"CLINICO EPIDEMIOLOGICAL AND MYCOLOGICAL STUDY OF PITYRIASIS VERSICOLOR"

Dissertation Submitted in

Partial fulfillment of the University regulations for

MD DEGREE IN

DERMATOLOGY, VENEREOLOGY AND LEPROSY

(BRANCH XX)

MADRAS MEDICAL COLLEGE

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

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CERTIFICATE

Certified that this dissertation title **"CLINICO EPIDEMIOLOGICAL AND MYCOLOGICAL STUDY OF PITYRIASIS VERSICOLOR"** is a bonafide work done by **Dr. R.SNEKAVALLI** Post graduate student of the Department of Dermatology, Venereology and Leprosy, Madras Medical College, Chennai – 3, during the academic year 2013 – 2016. This work has not previously formed the basis for the award of any degree.

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DECLARATION

The dissertation entitled **"CLINICO EPIDEMIOLOGICAL AND MYCOLOGICAL STUDY OF PITYRIASIS VERSICOLOR"** is a bonafide work done by **Dr. R. Snekavalli** at Department of Dermatology, Venereology and Leprosy, Madras Medical College, Chennai – 3, during the academic year 2013 – 2016 under the guidance of **Prof.Dr.A.RAMESH M.D.,DD., DNB.,** Professor, Department of Dermatology, Madras Medical College, Chennai -3. This dissertation is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai towards partial fulfillment of the rules and regulations for the award of M.D Degree in Dermatology, Venereology and Leprosy (BRANCH – XX)

Prof.Dr.A.RAMESH, M.D.,DD.,DNB.,
Professor,
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Chennai-03

DECLARATION

I, **Dr.R.SNEKAVALLI** solemnly declare that this dissertation titled **"CLINICO EPIDEMIOLOGICAL AND MYCOLOGICAL STUDY OF PITYRIASIS VERSICOLOR"** is a bonafide work done by me at Madras Medical College during 2013-2016 under the guidance and supervision of **Prof. K.MANOHARAN, M.D., D.D.,** Professor and head department of Dermatology, Madras Medical College,Chennai-600003.

This dissertation is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai towards partial fulfillment of the rules and regulations for the award of M.D Degree in Dermatology, Venereology and Leprology (BRANCH – XX).

PLACE :

DATE : **(DR. R.SNEKAVALLI)**

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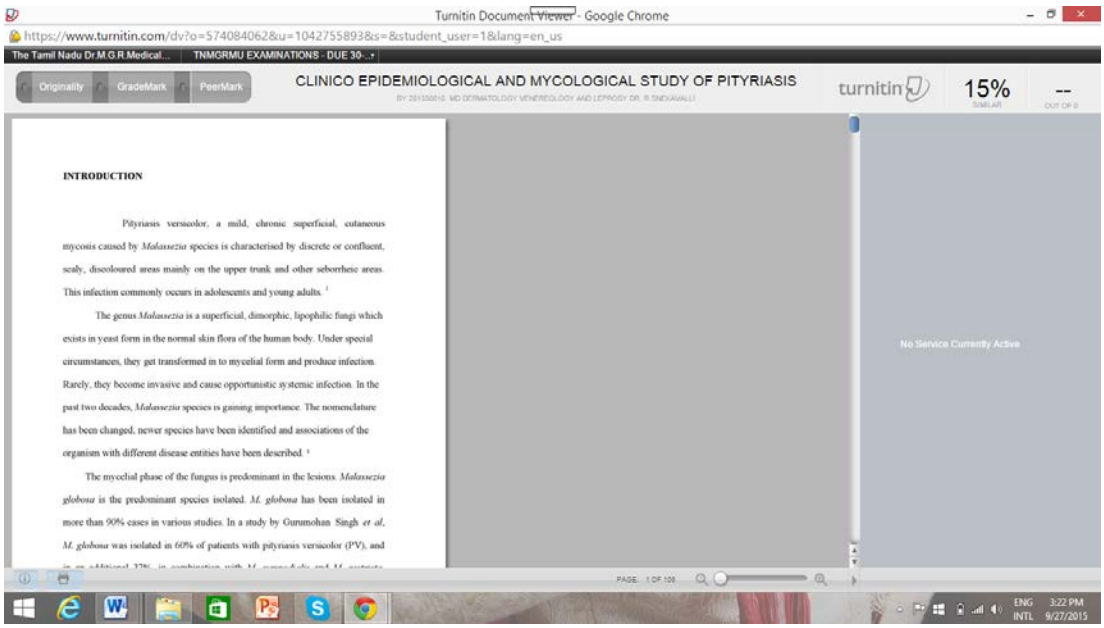
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INTRODUCTION
Pityriasis versicolor, a mild, chronic superficial, cutaneous mycosis caused by Malassezia species is characterised by discrete or confluent, scaly, discoloured areas mainly on the upper trunk and other seborrheic areas. This infection commonly occurs in adolescents and young adults. [1]
The genus Malassezia is a superficial, dimorphic, lipophilic fungi which exists in yeast form in the normal skin flora of the human body. Under special circumstances, they get transformed in to mycelial form and produce infection. Rarely, they become invasive and cause opportunistic systemic infection. In the past two decades, Malassezia species is gaining importance. The nomenclature has been changed, newer species have been identified and associations of the organism with different disease entities have been described. [1]
The mycelial phase of the fungus is predominant in the lesions. Malassezia globosa is the predominant species isolated. M. globosa has been isolated in more than 90% cases in various studies. In a study by Gurumohan Singh et al, M. globosa was isolated in 60% of patients with pityriasis versicolor (PV), and
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ABSTRACT

INDRODUCTION

Pityriasis versicolor, a mild, chronic superficial, cutaneous mycosis caused by *Malassezia* species is characterised by discrete or confluent, scaly, discoloured areas mainly on the upper trunk and other seborrheic areas. This infection commonly occurs in adolescents and young adults.

AIMS AND OBJECTIVES

- To study the epidemiology of pityriasis versicolor.
- To study the various clinical types.
- To study the association of pityriasis versicolor with other dermatological disorders and systemic diseases
- To identify the various species of *Malassezia*
- To study the clinicomycological correlation.

MATERIALS AND METHODS

100 patients with pityriasis versicolor were randomly selected. Detailed history was taken and clinical examination was done. Scraping was done for all the patients KOH positive scrapings were subjected to culture in SDA with olive oil overlay. Tween assimilation was done from positive cultures and species isolation done. Gram staining was done from positive cultures.

OBSEVATION AND RESULTS

Pityriasis versicolor is most commonly seen in the age group between 21-30years.Males outnumbered the females. PV was more common among the

students.Urban population was affected more than the rural population.Cosmetic concern was the main complaint. Duration of lesion in majority of the patients was 1 to 6 months. Diabetes and Seborrheic dermatitis was found to be the most commonly associated. Blood group A was commonly affected. Most common site was back and majority of the patients had more than 30%BSA.Achromicpityriasis versicolor was more common.*Malasseziaglobosa* was the most common species isolated.

CONCLUSION

There is no significant change in clinico-epidemiology and presentation of pityriasis versicolor.A blood group was the most common blood group associated with pityriasis versicolor which is different from the previous study in which O blood group is the commonest association. Hence, further studies in a larger population would provide more conclusive evidence with regard to the blood groups.

INTRODUCTION

Pityriasis versicolor, a mild, chronic superficial, cutaneous mycosis caused by *Malassezia* species is characterised by discrete or confluent, scaly, discoloured areas mainly on the upper trunk and other seborrheic areas. This infection commonly occurs in adolescents and young adults. [1]

The genus *Malassezia* is a superficial, dimorphic, lipophilic fungi which exists in yeast form in the normal skin flora of the human body. Under special circumstances, they get transformed in to mycelial form and produce infection. Rarely, they become invasive and cause opportunistic systemic infection. In the past two decades, *Malassezia* species is gaining importance. The nomenclature has been changed, newer species have been identified and associations of the organism with different disease entities have been described. [2]

The mycelial phase of the fungus is predominant in the lesions. *Malassezia globosa* is the predominant species isolated. *M. globosa* has been isolated in more than 90% cases in various studies. In a study by Gurumohan Singh *et al*, *M. globosa* was isolated in 60% of patients with pityriasis versicolor (PV), and in an additional 37%, in combination with *M. sympodialis* and *M. restricta*. More than one species of *Malassezia* can be isolated from a single clinical specimen.[3]

The chronic relapsing course of the disease and increased incidence of PV among patients on steroids and immunosuppressed patients are suggestive of failure on the host mechanism to mount a protective CMI response against the fungus. However, during active disease, they fail to generate a CMI response that would provide protection.[3]

The predisposing factors are warm climate, increased sweating, pregnancy, oral contraceptive pills, endocrine disorders and immunosuppressive states.[1]

The net outcome of the disease is determined by fungus – host relationship. This is particularly significant in the present era of advanced medical facilities, where the number of immunocompromised hosts is on the rise. [4]

The recognition of the significance of *Malassezia* has not been appreciated previously due to the difficulty in the isolation of these lipophilic yeasts in the routine laboratory setting. In the recent years, research on this genus has been expedited by the introduction of molecular methods, which have contributed to the understanding of its epidemiology, pathogenesis and management by robust interventional strategies, given that these yeasts are not only involved in superficial infections, but

also disseminated infections and that different species exhibit varying sensitivity patterns to the antifungal agents. To date, there are 14 species of *Malassezia* that have been isolated from human and animal skin.[5,6]

Pityriasis versicolor and seborrheic dermatitis are the two most common manifestations of *Malassezia,* though not alarming infections, known for their chronicity and recurrence and also for raising cosmetic concerns in those affected by these conditions.[6]

This part of our country is known for its hot and humid climate that favor the growth of *Malassezia* yeasts, and hence an understanding of the distribution of these yeasts and the dominant species prevalent, will serve to complement the previous studies conducted in India and elsewhere in determining the varying trends in distribution that may exist in different geographical locations.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

HISTORY

In 1846, Eichstedt and in 1847, Sluyter identified *Malassezia* as the causative agent of pityriasis versicolor though they were unable to isolate the organism. As the organism closely resembled *Microsporum audounii*, it was named as *Microsporum furfur* by Robin in 1953. In 1874, the French scientist, Louis Charles Malassez described budding 'spores' in the patients with seborrhoeic dermatitis.[7,8]

Bizzozero named the oval and spherical budding cells as *Saccharomyces ovalis*, and *Saccharomyces sphaericus* respectively. In 1889, in honour of Malassez, Baillon used the term *Malassezia furfur*.[9]

In 1904, Sabouraud described the genus *Pityrosporum* which had the budding yeast cells without hyphal elements. In 1913, Chalmers and Castellani isolated the oval lipophilic budding cells from normal skin and patients with seborrhoeic dermatitis and gave the name *Pityrosporum ovale*.[9]

Previously, the mycelial form of this fungi was called *Malassezia* and the yeast form was called *Pityropsorum orbiculare* (round cells with budding from narrow neck) or *Pityrosporum ovale* (oval yeast cells with budding from wide neck). In 1969, Sternberg and Keddie by using the fluorescent antibody technique identified antigenic components of *Malassezia* furfur and *Pityrosporum orbiculare* and found that both of them had similar features. In 1984, Yarrow and Ahearn stated that *Malassezia* gained priority over *Pityrosporum* and was accepted as the generic name for the fungus. The lipophilic nature of the genus *Malassezia* was discovered in 1939 by Rhoda Benham. Von Abbe in 1964 described the Dixon medium for the isolation of *Malassezia* yeasts which was subsequently modified for better yield. On the basis of the distinct cell surface antigens, Cunningham and colleagues in 1990 defined the serovars A, B and C. These serovars are now identified as *M. sympodialis, M. globosa* and *M. restricta*, respectively. In 1996, following taxonomic revision of the genus, four new species, *M. globosa, M. obtusa, M. restricta and M. slooffiae* were included.[4,6,9]

TAXONOMY

Phylum Basidiomycota

Order Malasseziales

Class Blastomycetes

Family Cryptococcaceae

Genus *Malassezia*[3]

In 1889, Baillon first identified *Malassezia furfur*. It was called by various names such as *Microsporum furfur, Malassezia tropica, Pityrosporum orbiculare, Malassezia macfadyeni, Microsporum furfur, Sporotrichum furfur, Oidium furfur*.[3]

Based on morphology, physiology, cellular characteristics, temperature requirements, biochemical reactions, dependence on long chain fatty acids, guanine and cytosine content of cellular DNA and ribosomal RNA sequences, the genus *Malassezia* has been found to have fourteen species.[10]

In the year 1925, Dodge identified *M.pachydermatis*. In 1990, Simmons and Gueho isolated *M. sympodialis*. Midgley, Gueho and Guillot, in the year 1996, reported four species namely *M. globosa, M.restricta, M. slooffiae and M. obtusa.* In 2002, *M. dermatis* was identified by Sugita,

Takashima, Nishikawa and Shinoda. They also identified *M. japonica* and *M.yamatoensis* in the year 2003 and 2004 respectively. Hirai, Kano, Makimura, Yamaguchi, Hasegawa reported *M.nana* in 2004. In 2007, Cabanes and Boekhout identified *M. caprae* and *M. equi.* Recently in 2011, F. J. Cabanes reported *M. cuniculi.*[11,12,13]

ECOLOGY

Malassezia usually inhabit the skin of only warm-blooded animals. *Malassezia* species require lipids for growth and survival and they are often mesophilic. [12] Canine ear infections are caused by *M. pachydermatis* .Many lipophilic species of *Malassezi*a have been isolated from pigs, monkeys, rhinoceros, bears and birds.[14]

Crespo Erchiga *et al* concluded that *M. sympodialis was* the most common species isolated from normal human skin, especially the trunk.[15] Midgley proposed that *M. globosa* had a higher incidence.[16]

The lipophilic yeast abounds in the skin of people from tropical and subtropical climates. Individual species have specific host preferences. *M. sympodialis* is the most common species that occur in humans as normal flora or at sometimes association with systemic disease. *M. furfur, M. globosa and M. restricta* also infect humans and produce diseases. *M. furfur*

is seldom found as normal flora or in disease states. *M. globosa* and *M. restricta* are mainly pathogenic, associated with pityriasis versicolor, pityriasis capitis or seborrhoeic dermatitis. In humans, the peak age for normal carriage or disease by *Malassezia* is in the early twenties, when the sebaceous gland activity is maximum. Colonization or disease by Malassezia is rare at the extremes of ages. *M. pachydermatis* was first isolated from rhinoceros. It is mainly found in domestic animals, like dogs, causing otitis externa, but occasionally can infect humans.*M. slooffiae* was demonstrated as normal flora on the trunk. This species is also pathogenic in pigs.[15]

The distribution of *Malassezia* as normal flora is related to density of sebaceous gland, and thus the scalp, face, central chest and back bear the highest number of fungi. Other sites, like the hair follicles and the external ear, are also the sites of colonization. The hair shaft, nail and mucosae are not affected. In an epidemiological study conducted by Rao *et al*, 97% of the normal adult population showed scalp carriage and 92% showed trunk carriage for *Malassezia*.. However, in various studies, hospitalized infants have shown a positive skin culture for *M. furfur*, the incidence varying from 37% to 84%. Many healthy infants develop a cutaneous flora comprising *Malassezia* species within the first 6 months of life.[15,17]

PITYRIASIS VERSICOLOR:

SYNONYMS

Pityriasis versicolor (PV) is also referred to as Tinea versicolor, Chromophytosis, , Dermatomycosis furfuracea, Tinea flavea and Liver spots.[3]

INTRODUCTION

The term “Pityriasis versicolor’ is derived from the Greek word ‘ptyra’ which means abnormal proliferation and the latin word ‘ versicolor’ that means many colours.[3]

PV is a mild ,common, benign superficial fungal infection of stratum corneum which is characterized by discrete or confluent, hypopigmented or hyperpigmented brawny scaly macules or patches present over the seborrheic areas caused by genus *Malassezia.* It was earlier called as tinea which is a misnomer and reserved for dermatophytic infections.[1,16]

In Sri Lanka, PV was considered to be a marker of beauty and locally called as‘Gomora’meaning ‘tears of liquid gold’.[16]

EPIDEMIOLOGY

Age

PV is more common in adolescents and young adults due to the increased sebum production. The amount of yeast that colonizes the stratum corneum is proportional to variation in sebum secretion which is in turn age-dependent. The percentage of yeast that colonizes the skin in the age group between 0-15yrs is 5 -15% and in the age group between 11- 20yrs is 56 – 90%. It is uncommon in older age group because sebaceous glands become nonfunctional with old age.[3]

Race

The incidence of PV is same in all races and it is more common in dark skinned individuals.[15]

Sex

Males and females are equally affected. There is no sexual preponderance. [15]

Seasonal Variation

In tropical countries, due to the increased temperature and humidity, PV is more common during spring and summer.[15] Infection is likely to be extensive and persistent with an incidence of about 35 – 40% in temperate climates. It is less prevalent in colder climate with an incidence as low as 1 – 4 %.[18]

Genetic Factors

It has been postulated that in pityriasis versicolor there is a genetically determined host susceptible factor, since a positive family history was present among blood relations. Conjugal cases can occur (infection does not arise from patient's own autologous flora rather than by transmission from other individuals).[3,18]

Predisposing Factors

Many systemic conditions favour the development of pityriasis versicolor. Some of them are

1. Cushing syndrome
2. Pregnancy
3. Oral contraceptive pill
4. Diabetes mellites
5. Tuberculosis
6. Organ transplant patients
7. AIDS
8. Immunosuppressive drugs[16]

Other environmental conditions conditions such as hot climate, high humidity, poor personal hygiene and overcrowding also plays a role.[19] Other factors like winter season, UV irradiation, airborne irritants and hair cosmetics are known to aggravate the condition.[6]

Malassezia is usually universal normal flora of the body but under certain special situations it overgrows and readily transforms to mycelial forms which is slightly more pathogenic.[16]

PATHOGENESIS

Malassezia species is universally present as normal flora of the skin. Under certain special conditions, it becomes pathogenic to the human skin and produces the disease. Weis *et al*, by electron microscopic studies showed that there is a change in the saprophytic yeast form to filamentous mycelial form which determines the pathogenesis of *Malassezia* infections. The endogenous or exogenous corticosteroids may lead to the development of PV due to reduction in the turnover of squamous cells. *Malassezia* are dependent on exogenous lipids, especially 12 to 14 carbon fatty acids for their growth. Human and animal skin rich in free fatty acids, serve as media for their growth and survival. In human beings, Malassezial antigens interact with neutrophils and monocytes resulting in stimulation of IL-8 and IL-1α and IL-8 respectively. [4] Metabolism of skin surface lipids by lipoxygenase enzymes of *Malassezia*, yields fatty acids that are toxic to the melanocytes.[20]

Many theories have been postulated to explain the pigment alterations associated with PV:

1. Studies by Nazzaro – Porro in 1993 stated that *Malassezia* metabolises oleic and vaccenic acids and produces lipid fractions like C_9 to C_{11} dicarboxylic acids. Azelaic acid, a dicarboxylic acid produced as a metabolite competitively inhibits tyrosinase in the melanin synthesis pathway.[20]
2. There is inhibition of the mitochondrial enzymes that in turn leads to degeneration and vacuolation of the melanocytes leading to smaller and fewer melanosomes thus producing hypopigmentation.[21]
3. Mayer and coworkers did experiments on the metabolism and nutritional requirements of *Malassezia* species in 1998 and stated that *M. furfur* has the ability to assimilate L- tryptophan as a nitrogen source leading to the colour change of the affected skin in PV. The presence of abnormal structure of stage 1 melanosomes which are not transferred to epidermal keratinocytes is responsible for hypopigmentation in PV cases.[21]
4. In 2000, Raabe *et al* studied the pigmentogenesis of *M. furfur* and stated that the indole pigment , pityriacitrin produced exclusively

by *M. furfur* acts as a UV filter, thereby preventing normal tanning.[11]

5. Weis *et al* confirmed the penetration of *Malassezia* into the keratinocytes, causing degradation of the keratin into an amorphous lipid–like substance, that acts as a potential ultraviolet light blocker which results in hypopigmentation.[10]
6. There is an increased space between melanocytes and keratinocytes.[8]
7. Hyperpigmentation in chromic type of PV is said to be multifactorial, namely presence of large number of the causative fungi, increased thickness of the keratin layer, increased turnover rate of cells in the stratum corneum, increased perivascular infiltration of lymphocytes and increased inflammatory responses that stimulate the melanocytes. Hyperpigmented lesions have been found to contain more hyphae and spores than the hypopigmented lesions.[21]
8. In studies by Sugita *et al,* hyperpigmented lesions were found to show an increased turnover rate of stratum corneum, i.e, 8 days compared to 15 days for normal areas. They also observed the presence of singly distributed, large melanosomes in contrast to multiple – packaged, smaller melanosomes in normal areas.[12]

METABOLITES OF *Malassezia furfur*[2]

Azelaic acid: a dicarboxylic acid that inhibits tyrosinase.

