"A CROSS-SECTIONAL STUDY OF PITYRIASIS VERSICOLOR WITH REFERENCE TO CULTURE POSITIVITY AT KLE'S DR. PRABHAKAR KORE HOSPITAL AND MRC, BELGAUM"

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DISSERTATION

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of the requirements for the degree of

Doctor of Medicine

In

DERMATOLOGY, VENEREOLOGY AND LEPROSY

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May 2011

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I hereby declare that this dissertation / thesis entitled "**A cross-sectional study of pityriasis versicolor with reference to culture positivity at KLE'S Dr. Prabhakar Kore Hospital and MRC, Belgaum**" is a bonafide and genuine research work carried out by me under the guidance of **Dr. B. Siddaramappa** **M.D., D.V.D.** Professor, Department of Dermatology.

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LIST OF ABBREVATIONS USED

1.	CAPD	=	Continuous ambulatory peritoneal dialysis
2.	M	=	Malassezia
3.	MDA	=	Modified Dixon Agar
4.	M.R.C	=	Medical Research Centre
5.	O.P.D	=	Out Patient Department
6.	PAS	=	Periodic Acid Schiff
7.	P	=	Pityrosporum
8.	SDA	=	Sabrouraud’s Dextrose Agar
9.	SPT	=	Skin Prick Test
10.	DMSO	=	Dimethyle Sulphoxide

ABSTRACT

Background and Objectives : To study the culture positivity rate in clinically diagnosed cases of pityriasis versicolor and to study the various clinical presentation in different skin colour people, risk factors and demographic characteristics of pityriasis versicolor.

Material and Methods : The present study is a one year cross- sectional study from November 2008 to October 2009. The patient's demographic data, location of lesion, risk factors were noted in the pre-tested and pre-designed proforma after taking consent. All the clinically diagnosed cases of pityriasis versicolor were subjected to skin scraping for KOH examination and culture for Malassezia growth.

Results : A total of 150 cases were studied, and Malassezia growth was obtained in 82 patients. M.sympodialis was the commonest species isolated. Maximum cases were in the age group of 21-30 years (39.33%). Male to female ratio was 4.17:1. In the study 60% of the patients were asymptomatic. The neck was the most common site involved and majority of patients had hypopigmented lesions.

Conclusion : M.sympodialis was the most common species involved in pityriasis versicolor in North Karnataka.

Key words : Pityriasis versicolor

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INTRODUCTION

Pityriasis (tinea) versicolor is a common, superficial fungal infection of the skin. It is more common than what might be inferred from dermatological statistics. It has been reported in upto 50 percent of general population.

Various other names have been given to this condition, including tinea versicolor, tinea flava, dermatomycosis furfuracea, chromophytosis, achromia parasitica, hodi-potsy, kleine-flechte and liver spots.

It is a chronically recurring fungal infection with no long term treatment or cure because pityriasis versicolor is seldom symptomatic and many persons having the disease do not seek medical attention.

It produces multiple hypopigmented and hyperpigmented macular scaly lesions, usually on the chest, back, neck or face, which is disfiguring and embarrassing to the patient. It is seen as hyperpigmented variety in fair people and hypopigmented variety in dark people.

Pityriasis versicolor has a wide geographic distribution. It is most commonly seen in hot and humid tropical regions like India.

During the summer, when the skin of some races tan and patches of pityriasis versicolor appear, the contrast between them increases, making the patient more aware of it. In addition, during the summer, many people wear less clothing, making the lesions more visible to others.

Most of the cases start during puberty or thereafter. There are case reports of patients manifesting the disease as early as neonatal life. It is less frequent in people over fifty years old.

The disease affects all races and both sexes, usually between the range of 16 and 40 years of age.

There is little evidence that the disease is contagious. Nonetheless, other factors that lead to an increased susceptibility should be considered, including genetic predisposition, poor general health, cancer, oral contraceptives, hyperhidrosis, pregnancy and other immunocompromised states.

The causative organism of pityriasis versicolor is the lipophilic yeast of Malassezia species.

Because of the lipid requirement of Malassezia yeast, both the yeast and the mycelia forms tend to be found most often on lipid-rich areas of the body including face, scalp and trunk. It is primarily located on the chest, back, upper arm, although facial involvement may also occur in some patients.

The etymology of the name for pityriasis versicolor comes from the Greek word "**Pytra**", meaning abnormal proliferation. "**Versicolor**" is derived from the Latin word meaning many colors. The early adaptation "Tinea Versicolor" evolved from a misconception that the disease was caused by a dermatophyte. Tinea versicolor has persisted in the American literature as the term for the disease, while in Europe the term pityriasis versicolor has been adopted.

Some patients may experience mild pruritus, although for most patients it is asymptomatic and is chiefly a cosmetic concern.

The diagnosis is relatively easy, and is essentially based on clinical examination of the skin lesions of pityriasis versicolor along with yellow or golden fluorescence on Wood's light examination and finding hyphae and spores on direct microscopy with potassium hydroxide. Culture of this organism is very difficult, not all cases are culture positive due to contamination.

There are a number of options available for treatment of pityriasis versicolor, and both topical and oral medications have been shown to be effective. Because of importance of the endogenous host factors in the development of pityriasis versicolor, relapse is common.

This study has been done to know the various clinical presentations in people with different skin colors, risk factors and demographic characteristics of pityriasis versicolor with reference to the cultural positivity rate.

OBJECTIVES

OBJECTIVES OF STUDY WERE

i. To study the culture positivity rate in clinically diagnosed cases of pityriasis versicolor.

ii. To study the various clinical presentations in different skin colour people, risk factors and demographic characteristics of pityriasis versicolor.

REVIEW OF LITERATURE

HISTORY

The lipophilic basidiomycetous yeast, i.e. Malassezia species have been recognized over a century as both normal inhabitants of human skin and as organisms involved with superficial diseases.[1]

In 1846, Eichstedt described the disease pityriasis versicolor, though he was unable to isolate the organism.[2]

Their role in conditions such as seborrheic dermatitis and scaly lesions of scalp was speculated for many years.[1]

Robin further described fungus in the scales of pityriasis versicolor in 1853. He considered it to be a dermatophyte and named it Microsporum furfur. In 1873, Rivolta described double contoured budding cells in a patient of psoriasis.[3] The term Seborrheic Eczema was used for the first time by Unna in 1887.[1]

In 1874, Malassez isolated yeast cells from human dandruff scales and consequently, spherical and oval forms of these yeasts were recognized.[4] In 1889, Baillon included this group of yeasts under the germs malassezia.[2]

In 1904, Sabouraud considered this organism as a cause of dandruff and gave it a new name, Pityrosporum Malassez.[2]

In 1913, Castellani and Chalmens for the first time isolated lipophilic oval budding yeast from normal skin and seborrheic dermatitis and coined the name Pityrosporum ovale.[5]

In 1927, Acton and Parya considered Pityrosporum to be a synonym of Malassezia, but no attention was given to this significant finding.[5] In 1939, Rhoda Benham discovered lipophilic nature of the genus Malassezia.[1]

