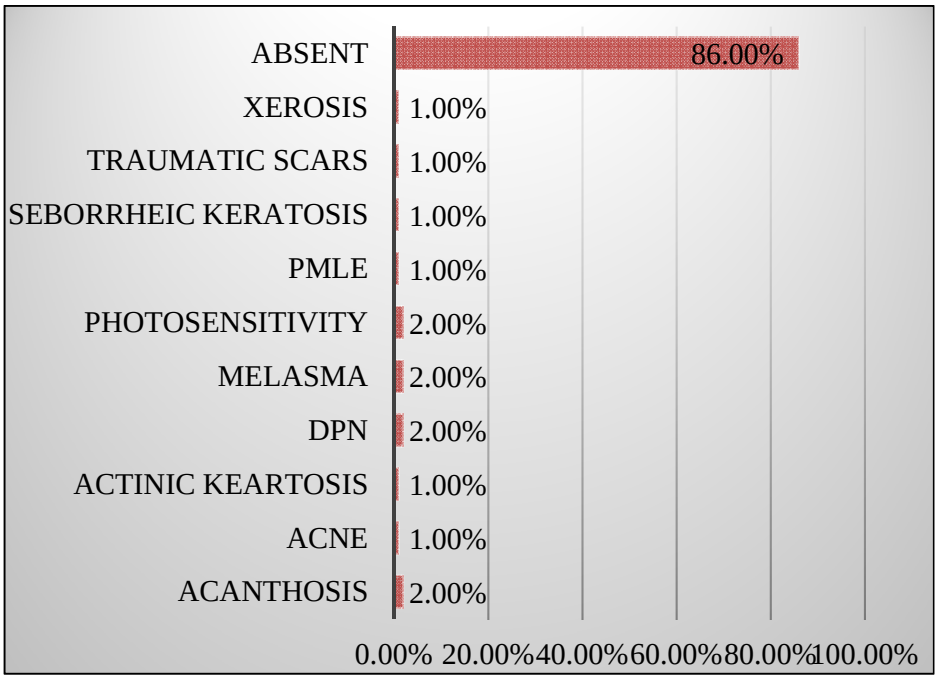


Graph 3: Incidence of family history in the study population

	Frequency	***Percent***
PRESENT	11	11.0
ABSENT	89	89.0
Total	100	100.0

Table 2: Distribution of the subjects by presence of family history

4. **ANY SKIN CONDITIONS** - associated skin disorders are less in this study. Nearly 86% of patients have no associated skin conditions. This was followed by 2% association with acanthosis nigricans, 2% association melasma, 2% of patient's photosensitivity is present. Of the total study population 1% subjects had xerosis, 1% subjects had shown acne, 1% subjects had shown actinic keratosis, 1% subjects had shown Dermatosis papulosa nigra, 1% subjects had shown PMLE, seborrheic keratosis and xerosis.



Graph 4: Distribution of the subjects by presence of associated skin conditions.

	Frequency	Percent
ACANTHOSIS	2	2.0
ACNE	1	1.0
ACTINIC KEARTOSIS	1	1.0
DPN	2	2.0
MELASMA	2	2.0
PHOTOSENSITIVITY	2	2.0
PMLE	1	1.0
SEBORRHEIC KERATOSIS	1	1.0
TRAUMATIC SCARS	1	1.0
XEROSIS	1	1.0
ABSENT	86	86.0
Total	100	100.0

Table 3: Distribution of the subjects by the presence of associated skin conditions.

5. **SYSTEMIC ILLNESS** -. In our study 81% of cases did not have any associated systemic illness followed by hypertension was noted in 4% of our cases. 2% of the study population had DM, 1% of the study population had coronary artery disease, and 1% of the study population had a history of covid & 1% of the study population had a hearing abnormality,1% of the study population had renal stones, and 1% of the study population had spondylitis.

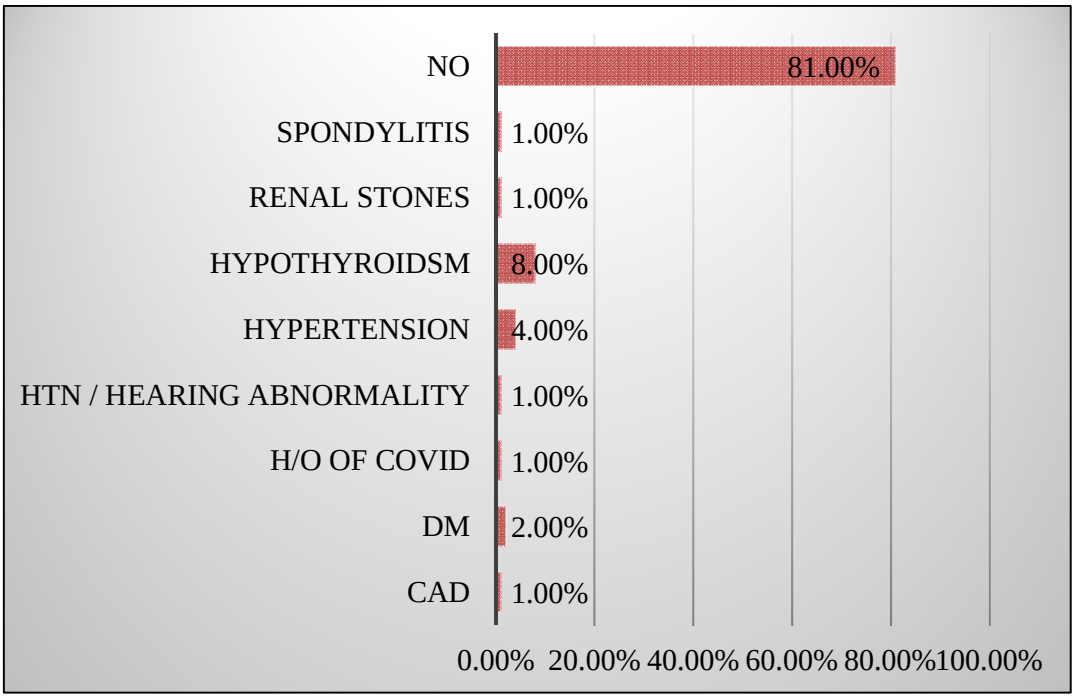


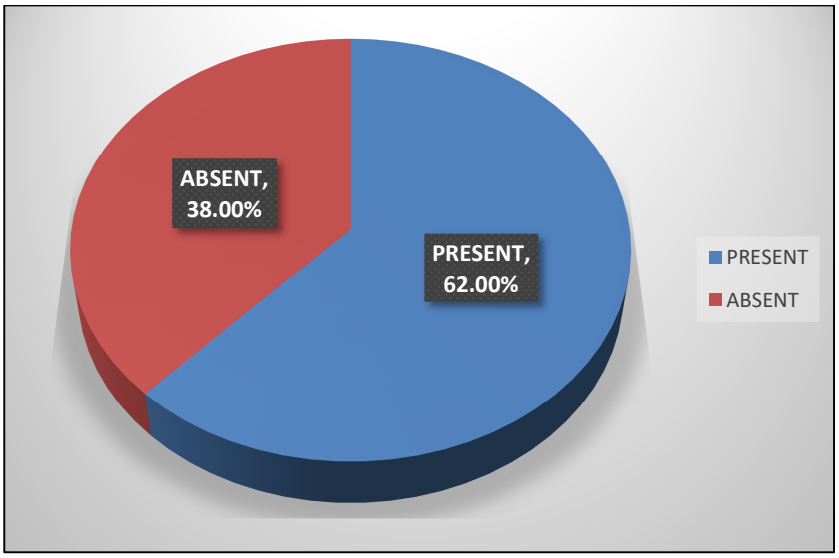
Chart 5: Distribution of the subjects by presence of systemic illness.

	Frequency	Percent
CAD	1	1.0
DM	2	2.0
H/O OF COVID	1	1.0
HTN / HEARING ABNORMALITY	1	1.0
HYPERTENSION	4	4.0
HYPOTHYROIDISM	8	8.0
RENAL STONES	1	1.0
SPONDYLITIS	1	1.0
NO	81	81.0
Total	100	100.0

Table 4: Distribution of the subjects by the presence of associated systemic illness.

6. CLINICAL ASSESSMENT IN THE PAST 1 YEAR

a. NEW LESIONS Majority of the study subjects i.e., 62 % (n=219) had new lesions in the past year. 38% of study subjects had no new lesions (n=133) in the past one year.

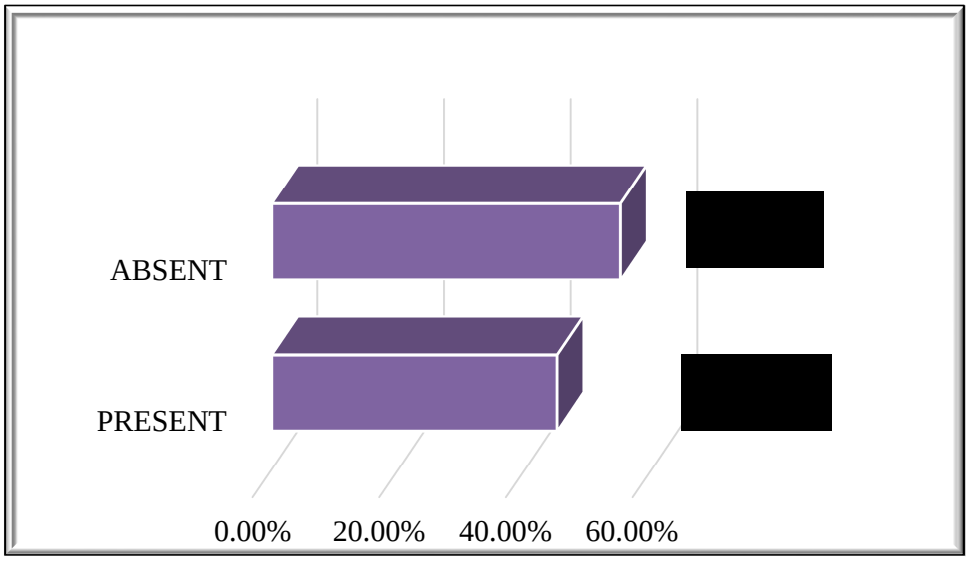


Graph 6: Distribution of the subjects by new lesions past year.

	Frequency	***Percent***
PRESENT	62	62.0
ABSENT	38	38.0
Total	100	100.0

Table 5. Incidence of new lesions past 1 year in the study patients.

b. PROGRESSION OF EXISTING LESIONS – The majority of the study subjects i.e., 45 % had progression of existing lesions in the past year. 55% of study subjects had no extension of lesions in the past year.