Malassezin: it is an aryl hydrocarbon receptor agonist that induces apoptosis in melanocytes.

Pityriacitrin: it is a yellow compound that helps in absorbtion of UV light.

Pityrialactone: an indole alkaloid (tryptophan derivative) that fluoresces under 366 nm UV light.

Pityriarubins: a red indole alkaloids that inhibit the neutrophil respiratory burst in vitro in a dose dependent manner and inhibit 5-lipoxygenase activity.[2]

Malassezia plays a definite role	Possible role of malassezia
• Pityriasis versicolor • Systemic intravenous catheter induced fungemia with or without embolism • Endocarditis • Interstitial pneumonia • Peritonitis in patients undergoing chronic	• Dandruff • Folliculitis • Neonatal cephalic pustulosis • Confluent and reticulate papillomatosis • Atopic dermatitis • Seborrheic dermatitis • Guttate psoriasis

ambulatory peritoneal dialysis	• Blepharitis • Balanitis • Onychomycosis • Lacrimal canaliculitis and dacryolith Sinusitis • Nipple discharge • Otitis externa[12]

CLINICAL FEATURES:

The clinical lesions of pityriasis versicolor have well defined borders with a clear cut edge and occurs in various shapes. The colour of the lesion may vary according to the interaction between the host and *Malassezia*. Clinical type and distribution depends on the skin colour of the patient, extent of colonization and degree of exposure of colonized areas to sunlight. Individuals may have either hypopigmented (Achromic pityriasis versicolor) or hyperpigmented lesions (Chromic versicolor) or both together.[1]

The morphology of the lesion may be macules, patches, follicular, perifollicular or papular. Atrophic form of PV has been reported by Kyle et al in 2014. They stated that Malassezia produce diffusible products that activate tissue histiocytes and result in dermal elastolysis.[1,22]

In children, paranasal distribution of PV is characteristic. The primary lesion is a sharply demarcated macule with characteristic fine, branny scaling

The macules may coalesce to form large confluent areas with scattered oval patches and outlying macules. They are usually asymptomatic but some of them may have mild itching or burning sensation. Patients mostly come to the hospital due to the cosmetic concern and fear of social stigma attached to hypopigmented lesions.[1]

DISTRIBUTION OF THE LESION

As the fungus is lipophilic, it occurs mostly on the seborrheic areas such as upper chest, neck, face, back, upper arm and abdomen. In the tropics, facial, palmar and scalp lesions are common. Sometimes pityriasis versicolor can occur in entirely different distribution affecting the flexural regions such as axillae, groin, thigh and genitalia when it is known as inverse pityriasis versicolor. Sometimes pityriasis rosea like lesions can occur. [2,15]

COUP D'ONGLE SIGN OF BESNIER

The one significant sign associated with the diagnosis of tinea versicolor is that of the *coup d' ongle* of Besnier (1831-1909), later named by

Balzer (1849-1929) as *le signe du copeau* (shaving, as of wood) or in German "*Hobelspanphänomen"*. It is also called as "scratch sign".[16]

The description is as follows:

"Sometimes the spots are smooth, sometimes powdery and manifestly branny, but this distinguishing mark is not evident in all of the stages of evolution of the parasite, and its diagnostic value is secondary; what is constant is the alteration in the consistency of the superficial horny layer of the epidermis, which, infiltrated with the *Microsporon (Malassezia*) is easily crumpled and detached either by the stroke of the curette, or more practically by the scrape of the fingernail. A scratch of the nail made a little vigorously, even without reaching the summit of the papillae and without causing the least degree of bleeding, easily produces the desquamative lamella which is almost pathognomic".[16]

ZIRELI'S SIGN

Clinical manifestations of pityriasis versicolor are characterized by multiple macular lesions, perifollicular at first, with light scaling. Stretching of the affected skin may help in better visualization of the scaling of lesions. This is known as Zireli's sign. [23]

UNUSUAL PRESENTATIONS OF MALASSEZIA:

PITYROSPORUM FOLLICULITIS(RAUSCH AND JACABS 1984)[1]

It is a delayed type of hypersensitivity to *Malassezia* species. Overgrowth of the yeast causes blockage of follicular ostia. It is proposed that hydrolysis of triglycerides, fatty acid synthesis and activation of the alternate complement pathway by the fungus induce inflammation, resulting in folliculitis. Pityriosporum folliculitis (PF) manifests as itchy perifollicular erythematous papules or pustules over back, chest and extremities and mimics steroid induced acne. Presence of itching helps to differentiate these two conditions. Most often PF is associated with SD. Histopathologically, there is a perifollicular mononuclear cell infiltrate around the infundibulum. In skin lesions, there is an increased number of T helper cells and Langerhans cells. *Malassezia* yeasts are seen, but mycelial forms are usually absent in contrast to PV. Topically, selenium sulphide wash may be useful. Systemic treatment includes tab. ketoconazole 200mg daily for four weeks or tab. fluconazole 150 mg once weekly for 2-4 weeks or itraconazole 200mg once a day for 2 weeks. [1]

CONFLUENT RETICULATE PAPILLOMATOSIS [1,24]

This rare disease of keratinization manifests as grey brown, pigmented confluent papules in the interscapular area, neck, abdomen and under the breast. Neighboring papules become confluent in the centre of the affected areas forming an irregular network. It was initially believed to be a genodermatosis but studies have demonstrated *Malassezia furfur* in the scales. Natarajan *et al* identified a novel Actinomycete, *"Dietzia papillomatosis"* as an etiological agent in CRP, but this finding is yet to be confirmed. Clinical response to antifungal agents is variable Topical vitamin D analogues and retinoids can be tried. Oral azithromycin and minocycline have also been used.[1]Electron microscopic features includes a marked alteration of the cornified cell structure resembling snake coil like or triangle like stacks and increase in the number of lamellar granules.[1]

SEBORRHOEIC DERMATITIS

'Sebum' means 'grease' and 'rhoea' means 'flow'. Seborrhoeic dermatitis is a chronic papulosquamous disorder seen predominantly in young adults, that manifests as erythematous, pruritic scaly lesions in the scalp and face, particularly, forehead, external auditory canal, nasolabial folds, retroauricular areas, eyebrows and moustache and also the presternal

area. The incidence is about 3 – 5% of the general population. Seborrhoeic dermatitis is the earliest clinical marker of HIV. In Acquired Immuno Deficiency Syndrome (AIDS) patients with low CD4 counts, seborrheic dermatitis is often severe and difficult to treat.

Therapeutic studies have confirmed the causative role of *Malassezia* in seborrheic dermatitis due to the response to ketoconazole .

The patients with SD have high skin surface levels of squalenes and free fatty acids (FFA). *Malassezia* species possess lipase activity, that converts triglycerides to free fatty acids. The FFA and the reactive oxygen species (ROS) which are formed have the antibacterial activity that alters the normal skin flora. This disturbance in flora, lipase activity and ROS may be linked to the pathogenesis of SD.[9,13]

Malassezia species produces increased amount of aryl hydrocarbon receptor(AhR) ligands and an interplay of the AhR and EGFR (Epidermal Growth Factor Receptor) has also been proposed to play an important role in SD.[25]

DANDRUFF (PITYRIASIS CAPITIS OR PITYRIASIS SIMPLEX)

It is a subclinical, inflammatory scalp disorder, episodic, recurrent or constant, that results in disruption of cohesion between corneocytes, visible as

scales. The disorder is most prevalent and severe among adolescents and young adults, and rare among children and the elderly. Environmental factors like winter season, UV irradiation, airborne irritants and hair cosmetics are known to aggravate the condition. Malassez (1874) had isolated the yeast forms of the species from dandruff scales. It has been hypothesized that a critical quantity of the yeasts are required for the clinical manifestation of dandruff and when it exceeds this, features of seborrheic dermatitis are seen. The oval yeast form of *M. restricta* has been isolated from dandruff scales. Toxin production and lipase activities of the yeast inducing a proinflammatory state and stimulating host immune response may be operative in the pathogenesis. Environmental factors have some additive effect on the pathogenicity of the fungus. There is a definite response to topical antifungals when *Malassezia* is associated with dandruff.[26]

FUNGEMIA

It is commonly seen in premature infants who are on total parenteral nutrition with intravenous lipid supplementation. They develop extensive vasculitis of small pulmonary arteries and also pneumonia. Fungemia can also result from catheterization by the proliferation of fungus from patient's skin and then dissemination. Fungemia occurs most commonly in immunocompromised host.[26]

DACRYOCYSTITIS

Malassezia species colonize the lacrimal sac and produce swelling of the lacrimal sac and obstruction leading to dacrolith formation and finally inflammation. [24]

PSORIASIS

Malassezia species produce an immunological reaction which plays a minor role in the pathogenesis of psoriasis.[24]

ATOPIC DERMATITIS

Malassezia furfur was isolated from skin lesions of atopic dermatitis. In such individuals, atopic eczema can be induced by *Malassezia* yeast. Also, IgE antibodies were found against *Malassezia furfur* in patients with atopic dermatitis.[24]

TRANSIENT ACANTHOLYTIC DERMATOSIS has been found to be associated with *Malassezia.*[24]

INFANTILE CEPHALIC PUSTULOSIS IN NEW BORN

Earlier, considered as neonatal acne, is characterized by non-follicular pustular eruption involving the face, neck and scalp. The incidence in hospitalized neonates is 3% .The diagnostic criteria include age of onset less than 1 month, cephalic location, smear positivity for *Malassezia*, elimination

of other causes of neonatal pustulosis, and response to topical ketoconazole. *M. furfur* is the most commonly found species. In the severe form, it is associated with *M.sympodialis*.[24]

Miscellaneous

The fungus has further been isolated from the nasal passages and nasopharynx in patients with maxillary sinusitis and osteitis. It has been found to produce onychomycosis and endocarditis. [24]

HIV INFECTION AND MALASSEZIA

The growth of *Malassezia* is known to be enhanced in immunocompromised conditions. The skin flora remains quantitatively normal in HIV infected patients. In an Indian study by Kaviarasan et al, the authors found the overall incidence of *Malassezia* infection among HIV infected patients to be 13.5%; they observed the incidence of PV, *Malassezia* folliculitis and SD to be 40%, 16% and 56% respectively. Among their patients, 9 of them had a clinical diagnosis of AIDS. [27] A higher incidence and severity of SD has been reported in AIDS patients (30-55% vs 1-3%) in the normal population. The incidence of SD in these patients can be correlated with CD4+ T cell counts; with > 200 cells it is 15% and with < 200 cells, it becomes 58%. Thus, it appears that immunosuppression in HIV infection

enhances *Malassezia* growth.[28] In the series of HIV infected patients reported by Kaviarasan et al, extensive PV and SD with an aggressive course were observed in advanced stages of immunosuppression, and the authors have proposed that the presence of such features can be used as a clinical marker of AIDS in resource poor countries. It has been proposed that aggravation of SD in HIV infected patients is not directly related to *Malassezia* growth. In disease states, the level of toxic products of *Malassezia* rises, increasing the prevalence and severity of SD. Moreover, the HIV infected state increases the level of interferons and tumor necrosis factor-α, which are known to alter the lipid metabolism, increasing serum triglyceride and cholesterol levels. This increases the patient's sensitivity to inflammatory mediators released by *Malassezia*. Though the genus *Malassezia* has been associated with many cutaneous and systemic disorders, its exact role in the causation of most of these conditions is yet to be elucidated.[29]

DIFFERENTIAL DIAGNOSIS FOR ACHROMIC PITYRIASIS VERSICOLOR

Seborrheic dermatitis is a chronic, recurrent, relapsing dermatitis with a distinctive morphology and it is considered to be an altered cell mediated response to *Malassezia* species.[1,4.]

Pityriasis alba, an endogenous eczema occurring in children which is characterized by hypopigmented patch over the face with indistinct borders. P.alba is associated with atopy and nutritional deficiency.[2]

Pityriasis rosea is characterized by erythematous patch which is oval or circular in shape with collarette of scales and exihibits a typical Christmas tree pattern.[2]

Indeterminate Hansen manifests as vaguely defined hypopigmented patch with minimal scaling without any loss of sensation over the face and gluteal region. It may either resolve spontaneously or progress to borderline Hansen disease.[2]

Neavus anaemicus is also known as pharmacological naevus. On diascopy, it merges with the normal skin. On Wood's lamp examination the lesion does not accentuate.[2]

Naevus achromicus is present since birth with serrated border and the lesions are nonscaly. On diascopy, the lesion will not merge with the surrounding skin.[2]

Early vitiligo is a depigmenting autoimmune disorder due to loss of epidermal melanocytes. The lesions of vitiligo are nonscaly, milky white with a well defined border.[2]

Macular syphilid is characterized by oval or round, faint pink lesions which occurs over nape of the neck, sides of the trunk and flexor aspects of the extremities. VDRL and TPHA will be positive.[2]

Ash leaf macules of tuberous sclerosis is present since birth or early infancy.The lesions are polygonal or ash leaf in shape which may be 1-100 in number. On Wood's lamp examination, lesions will get accentuated.[2]

Post kala azar dermal leishmaniasis is characterized by pinpoint macules and patches which is hypopigmented and later the lesions become raised. This condition mainly occurs after treatment of visceral leishmaniasis.[3]

Lichen sclerosus et atrophicus is characterized by porcelain white macules or plaques with induration and the patient will have itching.[2]

Resolving psoriasis- Psoriatic patients being treated with topical steroids or PUVA therapy have post inflammatory hypopigmentation.[2]

Resolving miliaria rubra on exfoliation may present with white coloured scaly macules occurring around eccrine orifices.[3]

DIFFERENTIAL DIAGNOSIS FOR CHROMIC PITYRIASIS VERSICOLOR

Erythrasma is a bacterial infection caused by *Corynebacterium minutissimum* mainly affecting the flexural areas. It manifests as

hyperpigmented, well defined maculopapular lesions with satellite lesions nearby. On Wood's lamp it produces characteristic coral red fluorescence. In axilla, vault involvement is characteristic of erythrasma in contrast to PV, where the vault is spared.[1,3]

Melasma presents as non scaly, blotchy, macular pigmentation with a reticulated appearance over the face. Most commonly seen in females.[1]

Lentigenes is characterized by hyperpigmented macular lesion which is non scaly.[1]

Junctional naevus is an acquired melanocytic naevus that presents as uniformly pigmented a macule or patch which is non scaly.[2]

Café au lait macules appear at birth or soon after birth and tend to disappear with age and manifests as nonscaly, brown coloured macules with irregular borders.[1]

Acanthosis nigricans is commonly seen in flexures and presents as verrucous lesion but they are soft to touch.[2]

Vagabonds disease is seen in pediculosis corporis.[1]

DIAGNOSIS OF PITYRIASIS VERSICOLOR

The diagnosis of pityriasis versicolor is essentially clinical. When there is a clinical suspicion, simple investigation like potassium hydroxide mount can be used .[30]

Culture is mainly done for academic and epidemiological purposes and for the confirmation of unusual manifestations of PV.

Pityriasis vesicolor can be detected by the characteristic fluorescence on Wood's lamp examination.[31]

WOOD'S LAMP EXAMINATION

The skin lesions of pityriasis versicolor gives a yellowish orange or golden yellow fluorescence which is due to the presence of coproporphyrin. The exact extent of skin involvement and subclinical infection may also be determined.[31]

SPECIMEN COLLECTION:

The nature of the specimen collection for *Malassezia* depends on the type of the disease involvement. In case of pityriasis versicolor, skin scrapings are sufficient. Rarely punch biopsy may be done.

Other specimens of *Malassezia* infections includes nail clippings for onychomycosis, peritoneal fluid for peritonitis, respiratory secretions for pneumonia , catheter tip and venous blood in case of fungemia. [32]

Collection of skin scrapings

The skin lesion of the patient is cleaned with 70% isopropyl alcohol in order to remove the surface contaminants. The area is allowed to dry and using the blunt end of a no. 15 flame– sterilized scalpel blade or the edge of a

glass slide, the skin scrapings are collected on an alcohol cleansed, flame-sterilized glass slide, from the active edges of the lesion.

The specimens from scalp includes the hair stubs, scales and the contents of the plugged follicles.[32]

Scotch tape method

This method is more useful when the scales are minimal but culture cannot be done by this method. Scales can be removed using clear adhesive tape also called as scotch tape or vinyl adhesive tape. The tape must be clear and should be pressed firmly over the affected area of the skin. It is then removed and kept over a glass slide.[32]

DIRECT MICROSCOPY

Potassium hydroxide (KOH) mount

The scales obtained by any of the above methods from affected area are examined under light microscope with 10% KOH. KOH digests the keratin and cellular debris and helps in visualization of fungal elements.[30]

Modifications of KOH mount[30]

Dimethyl sulfoxide (DMSO) may be added to the preparation, which allows better visualization by acting as a clearing agent.

Methylene blue may be added to delineate fungal elements.

Albert's stain stains the yeast cells and hyphae purple, delineating them from the background of keratinocytes.

Parker Quink's blue or black ink can be used with KOH to enhance the contrast

CHICAGO SKY BLUE INK 6B[33]

Direct microscopic examination of samples from the affected area using potassium hydroxide (KOH) wet mounts is the most widely-used laboratory method. Although rapid, the KOH wet mount lacks a color contrast and requires considerable skill to interpret. In busy clinics, it is useful to have a rapid, reliable, and easy to interpret diagnostic method.

Chicago sky blue (CSB) stain is a new contrast stain that has shown as a promising rapid and reliable diagnostic method for dermatomysoses and pityriasis versicolor. It contains 1% CSB 6B and is used together with KOH as the clearing agent. Use of Chicago sky blue stain along the KOH wet mount helps to detect all cases of pityriasis versicolor at the 30-minute examination with no additional cases detected on Day 2. This suggests a 30-minute examination is adequate for detecting *M. furfur*. Fungal elements stain blue.[33]

Calcoflour white stain(CFW)

CFW is a water-soluble, non-toxic, colourless fluorescent whitener

and textile dye that selectively binds to cellulose and chitin of the fungal cell wall. When viewed under fluorescent microscope, it fluoresces light blue on exposure to UV light (340 – 365 nm).[34]

CFW stain is superior to conventional KOH mount in that it provides a better differentiation of hyphal elements and collagen fibres.

Appearance in direct microscopy

Under direct microscopic examination of 10% KOH, modified KOH or CFW preparation, all the *Malassezia* species are seen as clusters of round or oval 2 – 7 µ yeast cells and short (about 20 – 25 µ), straight or angulated, stout, aseptate, hyaline hyphal elements along with blastospores showing the characteristic 'banana and grapes' or 'spaghetti and meat ball' appearance or 'foot print shaped'appearance.[1,5]

Punch biopsy

Punch biopsy of skin can be done from the affected area and stained with haematoxylin and eosin. Special stains such as Giemsa, Periodic acid Schiff stain and Gomorri's methanamine silver help to confirm the diagnosis.[35]

Histopathology reveals the presence of yeast cells and hyphal elements in the stratum corneum. There is thickening of stratum corneum and patchy

perivascular lymphocytic inflammatory infilterate in the dermis. Hyperkeratosis, increased density of the fungus and increased dermal inflammatory infilterate are characteristically seen in the hyperpigmented type of PV.[35,36]

CULTURE:

All the species of Malassezia are lipophilic and require lipid supplemented media except *M.pachydermatis*. But culture is not mandatory in the diagnosis of *Malassezia* infections.[34]

Growth requirements

All *Malassezia* species except *M. pachydermatis* require fatty acids for their growth in vitro particularly of saturated or unsaturated fatty acids containing C_{12} to C_{24} series. Any media that contains glycine, glucose, mineral oils, glycerol monostearate or Tween 80 supports the conversion of yeast to mycelial form. Glycine has a unique positive effect on the growth of *M. furfur*.[34]

The ideal temperature for the growth of all *Malassezia species* is 30 - 35˚C.Temperature above this range inhibits most of the species. Preservation of the culture is done by freezing at -80˚C, as *Malassezia* cannot withstand temperatures of 4 - 8˚C.[34]

CULTURE MEDIA [1,3,4,34]

The following media are used for the isolation of *Malassezia* species:

1. **Sabouraud Dextrose Agar** with actidione and overlay of olive oil

2. Modified Dixon's agar

This agar gives substantial growth of the fungi with different morphological patterns for identification. It contains 3.6% malt extract, 0.6% peptone,1% Tween40, 2% desiccated ox bile, 0.2% glycerol, 0.2% oleic acid.

3. Leeming and Notman medium

It has whole fat cow's milk, and produces high recovery rate and longer shelf life.

4. GYP-S agar

It gives quicker growth and quantitation of colonies.This media consists of peptone, glucose, yeast extract, olive oil, Tween 80,glycerol monostearate

5. A minimal medium containing a lipid source and L- tryptophan is used specifically for *M.furfur* which produces a diffusible brown pigmentation.