In the following years, there were controversies regarding the generic name of the fungus, Pityrosporum or Malassezia.[2]

In 1951, Morris Gordon isolated a round, double contoured budding yeast that produced oval buds in scales of pityriasis versicolor as well as on the normal skin.[7] He renamed this organism P.orbiculare on the basis of morphology and presumed to be distinct organisms from P.ovale.[7] In 1964, Dixon agar was used for the isolation of these species, which was subsequently modified to obtain better results.[1]

In 1969, Sternberg and Keddie detected the same antigenic components i.e. Pityrosporum orbiculare and Malassezia furfur by fluorescent antibody technique.[2]

In 1984, Yarrow and Aheam – Malassezia gained priority over Pityrosporum and was accepted as the generic name for the fungus.[2]

ETIOLOGY

Malassezia furfur has been shown to be a common endogenous saprophyte of the normal skin. Malassezia comprises lipophilic yeast found in the flora of human skin and other mammals.[3]

With some reservations, authorities today acknowledge the role of Pityrosporum (orbiculare and/or ovale) with pityriasis versicolor and its identity with Malassezia furfur. Morphologic similarities between M.furfur and Pityrosporum have been demonstrated on electron microscopy by Dorn in 1963 and Rochnert K. in 1977.[3]

Pityriasis versicolor is caused by Pityrosporum ovale and Pityrosporum orbiculare, which are lipophilic and lipid dependent human flora, when transformed into the mycelia phase, are known as Malassezia furfur. They are fastidious, requiring the addition of lipid substances to culture media,[4] grows best in the temperature average of 32-37°C in an aerobic environment.[5]

P.orbiculare and P.ovale are similar in macromorphology, but different in micromorphology. P.orbiculare tends to produce single spherical buds on a narrow base, while P.ovale develops single oval to cylindrical buds on a broad base.[6]

They are distributed unevenly on the human body, P.ovale resides more in the scalp and P.orbiculare occurring more often on the trunk. Both forms are commonly found together in scrapings of pityriasis versicolor patches. Observations point that the two forms maybe the same organism.[7]

Transformation from one to the other, as well as antigenic similarities have been noted.[4]

Three distinct forms of Malassezia furfur were described by Cunningham et al. They differed morphologically, physiologically and serologically with distinct cell membrane antigens. They were designated as serovars A, B and C. A study described no difference between the distribution of serovars on lesional skin when compared with controls.[8]

STRUCTURE, PHYSIOLOGY AND BIOCHEMISTRY

Malassezia is able to exist in both yeast and mycelia form, with the yeast being most commonly associated with normal skin. The yeast forms also predominate in culture, although hyphae may be seen with some species. Malassezia species undergo asexual reproduction by monopolar, enteroblastic budding from a characteristic broad base. The mother and daughter cell are divided by a septum and the daughter cell separates by fission, leaving a bud scar or collarets through which successive daughter cells will emerge. The cell wall of the genus Malassezia is poorly characterized. It is very thick in comparison with other yeasts and constitutes 26% to 37% of the cell volume. Mainly contains sugars, proteins and lipids, with small amounts of nitrogen and sulphur.[31]

Recent work confirmed the presence of multiple layers in cell wall. Also demonstrates the presence of an outer lamellar layer around the cell wall. The lamellar layer is "membrane-like" with an electron-transparent middle layer enclosed by two electron – dense lines. The structure of the layer varied with different lipid sources in the medium and stained with Nile blue sulfate, suggesting that it contained lipid.

The lamellar layer may play role in adhesion of the organism to both human skin and indwelling catheters. The cytoplasmic membrane adheres closely to the inner surface of the cell wall.[31]

The number and shape of the mitochondria in each cell may vary. The nucleus has a well – defined limiting membrane surrounded by a granular homogenous nucleoplasm. Vacuoles present in the cell contain lipid and vary in size according to the age of the cell.[31]

Three distinct forms of the organism have been identified using electronic microscopic techniques: Conidia, budding yeast and mycelia.[33] The cell wall of

conidia are smooth on the outer surface. The inner surface protrudes slightly, pushing the cytoplasm. Malassezia may exhibit a thinner cell wall when grown in culture than what it develops in vivo.[34,35,36] The mycelium is segmented by septa and fibrils occurring in septum. Internally, the organisms contain large vacuoles, most of which appear empty, have few mitochondria and an endoplasmic reticulum that can be clearly distinguished.[10]

The physiology of Malassezia species is poorly understood. In 1939, Benname noted that Malassezia is unable to ferment sugars. The organism can use lipid as the sole source of carbon, does not require vitamins, trace elements or electrolytes and preferentially uses methionine as the sole sulphur source, but it can also use cystine or cysteine. It is able to use many amino acids, as well as ammonium salts, as nitrogen sources. Although the organism is normally grown in vitro under aerobic conditions, it is also able to grow under microaerophilic and anaerobic conditions.[31]

The growth requirement of Malassezia for lipid was first noted in 1939, but was studied in detail by Shifrine and Marr. The lipid source used during growth affects the fatty acid composition of the organism, suggesting that the fatty acids are not used as empty source, rather are incorporated directly into cellular lipids without being further metabolized. Wilde and Stewart further found that the lipid present on normal human scalps were able to fulfill the lipid requirement of the organism.[31]

Malassezia species express a range of enzymes and metabolites. They have lipolytic activity both in vitro and vivo, indicating the production of a lipase. Ran et al found that the pH optimum was 5.0 and that lipase production was greatest during the logarithmic phase of growth, perhaps demonstrating its importance in the hydrolysis of lipid for cell growth.[31]

Malassezia species also produce a phospholipase. This phospholipase activity is able to cause the release of arachidonic acid from HEP-2 cell lines. Due to their involvement in inflammation in skin, it has been suggested Malassezia species may also trigger inflammation.

The taxonomy and nomeculature of genus Malassezia was controversial for many decades.[31]

Indeed until 1990 only three species were recognized : M.furfur, M.sympodialis and M.pachydermatis, a non-lipid dependent species.[37]

The species M.globosa, M.restricta, M.obtusa and M.slooffiae were described in 1995 by morphology, ultrastructure, physiology and molecular techniques.[37]

In the last few years, M.dermatis, M.japonica, M.nana and M.yamatoensis have been reported as Malassezia species.[37]

Recently, Cabanes et al described two new species M.equina and M.caprae, isolated from domestic animals.[37]

Nine of the 13 species namely M.furfur, M.sympodialis, M.globosa, M.restricta, M.slooffiae, M.obtusa, M.dermatis, M.japonica and M.yamatoensis are associated with normal human flora and pathologies, four species M.pachydermatis, M.nana, M.equina and M.caprae are associated with animals.

New physiological patterns for identification have been recently available for molecular biology and sequencing techniques has allowed the species to be distinguished more clearly.

Despite the difficulty in isolating, maintaining and identifying these yeasts, different characteristics of the genus such as macroscopic and microscopic morphology and some physiological aspect allow them to be differentiated like :-

a. Presence/ absence of catalase.
b. Selective growth on cremophor EL.

c. β-glucosidase activity.

d. Pigment production on pigment producing medium.