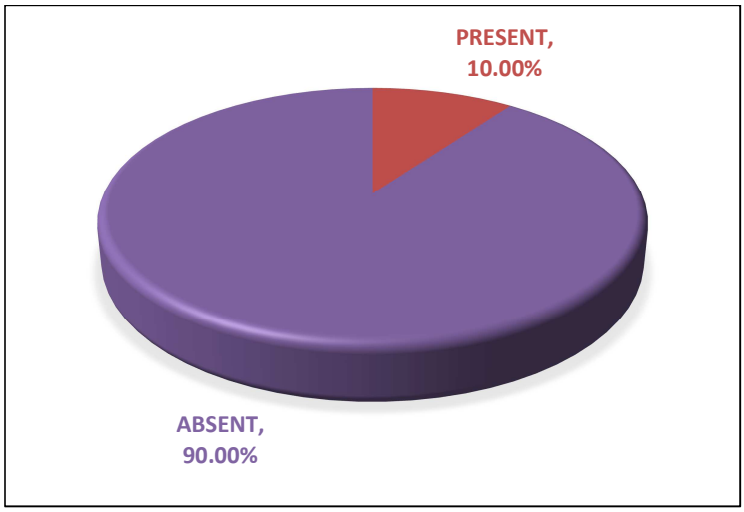


Graph 7: Distribution of the subjects by the progression of existing lesions in the past 1 year.

	Frequency	Percent
PRESENT	45	45.0
ABSENT	55	55.0
Total	100	100.0

Table 6. Incidence of increase in the size of lesions over 1 year

c. **KOEBNER PHENOMENON** - 10 % of the study subjects had new lesions following trauma in the past year. 90% of the study subject had no new lesions at the trauma site (n=133) in the past year, that is no Koebner's phenomenon is present in them.

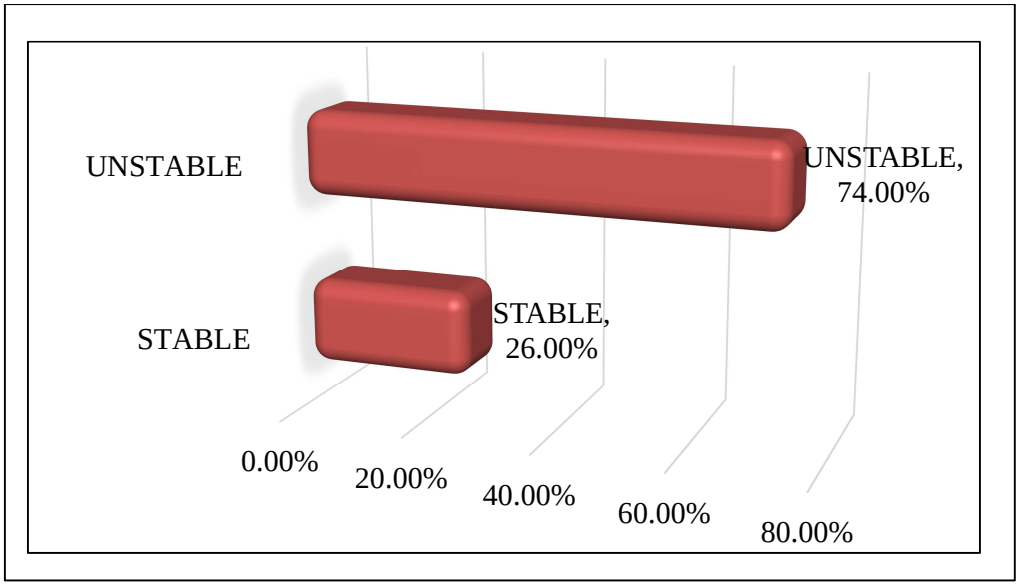


Graph 8: Distribution of the subjects by the history of Koebner's phenomenon.

	Frequency	***Percent***
PRESENT	10	10.0
ABSENT	90	90.0
Total	100	100.0

Table 7: Incidence of Koebner's phenomenon in the study population

d. CLINICAL ASSESSMENT ACTIVITY on clinical examination out of 100 patients 74 patients showed unstable lesions and 26 had stable lesions.



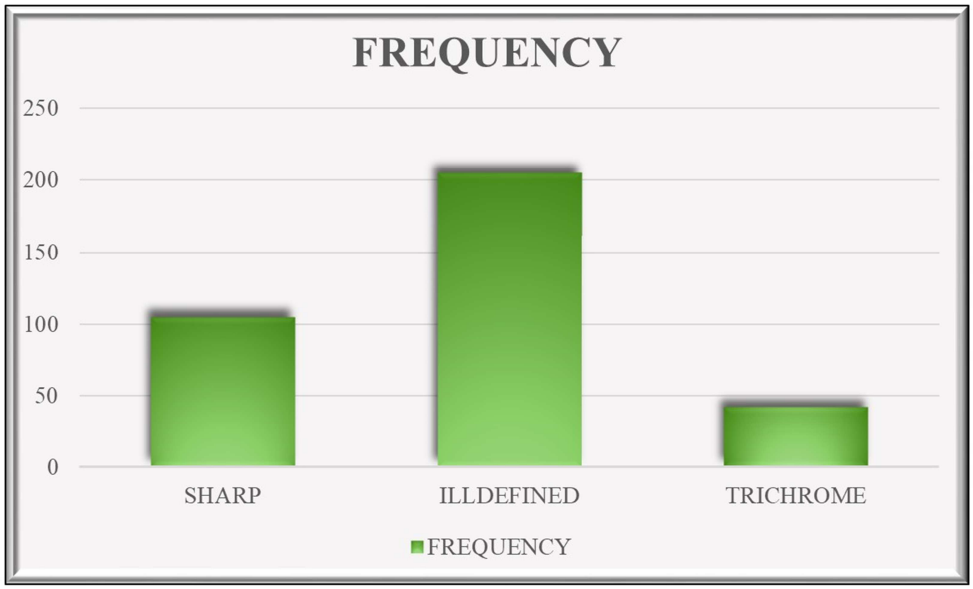
Graph 9: Distribution of the subjects by the clinical activity of vitiligo.

	Frequency	Percent
STABLE	26	26.0
UNSTABLE	74	74.0
Total	100	100.0

Table 8: Distribution of study subjects by clinical stability of lesions

7. Dermoscopic assessment of lesions based on BPleFoSK criteria

a. Border: On dermoscopic examination, out of 352 lesions examined 205 lesions showed ill-defined borders and 105 lesions showed sharp borders and 42 lesions showed trichrome borders.

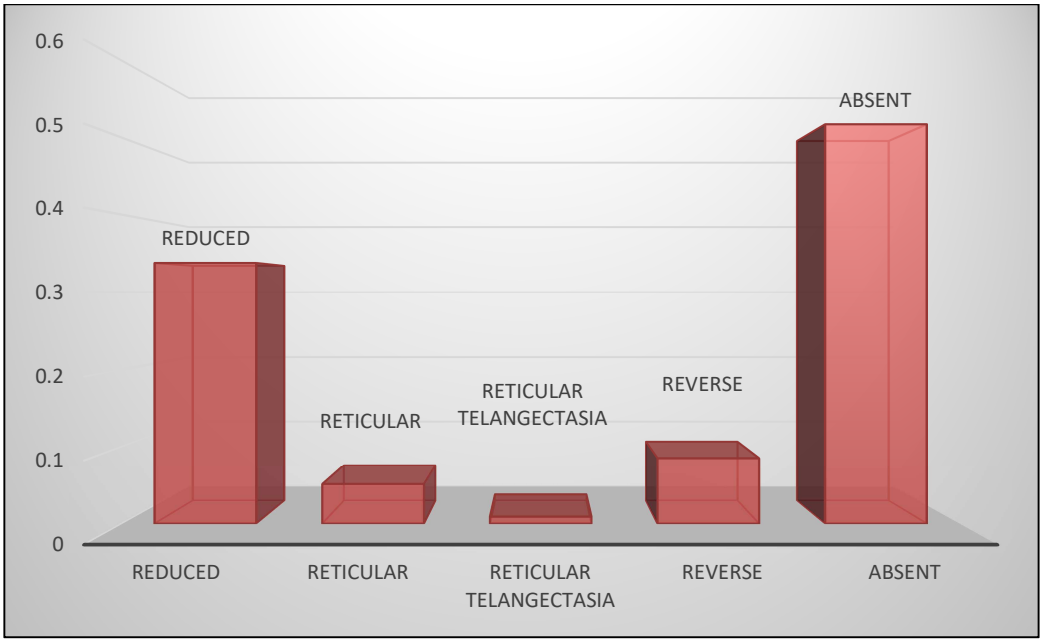


Graph 10: Distribution of the lesions based on borders.

	Frequency	Percent
ILLDEFINED	205	58.2%
TRICHROME	42	11.9%
SHARP	105	29.8
Total	352	100.0

Table 9: Dermoscopic assessment of lesional borders.

b. **PIGMENT NETWORK** on dermoscopic examination out of 352 lesions examined, 51.7% showed absent pigmentation network and 33.8% showed reduced pigment network, 8.5% of study subjects have shown reverse pigmentation and 0.9% had shown reticular telangiectasia.

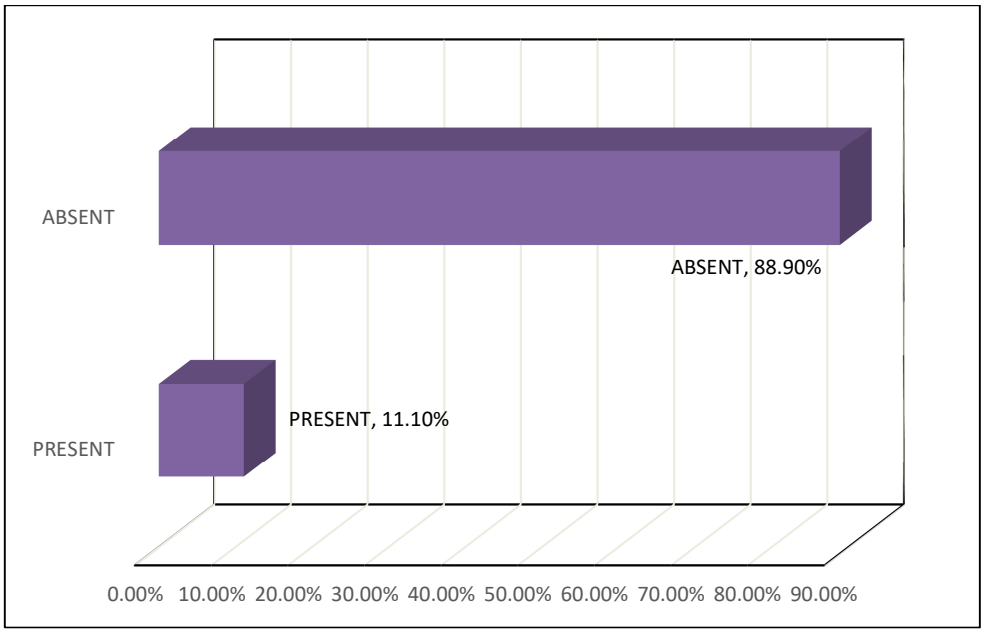


Graph 11: Distribution of the lesions by pigment network.