Malassezia species are part of normal commensal flora of human skin. So it is necessary that the culture media must be supplemented with

cycloheximide and chloramphenicol.[3]

COLONY MORPHOLOGY ON MODIFIED DIXON'S AGAR [2,32]

The morphological features of the colonies of commonly isolated *Malassezia* species on modified Dixon's agar are as follows:

***Malassezia furfur* :**

M. furfur generates cream coloured, thick, convex, umbonate, 4 – 5 mm colonies with a smooth to rough surface.

***Malassezia pachydermatis*:**

M. pachydermatis forms cream coloured, thick, convex colonies with a matte surface and brittle texture, making it difficult to emulsify.

***Malassezia sympodialis*:**

M. sympodialis forms cream to buff coloured, flat, easily emulsifiable colonies with a slight central elevation and a smooth shiny surface.

Malassezia globosa:

M. globosa produces 4 mm cream to buff colonies with a rough and deeply folded surface and a brittle texture and hence are difficult to emulsify.

***Malassezia obtusa*:**

M. obtusa is a slow growing species and forms small colonies with a sticky texture.[37]

***Malassezia restricta*:**

M. restricta is slow growing and forms irregular, small, 2 mm sized, cream coloured colonies with a hard texture.

***Malassezia slooffiae*:**

M. slooffiae forms 3 mm colonies, with finely folded margins and a brittle texture.

Ideally all the inoculated culture media must be incubated for a period of 7 – 10 days at a temperature range of 30 - 35˚C (preferably at 32˚C), and examined daily for growth.[3]

BIOCHEMICAL CHARACTERIZATION:

Conventional assimilation and fermentation tests employed in the speciation of other yeasts cannot be used for these non–fermentative, lipophilic species.[3] The various tests used to identify *Malassezia* species are as follows:

Urease test:

All species of *Malassezia* produce urease enzyme that hydrolyzes urea. This test can be done by using Christensen's urea agar or broth medium. Positive test is indicated by the colour of the medium turning into pink.[4]

Catalase test:

Malassezia species is catalase positive. 20 – 30% hydrogen peroxide is used for this test. On adding the agent *M. restricta* is the only species that is catalase negative.[4]

Esculin hydrolysis test:

Malassezia species contains beta glucosidase enzyme present which has the capacity of hydrolyzing esculin. Esculin is converted in to esculetin that reacts with iron in the medium producing black colour of the medium.

Positive test is seen in *M. sympodialis, M.cuniculi, M.caprae* and *M.obtusa*

Weakly positive or absent in *M. furfur*

Variable - *M. pachydermatis*.[32]

Growth at 42˚C:

The capability of growth at a temperature of 42˚C is determined. *M. sympodialis, M. pachydermatis, M.japonica, M.slooffiae, M. cuniculi, M. furfur and M.dermatis* shows positive growth.[3,32]

Tween assimilation test:

Tween compounds are polymers of ethylene oxide linked to sorbitan and a lipophilic group. They are water soluble and nonionic surfactants. Each *Malassezia* species has the ability to utilize different Tween

compounds, (i.e, 10% Tween 20, 0.5% Tween 40, 0.5% Tween 60 and 0.1% Tween 80) which acts as a source of lipid:[3,32]

Tween 20 - Polyoxyethylene (20) sorbitan monolaureate

Tween 40 - Polyoxyethylene (20) sorbitan monopalmitate

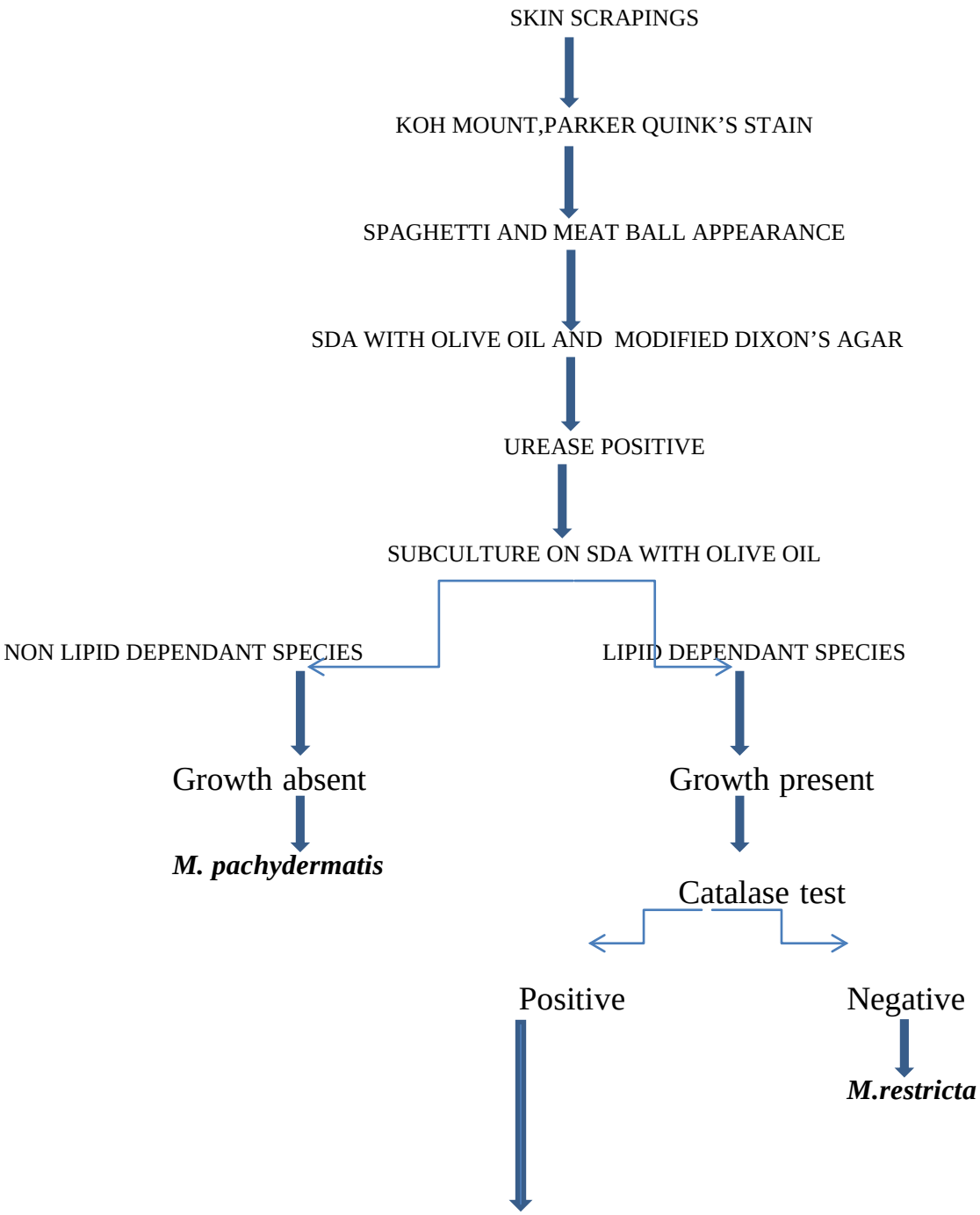
Tween 60 - Polyoxyethylene (20) sorbitan monostearate

Tween 80 - Polyoxyethylene (20) sorbitan monooleate[3,35]

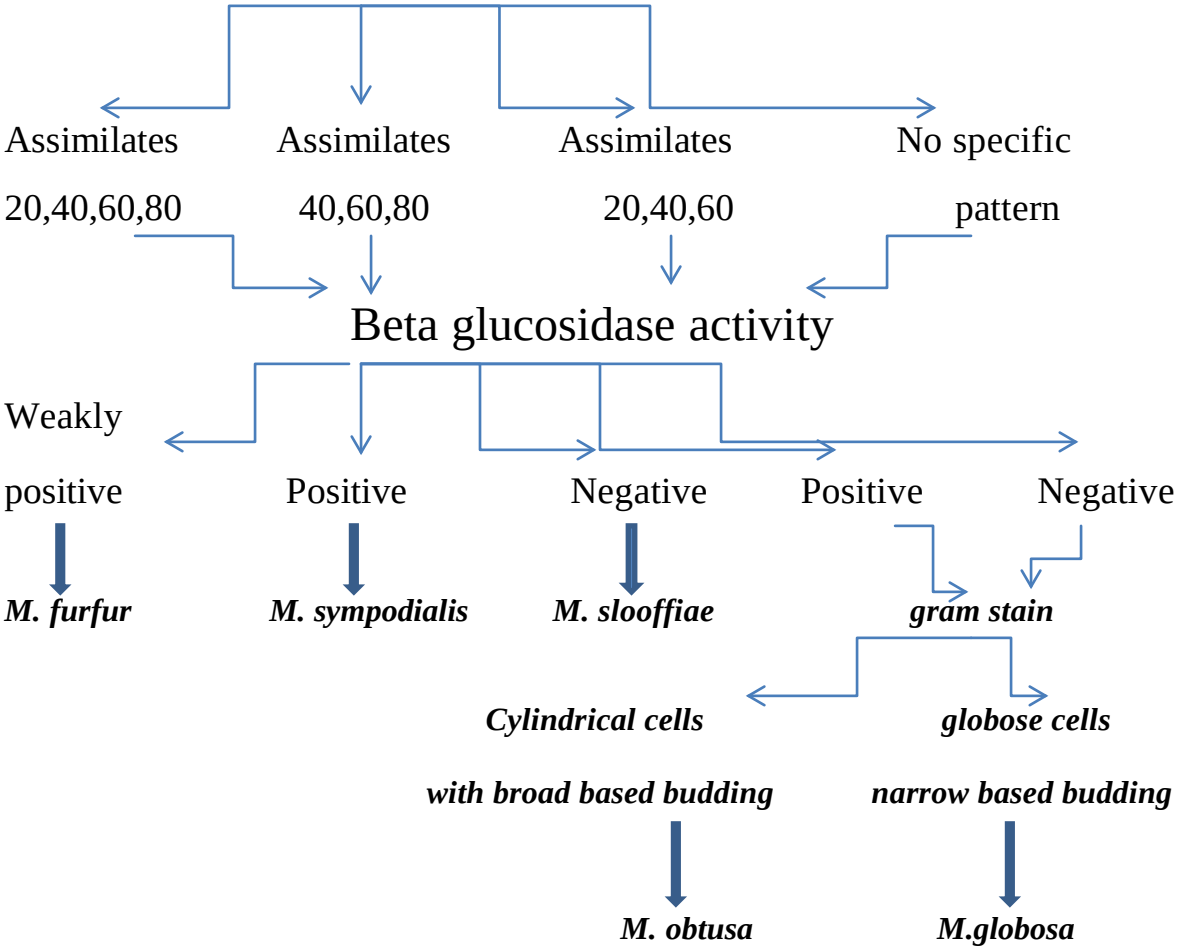
CHARACTERISATION OF MALASSEZIA SPECIES:

Based on the results of the above biochemical tests, the 14 species of *Malassezia* can be identified as follows according to the scheme suggested Mayser *et al* and Gueho *et al*.[10]

SCHEMATIC REPRESENTATION OF IDENTIFICATION OF MALASSEZIA ISOLATES:



Tween assimilation patterns using Tweens 20, 40, 60, 80


Assimilates 20,40,60,80
Assimilates 40,60,80
Assimilates 20,40,60
No specific pattern
Beta glucosidase activity
Weakly positive
Positive
Negative
Positive
Negative
M. furfur
M. sympodialis
M. slooffiae
gram stain
Cylindrical cells with broad based budding
globose cells narrow based budding
M. obtusa
M.globosa

BIOCHEMICAL CHARACTERIZATION [3,32]

	Urease	Catalase	Esculin hydrolysis	Growth at 42˚C	Tween 20	Tween 40	Tween 60	Tween 80
M. furfur	+	+	-/W+	+	+	+	+	+
M.pachy dermatis	+	+/V	V	+	+/V	V	V	V
M. sympodialis	+	+	+	+	+/-	+	+	+
M. globosa	+	+	-	-	+	-	-	-
M. obtuse	+	+	+	-	-	-	-	-
M. restricta	+	-	-	-	-	-	-	-
M. dermatis	+	+	V	+	+	+	+	+
M. japonica	+	+	V	+	-	+	+	-
M. yamatoensis	+	+	V	-	+	-	-	-
M. sloofiae	+	+	-	+	-	-	-	W+
M. nana	+	+	-	V	+	+	V	V
M. caprae	+	+	+	-	V	V	V	V
M. cuniculi	+	+	+	+	-	-	-	-
M. equine	+	+	-	-	V	V	V	-

V – Variable; W+ Weakly positive

IMMUNODIAGNOSI

Cell mediated immunity response to specific fungal antigens is assessed by lymphocyte blastogenesis. Solid phase ELISA technique can be used to measure antibody titers specific to *Malassezia* species.[38]

ANIMAL PATHOGENICITY

Experimental studies conducted in common laboratory animals like guinea pigs and Swiss white mice have resulted in the development of experimental dermatitis showing hyperkeratosis at pilous bulbs and follicular ostia.[38]

MOLECULAR DIAGNOSIS [3]

Molecular testing methods give exact identification of the *Malassezia* species for diagnostic and epidemiological purposes. This helps in rapid screening of large number of isolates in epidemiological surveys. Various methods are as follows:

- Estimation of the G+C content of chromosomal DNA
- 25S rRNA sequencing and karyotyping,
- Restriction fragment length polymorphism (RFLP)
- Multilocus enzyme electrophoresis (MLEE)
- PCR fingerprinting
- Randomly amplified polymorphic DNA analysis (RAPD)

MANAGEMENT OF PITYRIASIS VERSICOLOR

As *Malassezia* species are endogenous to the skin flora, this condition is particularly difficult to eradicate. Our aim is to prevent recurrence of infections. In the meantime, there are a number of topical and oral antifungal treatments that are effective in alleviating clinical symptoms and producing mycological cure. Relapse may occur. Patient must be instructed regarding the pigmentary changes as it may take some time to resolve.[39]

TOPICAL TREATMENT[40]

Various topical antifungal agents used are as follows :

Topical imidazoles

2% clotrimazole, 1% miconazole, 1% econazole, 2% fenticonazole, 1% oxiconazole, 1% bifonazole.

Topical triazoles

1% fluconazole, 2% ketoconazole

Topical allylamines

1% terbinafine , 1% naftifine[41]

Topical benzylamines

1% butenafine

Hydroxy piridone

1% ciclopiroxolamine [42]

Non specific agents [43]

Topical keratolytics were used in the treatment pityriasis versicolor in previous days. They are non specific and they try to remove infected stratum corneum either physically or chemically. It includes Whitefield ointment (salicylic acid 3%, benzoic acid 6% with 24% coconut oil in a paraffin base),Sulphur with salicylic acid ointment.2.5% selenium sulphide supplied as shampoo,1% tolnafate ointment,20% sodium hypochlorite lotion with a contact time of 5-7mins.,1% zinc pyrithione as shampoo,2.5% benzoyl peroxide,50:50 propylene glycol in water

Creams are ideal for limited involvement while lotions are best suitable for extensive area of involvement. Ketoconazole foam is a newer option for treatment and may be preferable than shampoo or cream in case of extensive involvement, as easier application may lead to increased patient compliance.[39]

Treatment once or twice daily for 14 days with topical ketoconazole cream or foam, and once weekly use of ketoconazole shampoo may be effective treatment for PV, with cream or foam showing long-term efficacy.[39] Similarly, topical terbinafine cream could be applied twice daily for 7 days.Treatment efficacy of topical formulations may be lower in more tropical climates.

Some of the newer topical immunomodulatory agents such as calcineurin inhibitors like tacrolimus 0.1% for adults and 0.03% for children pimecrolimus 1% are being tried .[41]

Systemic therapy [43]

Indications:

- Recurrent lesion
- Extensive lesion
- Chronicity
- Scalp infection
- Unusual presentations

Systemic treatment includes

Tab.Ketaconazole 200mg once daily for 15 days (or)

Tab.Itraconazole 200mg once daily for 15 days [44] (or)

Tab.Fluconazole 50mg daily for 15 days.[2]

Pulse therapy [2,43]

Tab. Ketoconazole 400mg on 2 consecutive days in a week for 2 weeks(or)Tab. Itraconazole 600mg stat(or)Tab.Fluconazole 400mg single dose repeated after a week.

Prevention of recurrence:

Tab. Ketoconazole 200mg once a day for 5 days for 3- 6 months

or

Tab. Itraconazole 200mg once a day for 5 months for 3 – 6 months [6]

along with short contact topical application of selenium sulphide for 10 minutes on the 1st and 2nd day.

Pramiconazole is a relatively new triazole that disrupts ergosterol synthesis in fungal cells. It has been shown to be active *in vitro* against dermatophytes, Candida species, and *Malassezia* species. At concentrations <1 µg/mL, pramiconazole activity was twice that of itraconazole against

Candida species, and 10 times greater than ketoconazole against Malassezia species. Recommended dosage is 200 mg pramiconazole daily for 2 days. Diarrhea and nausea were the most common adverse effects, with the study drug formulation (hydroxypropyl-ß-cyclodextrin) likely contributing to this.[46] Overall, pramiconazole may be a promising treatment for PV however, it remains to be determined the clinical efficacy of pramiconazole in relation to existing oral antifungals.[45]

Amphotericin B or fluconazole is used in the treatment of fungemia and also timely removal of the intravascular catheter and stoppage of parenteral lipid infusion is needed.[3]

AIMS AND OBJECTIVES

AIMS AND OBJECTIVES OF THE STUDY

- To study the epidemiology of pityriasis versicolor.
- To study the various clinical types.
- To study the association of pityriasis versicolor with other dermatological disorders and systemic diseases
- To identify the various species of *Malassezia*
- To study the clinicomycological correlation.

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MATERIALS

AND METHODS

MATERIALS AND METHODS

This study was conducted in the mycology section of department of dermatology, madras medical college and Rajiv Gandhi Government General Hospital Chennai. Hundred patients with pityriasis versicolor who attended the mycology OPD between September 2014 and August 2015 were selected for the study.

Inclusion Criteria:

1. Patients having clinical signs of pityriasis versicolor
2. KOH positivity for Malassezia
3. Not taken topical or oral antifungal medication 3 months prior to study

Exclusion Criteria:

1. Patients who have taken topical or systemic antifungal treatment within 3 months prior to the study.
2. KOH negativity for fungus.

Procedure:

100 randomly selected patients with clinical diagnosis of pityriasis versicolor who have not taken any topical or systemic antifungal treatment in the previous three months were selected for the study.

Detailed case history of each patient with reference to age, sex, occupation, marital status, seasonal variation, family history, history of exposure to sexually transmitted disease in those with high risk behaviour, other associated systemic conditions such as diabetes, tuberculosis, endocrine diseases, pregnancy, immunosuppressive states (HIV, transplant patients, internal malignancy) and malnutrition was taken. History with regard to other associated dermatological disorders such as acne, seborrhoeic dermatitis, dermatophytosis, candidiasis, acanthosis nigricans etc was taken.

Clinical features like site of involvement, percentage of body surface area, type of the lesion, other associated systemic and cutaneous disorders were noted. Blood haemogram, blood sugar, renal and liver function tests, serum cholesterol, blood grouping and typing were done. Blood VDRL for syphilis and ELISA for HIV infection were done in patients with extensive pityriasis versicolor.

Skin scrapings of each patient was examined in 10% KOH for the presence of hyaline, short, straight, angulated, aseptate hyphae with blastospores . All the positive specimens were subjected to culture. The selected site was cleaned with 70% alcohol and then scraping was done and scales were inoculated into modified Sabouraud's Dextrose Agar medium

with chloramphenicol, actidione with olive oil overlay and kept at 37 degree Celsius in an incubator for 48 -72hrs. Macroscopic appearance of the colony was noted. Microscopic appearance of the colony in Gram's stain and lacto phenol cotton blue was observed. Tween assimilation test was done for the isolation of species.

Follow Up

Patients were started on topical and/ systemic treatment depending on the extent of involvement and recurrence. They were advised to come after one week with all the investigation reports.

OBSERVATION AND

RESULTS

OBSERVATIONS AND RESULTS

Table 1: Age and gender distribution in the study population(n=100)

	Total no. of cases		Male Patients		Female Patients	
Age in years	**Number**	**%**	**Number**	**%**	**Number**	**%**
11-20	22	22	16	16	6	6
21-30	43	43	27	27	16	16
31-40	27	27	20	20	7	7
41-50	4	4	3	3	1	1
51-60	3	3	3	3	0	0
61-70	1	1	1	1	0	0
TOTAL	**100**	**100**	**70**	**70**	**30**	**30**

Out of the 100 patients with pityriasis versicolor, 43(43%) patients were between the age group of 21-30 years, followed by 27 patients (27%)belonging to 31-40 years. Out of the 43patients(43%), 27were males(27%), 16 were females (16%). The youngest age was a 13 year old male and the oldest was a 65 year old male.