Assimilation of Tween 20, 40, 60 and 80 by M.furfur, M.sympodialis and M.slooffiae yields a specific pattern that easily differentiates them from other species.[37]

The organism is found in 90-100% of subjects as normal flora. It appears to be somewhat opportunistic, although the factors that enhance susceptibility have yet to be completely defined.[4]

Simple overgrowth does not appear to be the etiology of pityriasis versicolor, although some quantitative cultures of skin lesions have shown higher number of P.orbiculare when compared with both non-lesional skin and skin of normal healthy volunteers.[4]

Other authors have suggested genetic predisposition, poor nutritional status, poor health, chronic infections and pregnancy as predisposing factors. However, the low incidence of conjugal cases suggests the individual predilection. The disease is worldwide and common to both sexes and prevalent in tropics and sub-tropics. The disease is seen in young adults and usually establishes in early twenties. As these are lipophilic yeasts, there has been a suggestion that oil baths, skin lubricants and many such factors contribute to produce this condition. Role of hormones has been suggested by few authors. According to them, patients receiving hormone therapy and young women taking oral contraceptives are more prone to develop this condition.[24]

Antibodies to Pityrosporum have been seen on the sera of healthy persons and persons with pityriasis versicolor, indicating that it is of not much of significance. Yeasts have lipase activity in-vivo and in-vitro and this may be important in other dermatoses where this organism is suspected to play role.[24]

EPIDEMIOLOGY

Pityriasis versicolor is commonly seen in wide geographical regions. The true incidence cannot be estimated correctly because the disease is not very troublesome and quite often passes unnoticed and a good number of patients therefore do not consult a dermatologist.

Pityriasis versicolor is most commonly a disease of teenagers and young adults. Because of the lipid requirements of Malassezia, the yeasts are rarely found on the skin of young children and older individuals, but are usually present during the years when sebum production is highest.[2] Despite the fact that most adults have the yeast on their skin, conversion from the yeast to the mycelia form of the organism is required for pityriasis versicolor to develop.[10]

Systemic infections with Malassezia have been reported. These are generally nosocomial, occur in neonates and less frequently in adults receiving total parenteral nutrition supplements with lipids.[10]

The fungus is abound in the skin of subjects from tropical and subtropical climates.

Individual species have specific host preference.

- M.pachydermatis was first isolated from the Rhinoceros and is mainly found in domestic animals like dogs causing otitis externa, but occasionally can infect humans.
- M.sympodialis is the most frequent species occurring in humans as normal flora or sometimes in association with disease.
- M.furfur, M.globosa and M.restricta also affect humans.
- M.furfur is seldom found as normal flora or in diseased state.

- M.globosa and M.restricta are mainly pathogenic and are associated with pityriasis versicolor and pityriasis capitis or seborrheic dermatitis respectively.
- M.slooffiae can be demonstrated as normal flora on trunk. This species is also pathogenic in pigs.[2]

In tropical countries, cases of pityriasis versicolor are more prevalent, reaching as high as 50% in some areas.[13]

Its distribution as normal flora is related to sebaceous gland density and thus the scalp, face, central chest and back bears the highest number of fungi. Other sites like the hair follicles and the external ear, are also sites of colonization. The hair shaft, nail and mucosa are not affected.[2]

In an epidemiological study, 97% of the normal adult population showed scalp carriage and 92% showed trunk carriage for Malassezia. Colonization or disease by Malassezia is rare at the extremes of ages.[11,14]

Among children, pityriasis versicolor is generally rare, although cases are more common in tropical climates. Pediatric cases of pityriasis versicolor have been reported more frequently from Europe compared with North America. In children, facial involvement may be more common than in adults. Pityriasis versicolor has been rarely reported in children less than 2 years of age and in most of these cases the infants were premature and placed in intensive care, following birth.[10]

In a study M.sympodialis was found to be the predominant species in healthy human skin. M.restricta was seen to be associated with pityriasis capitis.[2] M.pachydermatis is known as an agent causing external otitis media in cats and dogs, but occasionally can infect humans.[15]

In different studies, hospitalized infants have shown a positive skin culture for M.furfur, the incidence varying from 37% to 84%.[10] Many healthy infants develop a cutaneous flora comprising malassezia species within the first 6 months of life.[47]

Hospitalized elderly may be at a higher risk for developing pityriasis versicolor.[17]

Studies conducted in India showed, that the incidence of pityriasis versicolor was 33.95% of all superficial mycoses in Western Orissa,[23] 14% in Lucknow,[29] whereas it was less in Chennai being 5.74%.[25]

PATHOGENESIS

The genus Malassezia is responsible for a variety of superficial cutaneous as well as systemic fungal infections and pityriasis versicolor is the most common presenting disease.[1]

Other infections in which this fungus plays an important etiological role are seborrheic dermatitis and folliculitis.[1]

Apart from these diseases it may cause certain systemic fungal infections as well. The recent developments in molecular technology have considerably helped to resolve most of the intricacies pertaining to taxonomy of this genus.[1]

Under the influence of pre-disposing factors, P.orbiculare (P.ovale) changes from its saprophytic yeast form to its pathogenic mycelia forms. P.orbiculare produces in-vitro dicarboxylic acids with a tyrosinase inhibitor effect. Dicarboxylic acids, apart from inhibitory effect, may also have cytotoxic effect on the melanocytes and are responsible for hypopigmentation seen in pityriasis versicolor.[43]

It has been also suggested that the lipoperoxidation process produced by Pityrosporum may account for the clinical hypopigmented appearance of the skin patches.[4]

In-vitro, strong inhibitory action, on the dopa tyrosinase reaction has been shown by culture extracts containing dicarboxylic acids, such as azelaic acid. Ultrastructural studies have shown severe melanocyte damage, varying from altered melanosomes and mitochondria to actual degeneration. Damage to melanocytes may explain why repigmentation may take months or years to occur.[4]

Another explanation may be that scales of pityriasis versicolor prevents tanning from occurring. Therefore, for a period of time after treatment the affected

area remains hypopigmented until the lesion tans. The pathogenesis of hyperpigmentation in pityriasis versicolor lesion is not fully understood.[4]

Two theories are:-

- Increased thickness of the keratin layer in hyperpigmented lesions.
- More pronounced inflammatory cell infiltrate in hyperpigmented lesions, acting as a stimulus to melanocytes, resulting in production of more pigment.[4]

Malassezia species produces an enzyme with lipoxygenome activity, as demonstrated by its ability to oxidize free and esterified unsaturated fatty acids, squalene and cholesterol. The resultant production of lipoperioxides may damage cell membranes and consequently interfere with cellular activity- (a mechanism that has been proposed to cause the alteration in skin pigmentation).[31]

Conversion of yeast to its mycelia forms is due to certain predisposing factors. These can be classified as exogenous and endogenous factors. The exogenous factors include heat and moisture that may cause, the disease to be more prevalent in tropics.[4]

Other factors include occlusion of the skin by either clothing or cosmetics. Occlusion results in carbon dioxide concentration, an altered microflora and an altered pH range. Infection has been experimentally induced by occlusive dressing.[4]