	Frequency	Percent
REDUCED	119	33.8
RETICULAR	18	5.1
RETICULAR TELANGECTASIA	3	0.9
REVERSE	30	8.5
ABSENT	182	51.7
Total	352	100.0

Table 10: Dermoscopic assessment of pigment network of lesions

c. **PERIFOLLICULAR HYPERPIGMENTATION** on dermoscopic examination out of 352 lesions examined 88.9% showed absent perifollicular hyperpigmentation and 11.1% that is 39 of study population have shown perifollolicular pigmentation.

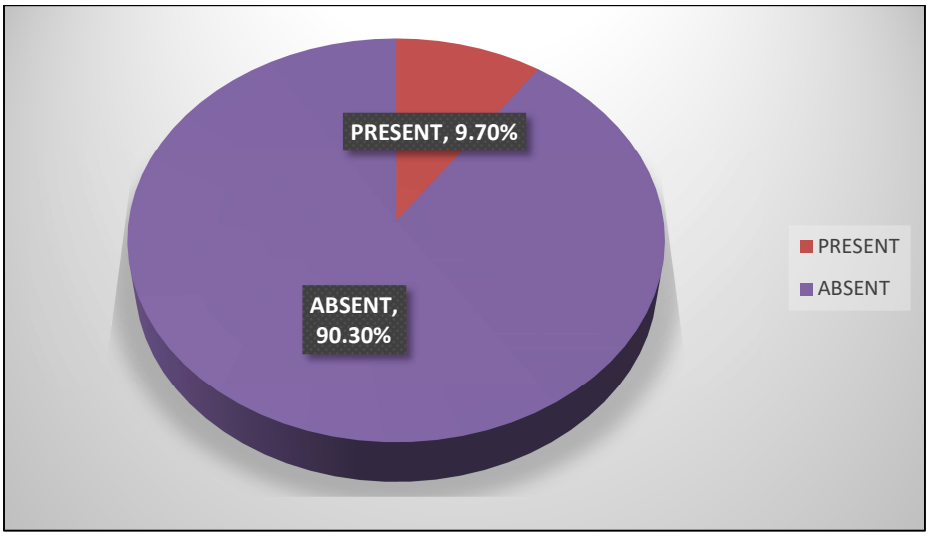


Graph 12: Distribution of the lesions by presence of perifollicular Hyperpigmentation.

	Frequency	Percent
PRESENT	39	11.1
ABSENT	313	88.9
Total	352	100.0

Table 11: Distribution of the lesions by presence of perifollicular hyperpigmentation.

d. **PERILESIONAL HYPERPIGMENTATION** on dermoscopic examination out of 352 lesions 90.3% showed absent perilesional hyperpigmentation and 9.7% had showed absent perilesional hyperpigmentation.

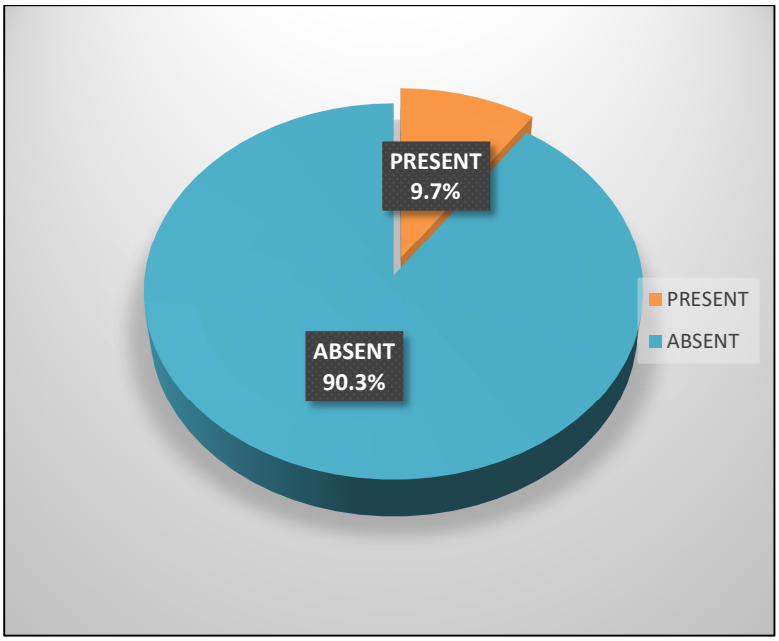


Graph 13: Distribution of the lesions by presence of perilesional hyperpigmentation

	Frequency	Percent
PRESENT	34	9.7
ABSENT	318	90.3
Total	352	100.0

Table 12: Distribution of the lesions by presence of perilesional hyperpigmentation.

e. SATELLITE LESIONS: Dermoscopic examination of 352 lesions, 9.7% showed presence of satellite lesions& 90.3% had no satellite lesions. Absent satellite lesions are indicator of stability by a sensitivity of 99.6%.[108]

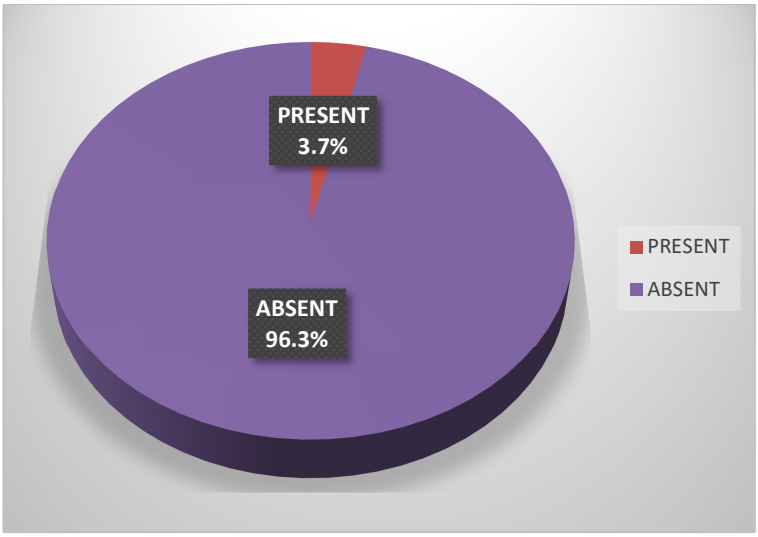


Graph 14: Distribution of the lesions by presence of satellite lesions.

PRESENT	34	9.7
ABSENT	318	90.3
Total	352	100.0

Table 13: Distribution of the lesions by presence of satellite lesions .

f. **MICROKOEBNERS** - dermoscopic examination out of 352 lesions examined showed microkoebners in 3.7% of lesions. Absent microkoebners is indicator of stability by a sensitivity of 100%[108].

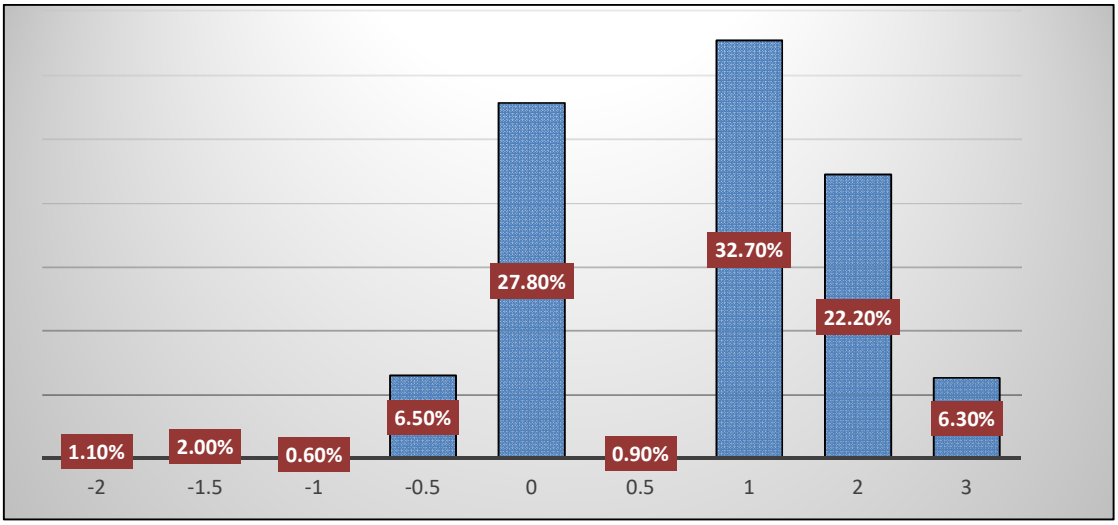


Graph 15: Distribution of the lesions by presence of microkoebners phenomenon.

	Frequency	Percent
PRESENT	13	3.7
ABSENT	339	96.3
Total	352	100.0

Table 14 : Distribution of the lesions by presence of microkoebners phenomenon

G. DERMOSCOPIC ASSESSMENT ACTIVITY

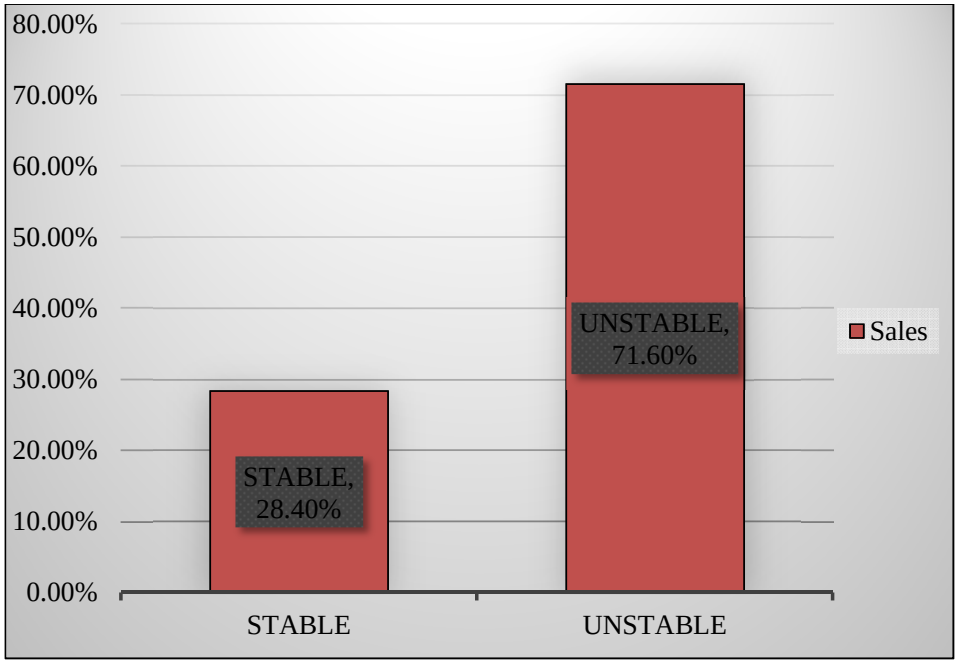


Graph 16: Distribution of the subjects by dermoscopic scoring for stability according to BPleFoSK criteria.

BPleFoSK scoring	Frequency	Percent
-2.0	4	1.1
-1.5	7	2.0
-1.0	2	.6
-.5	23	6.5
0	98	27.8
.5	3	.9
1.0	115	32.7
2.0	78	22.2
3.0	22	6.3
Total	352	100.0

Table 15: Distribution of the subjects by dermoscopic scoring for stability according to BPleFoSK criteria

Out of 352 lesions on dermoscopic examination 100 were scored for stable vitiligo and 252 showed features of unstable vitiligo.