Fig 1.Age and gender distribution in the study population(n=100)

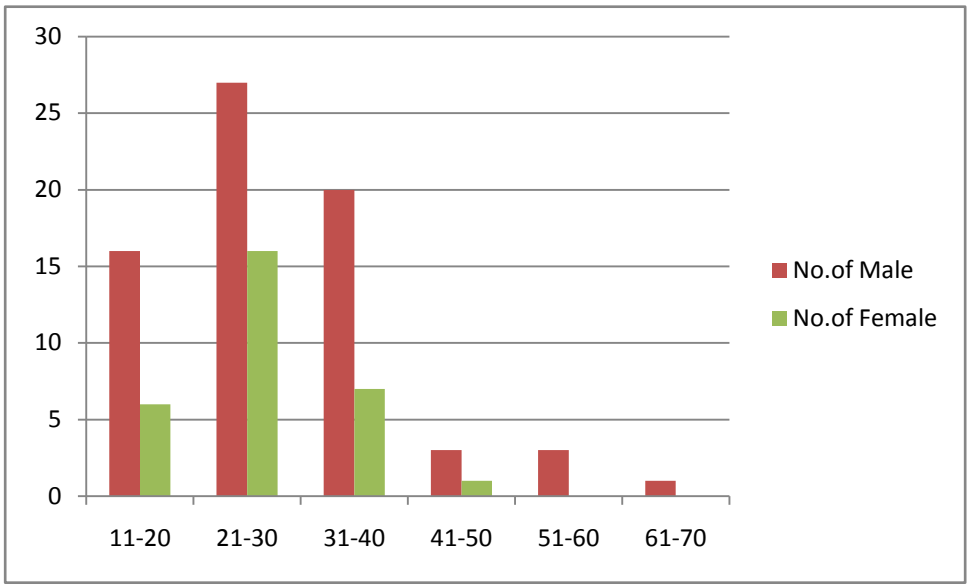


Table 2:Gender distribution of Pityriasis versicolor in the study population (n = 100)

Sex	No.of patients	%
Male	70	70
Female	30	30
Total	**100**	**100**

Out of the hundred patients, 70were males(70%) and remaining 30 were females (30%).

Figure 2:Gender distribution in the study population (n = 100)

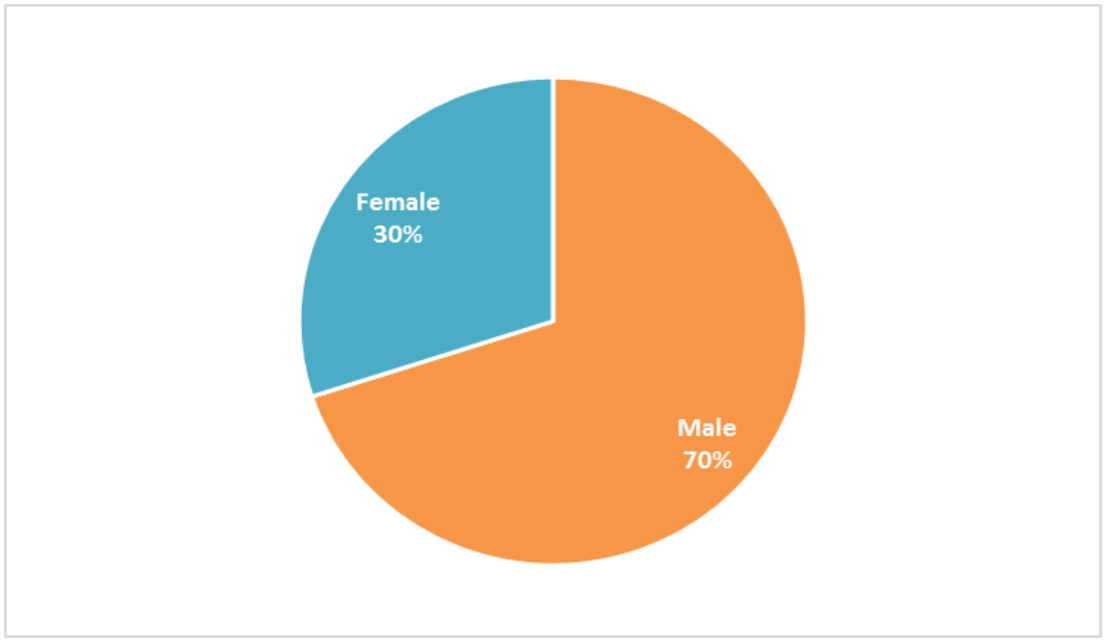


Table 3: Distribution of occupation among pityriasis versicolor cases (n=100)

Occupation	No.of patients	%
Student	29	29
Manual labourer	16	16
Farmer	9	9
Plumber	9	9
Driver	7	7
Electrician	1	1
Housewife	8	8
Housekeeper	1	1
Shop Keeper	8	8
Painter	1	1
Tailor	7	7
Porter	1	1
Security	1	1
Teacher	1	1
Cook	1	1
Grand Total	**100**	**100**

Among the 100 patients, 29 of them were students(29%) and 16 of them were working as manual labourers(16%). There were 9 farmers (9%) and 9 plumbers (9%).

Figure 3:Occupation distribution among pityriasis versicolor cases (n=100)

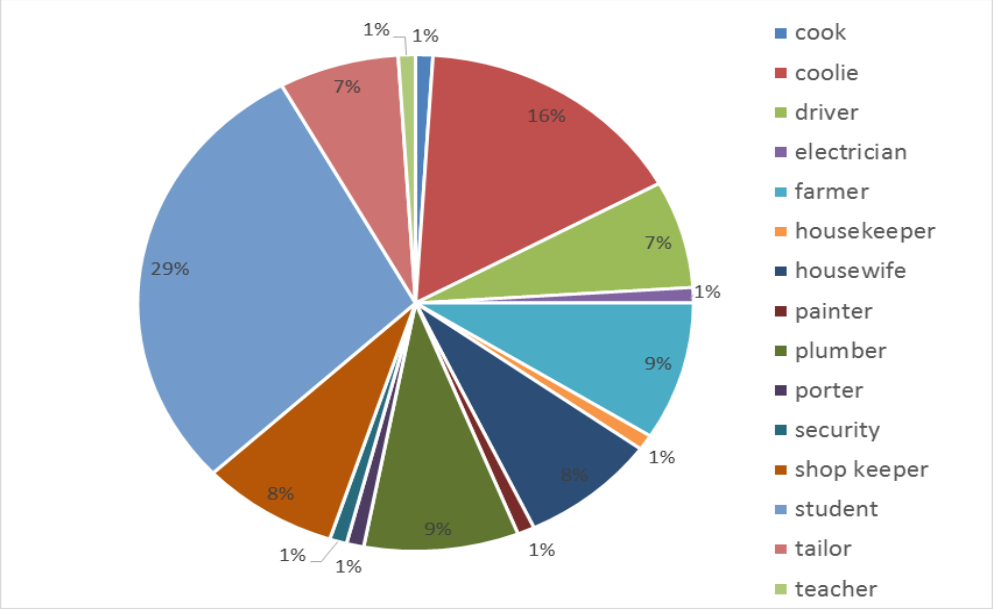


Table4 :Distribution of urban and rural population among pityriasis versicolor patients (n=100)

Residence	No.of Patients	%
Urban	65	65
Rural	35	35
Total	**100**	**100**

In this study ,65 patients (65%) belonged to urban population while 45patients (45%) were from rural areas.

Figure 4 :Distribution of urban and rural population among pityriasis versicolor patients (n=100)

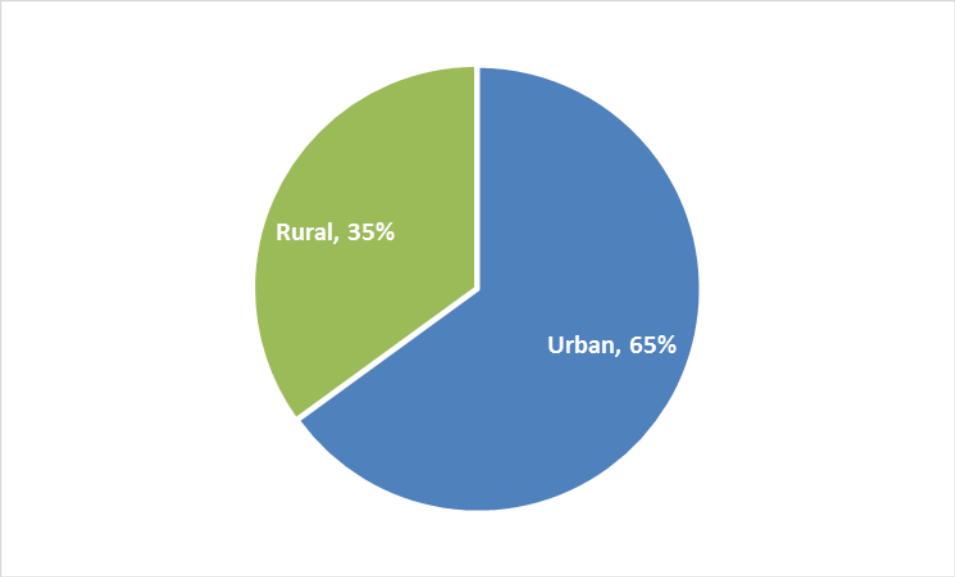


Table 5: Marital status among pityriasis versicolor patients (n=100)

Marital Stage	No.of Patients	%
Married	51	51
Unmarried	49	49
Total	**100**	**100**

In this study, 51patients(51%) were married and others, 49 patients(49%) were unmarried. There was not much of a difference in the marital status of the patients.

Figure 5: Marital status among pityriasis versicolor patients (n=100)

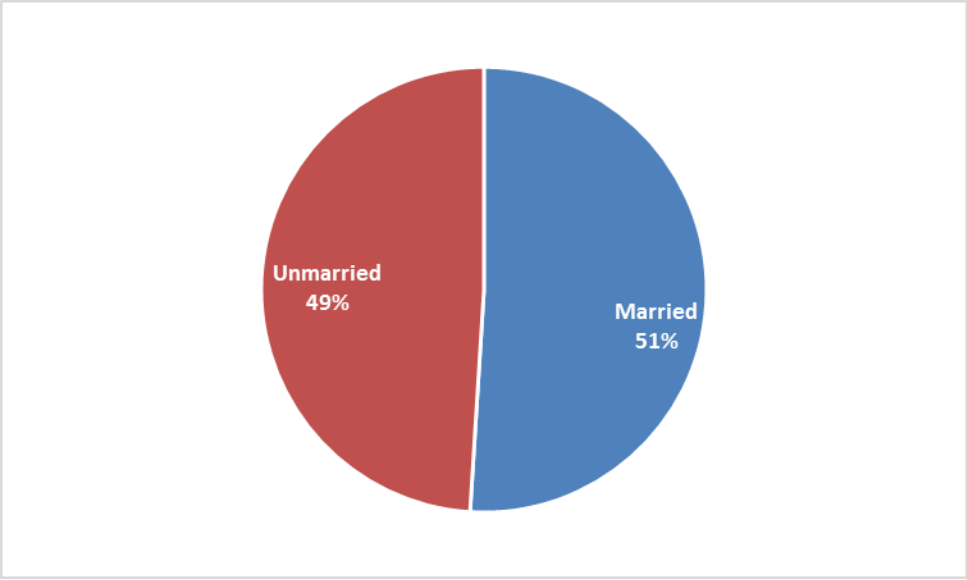


Table 6: Family history in the study group (n = 100)

FAMILY HISTORY	No.of Patients	%
Present	31	31
Absent	69	69
Total	100	100

Family history of similar infection was present in 31patients(31%) and remaining 69 patients(69%) had no such history

Table 7: Frequency of sweating among pityriasis versicolor patients (n=100)

EXCESSIVE SWEATING	No.of Patients	%
Present	58	58
Absent	42	42
Total	100	100

History of excessive sweating was present in 58 patients (58%) while the remaining 42(42%)patients had no such history.

Table 8:Frequency of occurrence of PV in the study population(n =100)

Frequency of Occurrence	**No.of Patients**	**%**
First Episode	65	65
Recurrence	35	35
Total	100	100

Out of the hundred patients, 65 patients(65%) had pityriasis versicolor for the first time and 35patients (35%) had recurrence.

Figure 6: Distribution of patients with pruritus, family history and history of recurrence of PV in the study population (n = 100)

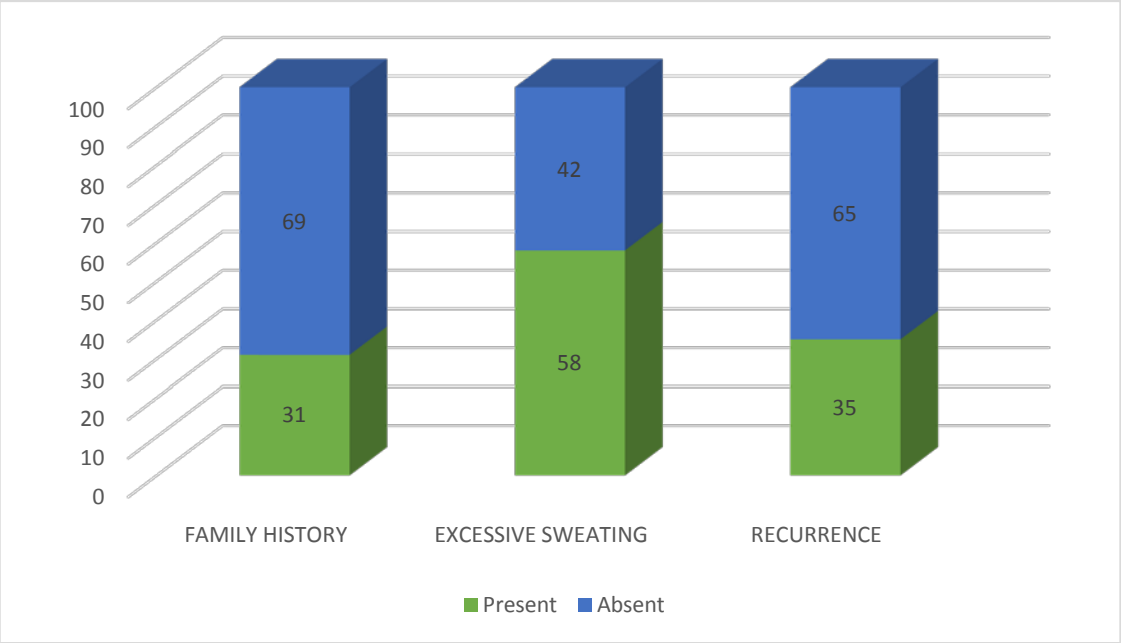


Table 9: Duration of lesion among pityriasis versicolor patients(n=100)

DURATION	**No.of Patients**	**%**
1 Week to 1 Month	8	8
Above 1 Month to 6 Month	64	64
Above 6 Month to 1 year	19	19
More than 1 year	9	9
Total	**100**	**100**

In this study, duration of the disease between 1 month to 6 months was noted in 64 patients(65%), 19 patients(19%) had duration between 6 months to 1 year and 9 (9%) patients had disease for more than 1 year.

Figure 7: Duration of PV among study population(n=100)

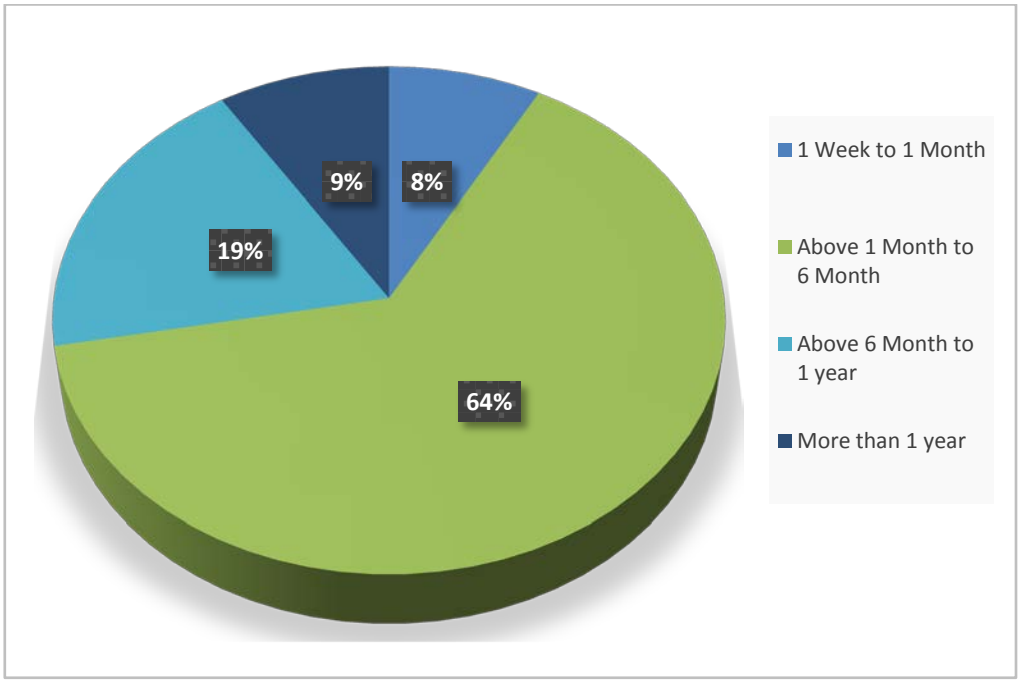


Table 10 : Symptoms among patients with PV in the study population (n = 100)

SYMPTOMS	No.of Patients	%
Cosmetic	68	68
Pruritis	32	32
Total	**100**	**100**

Out of the 100 patients, 68 patients (68%) had come due to cosmetic concern and 32 patients(32%) patients had pruritis.