Endogenous factors contributing to the development of pityriasis versicolor include malnutrition, use of contraceptives, use of systemic corticosteroids or immunosuppressants and hyperhidrosis, resulting in the disease being more common in the summer than in winter months. Athletes may be particularly prone to pityriasis versicolor, but whether this is due to excessive sweating is still unclear. Although hygiene is not an important factor, application of cream or lotions to the skin may exacerbate the tendency to develop lesions in those who are prone.[10]

Lesions restricted to the irradiated field have been reported in association with radiation therapy.[44] Pityriasis versicolor has been reported in patient undergoing lithium therapy for mood disorders.[45]

It is unusual for older adults to suffer from pityriasis versicolor. This is thought to be due to the reduction in sebum production that occurs with increasing age.[46]

In a study, Malassezia colonization was found in 32% of the population, ear being the most common site of colonization. Risk factors to which the neonates are exposed during their stay in the hospital have been analyzed to determine what influences the colonization of Malassezia. It indicated that factors predisposing to the colonization included extended stay in hospital and prematurity. The strains or species that form the commensal flora of the children become adapted to the skin and infection occurs only when a different strain or species gains systemic access.[47]

Although it is not contagious according to the study conducted by Faergemann and Fredriksson, positive family history was seen in 18.8% of cases.[48]

Table 1 : Shows conditions associated with an increased frequency of pityriasis versicolor[49]

Adrenalectomy
Cushing's disease
Diabetes
Pregnancy
Malnutrition
Severe burns
Striae
Parenteral steroid therapy
Immunosuppression
Oral contraceptives
Hyperhidrosis
Ichthyosis

Table 2 : Condition where Malassezia has a definite role[2]

Cutaneous	• Pityriasis versicolor.
Systemic	• I.V catheter induced fungemia with or without embolism • Endocarditis • Interstitial pneumonia • Peritonitis in patients undergoing continuous ambulatory peritoneal dialysis

Other conditions where it has been associated

• Dandruff	• Folliculitis
• Neonatal cephalic pustulosis	• Confluent and reticulated papillomatosis
• Atopic dermatitis	• Guttate psoriasis
• Seborrheic dermatitis with blepharitis	• Balanitis
• Onychomycosis	• Lacrimal canaliculitits with dacrolithiasis
• Sinusitis	• Otitis externa
• Nipple discharge	

In both normal and pathogenic state, yeast resides within the stratum corneum and hair follicles, where free fatty acids and triglycerides from sebum and keratinized epidermis provide nourishment.[4]

A variable quantity of Malassezia, specific IgG antibodies are found in normal adults. In diseased state, inflammatory and regulatory cytokines of both Th_1 and Th_2 types are generated.[2]

The current view about the pathogenicity of Malassezia is that the organism may produce a lipid soluble low molecular weight (<1-2 KDa) substance under

certain growth conditions which acts as a chemotactic agent for leukocytes and thus induces inflammation.[2]

In a study by Faergemann et al, there was an increase in NK1+ and CD16 – cells along with complement activation in patients with seborrheic dermatitis and Malassezia folliculitis indicating an irritant, non-immunogenic stimulation of the immune system. The cause of this irritant response is likely due to products released by Malassezia.[55]

PITYRIASIS VERSICOLOR

It is the only skin infection where Malassezia plays a definite causative role. It is a chronic, superficial, non-inflammatory infection of the skin occurring principally on the trunk and proximal extremities of young adults.[2]

In a study M.sympodialis was found to be the predominant species in healthy and diseased human skin and was found mainly over trunk, while M.globosa was found in pityriasis versicolor scales as well as in healthy skin and M.restricta was found to be associated with pityriasis capitis.[50]

- In another study, M.globosa was the only isolated species in 60% of the cases in pityriasis versicolor and 37% in addition with M.sympodialis and M.restricta.[19]
- Dutta et al had reported M.globosa as the main species isolated from patients with pityriasis versicolor in North India.[51]
- Kindo et al, showed that in South India, M.sympodialis is the commonest agent (58.3%) followed by M.globosa (39.6%).[52]
- Chaudhary et al showed that in Central India the main species was M.globosa (57.5%), followed by M.sympodialis (17.2%), M.furfur (6.9%) and M.obtusa and M.restricta (3.4%).[53]

More than one species of Malassezia can be isolated from a clinical specimen.[2]

DANDRUFF (PITYRIASIS CAPITIS OR PITYRIASIS SIMPLEX)

It is a subclinical, inflammatory scalp disorder which is episodic, recurrent or constant in nature, which results in disruption of cohesion between corneocytes, visible as scales. The disorder is most prevalent and severe among adolescents and young adults, rare among children and elderly. Environmental factors like winter season, U.V irradiation, airborne irritants and hair cosmetics are known to aggravate the condition.

Malassez had isolated the yeast forms of this species from dandruff scales. It has been hypothesized that a critical quantity of the yeasts is 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 activity of the yeast induces a pro-inflammatory state and stimulates host immune response that may result in disease. 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.[2]

SEBORRHEIC DERMATITIS

Distribution of Malassezia species in seborrheic dermatitis lesions represent complex interaction of the fungus with the skin microenvironment.[54]

It is a chronic dermatitis with greasy scales over seborrheic areas in children and adults. The total count of the fungus has been found to be raised in lesions of seborrheic dermatitis. M.sympodialis and M.restricta are the Malassezia species found commonly in seborrheic dermatitis lesion.[2]

PITYROSPORUM FOLLICULITIS

In most cases, this condition is associated with seborrheic dermatitis. Overgrowth of the yeast causes blockage of follicular ostia. It is possible that hydrolysis of triglycerides, fatty acid synthesis and activation of the alternate complement pathway by fungus induce inflammation, resulting in folliculitis.[2]

Increased number of NK1+ and CD10+ cells as well as increase in complement state indicates that an irritant or non-immunogenic stimulation of the immune system is important in both seborrheic dermatitis and Pityrosporum folliculitis.

Reaction seen with the interleukins is complex, showing increase in the production of inflammatory interleukins IL-K, IL-1β, IL-6 and TNF-α, as well as interleukins responsible for both TH_1 and TH_2 responses were also seen.

IL-12 not only stimulates a TH_1 reaction but it also stimulates NK cell proliferation, which may explain the increase in NK1+ and CD16+ cells.

ICAM-I cells have been found to be increased in lesional skin in both patients with seborrheic dermatitis and Pityrosporum folliculitis.[55]

ATOPIC DERMATITIS

Yeasts of Malassezia species have been implicated in atopic dermatitis also. It is a common inflammatory skin disease characterized by a chronically relapsing course having distinctive clinical appearance and severe pruritus.

Most patients with atopic dermatitis have elevated serum IgE and positive skin prick test (SPT) reaction to a wide variety of allergens.

Positive skin prick test reaction to M.sympodialis extract were found in 51% of the patients in a study. Malassezia extracts contain a wide range of IgE binding components.