Graph 17: Distribution of the subjects by dermoscopic stability of lesions.

	Frequency	***Percent***
STABLE	***100***	***28.4***
UNSTABLE	***252***	***71.6***
Total	***352***	***100.0***

Table 16: Distribution of the subjects by dermoscopic stability of lesions.

8. **CO-RELATION:** As shown in the below table, correlation was present between Clinical and Dermoscopic assessments in 248 lesions (70.5%), whereas in the remaining 104 lesions (29.5%) there was no correlation between Clinical and Dermoscopic assessments.

	Frequency	Percent
PRESENT	248	70.5
ABSENT	104	29.5
Total	352	100.0

Table 17. Distribution of the subjects showing Clinical and Dermoscopic correlation

9. **Clinical Assessment activity and Dermoscopic Assessment activity**

		Dermoscopic Assessment activity		Total
		STABLE	UNSTABLE	
Clinical Assessment activity	STABLE	45	49	94
	UNSTABLE	55	203	258
Total		100	252	352

Table 18: summary

As shown in the above table, in 45 cases both assessments recorded stable activity and in 203 cases both assessments recorded unstable activity. But in 49 cases clinical assessment recorded stable activity while dermoscopic assessment recorded unstable activity. In 55 cases clinical assessment recorded unstable activity while dermoscopic assessment recorded stable activity. Hence correlation was present between both assessments in 45+203=248 cases while in remaining 55+49 =104 cases correlation was absent. Kappa value was 0.260 which indicated mild agreement between clinical and dermoscopic assessments which was statistically significant (P value <0.05). Henceforth dermoscopy is a better indicator of assessment of stability. It can predict stability in clinically unstable lesions and thus aids in management modification, helps in planning the surgery, and can act as visual indicator in patient counselling.

DISCUSSION

Vitiligo being a common depigmentary disorder is associated with a lot of social stigma. Therefore, timely diagnosis, stability assessment and medical treatment of vitiligo becomes important to prevent spread, stabilize the disease & aids in planning for surgical treatment in required cases. Dermoscopy being a non-invasive tool can be used in diagnosis of vitiligo, finding stability of vitiligo and differentiates it from other common hypopigmentary disorders.

Our study was a hospital-based cross sectional study conducted over a period of 12 months from January 2021 to December 2021 in the department of Dermatology, Venereology and Leprosy, KLE's Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi.

A total of 100 patients screened by woods lamp clinically diagnosed with vitiligo, who satisfied the inclusion and exclusion criteria were included in the study. After obtaining their consent, all the patients were made to undergo Wood's lamp, and dermoscopic examination (ILLUCO IDS-1100 dermatoscopy). Appropriate photographs of the patients were taken. After compiling these records, the data was analyzed.

AGE

Out of them, 41 were males and 59 were females with female to male ratio of 3:2. Minimum age observed in the sample was 3 years and maximum observed was 73 years. The average age of presentation with standard deviation was 33.97±17.158 years and was near comparable to Houssien, Abdullah & Houssien, Rana where +/- SD was 40±17.[109]

This signifies that, it remains as social stigma among all age groups and patients are seeking treatment.

SEX

There was a female predominance observed in our study, i.e., out of 100 patients, there were 59 females and 41 males (female to male ratio of 2:3). This study is comparable with Anaba, Ehiaghe, Gender Based Differences in Epidemiologic and Clinical Profile of Adult Vitiligo[110.] The female preponderance in our study may be because female patients seek medical care for vitiligo more than males due to social stigma and cosmetic concerns.

FAMILY HISTORY

At present, the consensus is that a familial incidence between 20% and 30% occurs; the reported findings on this score in India and abroad vary between 7.5% and 41% respectively by Dutta AK. Vitiligo: Neural and immunologic linkages[111], which is in support of this study with a family history of 11%. The low rate of family history may be attributed to the small sample size included in our study.

ANY SKIN CONDITIONS - associated skin disorders are less in this study. Nearly 86% of patients have no associated skin conditions.

SYSTEMIC ILLNESS - Other autoimmune diseases are increased in comparison with the general population with 8% with of cases showing hypothyroidism. Most patients with vitiligo will not develop autoimmune disease, but a significant minority will develop hypothyroidism. Patients with vitiligo should be regularly screened yearly for thyroid disorders. In our study 81% of cases did not have any associated systemic illness followed by hypothyroidism in 8%, hypertension in 4% of cases.

20% had at least 1 comorbid autoimmune disease in a study of autoimmune diseases with vitiligo by Liza gill[112] which is in not in line with our study.

CLINICAL ASSESSMENT ACTIVITY

a. **NEW LESIONS** Majority of the study subjects i.e., 62 % (n=219) had new lesions in the past one year. 38% of study subjects had no new lesions(n=133) in the past one year. According to Gandhi et al.[50] the incidence of new lesions over the preceding 1 year was noted in 24 (43.6%) patients.

b. **PROGRESSION OF EXISTING LESIONS** – The majority of the study subjects i.e.., 45 % had progression of existing lesions in the past one year. 38% of study subjects had no extension of old lesions in the past one year. Gandhi et al.[50] had reported a similar rate in 109 (68.12%) patients in their study.

c. **KOEBNER PHENOMENON** minority of the study subjects i.e., 10 % had new lesions following trauma in the past one year. 90% of study subject had no new lesions at site of trauma (n=133) in the past one year. The prevalence of the Koebner phenomenon is 21 to 62% in vitiligo by Khurram H [113] which is comparatively more in comparison with our study.

Lower response to treatment also appears to occur in vitiligo patients who exhibited Koebnerisation. **Van Geel N[114]** concluded that Koebnerisation might function as a clinical indicator of disease activity in vitiligo, as well as serve as a predictor of treatment response. Clinical examination of 100 patients, 74 patients showed unstable lesions and 26 patients had stable lesions.

Dermoscopic assessment -

a. **Border** - On dermoscopic examination out of 352 lesions examined 205 lesions showed ill-defined borders, 42 lesions showed trichrome borders and 105 lesions showed sharp borders which is denoted as stable parameter with high specificity by Balakrishna Nirmal [108].

b. **PIGMENT NETWORK -** On dermoscopic examination out of 352 lesions examined 182 showed absent pigmentation and 119 showed reduced pigmentation. Rapidly evolving vitiligo results in reverse pigment network. It was seen in 8.5% of our cases which is lesser than described by thatte et al.[34] as 20 %.

c. **PERIFOLLICULAR HYPERPIGMENTATION** on dermoscopic examination out of 352 lesions examined 88.9% showed absent perifollicular hyperpigmentation and 11.1% showed perifollolicular pigmentation. Perifollicular pigmentation is indicated as a stable parameter by chuh and Zawar[115].

d. **PERILESIONAL HYPERPIGMENTATION -** On dermoscopic examination out of 352 lesions 90.3% showed absent perilesional hyperpigmentation and 9.7% showed perilesional hyperpigmentation. Perilesional hyperpigmentation has high sensitivity < 89.2%> and specificity < 85.4%> by Meng& Wali.[116,117]

e. **SATELLITE LESIONS –** On dermoscopic examination out of 352 lesions, satellite lesions were observed in 9.7% of lesions studied. Absent satellite lesions are an indicator of stability by a sensitivity of 99.6%[108].

f. **MICROKOEBNERS** - dermoscopic examination out of 352 lesions in showed microkoebners in 3.7% of lesions examined. Absent micro-Koebner is indicator of stability by a sensitivity of 100%[108].

Using BPleFoSK criteria for dermoscopic assessment of stability out of 352 lesions on dermoscopic examination 100 lesions(28.4%) have scored for stable vitiligo and 252 lesions (71.5%) showed features of unstable vitiligo. In 45 lesions both assessments recorded stable activity and in 203 lesions both assessments recorded unstable activity. But in 49 lesions clinical assessment recorded stable activity while dermoscopy assessment recorded unstable activity. In 55 lesions clinical assessment recorded unstable activity while dermoscopic assessment recorded stable activity. Hence correlation was present between clinical & dermoscopic assessments in 45+203=248 lesions while in the remaining 55+49 =104 lesions clinical & dermoscopic correlation was absent.

The following classifications has been suggested to interpret the strength of the agreement based on the Cohen's Kappa value (Altman 1999, Landis JR (1977)).

VALUE OF K	STRENGTH OF AGREEMENT
< 0	Poor
0.01 - 0.20	Slight
0.21-0.40	Fair
0.41-0.60	Moderate
0.61-0.80	Substantial
0.81 - 1.00	Almost perfect

Table 19: Interpretation of K Values.

The Kappa value in our study is 0.260 which indicated mild strength of agreement between clinical and dermoscopic assessments which was statistically significant (P value <0.05). Henceforth dermoscopy is a better indicator of assessment of stability. It can predict stability in clinically unstable lesions and thus aids in management modification, reduces the delay in planning the surgery, and can act as a visual indicator in patient counseling. Altogether, the sensitivity and specificity of dermoscopy were unarguably high, hence making it an important prognostic tool in the assessment of vitiligo.

CONCLUSION

The purpose of the current analysis was to assess the dermoscopic characteristics of vitiligo and their relationship to clinical stability of the lesion. Consequently, we concluded the following in our present study:

- One hundred patients were enrolled in the study and 352 lesions were examined and analyzed.
- Age group of 3–73 years of age were the commonest age group affected with disease.
- Females are affected more than males and the difference between the sexes is statistically significant.
- Accentuation (bright bluish white fluorescence) on Woods lamp examination was noted in almost all the lesions in all the patients evaluated in this study.
- Patients who were diagnosed with unstable vitiligo clinically were diagnosed with unstable vitiligo on dermoscopic examination as well.
- However, in 49 (13.9%) lesions which were clinically stable vitiligo lesions, were diagnosed as unstable vitiligo lesions on dermoscopy examination and 55(15.6%) lesions which were clinically unstable vitiligo lesions were diagnosed as stable vitiligo lesions following dermoscopic examination.
- In patients with depigmentary disorders, the use of dermoscopy should be encouraged in daily clinical practice to establish an accurate diagnosis early and avoid unnecessary biopsies.
- Dermoscopy is a better indicator of assessment of stability. It can predict unstability in clinically stable lesions and thus aids in management modification, helps in planning the surgery, and can act as visual indicator in patient counselling.

SUMMARY

The present study aimed to evaluate the dermoscopic features of vitiligo and their association with clinical stability in children.