Figure 8: : Symptoms among patients with PV

in the study population (n = 100)

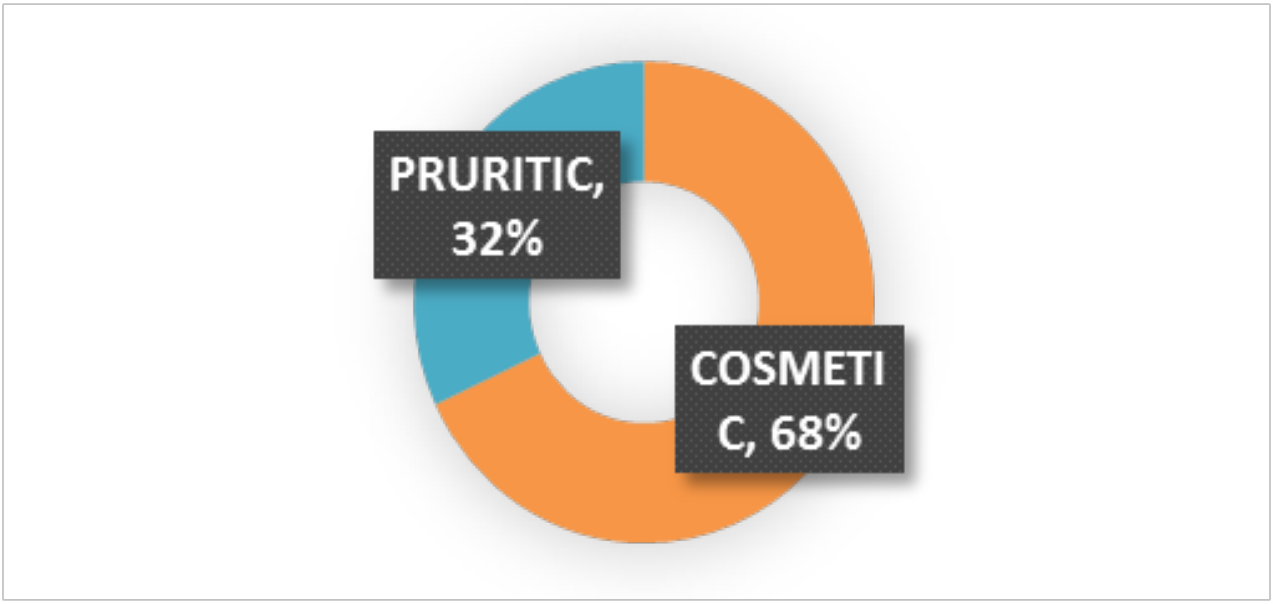


Table 11: Associated systemic conditions among pityriasis versicolor patients (n=100)

SYSTEMIC ASSOCIATIONS	No.of Patients	%
Diabetes mellitus	11	39.3
Hypertension	7	25.0
Chronic kidney disease	2	7.1
Pulmonary tuberculosis	2	7.1
Renal transplant	2	7.1
Bronchiectasis	1	3.6
DCLD	1	3.6
Hypothyroidism	1	3.6
Nephrotic Syndrome	1	3.6
Total	**28**	**100**

Among the 100 patients, 28(28%) patients had systemic association. Diabetes mellitus was present in 11 patients (39.3), followed by hypertension in 7 patients(25%), followed by pulmonary tuberculosis in 2 patients(7.1)

Figure 9: Associated systemic condition among pityriasis versicolor patients (n=100)

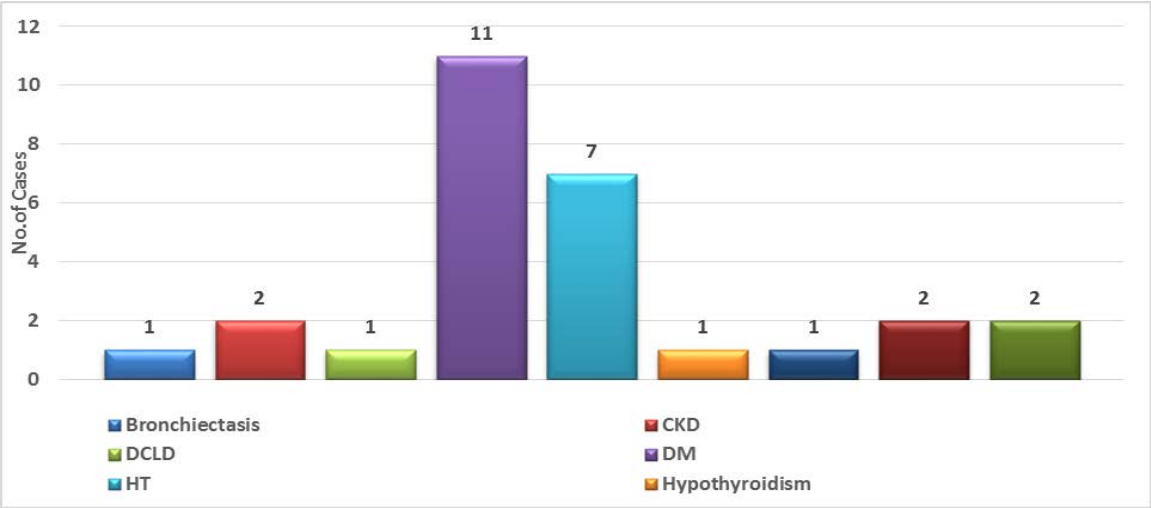


Table 12; Associated dermatological disorders with pityriasis versicolor patients (n=100)

ASSOCIATED DERMATOLOGICAL DISORDER	No.of Patients	%
Seborrheic dermatitis	21	36.20
Acne	14	24.13
Dermatophytosis	11	18.97
Candidiasis	2	3.45
Lichen planus	2	3.45
Congenital melanocytic naevus	1	1.72
IGH	1	1.72
Lichen amyloid	1	1.72
Pompholyx	1	1.72
Pyoderma	1	1.72
Seborrheic keratosis	1	1.72
Stasis dermatitis	1	1.72
Wart	1	1.72
Total	**58**	100

Out of the hundred patients, 58 patients(58%) had associated dermatological disorders, of which 21patients (21%) had seborrheic dermatitis, 14 patients(14%) had acne, 2 patients(2%) had candidiasis and lichen planus.

Figure10:Associated dermatological disorders among pityriasis versicolor patients(n=100)

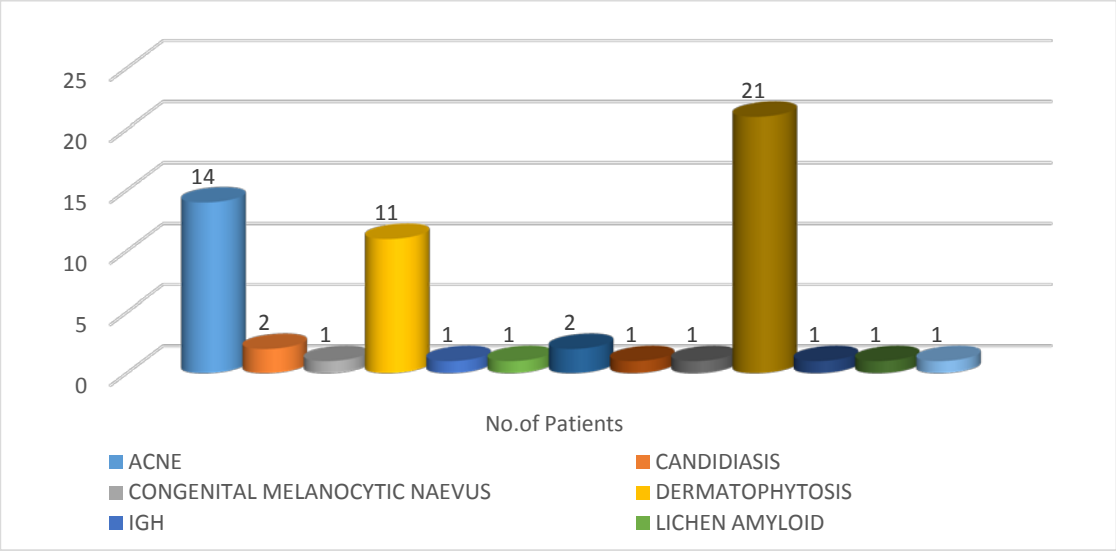


Table 13: Distribution of site of involvement of pityriasis versicolor (n=100)

SITE OF INVOLVEMENT	No.of Patients	%
Back	46	46
Face & Neck	26	26
Chest	17	17
Arms	9	9
Shoulder	2	2
Total	**100**	100

Among the 100 patients, 46 patients had lesions over back(46%), followed by face and neck in 26(26%), followed by chest in 17 patients (17%).

Figure 11 :Distribution of site of involvement of pityriasis versicolor patients (n=100)

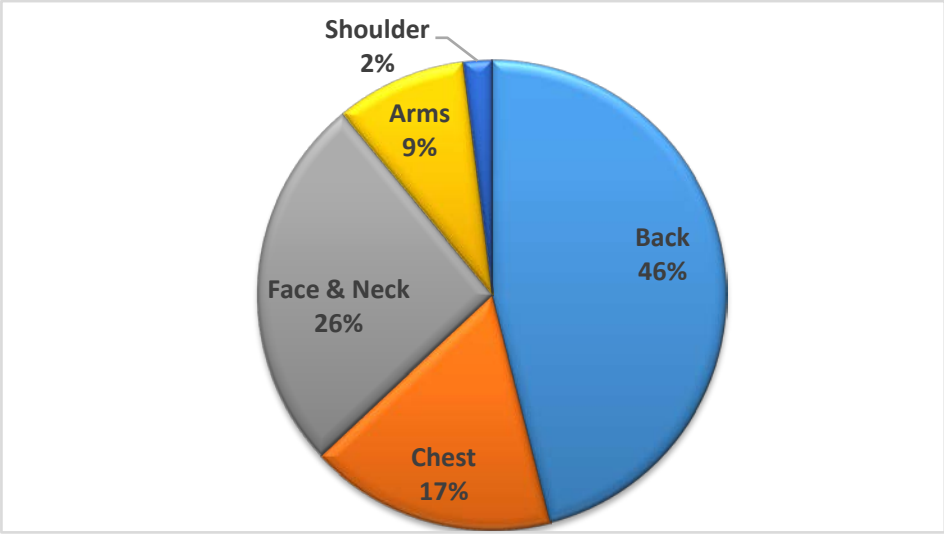


Table 14:Extent of area of involvement among pityriasis versicolor (n=100)

AREA OF INVOLVEMENT	No.of Patients	%
<10%	2	2
10-30%	26	26
30%-50%	70	70
>50%	2	2
Total	**100**	**100**

Out of the hundred patients, 70 patients (70%) had more than 30% body surface area involvement, followed by more than 10% in 26 patients (26%) and more than 50% BSA in 2 patients(2%).

Figure 12 :Extent of area of involvement among pityriasis versicolor patients(n=100)

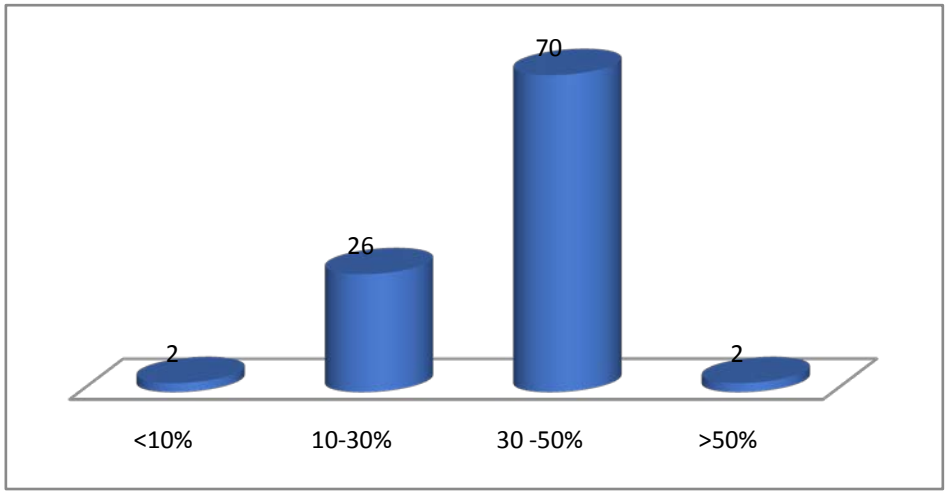


Table 15:Distribution of clinical types of pityriasis versicolor among study group (n=100)

CLINICAL TYPE	No.of Patients	%
Achromic	68	68
Chromic	23	23
Perifollicular	12	12
Both(mixed)	9	9
Paranasal	2	2

In this study, out of 100 patients, 68(68%) presented with achromic type of PV and the remaining 32 patients (32%) had chromic type of PV. 12 patients with achromic PV had perifollicular type and 2 patients had paranasal type of PV who were adoloscents. Nine patients had mixed type with both achromic and chromic type.

Figure 13 :Distribution of clinical types of PV among study group

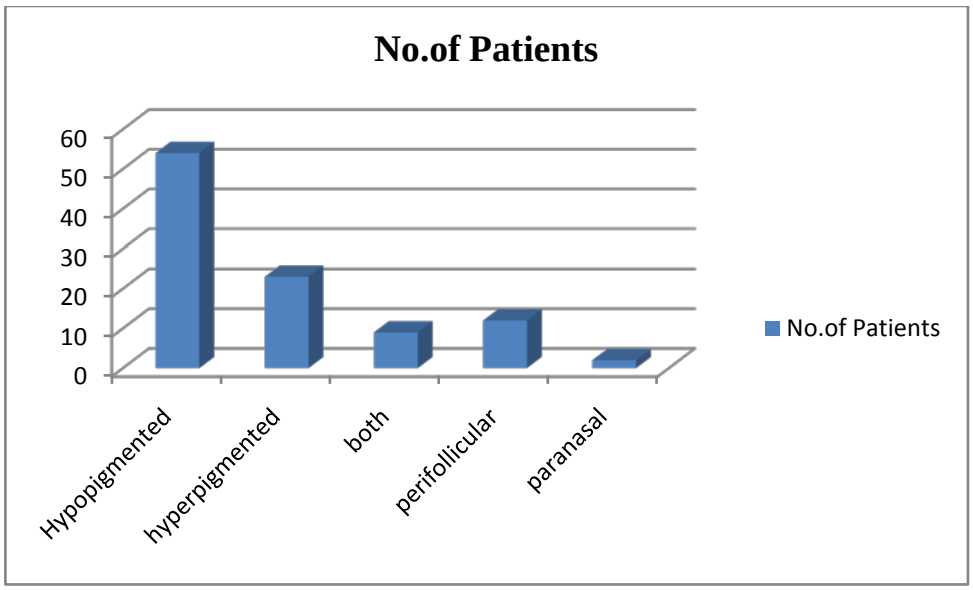


Table 16:Distribution of Blood grouping & typing among pityriasis versicolor cases(n=100)

BLOOG GROUPING & TYPING	No.of Patients	%
A	52	52
O	38	38
B	9	9
AB	1	1
Total	**100**	**100**

In this study A blood group A was found in 52 patients (52%) . 38 patients(38%) had blood group O, 9 patients(9%) had blood group B and one patient had AB blood group(1%) respectively.

Figure14: Distribution of Blood grouping & typing among pityriasis versicolor cases

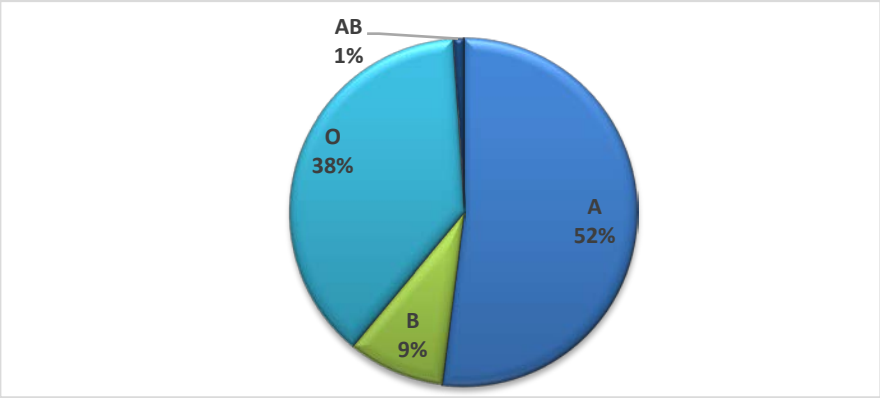


Table17 : Isolation rate of *Malassezia* spp. from pityriasis versicolor cases in the study population (n = 100)

Culture for *Malassezia* spp	No.	%
Positive	76	76
Negative	24	24
Total	**100**	**100**

Out of the 100 samples of KOH positive skin scrapings, 76 (76 %) yielded growth on Sabouraud dextrose agar with olive oil overlay and the remaining 24 (24%) were culture negative.

Figure 15:Isolation rate of *Malassezia* spp. from pityriasis versicolor cases in the study population (n = 100)

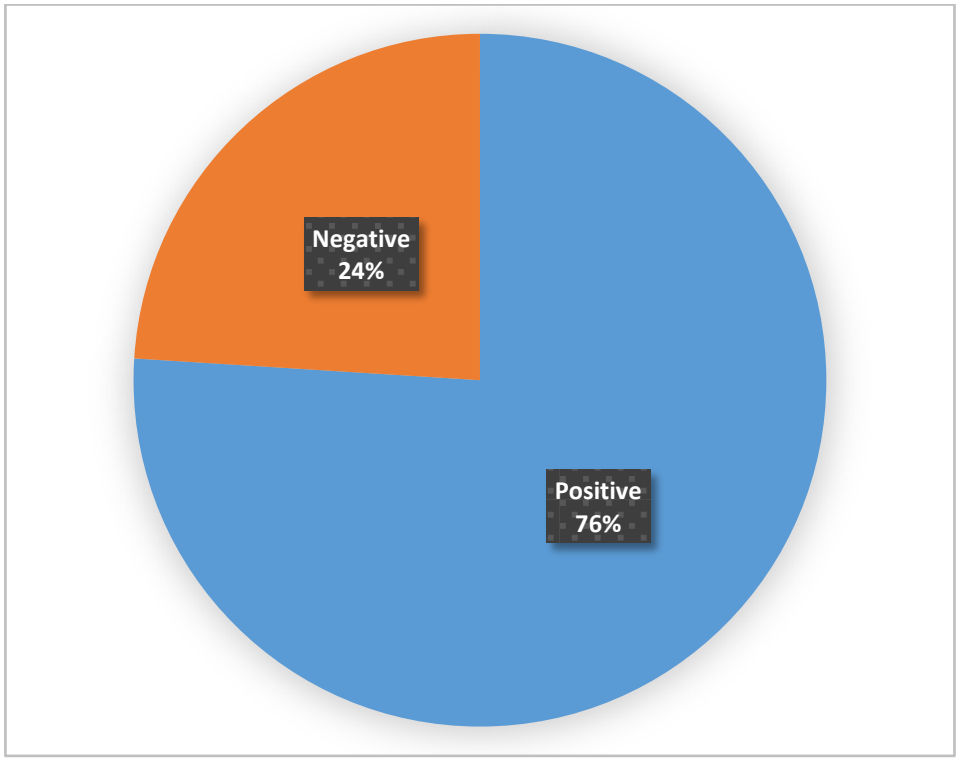


Table 18: Distribution of *Malassezia* species among the culture positive PV cases in the study population (n = 76)

***Malassezia* species**	**No.of Patients**	**%**
M. globosa	37	48.7
M. sympodialis	19	25
M. furfur	8	10.5
M. restricta	6	7.9
M. obtusa	5	6.6
M.sloofiae	1	1.3
Total	**76**	**100**

Out of the 76 isolates, *Malassezia globosa* was the most common species isolated in 37 patients(48.7%) , followed by *Malassezia sympodialis* in 19 patients(25%), *Malassezia furfur* in 8 patients(10.5%), *Malassezia restricta* in 6(7.9%) and *Malassezia obtusa* in 5 patients(6.6%) .

Figure16: Distribution of *Malassezia* species among the culture positive PV cases in the study population (n = 76)

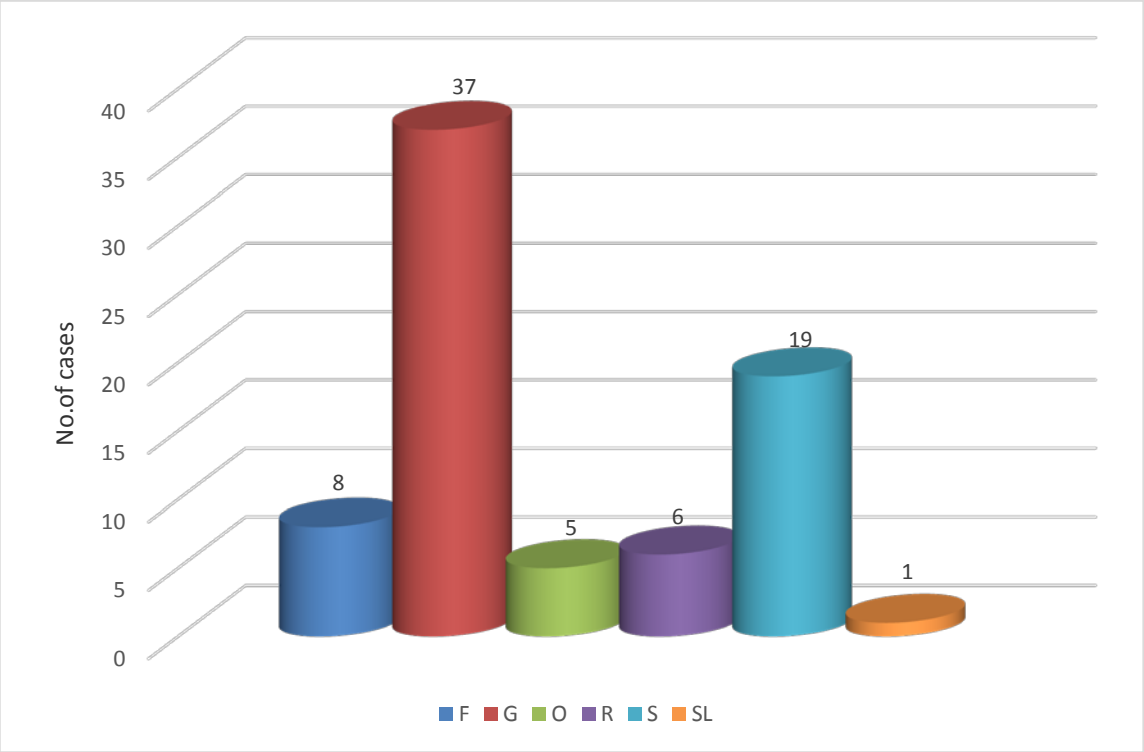


Table 19: Correlation between type of PV and isolation rate of various *Malassezia* spp. in culture positive PV cases (n = 76)

CULTURE	Total	Chromic	%	Achromic	%
M. globosa	37	14	48.28	23	48.94
M. sympodialis	19	7	24.14	12	25.53
M. furfur	8	3	10.34	5	10.64
M. restricta	6	1	3.45	5	10.64
M. obtusa	5	3	10.34	2	4.26
M.sloofiae	1	1	3.45	0	0.00
Total	**76**	**29**	**100**	**47**	**100**

Malassezia globosa (48%) was isolated from 23 patients with (23%) achromic type of PV and 14 patients with (14%)chromic type of PV followed by *M. sympodialis* in 12 patients(25%) with achromic type and 7 patients with (7%) chromic type of PV.