By its growth on human skin Malassezia could be suspected mainly to elicit eczema. Thus the atopy patch test may be considered as a clinically relevant and important test for the diagnosis of Malassezia hypersenstivity.[56]

NEONATAL CEPHALIC PUSTULOSIS

Previously considered as neonatal acne, it is a newly described benign clinical entity occurring in neonates. There is a non-follicular pustular eruption involving the face, neck and scalp. Its incidence is 3% in hospitalized neonates. The diagnostic criteria for differentiating this condition from others like milia, sebaceous gland hyperplasia and neonatal acne are: age of onset less than one month, cephalic

location, smear positive for Malassezia, elimination of other causes of neonatal pustulosis and response to topical ketoconazole.

M.furfur is the most commonly isolated species, M.sympodialis can occur in severe form. Neonate sebum production due to maternal hormonal influences facilitates the growth of the yeast, which might have been seeded from the mother or from health care worker.[2]

CONFLUENT AND RETICULATE PAPILLOMATOSIS

It is a genetic defect of keratinization where there is an abnormal response to colonization by Malassezia and/or follicular bacteria. The condition is commonly seen around puberty as asymptomatic, flat, dry papules varying in size from 1-5 mm on the mid-back, chest, sides of the neck and shoulders. The response to topical or systemic antifungals is variable.[2]

OTHER SUPERFICIAL DISEASE

Malassezia has been associated with a wide range of other skin diseases, including acne vulgaris, dacrocystitis, seborrheic blepharitis, onychomycosis, nodular hair infection and psoriasis. In many of these reports, the isolation of Malassezia was taken as proof of its involvement in the disease, an assumption that may not be correct due to its presence on the skin as a commensal. Malassezia has also been implicated in some more deep-seated infections including mastitis, sinusitis, septic arthritis and malignant otitis externa.[31]

SYSTEMIC DISEASE

Peritonitis : The first time that Malassezia was associated with a deep-seated infection was in 1979, when it was isolated from a patient undergoing continuous ambulatory peritoneal dialysis (CAPD) who developed peritonitis. After several episodes of apparently "sterile" peritonitis, lipid supplementation of cultures of

peritoneal dialysate fluid grew Malassezia. Since this initial report, there have been three further reports of peritonitis due to Malassezia in CAPD patients.[31]

Catheter - related fungemia : Systemic infection by Malassezia is becoming an increasingly recognized entity among seriously ill, low-birth-weight, hospitalized neonates or adults receiving infusion of intravenous lipid preparations as a part of parenteral alimentation. M.furfur is the commonest species isolated. M.pachydermatis has also been reported. The source of fungus is usually patient's own skin flora or hands of health care workers. Intralipid solution has long-chain fatty acids, which facilitate the growth of the organism, depending on host immune response, there is further systemic spread. Examination of the removed catheter reveals adherent fungi, maximally along the distal lumen, often visible as white clumps.[2]

In the presence of fungemia, embolization occurs, the lungs being involved frequently. The yeast can be demonstrated in the walls of small pulmonary arteries or in the areas of an infarct. Blood culture is frequently positive. Clinical symptoms may be subtle or asymptomatic. Morbidity associated with Malassezia fungemia is difficult to assess as the condition is under-diagnosed and patient usually suffers from other major illnesses.[2]

HIV INFECTION AND MALASSEZIA

The growth of the yeast is known to be enhanced in immunocompromised conditions. The skin flora remains quantitatively normal in HIV infected patients. It has been proposed that the level of toxic products of Malassezia rises, increasing the prevalence and severity of seborrheic dermatitis. Moreover, the HIV infected state increases the level of Interferons and TNF–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.[2]

INVESTIGATIONS

Generally, it is relatively easy to diagnose pityriasis versicolor. However, the varied presentation of the lesions may confuse the clinician.

The differential diagnosis includes vitiligo, melasma, tinea corporis, seborrhoeic dermatitis, pityriasis rosea, pityriasis alba, erythrasma, confluent and reticulated papillomatosis of gougerot and carteaud, pityriasis rotunda, secondary syphilis and pinta.[10]

DIRECT EXAMINATION

In 1903, a Baltimore Physicist, Robert W. Wood, devised the lamp containing high-pressure mercury with Wood's filter made up of 9% nickel oxide and barium silicate, which transmitted the invisible part of the light spectrum. Margarot and Deveze (1925) used the Wood's lamp for the first time in medical mycology. Wood's lamp is small, durable, inexpensive, safe and very easy to use.[90]

PRINCIPLE

It works on a simple principle, the rays from the ultraviolet lamp pass through Wood's filter which screens out the short wave rays, the visible ultraviolet rays and transmits only long wave ultraviolet radiation. When these waves come in contact with some chemicals, it creates a phenomenon of fluorescence. Filtered ultraviolet rays causes a luminescence which is more clearly seen in a darkened room.

The fluorescence of normal skin is very faint or absent and is mainly due to constituents of elastin, aromatic amino acids and precursors or products of melanin.[87]

The Wood's light is produced with a high pressure mercury lamp emitting through a "Wood's filter" made of silicate with nickel oxide, which is opaque to all radiation beyond a wavelength between 320 nm and 400 nm [ultraviolet A (UVA)], with peak emission at 365 nm.

Wood's light is strongly absorbed by melanin, making it a useful tool in the evaluation of pigmented lesions. A lesion with an increased concentration of epidermal melanin appears darker than surrounding normal skin, with more contrast than normally discernible by visible light examination.

The ability of Wood's light to produce characteristic fluorescence in pathologic conditions extends its application to skin infections and porphyria. The green fluorescence of ectothrix-type tinea capitis, can help make the correct bedside diagnosis, it can also serve as a guide to the scalp site that has a higher likelihood of yielding a positive culture, although not all cases clearly fluorescence. Typical fluorescence may also be seen in erythrasma ("coral red"), tinea versicolor (yellow to orange) and Pseudomonas aeruginosa infection in burn patients (green), particularly if the examined area has not been cleansed recently. In porphyria cutanea tarda, Wood's light can be used to screen for pink-orange fluorescence in the urine.

The examiner should be aware of false-positive sources of fluorescence such as scales, ointments, dried soaps, threads of fiber and scars.[80]

TECHNIQUE

The use of Wood's lamp does not require great skill. However, some practical points should be kept in mind to avoid misinterpretation of results.

- Lamp should ideally be allowed to warm up for about one minute.
- The examination room should be perfectly dark, preferably a windowless room or a room with black occlusive shades.
- The light source should be 4 to 5 inches from the lesion.
- Washing the area before subjecting it for Wood's lamp examination should be avoided since it may yield false negative results due to dilution of the pigment.