- A total of 100 patients were recruited.
- Female preponderance was noted (M: F = 2:3).
- Age group of 3–73 years were commonly affected.
- Family history is 11% in our study.
- 86% of patients have no associated skin conditions.
- 81% of cases did not have any associated systemic illness followed by hypothyroidism in 8%, hypertension in 4% of cases.
- Woods lamp accentuation was noted in most of the lesions in all patients.
- Increase in the size of lesions over the preceding 1 year was seen in 45% of patients with vitiligo lesions
- Incidence of new lesions over the preceding 1 year was noted in 62% of patients with vitiligo.
- Koebner's phenomenon is present in 10 % of patients
- Vitiligo was clinically classified as unstable in 74% of patients and stable in 26% of patients.
- Dermoscopically, vitiligo was classified as unstable in in 70.2% of patients and stable in 29.8 % of patients.
- The findings in patients with stable vitiligo included well defined sharp border (29.8%) followed by absent pigmentation in 182 (51.7%) marginal hyperpigmentation (9.7%), perifollicular hyperpigmentation (11.1%),

- Most of the dermoscopic features were observed in patients with unstable vitiligo.
- Patterns of satellite lesions are seen in 9.7%, comet tail in 3.7% of study population is noted.
- Patients who were clinically diagnosed with unstable vitiligo were diagnosed with unstable vitiligo on dermoscopic examination as well.
- However, 13.9% of patients who were clinically diagnosed with stable vitiligo were diagnosed with unstable vitiligo on dermoscopic examination.

LIMITATIONS

The study's shortcomings were its limited sample size, which could have reduced the power of the study. Additionally, biopsy was not performed and, therefore, tissue diagnosis was not available for confirmation of the clinical and dermoscopic conclusions.

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ANNEXURE – I

INFORMED CONSENT FORM

I.D.NO. | | | |

The study is conducted by DR_____Post Graduate (M.D) student in Dermatology under the guidance of **DR. _____** Associate Professor, Department of Dermatology, Venereology and Leprosy, JNMC, BELAGAVI.

Respected Sir/Madam,

We invite you to participate in our study as you are eligible for the same. During the study you will be asked some questions in detail regarding your present complaints.

Purpose of the study: Vitiligo is a common depigmentary condition wherein the changes can be seen using an instrument called dermoscope. Hence this study intends to observe those changes/findings using the dermoscope. You are being requested to participate in this study because you have been diagnosed to have vitiligo.

Procedure: Should you choose to participate, you will be asked to give a detailed history of your disease and undergo physical examination. Following this, Wood's lamp examination and dermoscopic examination of the lesions will be done along with appropriate clinical picture documentation.

Risks and Benefits: The result of you taking part in this research would help health care providers towards a better understanding of the disease, and thus we will be able to provide improved patient care.

Alternatives: If you decide not to participate in this study, you will still be receiving the usual standard care for your disease.

Privacy and confidentiality: Your privacy will be respected and all information collected about you during the course of this study will be kept confidential. Your identity will remain undisclosed.

Relations with the Institutional policy: The J N Medical College will provide, within the limitations of the laws of the State of Karnataka, facilities and medical attention to patients who suffer injuries as a result of participating in this project.

Financial incentives: You shall not be receiving any payment or any financial incentives for participating in this study.

Authorization to publish results: The results of this study may be published for scientific purpose or presented to a scientific group. Your identity, however, will be maintained confidential at all times.

Voluntary participation: In case you need further information regarding your rights as a study participant, you may please contact Dr. Harsha Hedge**,** chairman of the ethical committee, J N Medical College, IEC & Scientist D, ICMR, National Institute Of Traditional Medicine, Belagavi, Telephone No: 9480422500.

STATEMENT OF CONSENT

I.D.NO: | | | |

I Mr/Ms/Mrs ---------- volunteer and consent to participate in this study. I have read the consent document or it has been read to me in my vernacular language. I accept to participate in the study. All the information regarding this study is provided to me and I have understood the same. I have been given the opportunity to ask questions and obtain appropriate answers.

Participant's name:

Signature or left thumb print of participant:

Witness name:

Signature of witness:

Signature of the investigator:

Date:

ANNEXURE - II

PROFORMA

ONE YEAR HOSPITAL BASED CROSS SECTIONAL STUDY OF DERMOSCOPIC FINDINGS IN VITILIGO CORRELATING WITH LESIONAL ACTIVITY"

KLE's Dr. Prabhakar Kore Hospital & MRC, Belagavi.

Case No. **OP/IP No.**

Name:

Age:

Sex:

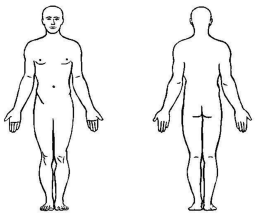
Occupation:

Income:

Address with phone number:

* **Presenting complaints and duration:**

A. white patch **duration in years** **sites involved**



B. **Family History:**

C. Associated skin conditions

D. Clinical assessment of disease activity according to standard guidelines of care for vitiligo surgery by devinder prasad and somesh gupta

* PARAMETERS FOR ESTABLISHING STABILITY OF VITILIGO

a. History of progression: Absence of new lesion

b. Extension of old lesions: No extension of old lesions

c. Koebner phenomenon: Absence of Koebner phenomenon either based on history or by checking for experimentally induced vitiligo.

Investigations

E. WOOD'S LAMP EXAMINATION

- **BRIGHT BLUE ACCENTUATION YES NO**

F. DERMOSCOPY PARAMETERS SCORE

1.	**Border sharp(sharp/ill-defined/trichrome)**	**+1**
2.	**Pigment network (absent / reticulate/reduced/reverse)**	**+1**
3.	**Perilesional hyperpigmentation**	**+1**
4.	**Perifollicular hyperpigmentation**	**+1**
5.	**Satellite lesions/tapioca sago/polka dot**	**-1.5**
6.	**Micro koebner phenomenon/comet tail**	**-2**

a/c to BPLeFoSK criteria, predictors of stability are sharp border, absent / reticulate pigment network, perilesional, perifollicular hyperpigmentation are markers of stability.

A cut of score of ≥1.5 is marked as stable vitiligo.

Diagnosis:-

Signature:

ANNEXURE III – PHOTOGRAPHS

Fig: 1a

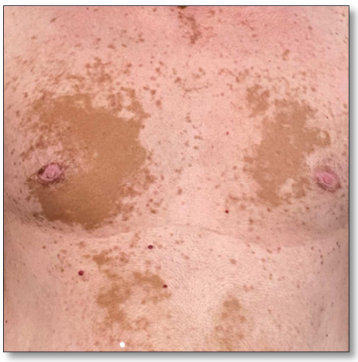


Fig: 1b

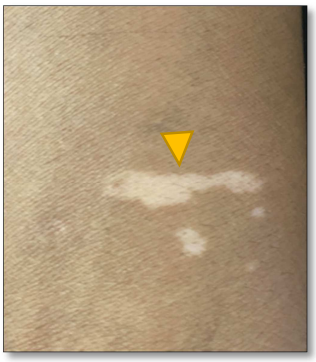


Fig:1c

Figure 1a – clinical image of stable vitiligo. Figure 1b - clinical image of unstable vitiligo. Figure 1c – clinical image of koebners phenomenon.(based on clinical criteria of stability).

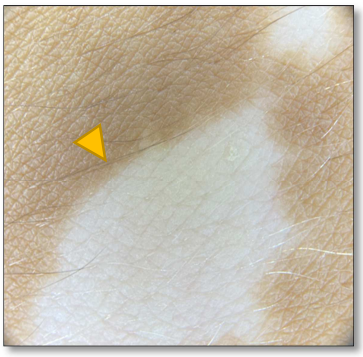


Fig: 2a

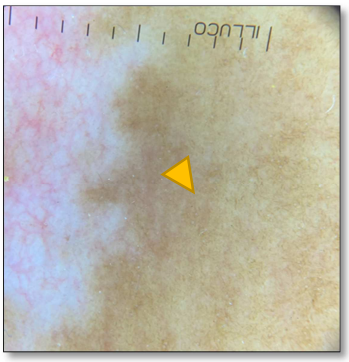


Fig:2b

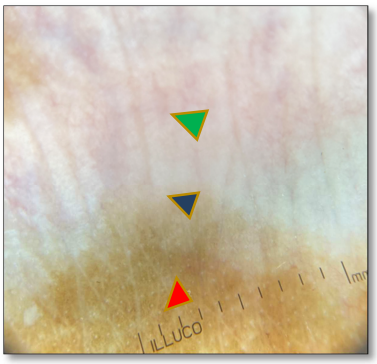


Fig:2c

Dermocopy images of vitiligo lesions captured by ILLUCO IDS -1100 model, under 10X magnification in polarised mode.

Figure 2a – well defined sharp borders (yellow arrow head).

Figure 2b – ill-defined borders (yellow arrow head).

Figure 2c – trichrome border (from center to periphery green arrow head- absent pigmentation followed by blue arrow head – reduced pigmentation followed by red arrow head - normal skin pigmentation).

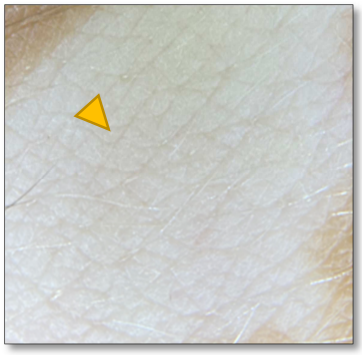


Fig:3a

Fig:3b

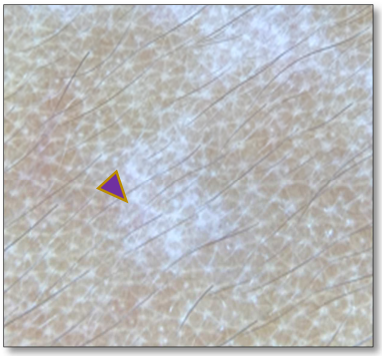


Fig: 3c

Fig: 3d

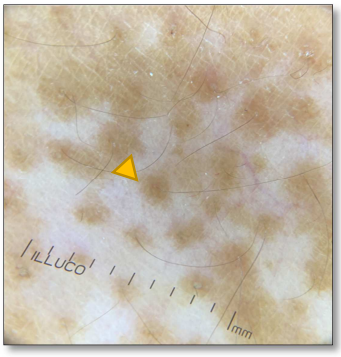
Dermocopy images of vitiligo lesions(polarised x 10) showing types of pigment networks.

Figure 3a – absent pigment network due to complete loss of pigment cells.

Figure 3b –green arrow - reduced pigment network due broken pigment network with ill defined borders. Appreciated when compared to white glow area i.e is absent pigment netwok.

Figure 3c – purple arrow -reverse pigment network/negative pigment networkdue to reticular white lines with central dark areas seen in rapily evolving vitiligo.

Figure 3d – (blue arrow)brown thin lines in reticular pattern with intervening pale areas. Brown reticular pattern corresponds to slope of rete ridges and pale white areas correspond to tip of rete ridges. Yellow arrow head – correspond to ecrine sweat glands.



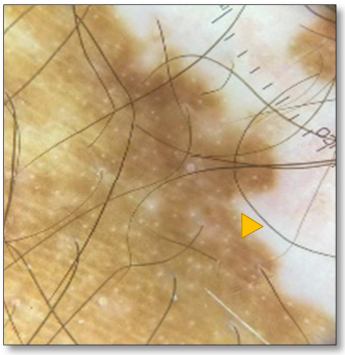


Fig: 4a **Fig: 4b**

Dermocopy images of vitiligo lesion margins (polarised x 10) showing,

Figure 4a – perifollicular hyperpigmentation.