Figure 17: Correlation between type of PV and isolation rate of various *Malassezia* spp. in culture positive PV cases (n = 76)

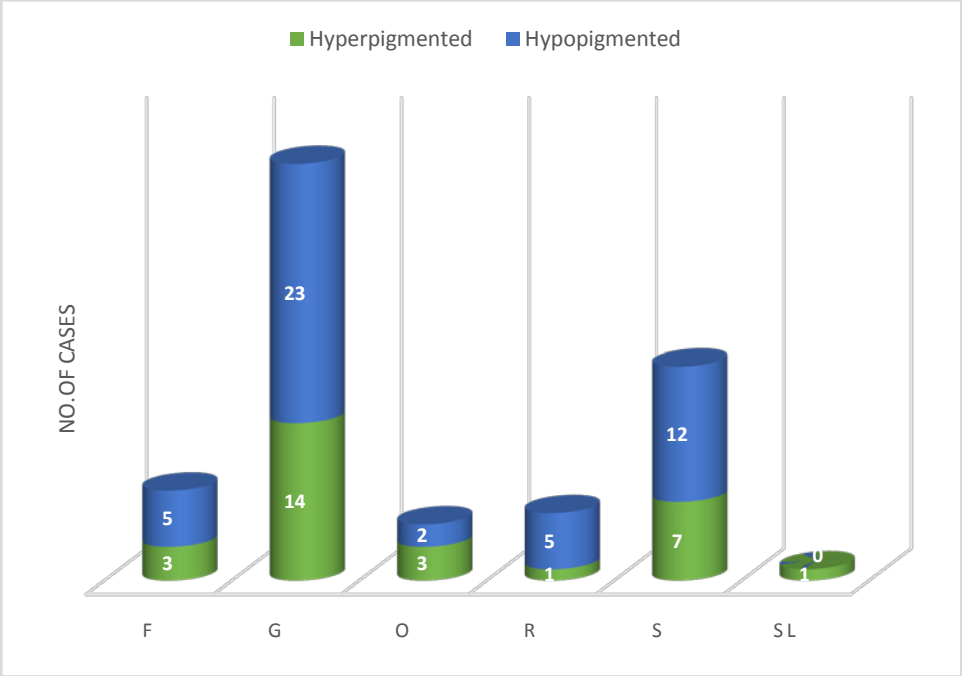
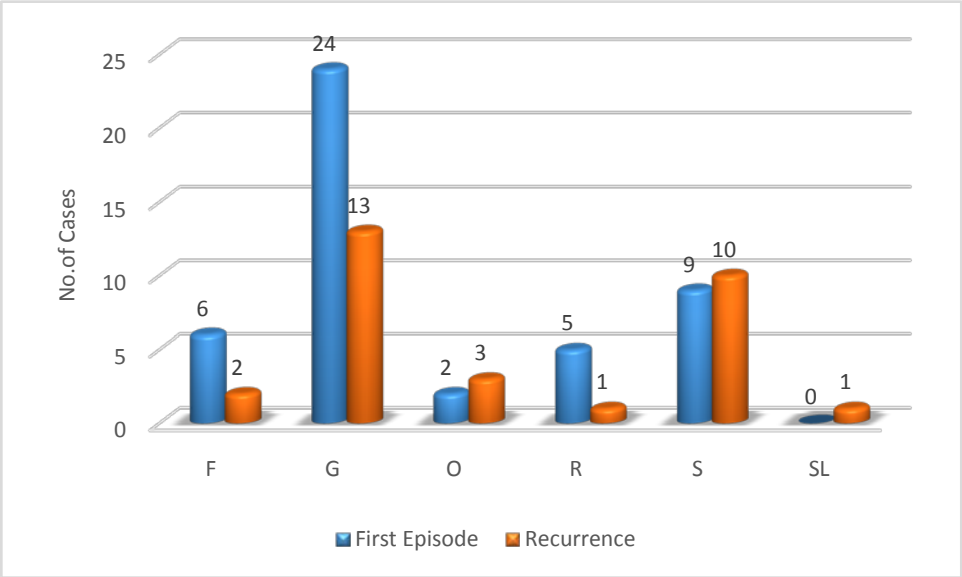


Table 20: Correlation between the PV episode and *Malassezia* spp. Isolated from culture positive PV cases in the study population (n= 76)

CULTURE	Total	First Episode	%	Recurrence	%
M. globosa	37	24	52.17	13	43.33
M. sympodialis	19	9	19.57	10	33.33
M. furfur	8	6	13.04	2	6.67
M. restricta	6	5	10.87	1	3.33
M. obtuse	5	2	4.35	3	10.00
M.sloofiae	1	0	0.00	1	3.33
Total	**76**	**46**	**100**	**30**	**100**

In 24 patients (52%) with first episode PV and in 13patients (43%) with recurrent lesions, *M. globosa* was the most common species isolated followed by *M. sympodialis* in 9 patients (19%) with first episode and 10(33.3%) patients with recurrence.

Figure 18: Correlation between the PV episode and *Malassezia* spp. isolated from culture positive PV cases in the study population (n= 76)



COLOUR PLATES

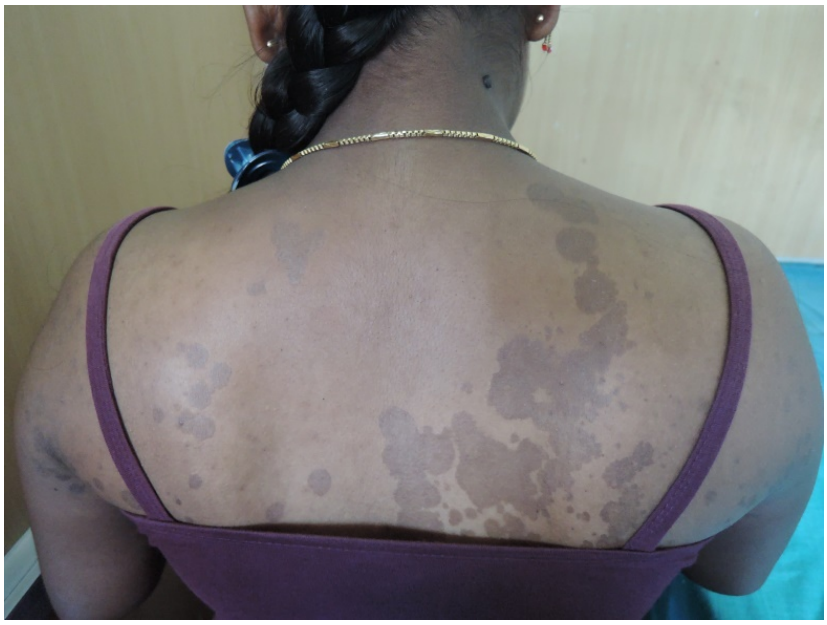


Fig.19 Clinical photograph of chromic type of pityriasis versicolor over back

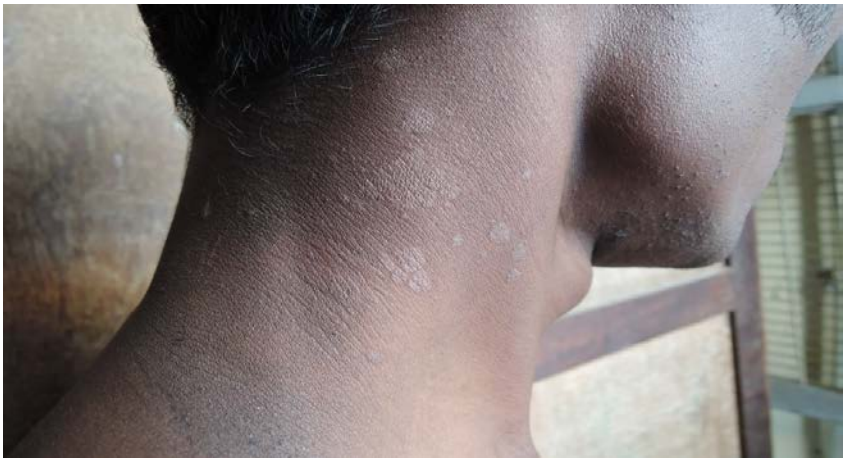


Fig.20 Clinical photograph of chromic PV over neck

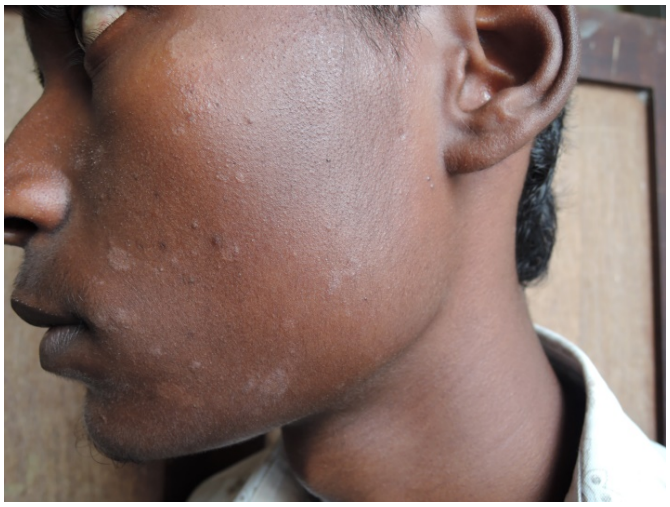


Fig.21 Clinical photograph of achromic type of pityriasis versicolor over face

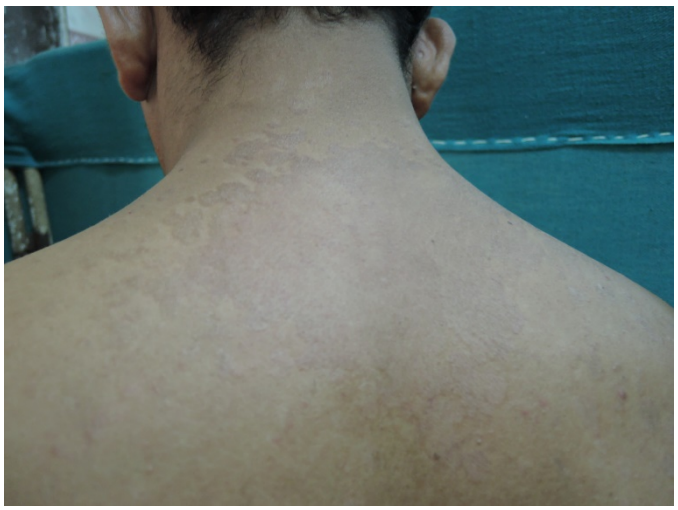


Fig.21 Clinical photograph of chromic type of pityriasis versicolor over back

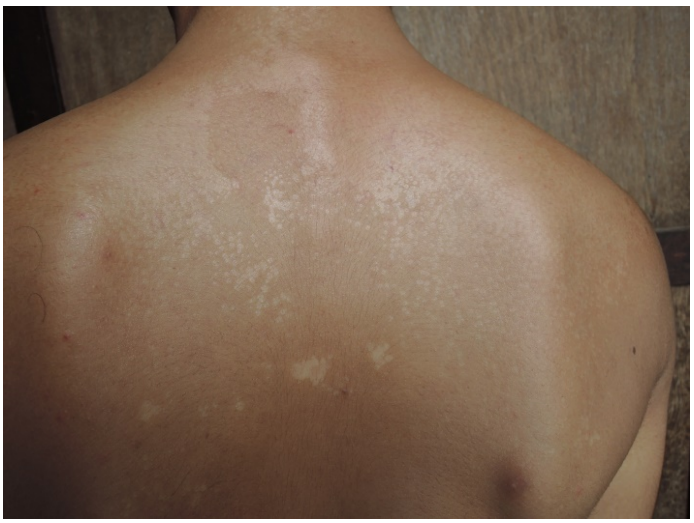


Fig.21 Clinical photograph of perifollicular type of pityriasis versicolor over back

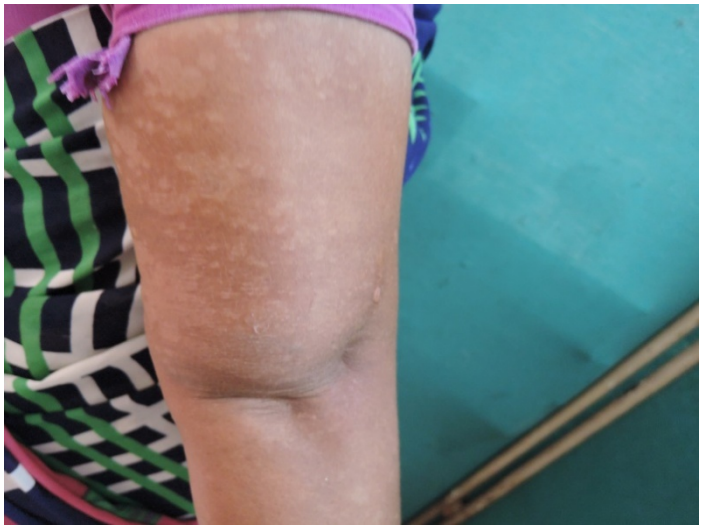


Fig.22 Clinical photograph of achromic type of pityriasis versicolor over arm

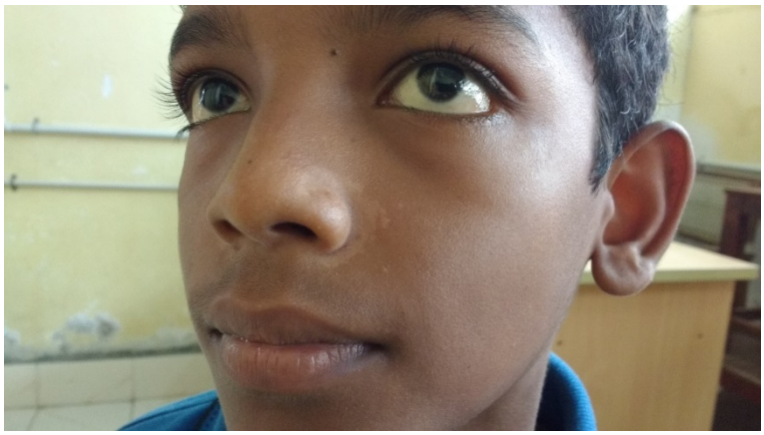


Fig.21 Clinical photograph of paranasal type of pityriasis versicolor over face

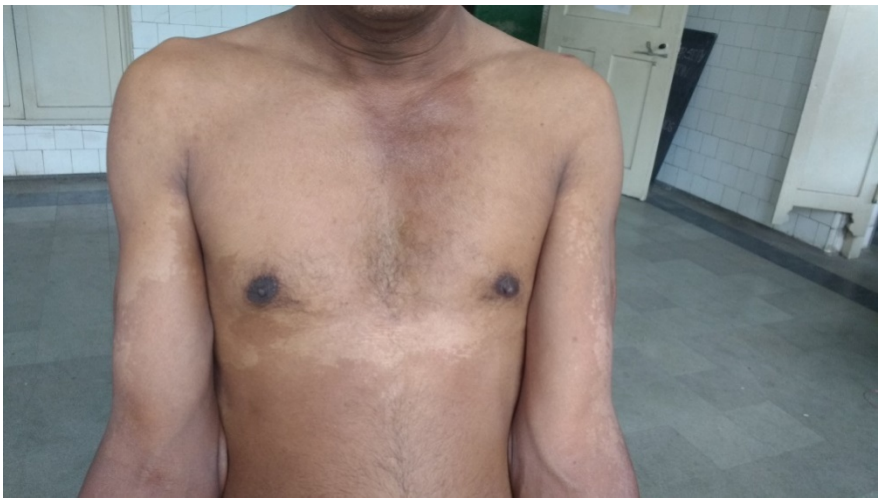
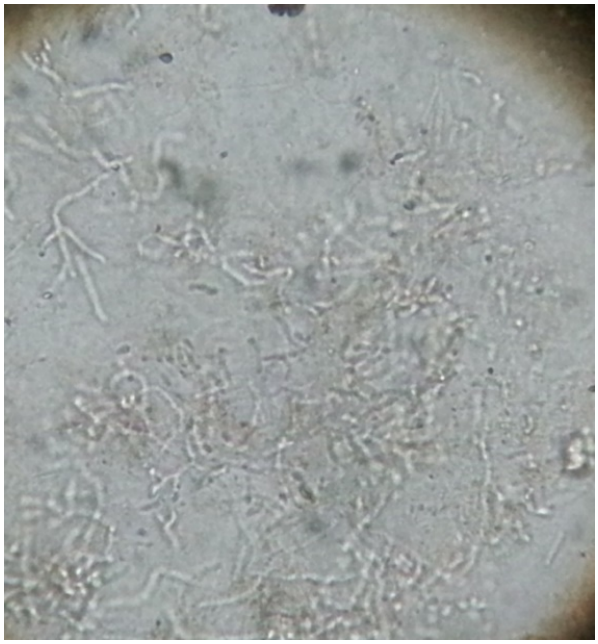


Fig.21 Clinical photograph of achromic type of pityriasis versicolor over chest and arms

KOH MOUNT



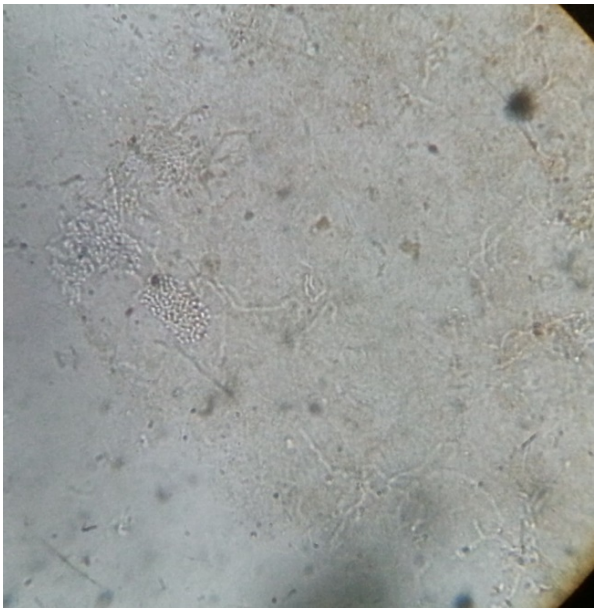


Fig.21,22. Short , straight or angulated, stout, aseptate, hyaline hyphal elements showing 'spaghetti and meat ball' appearance