- Topical medicaments, lint and soap residues should be wiped off from the site to be examined since they may cause fluorescence under Wood's light. Common sources of error are bluish or purplish fluorescence produced by ointments containing petrolatum, green fluorescence by salicylic acid containing medicaments and light reflected from examiner's white coat producing light blue fluorescence. Malassezia furfur emits a yellow-white or copper-orange fluorescence. Woods lamp can detect subclinical infection and the extent of infection.[88,89,90]

Fluorescence includes the areas immediately surrounding clinically visible lesions, indicating that the infection is spreading.[92,93] However, Wood's light provides a positive response in only one-third of cases,[94] most likely those involving Malassezia furfur.[95,96]

These patches will be difficult to detect if the patient has taken a recent bath. An important diagnostic clue may be the loosening of hardly noticeable scales, with finger nails (coup d'ongle).[97]

MICROSCOPIC EVALUATION

Mycological examination can be used to confirm the diagnosis of pityriasis versicolor. Specimens for light microscopic examination should be taken from the center as well as from the edges of the lesions. However, better results are obtained if scraping is done from the center of lesion, as these areas are most likely to contain a large number of micro-organism.[98] Skin scraping may be collected directly onto the microscope slide with a no. 15 Parker blade or the edge of a second glass slide [99]

Alternatively, the scotch tape stripping technique can be used. Tape is pressed firmly on scaly lesions, left on a few minutes and then briskly removed. This tape is placed on a glass slide and examined under the microscope. A drawback of the tape method is the adherence of dust and cosmetics, which tend to darken the visual field and render recognition of the fungus more difficult. Scotch tape no.681 is durable, resistant to moisture, chemicals and solvent and is the tape least affected by such procedure.[100]

Prior to performing light microscopic examination, the addition of 10-15% potassium hydroxide to the sample helps dissolve the keratin and debris, facilitating examination of fungal elements. Gentle heating of the glass slide speeds dissolution of keratin, although if left at room temperature, the sample should be ready to view under the microscope in 15-20 mins.[10]

Characteristic appearance on microscopy reveals multiple round or oval, unipolar budding yeast cells and short septate hyphae, typically has been linked to "**spaghetti and meatballs**" or "**bananas and grapes".** As both hyphae and spores are present.[10,18]

These characteristic cells are more easily demonstrated if stained with methylene blue or potassium hydroxide with parker 51 super chrome blue-black ink.

Heating the slide is not necessary if potassium hydroxide and ink are used to stain the specimen. The mycelia and yeasts stain dark-blue and the surrounding background light blue.[102]

Albert solution: Toluidine blue 0.15gm, malachite green 0.2g, glacial acetic acid 1mL, 95% ethanol 2ml and distilled water 110 ml can be used instead of potassium hydroxide.[103]

CULTURE

The organism is lipophilic and requires lipid in the medium to be grown in vitro. Sabouraud's dextrose agar (SDA) overlaid on the surface with sterile olive oil or lanolin readily supports the growth of this lipophilic, yeast like organism. To this media, streptomycin, penicillin and cycloheximide (actidione) are added to inhibit bacterial growth. The tubes are incubated at a temperature of 37ºC. Colonies will grow on SDA with olive oil within. Although only rarely are hyphae seen in culture.[18]

Among Malassezia species, only M.pachydermatis is capable of growing on Sabouraud's agar without lipid supplement.[107]

Malassezia species are identified according to their macroscopic and microscopic features and physiological characteristics.[12]

Modified Dixon Agar (MDA), a more specialized media, permits better visualization and isolation of the colonies can also be used.[53]

MDA medium comprises 3.6% malt extract, 0.6% mycologic peptone, 2% Ox bile, 1% Tween 40, 0.2% glycerol, 0.2% oleic acid, 1.2% agar, 0.5% chloramphenicol and 1g/L cycloheximide.[51]

Based on studies conducted within the last few years, it appears that the organism most frequently associated with pityriasis versicolor may not be M.furfur, possible candidates include M.globosa[82] and M.sympodialis.[105]

Mayser and collaborators however have pointed out that Indole compounds apparently responsible for a fluorescence, often seen in pityriasis versicolor lesions illuminated with a Woods lamp are found in-vitro only by M.furfur, suggesting that this species is etiological in at least some lesions.[95,96]

Providing a pure culture and discriminating a species from mixed sample is difficult. This might be due to the fact that fast growing species usually cover other species in the culture. Besides, because of the hydrophobic characteristic of Malassezia yeast, preparing homogenous suspension is very difficult to separate them by culture.[14]

Culture media which can be used : [32]

- Modified Leeming and Notman agar composed of (per lit) 10gm of peptone, 10gm of glucose, 2gm of yeast extract, 8gm of Ox bile, 10ml of glycerol, 0.5gm of glycerol monostearate.
- 5ml of Tween 60, 20ml of olive oil and 15gm of agar.
- CHROM agar Malassezia medium (CHROM) composed of 56.3gm of CHROM agar Malassezia basal medium and 10ml of Tween 40.
- SDA composed of 10gm of mycological peptone, 40gm of glucose and 15gm of agar.
- Cremophor EL agar composed of 65gm of SDA and 10ml of cremophor EL.
- Tween 60 Esculin agar composed of 10gm of peptone, 10gm of glucose, 2gm of yeast extract, 5ml of Tween 60, 0.5gm of ferric ammonium citrate, 1gm of esculin and 15gm agar.

According to a study, the different phenotypic characteristic of species of Malassezia on CHROM agar was seen as following : [32]

TABLE NO. 3 Biological features and incidence of a typical phenotype of nine species of Malassezia

BIOCHEMICAL TEST

1. Catalase reaction

Production of gas bubbles on adding a drop of hydrogen perioxide indicates a positive reaction. Only M.restricta lacks catalase activity.[53]

2. Tween assimilation test

The possibility of utilizing different Tween compounds as a unique lipid supplement by Malassezia is the feature that is used to evaluate different species. Yeast suspension is made in sterilized distilled water and poured into plate containing SDA at 45°C. The inoculums are then spread evenly. After solidification of each plate, four wells are made and filled with 30µl of a Tween compound i.e., Tween 20, 40, 60 and 80 respectively. These plates are then incubated for a week at 31°C. Growth is estimated around the individual wells after 2, 4 and 7 days.

3. Splitting of Esculin

β-glucosidase activity of different Malassezia species is analyzed. A loop of fresh yeast is inoculated deeply in the Esculin agar tubes and incubated for 5 days at 32°C. The splitting of Esculin is revealed by darkening of the medium.

This test is used to distinguish M.furfur, M.sloofiae and M.sympodialis from other Malassezia species.[107]

4. Assimilation of Glycine as a Nitrogen source

M.furfur is the only species which assimilates glycine. Growth within 2 to 3 days shows glycine assimilation.

Other media suggested for culturing Malassezia are Martin-Scott's medium (1952) containing sodium tauroglycocholate, "Caprilli's medium" with yeast extract and synthetic formulation incorporated with neopeptone agar, glucose, yeast extract, glycerol monosterate, Tween-80, chloramphenicol, gentamicin and olive oil. Sabouraud's agar covered with olive oil is simple to prepare and gives reliable results.