Figure 4b – perilesional hyperpigmentation.

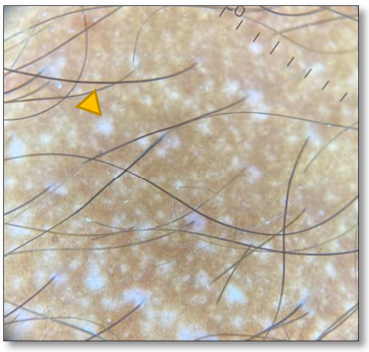


Fig: 5

Dermocopy images of (polarised x 10) margin of vitiligo lesions showing,

Figure 5 – satellite/ tapioca sago/ sabudana/ polka dot apperence descibes multiple hypo pigmented dots surrounding a primary lesion.

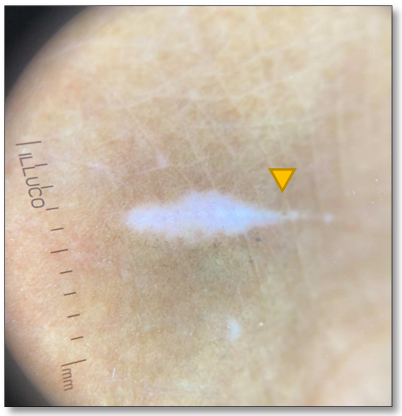


Fig: 6

Dermocopy images of (polarised x 10) margin of vitiligo lesions showing,

Figure 6 – micro koebners/ comet tail appearance showing areas of depigmentation in linear pattern along the lines of trauma.

ANNEXURE IV - KEY TO MASTER CHART

- COLOUMN A – SERIAL NUMBER
- COLOUMN B – OUT PATIENT NUMBER
- COLOUMN C – NAME OF THE SUBJECT
- COLOUMN D – AGE OF THE SUBJECT
- COLOUMN E – SEX OF THE SUBJECT
- COLOUMN F – FAMILY HISTORY OF THE DISEASE
- COLOUMN G – ASSOCIATED SKIN CONDITIONS IF ANY
- PMLE – POLYMORPHOUS LIGHT ERUPTION
- DPN – DERMATOSIS PAPULOSA NIGRA
- COLOUMN H – ASSOCIATED SYSTEMIC ILLNES IF ANY
- HTN – HYPERTENSION
- DM – DIABETES MELITUS
- COLOUMN I – SITE OF LESION
- H&N – HEAD AND NECK
- UL – UPPER LIMB
- LL – LOWER LIMB
- COLOUMN J – NUMBER OF LESIONS EXAMINED
- COLOUMN K – BRIGHT BLUE FLUORESENCE IF PRESENT
- COLOUMN L – PRESENCE OF CLINICALLY NEW LESIONS
- COLOUMN M – PROGRESSION OF EXSISTING LESIONS
- COLOUMN N – KOEBNERS PHENOMENON
- COLOUMN O – CLINICAL ACTIVITY OF LESION BASED ON CLINICAL CRITERIA OF STABILITY

- COLOUMN P – DERMOSCOPIC EXAMINATION OF BORDERS
- COLOUMN Q – DERMOSCOPIC EXAMINATION OF PIGMENT NETWORK
- COLOUMN R – DERMOSCOPIC EXAMINATION OF PERIFOLLICULAR PIGMENTATION
- COLOUMN S – DERMOSCOPIC EXAMINATION OF PERILESIONAL PIGMENTATION
- COLOUMN T – DERMOSCOPIC EXAMINATION OF SATELLITE LESIONS
- COLOUMN U – DERMOSCOPIC EXAMINATION OF MICRO KOEBNERS PHENOMENON
- COLOUMN V – DERMOSCOPIC SCORING ACCORDING TO BPleFoSK CRITERIA
- COLOUMN W – DERMOSCOPIC ASSESSMENT OF LESIONS
- COLOUMN X – CLINICAL AND DERMOSCOPIC CORELATION

CLINICAL & DERMOSCOPIC CORELATION OF DISEASE ACTIVITY IN VITILIGO																							
										WOODS LAMP	LINICAL ASSEMENT PAST 1 YEAI				DERMOSCOPIC ASSEMENT								
S.NO	O.P NO	NAME	AGE	SEX	FAMILY H/O	ANY SKIN CONDITIONS	SYSTEMIC ILLNESS	SITE	NO OF LESION	BRIGHT BLUE FLUORESENCE	NEW LESIONS	PROGRESSION OF EXISTING LESIONS	KOEBNER PHENOMENON	ACTIVITY	BORDER	PIGMENT NETWORK	PERIFOLLICULAR HYPERPIGMENTATION	PERILESIONAL HYPERPIGMENTATION	SATELLITE LESIONS	MICROKOEBNERS	SCORE	ACTIVITY	CO - RELATION
1		ACHYUT R HOLI		M	ABSENT	ABSENT	NO	UL	1	PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	PRESENT
								LL	1	PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	PRESENT
								CHEST	1	PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	PRESENT
								BACK	1	PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	PRESENT
2	4161967	AFTABPASHA M.P.	69	M	ABSENT	ABSENT	NO	LEFT HAND	3	PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	REDUCED	PRESENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	ABSENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	REDUCED	PRESENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	ABSENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	REDUCED	PRESENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	ABSENT
								RIGHT HAND	3	PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	REDUCED	PRESENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	ABSENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	REDUCED	PRESENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	ABSENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	REDUCED	PRESENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	ABSENT
3	4992423	AMIT GOUDA SIDDANGOUDA	18	M	ABSENT		NO	UL	1	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	ABSENT
								LL	2	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	ABSENT
										PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	0.5	UNSTABLE	ABSENT
4	6223294	ANITA SHANKAR KABBUR	42	F	ABSENT	ABSENT	HTN / HEARING ABNORMALITY	BACK	2	PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	ABSENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	ABSENT
5	5087187	ANAPPA YALLAPA VAGGANAV	36	M	ABSENT	ABSENT	NO	H&N		PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
								UL		PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
								TRUNK		PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
								LL		PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
6	4776468	ASMITA RAJENDRA DESHMUKH	20	F	ABSENT	ABSENT	NO	NECK	1	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	-0.5	UNSTABLE	PRESENT
								FACE	1	PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	-0.5	UNSTABLE	PRESENT
								TRUNK	1	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	-0.5	UNSTABLE	PRESENT
								LF LL	2	PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	-0.5	UNSTABLE	PRESENT
										PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	-0.5	UNSTABLE	PRESENT
7	4784073	ATIKA AMEN MULLA	22	F	ABSENT	ABSENT	NO	TRUNK		PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
								LEFT LL		PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
8	4784074	RASHMIKA	3	F	ABSENT	ABSENT	NO	H&N	2	PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	RETICULAR	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
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										PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
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										PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
9	6068192	BEERAPPA BASAPPA NEGINAL	24	M	ABSENT	ABSENT	NO	LL	3	PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	SHARP	ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	-0.5	UNSTABLE	PRESENT
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10	3348118	BHARATI SHIVANAND H	18	F	ABSENT	ABSENT	NO	H&N	1	PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	RETICULAR	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
								UL	1	PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	RETICULAR	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
								LL	2	PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	RETICULAR	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
11	3616191	DAYANAND KRISHNA PATIL	32	M	ABSENT	ABSENT	NO	TRUNK	7	PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	ABSENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	ABSENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	ABSENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	ABSENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	ABSENT
											ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	ABSENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	RETICULAR	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	ABSENT
12	5940622	DEEPA ANILKUMAR K	14	F	ABSENT	ABSENT	NO	UL		PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	-0.5	UNSTABLE	ABSENT
13	3954825	DEEPEN KANUBHAI THAKHAL	30	F	ABSENT	ABSENT	NO	UL		PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	REDUCED	ABSENT	PRESENT	ABSENT	ABSENT	1	UNSTABLE	ABSENT
								LL		PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	REDUCED	ABSENT	PRESENT	ABSENT	ABSENT	1	UNSTABLE	ABSENT
14	6019656	DEEPA PRABHAKAR AJAREKAR	22	F	ABSENT	ABSENT	PREGNANCY INDUCED HYPOTHYROIDISM	FACE		PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
								LEFT LL	3	PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	PRESENT	-2	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	PRESENT	-2	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	PRESENT	-2	UNSTABLE	PRESENT
15	5989208	GEETHA VIRESH	45	F	ABSENT	ABSENT	NO	FACE		PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	PRESENT
								UL		PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	PRESENT
								TRUNK		PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	PRESENT
								LL		PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	-0.5	UNSTABLE	ABSENT
16	3390069	GURUNATH MUSAPPA	31	M	ABSENT	ABSENT	HYPOTHYROIDSM	TRUNK	2	PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	ABSENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	ABSENT
								LL	2	PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	RETICULAR TELANGECTASIA	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	ABSENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	ABSENT
17	5260919	JAGADISH VEERAPPA	46	M	ABSENT	ABSENT	NO	UL	2	PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	RETICULAR TELANGECTASIA	ABSENT	PRESENT	ABSENT	ABSENT	2	STABLE	PRESENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	RETICULAR	ABSENT	PRESENT	ABSENT	ABSENT	2	STABLE	PRESENT
								TRUNK	1	PRESENT	ABSENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT

18	6383570	KAILASH CHOGARAM BHATI	16	M	ABSENT	ABSENT	NO	FACE	2	PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	PRESENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	PRESENT
19	3626623	KANCHAN BABAJI PATIL	50	M	ABSENT	ABSENT	NO	UL	4	PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	ABSENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	ABSENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	ABSENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	ABSENT
20	3626621	KASHAVA ANONY	51	F	ABSENT	ABSENT	HYPERTENSION	TRUNK		PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	RETICULATE TELANGECTASIA	PRESENT	ABSENT	ABSENT	ABSENT	2	STABLE	PRESENT
21	5839818	KASHAVA DEMAOOA HONGAL	45	F	ABSENT	ABSENT	HYPERTENSION	FACE		PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	ABSENT
								UL		PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
								TRUNK		PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
								LL		PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	ABSENT
22	6191951	KAVERI VIVEKANAND	11	F	PRESENT	ABSENT	NO	FACE	2	PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	ABSENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	ABSENT
								NECK		PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	REVERSE	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	ABSENT
23	6294305	KERTHI RASHAN JADHAV	8	F	ABSENT	ABSENT	NO	FACE	5	PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REVERSE	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	RETICULAR	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	RETICILAR	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	RETICULAR	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
24	5953673	KESHAV SHRINIVAS BALLARI	9	M	ABSENT	ABSENT	NO	FACE	2	PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	SHARP	ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	0.5	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	0	UNSTABLE	PRESENT
25	6326263	MAHADEVI BASANAGOUDA	44	F	ABSENT	ABSENT	H/O OF COVID	UL		PRESENT	PRESENT	PRESENT	PRESENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	-0.5	UNSTABLE	PRESENT
								TRUNK		PRESENT	PRESENT	PRESENT	PRESENT	UNSTABLE	SHARP	ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	0.5	UNSTABLE	PRESENT
								LL		PRESENT	PRESENT	PRESENT	PRESENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	-0.5	UNSTABLE	PRESENT
26	4445902	MAHADEVI MALLESHWAR JADHAV	38	F	ABSENT	ABSENT	NO	AREOLA	2	PRESENT	PRESENT	PRESENT	PRESENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	PRESENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
								LL	2	PRESENT	PRESENT	PRESENT	PRESENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	PRESENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
27	4445607	MALLAMA ARUN	38	F	ABSENT	ABSENT	HTN	RIGHT EAR	1	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
28	5700847	MALLAYYA BALAYYA C	54	M	ABSENT	ABSENT	NO	RIGHT FOOT	1	PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	ABSENT
29	5169062	MANUJNATH SHANKARAPPA	43	M	PRESENT	ABSENT	NO	TRUNK	2	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
										PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
								LL	4	PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	PRESENT	ABSENT	-1.5	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	PRESENT	ABSENT	-1.5	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	PRESENT	ABSENT	-1.5	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	PRESENT	ABSENT	-1.5	UNSTABLE	PRESENT
30	4856375	MEGHA MANJUNATH V	42	F	ABSENT	ABSENT	NO	UL	2	PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	ABSENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	ABSENT
								LL	2	PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	ABSENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	ABSENT
31	6054186	MALLAVVA ARUN LADI	43	F	ABSENT	ABSENT	HYPOTHYROIDSM	FACE	1	PRESENT	ABSENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	RETICULATE	ABSENT	ABSENT	PRESENT	ABSENT	-0.5	UNSTABLE	PRESENT
								UL	1	PRESENT	ABSENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REVERSE	ABSENT	ABSENT	PRESENT	ABSENT	-0.5	UNSTABLE	PRESENT
								TRUNK	4	PRESENT	ABSENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REVERSE	ABSENT	ABSENT	PRESENT	ABSENT	-0.5	UNSTABLE	PRESENT
										PRESENT	ABSENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REVERSE	ABSENT	ABSENT	PRESENT	ABSENT	-0.5	UNSTABLE	PRESENT
										PRESENT	ABSENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REVERSE	ABSENT	ABSENT	PRESENT	ABSENT	-0.5	UNSTABLE	PRESENT
										PRESENT	ABSENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REVERSE	ABSENT	ABSENT	PRESENT	ABSENT	-0.5	UNSTABLE	PRESENT
32	5700847	MALLAYA BALAYYA CHIKKAMATH	45	M	ABSENT	TRAUMATIC SCARS	NO	RIGHT TOE	1	PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
33	5169062	MANJUNATH SHANKARAPPA	43	M	ABSENT	ABSENT	NO	UL	1	PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	PRESENT	PRESENT	ABSENT	-0.5	UNSTABLE	PRESENT
								TRUNK	1	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	PRESENT	ABSENT	-1.5	UNSTABLE	PRESENT
								LL	1	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	PRESENT	ABSENT	-1.5	UNSTABLE	PRESENT
34	4856375	MEGHNA MANJUNATH VERNEKAR	42	F	ABSENT	ABSENT	NO	UL	1	PRESENT	ABSENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	2	STABLE	ABSENT
								LL	1	PRESENT	ABSENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	2	STABLE	ABSENT
35	6054187	MALLAMA LADI	43	F	ABSENT	MELASMA	NO	FACE	1	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
								UL	1	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
								TRUNK	1	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
								BACK	1	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
36	6026431	NALANI KASHINATH	38	F	ABSENT	MELASMA	NO	TRUNK	3	PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	SHARP	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	3	STABLE	ABSENT
										PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	SHARP	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	3	STABLE	ABSENT
										PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	SHARP	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	3	STABLE	ABSENT
37	6075276	NILESH K JADHAV	31	M	ABSENT	PMLE	NO	LL	2	PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	1	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	2	STABLE	ABSENT
39	5943114	SUSHILA APPASAHEB.	70	F	ABSENT	XEROSIS	NO	UL	2	PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	2	STABLE	PRESENT
										PRESENT	ABSENT	PRESENT	ABSENT	UNSTABLE	SHARP	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	2	STABLE	ABSENT
								LL	5	PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	2	STABLE	PRESENT
										PRESENT	ABSENT	PRESENT	ABSENT	UNSTABLE	SHARP	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	2	STABLE	ABSENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	2	STABLE	PRESENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	2	STABLE	PRESENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	2	STABLE	PRESENT
40	5943114	NEETHA KISHORE	45	F	ABSENT	NO	NO	FACE	1	PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
								UL	2	PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
										PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
								TRUNK	2	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
										PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
								LL	1	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
41	5943115	NEETHA K	46	F	ABSENT	NO	NO	FACE	1	PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
								UL	1	PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
								TRUNK	2	PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
42	6075276	NILESH K JADHAV	31	M	ABSENT	NO	NO	LL	3	PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	SHARP	ABSENT	PRESENT	ABSENT	ABSENT	PRESENT	1	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	SHARP	ABSENT	PRESENT	ABSENT	ABSENT	PRESENT	1	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	SHARP	ABSENT	PRESENT	ABSENT	ABSENT	PRESENT	1	UNSTABLE	PRESENT
43	6075278	NIKILESH JD	32	M	ABSENT	NO	NO	LL	3	PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	SHARP	ABSENT	PRESENT	ABSENT	ABSENT	PRESENT	1	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	SHARP	ABSENT	PRESENT	ABSENT	ABSENT	PRESENT	1	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	SHARP	ABSENT	PRESENT	ABSENT	ABSENT	PRESENT	1	UNSTABLE	PRESENT
44	5902250	OMKAR SAMBAJI MANE	18	M	ABSENT	NO	NO	LL	2	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	SHARP	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
										PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	SHARP	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT

45	6464945	PRITHAM PRAKASH	6	M	ABSENT	NO	NO	FACE	2	PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
46	6427292	PREMA JAGADISH	52	F	ABSENT	NO	NO	H&N	2	PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
								TRUNK	1	PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
47	5854844	PRIYANKA NARAYAN PATIL	28	F	PRESENT	NO	NO	TRUNK	2	PRESENT	PRESENT	ABSENT	PRESENT	UNSTABLE	ILLDEFINED	REVERSE	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
										PRESENT	PRESENT	ABSENT	PRESENT	UNSTABLE	ILLDEFINED	REVERSE	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
								LL	3	PRESENT	PRESENT	ABSENT	PRESENT	UNSTABLE	ILLDEFINED	REVERSE	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
										PRESENT	PRESENT	ABSENT	PRESENT	UNSTABLE	ILLDEFINED	REVERSE	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
										PRESENT	PRESENT	ABSENT	PRESENT	UNSTABLE	ILLDEFINED	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	2	STABLE	ABSENT
48	5902494	PUSHPA VIRUPAKSHI	32	F	PRESENT	NO	HYPOTHYROIDSM	UL	1	PRESENT	PRESENT	PRESENT	PRESENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	-1	UNSTABLE	PRESENT
								LL	2	PRESENT	PRESENT	PRESENT	PRESENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	PRESENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
49	514039	SAVITA RAMACHANDRA NAGRAL	39	F	ABSENT	NO	HYPOTHYROIDISM	UL	2	PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
50	5912363	RAJVEER SAGAR GAIKWAD	54	M	ABSENT	NO	NO	UL	1	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
								TRUNK	2	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
										PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
51	5983634	RAMESH PARAPPA	39	M	ABSENT	NO	NO	FACE	2	PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	PRESENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	PRESENT
								UL	2	PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	ABSENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	ABSENT
								TRUNK	4	PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	ABSENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	ABSENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	REDUCED	PRESENT	PRESENT	ABSENT	ABSENT	2	STABLE	PRESENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	ABSENT
52	5983634	RAMESH	40	M	ABSENT	NO	NO	LL	1	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	REVERSE	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
53	5983635	RAMESH ANONY	41	M	ABSENT	NO	NO	LL	1	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	REVERSE	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
54	5846039	RAMESH BALWANTH KULKARNI	45	M	ABSENT	NO	NO	LL	1	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	REVERSE	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
55	5863815	RAMANAIK BASANAIK MURED	60	M	ABSENT	NO	HYPOTHYROID	UL	2	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
										PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
								TRUNK	2	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
										PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
								LL	2	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
56	5663815	BASANAIK MURAD	60	M	ABSENT	NO	HTN	NECK	2	PRESENT	ABSENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REVERSE	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
										PRESENT	ABSENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REVERSE	ABSENT	ABSENT	ABSENT	PRESENT	-1	UNSTABLE	PRESENT
57	5896083	RASHMI M VEERESH	18	F	ABSENT	ACNE	SPONDYLITIS	SCALP	1	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
58	6032542	RESHMA SANJAY NAIK	42	F	ABSENT	PHOTOSENSITIVITY	NO	FACE	1	PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
								UL	2	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	REVERSE	ABSENT	ABSENT	PRESENT	ABSENT	-1.5	UNSTABLE	PRESENT
										PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	REVERSE	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
								TRUNK	2	PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REVERSE	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
								LL	2	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	REVERSE	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
										PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	REVERSE	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
59	6323036	rutuja ram desai	27	F	ABSENT	PHOTOSENSITIVITY	NO	FACE	2	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
										PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
								TRUNK	2	PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	3	STABLE	PRESENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	3	STABLE	PRESENT
								UL	2	PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	3	STABLE	PRESENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	3	STABLE	PRESENT
								LL	2	PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	3	STABLE	PRESENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	3	STABLE	PRESENT
60	5620991	SADDAMHUSAIN NOORAHMED QAZI	28	M	ABSENT	NO	NO	FACE	2	PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	ABSENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	ABSENT
								LL	2	PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	PRESENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	PRESENT
61	5620092	NOORAHMED	6	M	ABSENT	NO	NO	FACE	2	PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	PRESENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	PRESENT
62	6422121	SAGAR BHIMA BADIGAR	23	M	ABSENT	NO	NO	UL	2	PRESENT	PRESENT	PRESENT	PRESENT	UNSTABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	ABSENT
										PRESENT	PRESENT	PRESENT	PRESENT	UNSTABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	ABSENT
								LL	2	PRESENT	PRESENT	PRESENT	PRESENT	UNSTABLE	SHARP	REDUCED	PRESENT	ABSENT	ABSENT	ABSENT	2	STABLE	ABSENT
										PRESENT	PRESENT	PRESENT	PRESENT	UNSTABLE	SHARP	REDUCED	PRESENT	ABSENT	ABSENT	ABSENT	2	STABLE	ABSENT
63	6544123	SAMEENA BEGUM	28	F	PRESENT	NO	HYPOTHYROIDISM	TRUNK	3	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	SHARP	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	3	STABLE	ABSENT
										PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	SHARP	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	3	STABLE	ABSENT
										PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	SHARP	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	3	STABLE	ABSENT
								UL	2	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
										PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
								LL	3	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	SHARP	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	3	STABLE	ABSENT
										PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	SHARP	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	3	STABLE	ABSENT
										PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	SHARP	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	3	STABLE	ABSENT
64	3430526	SANGEETA SATYANARAYANA VALMIKI	8	F	PRESENT	NO	NO	LL	2	PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	REVERSE	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	ABSENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	REVERSE	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	ABSENT
65	5951994	SANJANA NARAYAN BHAGVAN	12	F	ABSENT	NO	NO	FACE	2	PRESENT	ABSENT	PRESENT	ABSENT	UNSTABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	ABSENT
										PRESENT	ABSENT	PRESENT	ABSENT	UNSTABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	ABSENT
								TRUNK	2	PRESENT	ABSENT	PRESENT	ABSENT	UNSTABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	ABSENT
										PRESENT	ABSENT	PRESENT	ABSENT	UNSTABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	ABSENT
								LL	1	PRESENT	ABSENT	PRESENT	ABSENT	UNSTABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	ABSENT
66	5951999	SANGEETA BOGAN	13	F	ABSENT	NO	NO	FACE	2	PRESENT	ABSENT	PRESENT	ABSENT	UNSTABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	ABSENT
										PRESENT	ABSENT	PRESENT	ABSENT	UNSTABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	ABSENT
								TRUNK	2	PRESENT	ABSENT	PRESENT	ABSENT	UNSTABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	ABSENT
										PRESENT	ABSENT	PRESENT	ABSENT	UNSTABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	ABSENT
67	5948744	SANTOSH TAYAPPA KUMBAR	33	M	ABSENT	ACANTHOSIS	NO	FACE	1	PRESENT	PRESENT	ABSENT	PRESENT	UNSTABLE	ILLDEFINED	REDUCED	PRESENT	ABSENT	PRESENT	ABSENT	-0.5	UNSTABLE	PRESENT
								UL	1	PRESENT	PRESENT	ABSENT	PRESENT	UNSTABLE	SHARP	REDUCED	PRESENT	ABSENT	PRESENT	ABSENT	-0.5	UNSTABLE	PRESENT
								LL	2	PRESENT	PRESENT	ABSENT	PRESENT	UNSTABLE	SHARP	ABSENT	PRESENT	ABSENT	PRESENT	ABSENT	-0.5	UNSTABLE	PRESENT