CULTURE

Fig.23,24,25 Yeasty pasty colonies of *malazeesia*

TWEEN ASSIMILATION TEST

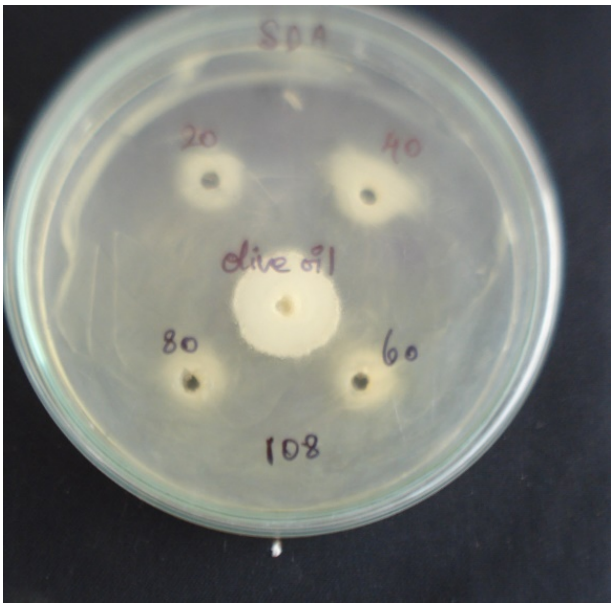


Fig 26 ***Malassezia globosa***

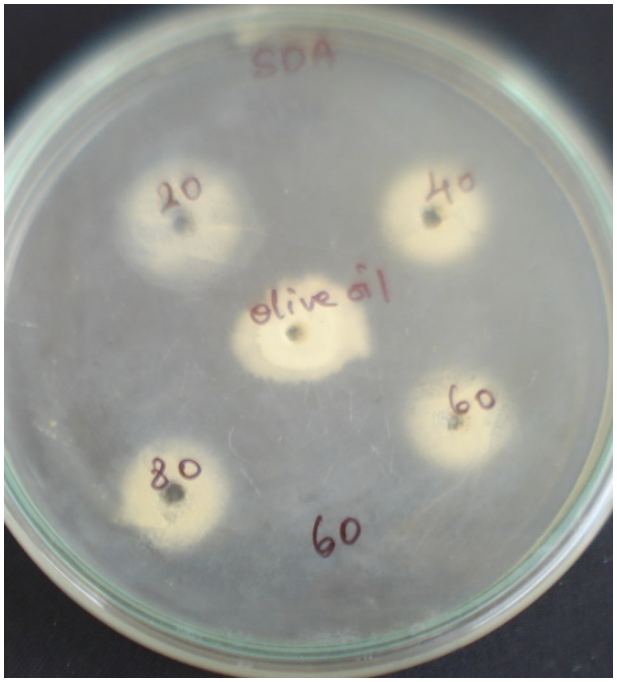


Fig 27 ***Malassezia sympodialis***

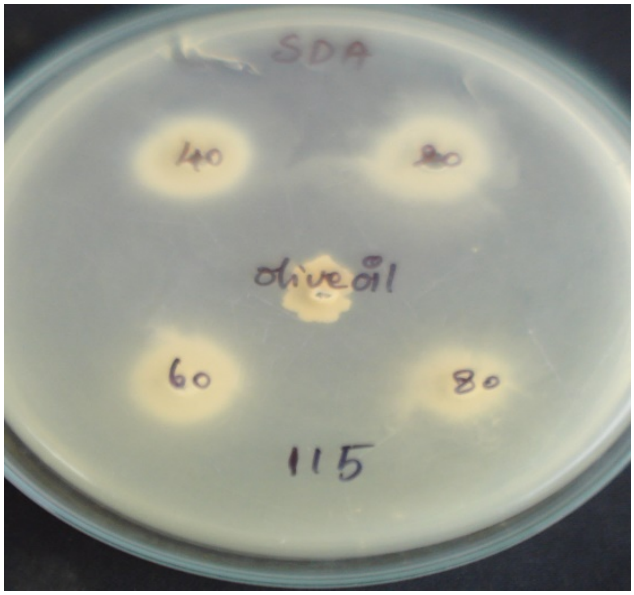


Fig 28 ***Malassezia furfur***

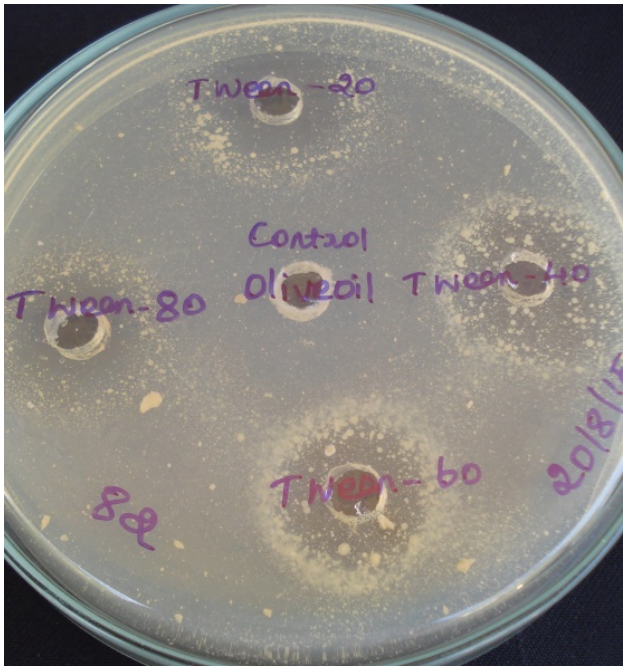


Fig 29 ***Malassezia sloofiae***

GRAM STAIN

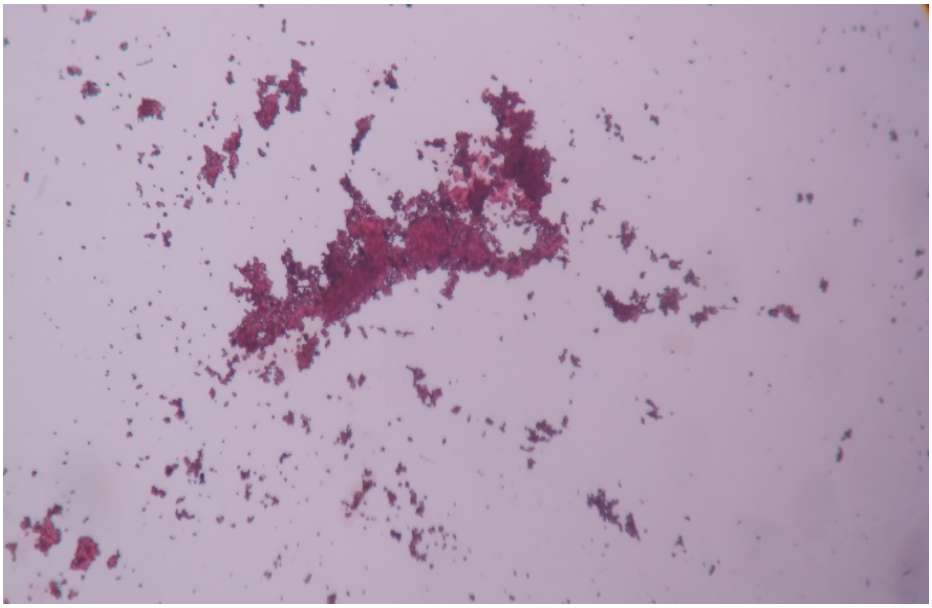
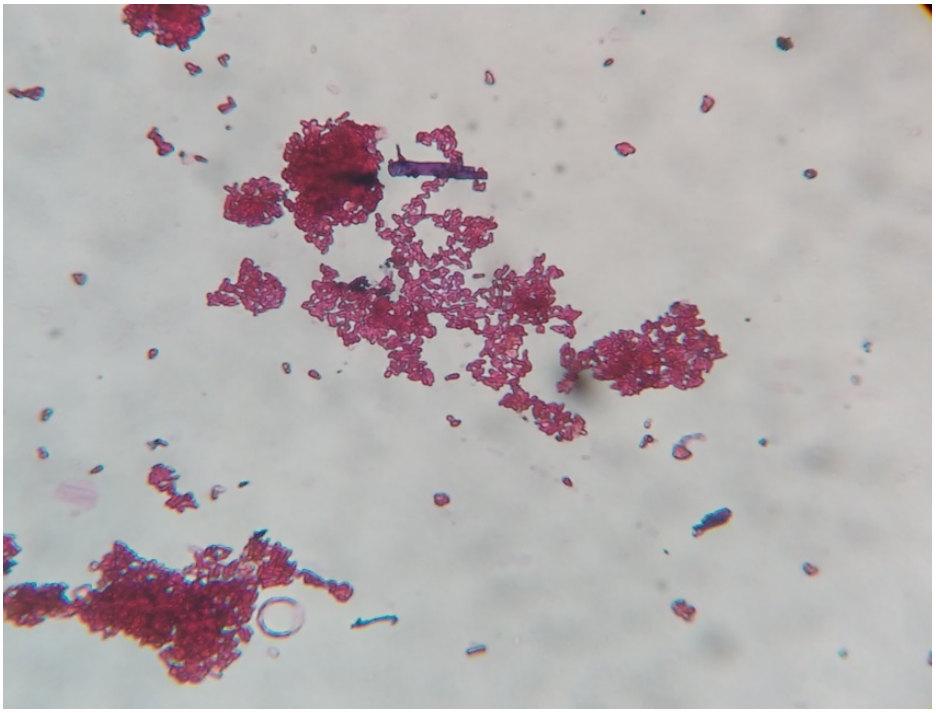


Fig 30,31. Gram stain showing characteristic spores and hyphae



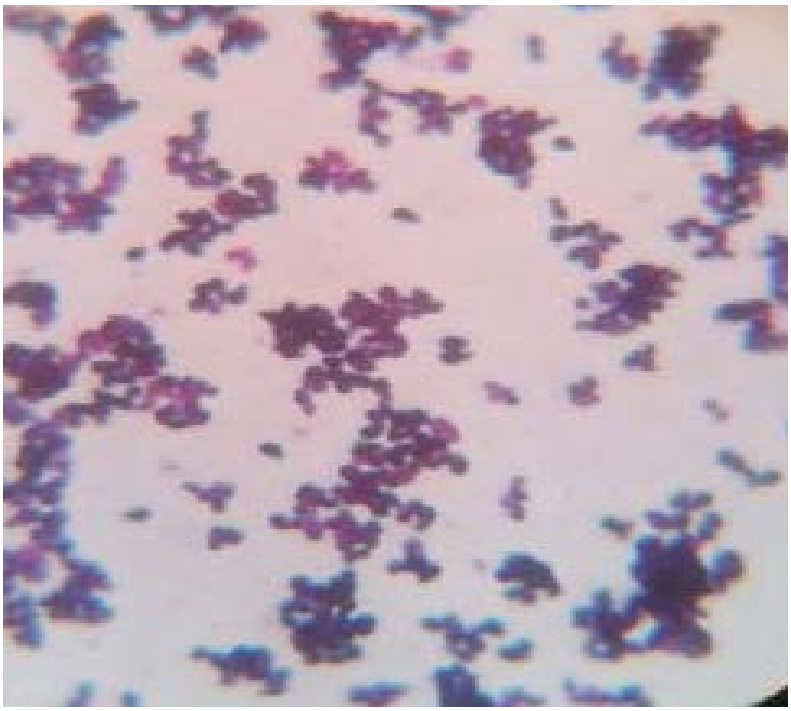


Fig 32. Gram stain showing characteristic spores and hyphae

DISCUSSION

DISCUSSION

AGE DISTRIBUTION

In this study, the age group most commonly affected with pityriasis versicolor was 21 - 30years (43%) followed by 30 – 39 years (22%). The youngest age in this study was a 13 years old male and oldest age was a 65 old male patient respectively. This is in accordance with the study by Sanjeev Grover *et al* in 2003, in which 39.6% of those affected with PV belonged to 21 – 30 years age group and 30% belonged to 31 – 40 years age group.[46] In 2002, Dutta *et al* and Gatha Rao *et al*, also reported a 39.6% and 30% incidence of PV respectively in the 21 – 30 years age group. This may be due to increased sebum production in the skin in this age group.[17,19]

SEX DISTRIBUTION

In the present study, males (70%) were more commonly affected with pityriasis versicolor than females (30%). This is similar to the study by Gatha Rao *et al* in 2002 and Gurumohan Singh *et al* in 1996 in which they observed 73.3% incidence and 75.3% incidence respectively.[17, 47] In contrast, Imwidthya *et al* in 1988 had observed a higher incidence in females which may be due to the increased cosmetic concern in the females residing in that part of the country [48]

The higher incidence in males in this study, may be attributed to increased outdoor activities of males for occupational purposes, putting them at a higher risk of sun exposure and humidity, which favours the growth of *Malassezia* yeasts.

DISTRIBUTION OF OCCUPATION AND RESIDENCE

In this study, majority of the patients were students(29%) followed by manual labourers (19%). This is in accordance with studies by Sudip *et al*, where majority were students (29.09%), followed by housewives (20%) and manual laborers (15.46%). [49] In our study, pityriasis versicolor was common among urban population (65%) compared to rural population(35%), as our institute caters predominantly to urban population.

FAMILY HISTORY

In the present study, a positive family history of lesions similar to pityriasis versicolor was observed in 31% of patients. Gatha Rao *et al* observed a positive family history in 38% of the PV patients. [17] Faergemann and Fredreichson in 1979 observed 18% of PV patients with history of similar lesions in other family members. [54] Though a positive family history has been noticed in more cases than chance would permit, whether it is genetically determined or due to increased exposure of the family members to the causative fungal agent, has to be evaluated.

SYMPTOMS AND SWEATING

In this study, 32 % of patients had pruritus. Similarly, in the study conducted by Gatha Rao *et al* in 2002, 30% of the patients had pruritus. [17] In the present study, 58% of the patients had excessive sweating.

DURATION OF THE INFECTION

In the present study, the duration of lesions in the majority (64%) of PV patients was between 1 to 6 months. According to Kristany *et al,* 72% of the PV patients presented with duration of lesions in the 1 month – 1year range.[51]

RECURRENCE

In the present study, 35% of the patients had history of recurrence. Kristany et al observed a 26% recurrence in Indonesian population. 51 On the contrary, Gatha Rao et al observed recurrent PV in only 3% of the Indian patients.17 Pityriasis versicolor tends to be recurrent in patients living in high humid conditions and in those with increased outdoor activities as they are more prone for increased sweating. Conditions like HIV infection, tuberculosis, prolonged steroid intake and malignancy predispose to recurrent PV.

DERMATOLOGICAL AND SYSTEMIC ASSOCIATIONS

Seborrhoeic dermatitis(36.2%) was the most common dermatological condition associated with pityriasis versicolor followed by acne(24.13%). All the three conditions have *Malassezia* species involved in their pathogenesis. Crespo Erchiga *et al* reported that seborrhoeic dermatitis (40%) was the most common condition associated with PV followed by dermatophytosis.[52]

Among the 100 patients , 28 % patients had systemic associations. Diabetes mellitus was the most commonly associated systemic disease(39.3%), followed by hypertension(25%) and pulmonary tuberculosis(7.1%). This is similar to the study conducted by Sudip et al, in which he found that diabetes mellitus (2.73%) was the commonest association but relatively this was in small proportion compared to this study.[49]

BLOOD GROUPING

Majority of the patients had blood group A (52%), followed by blood group O (38%). This is contrary to the study conducted by Kareema et al in 2014, blood group O was most commonly associated with PV in 74% of the patients followed by blood group A in 13.9% of patients and blood group B in 10.7% of patients.[53]

EXTENT OF INVOLVEMENT

Majority of the patients (70%) had more than 30% BSA involvement , followed by more than 10% BSA (26%) . The area of involvement is mainly in the sebum rich regions of the body.

SITE OF INVOLVEMENT

In this study, the major site of involvement of pityriasis versicolor was back (46%), followed by face and neck (26%)**.** This is similar to the study by Gatha Rao *et al* who reported 70% lesions in the back of trunk.[17] Gupta *et al* in 2001 and Imwidthaya *et al* in 1988 also observed PV lesions to be common on the back, followed by face.[48,54] Kristany *et al* in 2008 observed 76.5 % lesions were in the back of the trunk, followed by 16% in chest.[51] The increased involvement of the trunk, face and neck may be due to the presence of numerous sebaceous glands in these sites and also due to the hot and humid climate prevalent in South India, resulting in excessive sweating in these sites, which predisposes to PV.

CLINICAL TYPE

In the present study, achromic PV lesions (68%) were more common compared to the chromic lesions (23%)**.** This correlates with the

studies conducted by Imwidthya *et al,* Gatha Rao *et al* and Crespo Erchiga *et al,* who reported hypopigmented lesions with an incidence of 83%, 75% and 58% respectively. [17,48,52] Pityriacitrin and similar indole alkaloids produced by *M. furfur* and probably other *Malassezia* species induce apoptosis of human melanocytes and this is one likely explanation for the higher incidence of hypopigmented lesions.[17,51,52]

ISOLATION RATE

The isolation rate of *Malassezia* species from clinically diagnosed cases of pityriasis versicolor was 76% in this study. Faergemann *et al* in 1979 reported a 100% recovery rate, Dutta *et al,* 59% in 2002, Kindo *et al,* 68.75% in 2004. [19,50,55] The differences in the isolation rates in the various studies may be due to the differences in sampling techniques and the use of different media for culture like SDA with olive oil, modified Dixon's medium and Leeming and Notman agar and.[17,54]

SPECIES ISOLATION

In the present study, the most common species isolated from culture positive cases of PV was *M. globosa (48.7%)* followed by *M. sympodialis* (25%) and *M. furfur (*10%). This is in accordance with the studies by Crespo Erchiga *et al* in 1999, Dutta *et al* in 2002, and

Thayikannu *et al* in 2013 who observed the most common isolate to be *M.globosa*. [19,37,52] But Gupta *et al* in 2001 and Kindo *et al* in 2004 reported *M.sympodialis* to be the most common isolate followed by *M.globosa*.[54,55] Sanjeev grover *et al* in 2003 reported *M. furfur* to be the most common isolate recovered from PV lesions followed by *M.globosa.* [46]
In this study, *M. globosa* was the most common species isolated from both achromic (48.9%) and chromic (48.2%) types of PV. This is in concordance with the studies Thayikannu *et al,* who also reported *M. globosa* to be the most common isolate obtained from both achromic and chromic types of PV. [37] The higher esterase activity and lipase activity and hence the higher pathogenicity may be the likely cause for *M. globosa* being the most common isolated species.[37,47]

In this study, *M. globosa* was the most common species isolated from both first episode (52%) and recurrent attacks (43%) of PV. Similarly Gupta *et al* in 2001 and Kindo *et al* in 2004, reported *M. globosa* was the most common species isolated from both 55 % first episode and 40% recurrent lesions of PV followed by *M.sympodialis.*[54,55]

CONCLUSION

CONCLUSION

- Pityriasis versicolor is most commonly seen in the age group between 21 - 30years.
- Males outnumbered the females
- Pityriasis versicolor was more common among the students.
- Urban population was affected more than the rural population.
- Cosmetic concern was the main complaint
- Duration of lesion in majority of the patients was 1 to 6 months
- Diabetes was found to be the most common systemic associated disease
- Seborrheic dermatitis was the most common dermatological association Sweating was a major predisposing factor.
- Pityriasis versicolor was more commonly seen in patients with blood group A
- Majority of the patients had more than 30% of body surface area involvement.
- Most common site affected was back.
- Achromic pityriasis versicolor was more common than chromic PV. Follicular type was seen only in patients with achromic PV. Paranasal type was seen in the adolescent age group

- Culture was positive in 76% patients
- *Malassezia globosa* was the most common species isolated.
- *Malassezia globosa* was the most common species isolated in hyperpigmented and hypopimented lesions
- *Malassezia globosa* was the most common species isolated in both patients with first and recurrent episodes
- Other species isolated in this study were *Malassezia sympodialis*, *Malassezia furfur*, *Malassezia restricta*, *Malassezia obtusa and Malassezia sloofiae.*
- In this study, A blood group was the most common blood group associated with pityriasis versicolor which is different from the previous study in which O blood group is the commonest association. Hence, further studies in a larger population would provide more conclusive evidence with regard to the blood groups.

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ANNEXURES

INFORMATION TO PARTICIPANTS

Investigators : Dr.R.Snekavalli

Dr.A.Ramesh

Dr.R.Madhu

Name of Participant:

Title: **CLINICO EPIDEMIOLOGICAL AND MYCOLOGICAL STUDY OF PITYRIASIS VERSICOLOR**

You are invited to take part in this study. The information in this document is meant to help you decide whether or not to take part. Please feel free to ask if you have any queries or concerns

We are conducting a study on **CLINICO EPIDEMIOLOGICAL AND MYCOLOGICAL STUDY OF PITYRIASIS VERSICOLOR"** among patients attending Rajiv Gandhi Government General Hospital, Chennai and for that your participation may be valuable to us.

The purpose of this study is to determine the epidemiology, clinical profile, predisposing factors, common causative agents of pityriasis versicolor.

In this study history of patient will be taken,examination and routine blood test will be taken ,fungal culture will be done.

The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of Investigator Signature of the participant

PATIENT CONSENT FORM

Title of the study : CLINICO EPIDEMIOLOGICAL AND MYCOLOGICAL STUDY OF PITYRIASIS VERSICOLOR

Name of the Participant:

Name of the Principal investigator: Dr.R.Snekavalli

Name of the Institution : Rajiv Gandhi Government General Hospital, Chennai

Documentation of the informed consent

I _____ have read the information in this form (or it has been read for me). I was free to ask any questions and they have been answered. I am over 18 years of age and exercising my free power of choice, hereby give my consent to be included as a participant in the study.

1. I have read and understood this consent form and the information provided to me.
2. I have had the consent document explained to me.

3. I have been explained about the nature of the study.
4. My rights and responsibilities have been explained to me by the investigator.
5. I have informed the investigator of all the treatments I am taking or have taken in the past 1 year including any native (alternative) treatment.
6. I agree to cooperate with the investigator and I will inform her immediately if I suffer unusual symptoms.
7. I have not participated in any research study at any time .
8. I am aware of the fact that I can opt out of the study at any time without having to give any reason and this will not affect my future treatment in this hospital.
9. I hereby give permission to the investigators to release the information obtained from me as a result of participation in this study to the sponsors, regulatory authorities,Govt agencies and IEC.I understand that they are publicly presented.
10. My identity will be kept confidential if my data are publicly presented.
11. I have had my questions answered to my satisfaction.
12. I have decided to be in the research study

10. I am aware that if I have any question during this study, I should contact at one of the addresses listed above. By signing this consent form I attest that the information given in this document has been

clearly explained to me and apparently understood by me. I will be given a copy of this consent document.