Molecular methods such as nested PCR or PCR-REA are being developed to solve the problems arising due to time-consuming morphological and physiological techniques and difficulty in interpretation of some physiological patterns.[53]

HISTOPATHOLOGY

Histopathology with the Haematoxylin and Eosin stain shows the typical morphology of the yeast in the stratum corneum and sometimes in the perifollicular regions.[69]

The organisms may be located both intracellularly and between the keratinocytes, although hyphal form of the yeast actually invade cells. Intercellular invasion by the organism may result in the disruption of the normal, horizontal direction of the skin cells in the superficial and middle layers of the stratum corneum. Invasion of the host cells results in a 'clear zone' around the pathogen, which is due to loss of keratin structure in the cell.[10]

On electron microscopy, the thick walled hyphae of Pityrosporum are seen to be oval in cross section and are 1-3µm thick. They are much thicker than the horny cells. Nevertheless hyphae are seen not only outside the horny cells but quite frequently inside the horny cells also.[58]

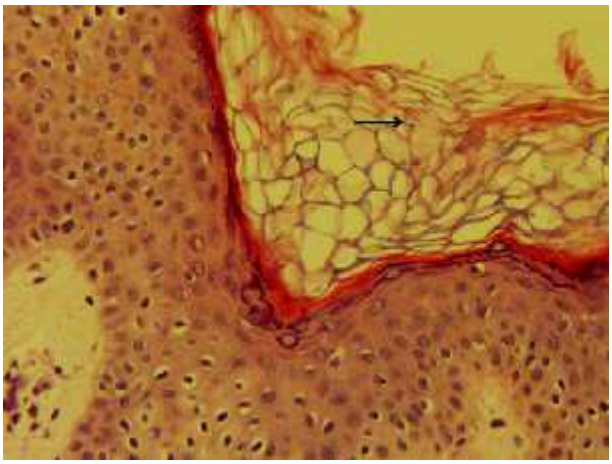


Figure 1 : H and E stain showing fungal filaments (arrow) at high power

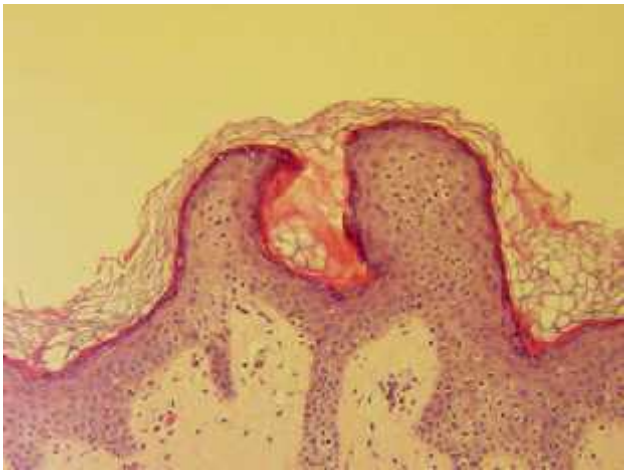


Figure 2 : Low power showing fungus in stratum corneum

Histologic differences between hyper and hypopigmented lesions have been seen, hyperpigmented lesions seen to contain more spores and hyphae.[69]

In hypopigmented lesions, the horny layer may be slightly hyperkeratotic, there is decrease in melanosomes in stratum spinosum and many patients first become aware that they have pityriasis versicolor when they develop hypopigmented lesions over sun exposed areas. Hypopigmentation has been observed at anatomical sites that have not been exposed to the sun, furthermore hypopigmentation has been seen in infants who have not been exposed to the sun.[10]

Indole pigments formed by M.furfur have recently been found to be potent ultraviolet filters.[84]

In hyperpigmented lesions, the stratum corneum may be thicker than in either normal or hypopigmented skin and both spore and hyphae are more numerous than in other samples.[101]

The inflammatory cell infiltrate is more pronounced in hyperpigmented skin and while the inflammation itself may be responsible for some changes in skin colour, melanocytes have themselves been shown to be stimulated by inflammation. Thus in the darkest lesions of pityriasis versicolor, it is possible that discolouration is a result of both inflammation and increased melanin products.[10]

Merkel cells may have an increased activity as they contain compound melanosomes and secretary granules.[69]

Several staining techniques are available for demonstration of this superficial fungus in histologic sections. These include the Periodic Acid Schiff (PAS) reaction, methenamine silver nitrate, Alcian blue and the Giemsa method. The presence of polysaccharide-rich cellulose and chitin in fungal cell walls results in a positive staining with the PAS reaction and the fungi stain deeply red. In contrast, the PAS reaction of glycogen is diastase resistant. Pretreatment of slides examined for pityriasis orbiculare with diastase is recommended to ensure the removal of glycogen granules, which may be confused with fungal spores. Methenamine silver nitrate stains the fungus black.[58]

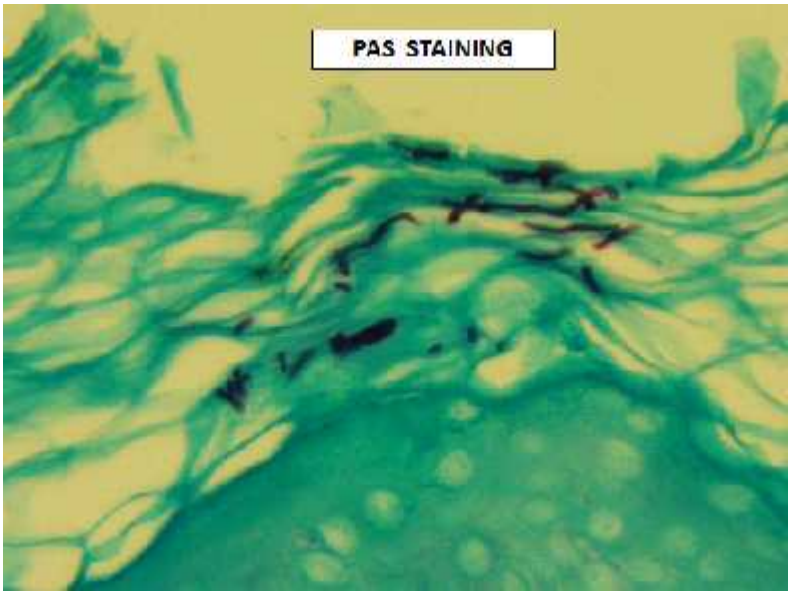


Figure 3 : PAS staining showing darkly stained fungal filaments

ANIMAL SEROLOGY

Ordinary animals appear immune to the fungus applied in scales. The disease has not been reported in animals.[69]

IMMUNOLOGY

Malassezia is a cutaneous commensal and thus its first point of contact with the immune system is likely to be via the skin immune system.

Skin acts as a physical barrier and presence of a commensal flora on the skin is an important nonspecific immune defense. In addition to Malassezia species, normal skin will have a resident population of other organisms, principally bacteria, including staphylococci and propiniobacteria. These will compete for nutrients and space, limiting the population size of each group and also competing out pathogens that may attempt to colonize the skin. Shedding of the cells of the epidermis occurs constantly and the rate of shedding is increased during inflammation. This shedding causes the loss of microorganisms, that are colonizing or infecting these cells and prevent invasion into the deeper layers of the skin.