										PRESENT	PRESENT	ABSENT	PRESENT	UNSTABLE	SHARP	ABSENT	PRESENT	ABSENT	PRESENT	ABSENT	-0.5	UNSTABLE	PRESENT
68	3903849	SAVITA CHANNABASAYYA HIREMATH	36	F	ABSENT	NO	NO	UL	4	PRESENT	PRESENT	PRESENT	PRESENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	PRESENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	PRESENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	PRESENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
								TRUNK	2	PRESENT	PRESENT	PRESENT	PRESENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	PRESENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
								LL	4	PRESENT	PRESENT	PRESENT	PRESENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	PRESENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	PRESENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	PRESENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
69	2995362	SHREYA SHIVAPPA SAIDAPUR	11	F	ABSENT	NO	NO	H&N	1	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
								LL	2	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
										PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
70	4314414	SHRIDEVI MARUTI KADAGOUDAR	16	F	ABSENT	NO	NO	FACE	1	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
								NECK	1	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
								UL	1	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
								TRUNK	2	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
										PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
								LL	4	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	PRESENT	PRESENT	ABSENT	ABSENT	3	STABLE	ABSENT
										PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	PRESENT	PRESENT	ABSENT	ABSENT	3	STABLE	ABSENT
										PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	PRESENT	ABSENT	PRESENT	ABSENT	-0.5	UNSTABLE	PRESENT
										PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	PRESENT	ABSENT	PRESENT	ABSENT	-0.5	UNSTABLE	PRESENT
71	6383558	SHRUTI PRAKASH MASTONMARDI	25	F	ABSENT	NO	NO	H&N	1	PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	REVERSE	PRESENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	ABSENT
								TRUNK	1	PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	REVERSE	PRESENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	ABSENT
72	5943114	SUSHILA APPASAHEB	70	F	ABSENT	NO	NO	UL	2	PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	PRESENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	PRESENT
								LL	1	PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	PRESENT
73	5943112	SANDYA	43	F	ABSENT	NO	NO	TRUNK	2	PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	2	STABLE	ABSENT
										PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	2	STABLE	ABSENT
								LL	2	PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	2	STABLE	ABSENT
										PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	2	STABLE	ABSENT
74	5943123	SIDHALINGAPPA	45	F	ABSENT	NO	NO	TRUNK	2	PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	2	STABLE	PRESENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	2	STABLE	PRESENT
75	543261	SRINIVAS SOMNATH	43	F	ABSENT	NO	NO	TRUNK	3	PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	3	STABLE	PRESENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	3	STABLE	PRESENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	3	STABLE	PRESENT
76	6136467	SUJATHA PRAKASH	49	F	PRESENT	NO	NO	FACE	1	PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
								TRUNK	3	PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
								LL	2	PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REVERSE	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
77	6408579	SUMAN TUKARAM	59	F	ABSENT	ACTINIC KEARTOSIS	NO	TRUNK	2	PRESENT	PRESENT	PRESENT	PRESENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	PRESENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
								UL	2	PRESENT	PRESENT	PRESENT	PRESENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	PRESENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
78	3616306	SUMITRA GURUNATH	24	F	ABSENT	DPN	NO	LL	2	PRESENT	PRESENT	PRESENT	PRESENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	PRESENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
79	3460979	SUVARNA RAMACHANDRA	38	F	PRESENT	ABSENT	NO	TRUNK	3	PRESENT	PRESENT	ABSENT	PRESENT	UNSTABLE	ILLDEFINED	RETICULATE	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
										PRESENT	PRESENT	ABSENT	PRESENT	UNSTABLE	ILLDEFINED	RETICULATE	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
										PRESENT	PRESENT	ABSENT	PRESENT	UNSTABLE	ILLDEFINED	RETICULATE	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
								UL	2	PRESENT	PRESENT	ABSENT	PRESENT	UNSTABLE	ILLDEFINED	RETICULATE	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
										PRESENT	PRESENT	ABSENT	PRESENT	UNSTABLE	ILLDEFINED	RETICULATE	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
								LL	3	PRESENT	PRESENT	ABSENT	PRESENT	UNSTABLE	ILLDEFINED	RETICULATE	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
										PRESENT	PRESENT	ABSENT	PRESENT	UNSTABLE	ILLDEFINED	RETICULATE	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
										PRESENT	PRESENT	ABSENT	PRESENT	UNSTABLE	ILLDEFINED	RETICULATE	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
80	6197624	TUKAPPA	23	M	ABSENT	ABSENT	RENAL STONES	LL	3	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	ABSENT
										PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	ABSENT
										PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	ABSENT
81	6197543	SAVAKKA	67	F	ABSENT	NO	NO	TRUNK	3	PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	PRESENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	PRESENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	PRESENT
								LL	4	PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	PRESENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	PRESENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	PRESENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	PRESENT
82	654378	SATYA PATIL	48	M	ABSENT	NO	NO	LL	2	PRESENT	ABSENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
										PRESENT	ABSENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
83	6547834	SWATHI KALLOLI	37	F	ABSENT	NO	NO	TRUNK	3	PRESENT	ABSENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
										PRESENT	ABSENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
										PRESENT	ABSENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
84	544673	SATHVIKA	12	F	ABSENT	NO	NO	UL	1	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
85	3518182	USHA CHANNAPPA	55	F	ABSENT	NO	NO	NECK	2	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	ABSENT
										PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	ABSENT
								LL	2	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	ABSENT
										PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	SHARP	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
86	321454	USHA KULKARNI	34	F	ABSENT	NO	NO	LL	2	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	SHARP	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	SHARP	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
87	6278145	VAISHNAVI SHARANAPPA	7	F	ABSENT	NO	NO	TRUNK	4	PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
88	5579291	veeksha ganapati bhutappagal	12	F	ABSENT	NO	NO	NECK	2	PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	ABSENT
										PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	ABSENT

89	6254009	VIRENDRA GIRISH WALI	18	M	ABSENT	NO	NO	UL	2	PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
								LL	3	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
										PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
										PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
90	338684	VEERESH SHIVAPURA V	14	M	ABSENT	NO	NO	FACE	2	PRESENT	ABSENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
										PRESENT	ABSENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
91	5929156	VIJAY TUKARAM GAWADE	42	M	ABSENT	NO	NO	TRUNK	3	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	REVERSE	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
										PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	REVERSE	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
										PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	REVERSE	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
92	5958479	YALLAPA MUKUND GAWADA	49	M	ABSENT	NO	NO	TRUNK	2	PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	ABSENT
										PRESENT	ABSENT	ABSENT	ABSENT	STABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	ABSENT
93	2900857	JAYAWANT LALAPPA	73	M	PRESENT	SEBORRHEIC KERATOSIS	CAD	UL	1	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
								LL	2	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
94	5665705	GOUSABI ABDULATIF DESAI	38	F	ABSENT	ACANTHOSIS	DM	NECK	1	PRESENT	ABSENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
95	4160373	PRABHAVATHI KARABASAPPA	40	F	PRESENT	DPN	HYPOTHYROIDISM	UL	2	PRESENT	ABSENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
										PRESENT	ABSENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
								TRUNK	1	PRESENT	ABSENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT
96	6572039	HARISH MAHADEV MUGAGI	9	M	ABSENT	NO	NO	FACE	2	PRESENT	ABSENT	ABSENT	ABSENT	UNSTABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	ABSENT
										PRESENT	ABSENT	ABSENT	ABSENT	UNSTABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	ABSENT
								UL	2	PRESENT	ABSENT	ABSENT	ABSENT	UNSTABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	ABSENT
										PRESENT	ABSENT	ABSENT	ABSENT	UNSTABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	ABSENT
								LL	2	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
										PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
97	6501871	AJIT ARUNAGOUDA PATIL	73	M	ABSENT	NO	DM	FACE	1	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	ABSENT
								UL	1	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	SHARP	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	2	STABLE	ABSENT
								LL	1	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	SHARP	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	3	STABLE	ABSENT
98	6473780	AMRUTA BHIMAPPA REDDY	6	F	PRESENT	NO	NO	FACE	1	PRESENT	PRESENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	2	STABLE	ABSENT
99	6191952	KAVERI H	11	F	ABSENT	NO	NO	UL	2	PRESENT	ABSENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
										PRESENT	ABSENT	ABSENT	ABSENT	UNSTABLE	ILLDEFINED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	1	UNSTABLE	PRESENT
100	6124172	PUSHPA K	32	F	ABSENT	NO	NO	TRUNK	1	PRESENT	PRESENT	PRESENT	ABSENT	UNSTABLE	ILLDEFINED	REDUCED	ABSENT	ABSENT	ABSENT	ABSENT	0	UNSTABLE	PRESENT