Participant's initials:_____

For adult participants:

Name and signature/thumb impression of the participant(or legal representative if participant incompetent)

_____ _____ _____

Name Signature Date

Name and signature of impartial witness (required for illiterate patients):

_____ _____ _____

Name Signature Date

Address and contact number of the impartial witness:

Name and Signature of the investigator or his representative obtaining consent:

_____ _____ _____

Name Signature Date

ஆராய்ச்சி தகவல் தாள்

ஆராய்ச்சியாளர்கள் : **டாக்டர். இரா. சிநேகவள்ளி**
டாக்டர். சி.ஜானகி
டாக்டர். இரா. மது

பங்கேற்பாளரின் பெயர் :

தலைப்பு : **அழகு தேமல் பற்றிய மருத்துவ மற்றும் காரணிகளை கண்டறியும் ஆய்வு.**

- தங்களை இந்த ஆராய்ச்சியில் பங்கேற்குமாறு கேட்டுக்கொள்கிறோம். இந்த படிவத்தில் உள்ள தகவல் மூலமாக இந்த ஆராய்ச்சியில் ஈடுபடலாமா என்பதை தீர்மானித்து கொள்ளலாம். தங்களுக்கு ஏதேனும் சந்தேகம் இருந்தால் எந்தவித தயக்கமும் இன்றி கேட்டுக்கொள்ளலாம்.
- அழகு தேமல் நோயின் முன்மொழியும் காரணிகள், வகைகள், காரணி பூஞ்சைகள் பற்றி கண்டறிவதே இந்த ஆராய்ச்சியின் நோக்கமாகும்.
- ஆராய்ச்சியில் நோயாளிகளின் அடையாளம் பாதுகாக்கப்படும், எவரிடமும் பகிர்ந்து கொள்ப்படமாட்டாது. ஆராய்ச்சித் தகவல்கள் பகிர்ந்து கொள்ளப்படும் போது தனிப்பட்ட முறையில் அடையாளம் காணக்கூடிய எந்தத் தகவலும் பகிர்ந்து கொள்ளப்படமாட்டாது.
- இந்த ஆய்வில் பங்கெடுத்துக் கொள்வது உங்கள் தனிப்பட்ட விருப்பம், எந்த நேரத்திலும் இந்த ஆய்விலிருந்து விலகிக் கொள்ள உங்களுக்கு முழு உரிமை உண்டு.
- இந்த ஆய்வின் இறுதியில் ஆய்வின் முடிவுகள் உங்களிடம் தெரிவிக்கப்படும்.

ஆராய்ச்சியாளரின் கையொப்பம் பங்கேற்பவரின் கையொப்பம்

தேதி :

பங்கேற்பாளரின் பெற்றோர் அல்லது காப்பாளரின்
பெயர், கையொப்பம் / இடதுகை பெருவிரல் ரேகை

.....	.....	.....
பெயர்	கையெழுத்து	தேதி

பாரபட்சமற்ற சாட்சியின் பெயர், கையெழுத்து / இடதுகை பெருவிரல் ரேகை

.....	.....	.....
பெயர்	கையெழுத்து / விரல்ரேகை	தேதி

ஆராய்ச்சியாளர் பெயர் மற்றும் கையெழுத்து

.....	.....	.....
பெயர்	கையெழுத்து	தேதி

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI-3

EC Reg No.ECR/270/Inst./TN/2013
Telephone No. 044 25305301
Fax : 044 25363970

CERTIFICATE OF APPROVAL

To
Dr. R.Snekavalli,
Postgraduate M.D.(Dermatology, Venereology and Leprosy),
Madras Medical College,
Chennai – 600 003.

Dear Dr. R.Snekavalli,

The Institutional Ethics Committee has considered your request and approved your study titled **"Clinico Epidemiological and mycological study of pityriasis versicolor" No.10092014.**

The following members of Ethics Committee were present in the meeting held on 02.09.2014 conducted at Madras Medical College, Chennai-3.

1.	Dr.C.Rajendran, M.D.,	: Chairperson
2.	Dr.R.Vimala, M.D., Dean, MMC, Ch-3	: Deputy Chairperson
3.	Prof.B.Kalaiselvi, M.D., Vice-Principal, MMC, Ch-3	: Member Secretary
4.	Prof.R.Nandhini, M.D., Inst.of Pharmacology, MMC	: Member
5.	Dr.G.Muralidharan, Director Incharge, Inst.of Surgery	: Member
6.	Prof.K.Ramadevi, Director i/c, Inst.of Biochemistry, MMC	: Member
7.	Prof.Saraswathy, M.D., Director, Pathology, MMC, Ch-3	: Member
8.	Prof.Tito, M.D., Director i/c, Inst.of Internal Medicine, MMC	: Member
9.	Thiru S.Rameshkumar, Administrative Officer	: Lay Person
10.	Thiru S.Govindasamy, B.A., B.L.,	: Lawyer
11.	Tmt.Arnold Saulina, M.A., MSW.,	: Social Scientist

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Member Secretary, Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003

PROFORMA

Case No :

Name : Age : Sex:: OP No :

Address : Occupation :

Chief Complaints:

Duration : Past history : previous episode single or muliple

Family History :

Marital History :

Sexual history:

Predisposing factors :
- Pregnanacy
- Diabetes
- TB
- Immunosuppressive therapy
- HIV
- Transplant patients
- Malnutrition
- Endocrine diseases

Associated dermatological disorders:
- Acne
- Seborrhoeic dermatitis
- Psoriasis
- Dermatophytosis
- Candidiasis
- Acanthosis nigricans
- Other dermatological conditions

General examination :
- Anemia

Systemic Examination :

Obesity / wt
Nutritional status
CVS
RS
Abdomen
CNS
Others

DERMATOLOGICAL EXAMINATION:

Sie of involvement: scalp/ face/ neck/ shoulder/ front of chest/ abdomen/ arm/ forearm/
Thigh/ legs/others
Palms and soles:
Mucosa:
Hair:

Percentage of surface area:

Type of the lesion: Achromic/ chromic/
peifollicular/plaque/popular/erythematous/pityrosporum folliculitis/others

Investigations

Blood haemogram

Blood sugar

RFT

LFT

Blood lipid profile

Blood grouping and typing

HIV ELISA, VDRL for syphilis

Potassium hydroxide mount

Culture

TREATMENT GIVEN; Topical:

Systemic:

ABBREVIATIONS

AhR	-	Aryl hydrocarbon Receptor (AhR)
AIDS	-	Acquired immunodeficiency syndrome
CFW	-	Calcoflour White stain
CSB	-	Chicago sky blue
DMSO	-	Dimethyl Sulfoxide
ELISA	-	Enzyme Linked Immunosorbent Assay
FFA	-	Free fatty acids
HIV	-	Human Immunodeficiency Virus
HPE	-	Histopathological Examination
KOH	-	Potassium hydroxide
PAS	-	Periodic Acid Schiff
PFGE	-	Pulsed Field Gel Electrophoresis
PV	-	PityriasisVersicolor
RFLP	-	Restriction Fragment Length Polymorphism
ROS	-	Reactive Oxygen Species
SDA	-	Sabouraud Dextrose Agar
SD	-	Seborrheic Dermatitis
UV	-	Ultraviolet

SL NO	AGE	SEX	OCCUPATION	RESIDENCE	MARIETAL STATUS	FAMILY HISTORY	EXCESSIVE SWEATING	DURATION	SYMPTOM	SYS ASSO	ASSO DERMAT	RECURRENCE	SITE	AREA OF	PV TYPE	BLD G&T	10%KOH	CULTURE
1	44	M	security	U	M	N	Y	9 M	P	DM	ACNE	Y	BACK	>30%	HYPO	A+	+	G
2	25	F	farmer	R	M	N	Y	4M	C		SD	Y	BACK	>30%	Both	A-	+	G
3	28	M	coolie	U	NM	N	Y	2Y	P		D	Y	FACE & NECK	>30%	HYPO	A+	+	G
4	26	M	plumber	R	NM	N	N	3W	C			Y	BACK	>50%	HYPO	B+	+	
5	29	M	tailer	U	M	Y	N	7W	C		LP	Y	BACK	>10%	HYPO	O+	+	S
6	43	M	shopkeeper	R	M	Y	N	5M	P		CANDIDIASIS	N	FACE & NECK	>10%	HYPER	O+	+	R
7	65	M	cook	U	M	N	N	8M	C	DM		N	FACE & NECK	>10%	HYPO	AB+	+	G
8	44	F	housewife	R	M	Y	N	3M	P	DM	LA	Y	FACE & NECK & CHEST	>10%	HYPO	O+	+	F
9	22	M	student	U	NM	N	N	5M	C		PYODERMA	N	FACE & NECK	>10%	HYPER	O+	+	G
10	19	M	student	R	NM	N	N	4M	C		IGH	N	BACK	>30%	HYPO	A+	+	
11	13	F	student	U	NM	N	N	1Y	C			Y	BACK	>30%	HYPO	A-	+	
12	17	M	student	R	NM	N	N	2M	C		CMN	Y	BACK	>30%	HYPO	A+	+	S
13	44	M	driver	U	M	N	Y	4M	C		SD	N	FACE & NECK	>10%	HYPO	O+	+	G
14	25	F	farmer	U	M	Y	Y	2Y	C		D	Y	BACK	>30%	HYPO	A+	+	O
15	28	M	coolie	U	NM	Y	Y	3W	C	RT	ACNE	N	FACE & NECK	>30%	HYPO	A+	+	F
16	26	M	plumber	R	NM	N	Y	7W	P		ACNE	Y	BACK	>10%	Both	O+	+	G
17	29	F	tailer	U	M	N	N	5M	C		D	N	FACE & NECK	>10%	HYPER	A+	+	G
18	43	M	shopkeeper	U	M	N	Y	8M	P	CKD	SD	Y	FACE & NECK & CHEST	>10%	HYPER	A+	+	
19	65	M	coolie	U	M	N	Y	3M	C	DM		Y	FACE & NECK	>10%	HYPO	O+	+	G
20	44	M	housewife	R	M	Y	N	5M	C	DM		Y	BACK	>30%	HYPO	O+	+	S
21	22	M	student	U	NM	N	N	4M	P		ACNE	Y	BACK	>30%	HYPO	O+	+	
22	19	M	student	U	NM	N	Y	1Y	C		ACNE	N	FACE & NECK	>10%	HYPO	O-	+	F
23	13	F	student	U	NM	N	N	2M	C	NS	D	Y	BACK	>50%	HYPO	O+	+	R
24	17	M	student	R	NM	N	Y	4M	C		ACNE	Y	FACE & NECK	>30%	HYPER	O+	+	G
25	44	M	driver	U	M	N	Y	2Y	C	HT		N	BACK	>30%	HYPER	O+	+	F

26	25	F	farmer	R	M	Y	N	3W	P			N	BACK	>10%	HYPER	A+	+	G
27	28	M	porter	U	NM	Y	Y	7W	C			N	FACE & NECK	>30%	HYPO	A-	+	G
28	26	M	plumber	R	NM	N	Y	5M	C		ACNE	Y	BACK	>30%	HYPO	A+	+	
29	29	F	tailer	U	M	N	N	9M	C			Y	BACK	>10%	HYPO	A-	+	
30	43	M	shopkeeper	U	M	N	N	3M	C			N	FACE & NECK	>30%	HYPO	O+	+	R
31	58	M	coolie	U	M	N	Y	5M	P	DM		Y	BACK	>30%	HYPO	O+	+	SL
32	44	F	housewife	R	M	Y	N	4M	C		SD	N	BACK	>30%	HYPO	O+	+	
33	22	M	student	U	NM	N	Y	1Y	P		SD	N	BACK	>30%	HYPO	A+	+	G
34	19	M	student	R	NM	N	Y	2M	C		ACNE	Y	FACE & NECK	>30%	HYPO	A-	+	S
35	13	F	student	U	NM	N	N	4M	C		D	Y	BACK	>30%	HYPO	A+	+	S
36	17	M	student	R	NM	N	Y	2Y	P		ACNE	N	BACK	>30%	HYPO	B+	+	G
37	44	M	driver	U	M	N	Y	3W	C	HT		N	BACK	>30%	HYPO	A+	+	R
38	25	F	farmer	U	M	Y	Y	7W	P		LICHEN PLANUS	Y	FACE & NECK	>10%	HYPER	A-	+	G
39	28	M	coolie	U	NM	Y	Y	4M	C		SD	Y	FACE ,NECK & ARM	>30%	HYPO	A+	+	
40	26	M	plumber	R	NM	N	Y	8M	C	CKD		N	FACE & NECK	>30%	HYPO	O+	+	G
41	29	F	tailer	U	M	Y	N	3M	P			Y	BACK	>30%	Both	A+	+	G
42	43	M	shopkeeper	R	M	N	N	5M	C	DM	SD	Y	BACK	>30%	HYPO	O+	+	
43	58	M	coolie	U	NM	Y	Y	4M	P	HT	POMPHOLYX	N	BACK	>30%	HYPO	A+	+	G
44	44	F	housewife	R	M	Y	Y	1Y	C			Y	FACE & NECK	>30%	HYPO	O+	+	
45	22	M	student	U	M	N	N	2M	C		ACNE	N	FACE & NECK	>30%	Both	B+	+	S
46	19	M	student	R	NM	N	N	4M	P		SD	N	FACE ,NECK & SHOULDER	>30%	HYPO	O+	+	G
47	13	F	student	U	NM	N	N	2Y	C			N	FACE & NECK	>30%	HYPO	O+	+	O
48	17	M	student	U	NM	N	N	3W	P		SD	N	FACE & NECK	>30%	HYPO	B+	+	
49	44	M	driver	U	M	Y	Y	7W	C			N	BACK	>30%	HYPO	O+	+	G
50	25	F	farmer	R	M	N	Y	5M	C		D	N	BACK	>30%	HYPER	O+	+	F
51	28	M	coolie	U	NM	N	Y	8M	P			Y	BACK AND CHEST	>30%	HYPER	A-	+	
52	26	M	plumber	R	NM	N	Y	3M	C		SD	Y	BACK	>30%	HYPO	A+	+	S
53	29	F	tailer	U	M	N	Y	5M	P			Y	BACK AND ABDOMEN	>30%	HYPO	A+	+	S
54	43	M	shokeeper	R	M	N	N	4M	C		SK	Y	BACK	<10%	HYPO	A+	+	G

55	66	M	coolie	U	M	N	Y	1Y	C	DM		N	BACK	>10%	HYPO	B+	+	
56	44	F	housewife	U	M	Y	N	2M	P	HT		N	BACK	>30%	HYPER	O+	+	S
57	22	M	driver	U	M	N	Y	4M	C		ACNE	N	BACK	>30%	HYPO	B+	+	
58	19	M	student	R	NM	N	N	2Y	P		SD	Y	BACK	>30%	HYPO	O+	+	
59	13	M	student	U	NM	Y	N	3W	C			N	BACK	>30%	HYPO	A+	+	G
60	17	M	student	U	NM	N	N	7W	C			N	BACK AND FACE	>10%	HYPER	A-	+	G
61	44	M	driver	U	M	N	Y	5M	P		ACNE	N	BACK	>30%	Both	A+	+	G
62	25	F	farmer	R	M	Y	Y	8M	C			N	BACK	>30%	HYPO	O+	+	F
63	28	M	coolie	U	M	N	Y	3M	P			N	FACE & NECK	>30%	HYPO	A+	+	S
64	26	M	plumber	U	NM	N	Y	5M	C		STD	Y	BACK	>10%	HYPER	O+	+	O
65	29	F	tailer	U	M	N	N	4M	C			N	FACE & NECK	>30%	HYPO	A+	+	
66	43	M	shopkeeper	R	M	N	Y	1Y	P		SD	N	BACK	>30%	HYPO	O+	+	R
67	58	M	coolie	U	M	N	Y	2M	C	PT		N	BACK	>30%	HYPER	O+	+	G
68	44	F	housewife	U	M	N	N	4M	P	DM	WART	N	BACK AND ARM	>30%	Both	B+	+	S
69	22	M	student	U	M	N	Y	2Y	C		SD	N	BACK	>30%	HYPO	A+	+	
70	19	M	coolie	R	NM	Y	Y	7W	C		SD	N	BACK	>10%	HYPO	A-	+	F
71	13	F	student	U	NM	Y	N	7W	P		ACNE	Y	BACK	>30%	HYPER	A+	+	S
72	17	M	student	U	NM	N	N	5M	C		ACNE	N	FACE & NECK	>30%	HYPO	B+	+	O
73	44	M	teacher	U	M	N	Y	8M	P	PT		Y	FACE & NECK	>30%	HYPO	A+	+	G
74	25	F	farmer	U	M	Y	N	3M	C		D	N	FACE & NECK	>30%	HYPO	A+	+	G
75	28	M	painter	U	NM	N	Y	5M	C			Y	BACK	>30%	HYPER	A-	+	O
76	26	M	plumber	U	NM	N	Y	4M	P		SD	N	FACE & NECK	>10%	HYPO	A+	+	
77	29	F	tailer	U	M	N	N	1Y	C		D	N	FACE & NECK	>30%	Both	O+	+	S
78	43	M	shokeeper	R	NM	N	Y	2M	P	B	D	Y	BACK	>30%	HYPER	A+	+	G
79	58	M	coolie	U	M	N	Y	3M	C	DM		N	BACK	>10%	HYPO	A-	+	G
80	44	F	housewife	R	M	Y	Y	2Y	C	HT		N	BACK	>30%	HYPER	O+	+	G
81	22	M	student	U	NM	N	N	3W	C		SD	N	FACE & NECK	>30%	HYPO	A+	+	

82	19	M	coolie	U	NM	Y	Y	7W	C		SD	N	FACE & NECK	>30%	Both	A-	+	G
83	13	F	student	U	NM	Y	N	5M	P		CMN	N	BACK	>30%	HYPER	A+	+	S
84	17	M	student	U	NM	Y	Y	7M	C		ACNE	Y	BACK	>30%	HYPO	O+	+	
85	44	M	electrician	U	M	N	Y	3M	C	HT		N	BACK	>10%	HYPER	O+	+	G
86	25	F	farmer	R	M	Y	Y	5M	C	HYP		Y	FACE & NECK	>30%	HYPO	B+	+	
87	28	M	coolie	U	NM	N	Y	4M	C			N	BACK	>30%	HYPO	O+	+	G
88	26	M	plumber	R	NM	N	N	1Y	C		D	Y	BACK	>10%	HYPER	O+	+	F
89	29	F	housekeeper	U	NM	Y	Y	6M	C			N	BACK	>30%	HYPO	A+	+	S
90	43	M	shokeeper	R	M	N	Y	4M	C			N	BACK	>30%	Both	A+	+	S
91	58	M	coolie	U	M	Y	Y	2Y	P	HT		Y	BACK	>10%	HYPER	A+	+	G
92	44	F	housewife	R	M	Y	N	2W	C	DCLD		N	BACK	>30%	HYPO	O+	+	G
93	22	M	student	U	NM	N	N	7W	C			Y	BACK	<10%	HYPO	O+	+	
94	19	M	student	R	NM	N	N	5M	C		SD	Y	BACK	>30%	HYPO	O+	+	S
95	13	F	student	U	NM	Y	Y	8M	C			Y	BACK	>30%	HYPER	A+	+	S
96	17	M	student	R	NM	Y	Y	3M	P		SD	N	BACK	>30%	HYPO	A+	+	G
97	44	M	driver	U	M	N	N	4M	C	DM	SD	N	BACK	>10%	HYPO	A+	+	R
98	25	F	farmer	R	M	Y	Y	4M	P		D	Y	BACK	>30%	HYPO	A+	+	G
99	28	M	coolie	U	NM	N	N	1Y	C			N	BACK	>10%	HYPO	A-	+	
100	26	M	plumber	U	NM	N	Y	3M	C	RT	D	N	BACK	>10%	HYPO	A+	+	S

KEY TO MASTER CHART

SEX

M	MALE
F	FEMALE

RESIDENCE

U	URBAN
R	RURAL

FAMILY HISTORY, EXCESSIVE SWEATING, RECURRENCE

Y	YES
N	NO

DURATION OF INFECTION

M	MONTH
Y	YEAR
W	WEEK

SYMPTOM

P	PRURITIC
C	COSMETIC

SYSTEMIC ASSOCIATION

DM	DIABETES MELLITES
RT	RENAL TRANSPLANT
PT	PULMONARY TUBERCULOSIS
DCLD	DECOMPENSATED LIVER DISEASE
CKD	CHRONIC KIDNEY DISEASE
HT	HYPERTENSION
HYP	HYPOTHYROIDISM
NS	NEPHROYIC SYNDROME
B	BRONCHIECTASIS

ASSOCIATED DERMATOLOGICAL DISORDER

SD	SEBORRHEIC DERMATITIS
LP	LICHEN PLANUS
LA	LICHEN AMYLOID
CMN	CONGENITAL MELANOCYTIC NEVUS
D	DERMATOPHYTOSIS
IGH	IDIOPATHIC GUTTATE HYPOMELANOSIS
SK	SEBORRHEIC KERATOSIS
STD	STASIS DERMATITIS

TYPE OF PV

HYPO	HYPOPIGMENTED
HYPER	HYPERPIGMENTED

CULTURE

G – *Malassezia globosa*

S - *Malassezia sympodialis*

F – *Malassezia furfur*

SL - *Malassezia sloofiae*

R - *Malassezia restricta*

O - *Malassezia obtusa*