Other factors involved in the non-specific immune system of the skin are :-

a. The lipids found on adult scalp and adult hair, which are fungistatic for certain dermatophytes.
b. Fungicidal proteins present in the epidermis.
c. Inhibitory effect of unsaturated transferrin on various fungi.[31]

SPECIFIC IMMUNE RESPONSES

In addition to non-specific immune functions, the skin is involved in specific immune responses. The skin immune system consists of both cellular and humoral components. The cellular skin immune system includes keratinocytes, Langerhans cells, monocytes, mast cells, endothelial cells and lymphocytes, while humoral components include complement proteins, IgG, IgA and various cytokines.[31]

HUMORAL IMMUNE RESPONSES TO MALASSEZIA IN NORMAL INDIVIDUALS

Immunoglobulins specific to the yeast phase of Malassezia can be readily detected in normal individuals with no history of skin diseases. Several groups have studied this humoral response in healthy individuals with no history of skin disease. Demonstration with ELISA is a more sensitive method than immuno-fluorescence.

IgM was similar for all age groups, but lower in old age group, IgG did not differ significantly between age groups. IgA levels were low in all groups. Antibodies to Malassezia can be detected by immuno-electrophoresis

The majority of individuals have some antibodies to Malassezia, even from a relatively young age. Antigen is presented to the immune system over a sufficient period to initiate both naive (IgM) and anamnestic (IgG) responses. Levels of IgA are generally low, suggesting that mucosal sensitization by Malassezia is not an important route.

In the commensal state, Malassezia usually occurs as yeast cells, although mycelium may also be seen. No investigators have determined the humoral response to mycelium.

Recently Saadatzadeh, induced the mycelia phase and whole mycelia antigen in indirect immuno-fluorescence. All the classes of immunoglobulins were detected, with the highest titers being found for IgG. Appreciable levels of IgM, IgA and the IgG subclasses were also found.[31]

CELLULAR IMMUNE RESPONSES TO MALASSEZIA IN NORMAL INDIVIDUALS

Cellular immunity is known to be of major importance in the host defense against fungal infection. The higher incidence of Malassezia associated dermatoses in patients with cellular immunodeficiencies suggests that cellular immunity is also important in maintaining the organism as a commensal.

The incidence of pityriasis versicolor is known to be increased in renal transplant recipients and patients receiving steroids, folliculitis is seen in bone marrow transplant recipient and the incidence of seborrheic dermatitis is very high in patients with AIDS.

Cunningham studied the cellular immune response to whole yeast cells of Malassezia serovars A, B and C using the leukocyte migration inhibition (LMI) of LT assay. There were positive migration inhibition responses and responses did not differ significantly between the age groups. However, the responses to serovars B and C were generally higher than those to serovar A.

Malassezia elicits significant cellular immunity in normal individuals and levels of cellular immunity remains fairly constant throughout life.[31]

In those individuals in whose pityriasis versicolor is present on a relatively chronic basis, a defect in cell mediated immunity may exist.[10]

Pityriasis versicolor produces fewer lymphokines in comparison to normal controls. In addition, a large number of Langerhans cells are found in the epidermis in pityriasis versicolor lesions and an increase in T-cells have been reported in both the dermis and epidermis of lesional skin. The majority of these T-cells are thought to be helper / inducer cells, although some suppressor / cytotoxic cells are also present. Patients also exhibit a diminished lymphocyte transformation response to the fungus.

In addition to the role of the immune system in the development of pityriasis versicolor, other endogenous factors have been suggested with the exception of M.pachydermatis. The Malassezia species are all obligatorily lipophilic species, requiring medium or long-chain (C_{12-24}) fatty acids in the culture medium.

In-vitro, the addition of glucose or asparagine to the culture medium promotes growth of the yeast. Cholesterol and cholesterol esters added to culture also stimulate the induction of hyphae.[10]

CLINICAL FEATURES

Pityriasis versicolor is a mild, chronic infection of the skin caused by Malassezia and characterised by discrete confluent, scaly, discoloured or depigmented areas, mainly on the upper trunk.[59]

Cutaneous infections with Malassezia may take three forms:

a) Papulosquamous pityriasis versicolor

b) Folliculitis

c) Inverse pityriasis versicolor.[60]

The lesion of inverse pityriasis versicolor are encountered predominantly in flexural area. These are sharply demarcated confluent erythematous patches that can be confused with inverse psoriasis, seborrheic dermatitis, erythrasma, candiasis and dermatophyte infection.[60]

The most common presentation is scaly hypopigmented or hyperpigmented macules observed in characteristic areas of the body, including the chest, back, abdomen and proximal extremities.[60] Less common areas of involvement include the face, scalp and genitalia.[60]

Rarely, the eruption can be limited to the lower limb, popliteal fossa, forearms, axilla, genitalia or to a radiotherapy field. The colonization of the ear by M.furfur has been linked to ear wax. Two ear wax phenotypes have been recognized: wet and dry.[4]

The coexistence of hypopigmented and hyperpigmented lesions suggests the involvement of factors other than the normal pigmentation of the skin in the production of the pigmentary changes of lesions of pityriasis versicolor.[57]

The primary lesion of pityriasis versicolor is a sharply demarcated macule, sometimes slightly erythematous, but characterized essentially by fine, branny scaling.[50]

Clinically, the lesions present as flaky round or oval macules, although in larger lesions the flaking may be apparent only at the outer border of the macule. These lesions may be hypopigmented or hyperpigmented, a single patient may have both types of lesions. The colour of the hyperpigmented lesions varies from pink or tan to dark brown or black.[61]

Macules may coalesce into large patches with an adherent fine scale.[62] Distribution of the lesions generally parallels the density of sebaceous gland distribution.[63]

The presenting complaint is usually a cosmetic one, because lesions often fail to tan with sun exposure. Pruritus is mild or absent.[60] Lesions of the face are much more common in children than adults.[22]

Infants in tropical areas may get severe hypopigmented lesions of pityriasis versicolor in the diaper area. These lesions are called Achromia parasitica or Tinea versicolor alba.[57]

Occasionally atrophy may be seen in the patches of pityriasis versicolor which warrants differentiation from parapsoriasis or mycosis fungoids, anetoderma, lupus erythematosus and steroid atrophy. This variant has been termed as Atrophic pityriasis versicolor.[69]

In atrophic pityriasis versicolor, the infection causes degeneration of the cutaneous elastic network with the subsequent appearance of small whitish plaque with a slight thinner skin, the pathogenesis is unclear but could be a sequel of delayed type of hypersensitivity. There is release of leukotrienes which disturbs collagen metabolism It may be mistaken clinically for other atrophic dermatoses.[76]

The distribution also appears to occur to covered areas, thus supporting the theory, that the occlusion of glands plays a role in the etiology of disease.

The macules and patches, as implied by the name "Versicolor", may be hypopigmented, hyperpigmented, leukodermal, erythematous or dark brown. The individual patches usually display a fine scale, although at times papules or annular plaques may be evident.[4]

The sites most commonly involved are the upper part of the trunk, with a marked predilection for the sternal region, back and neck.[21]

The disease is less common on the face, scalp and lower extremities. Facial lesions are commoner in children than in adults and in females than in males.[22]

The colour of the scales may vary from pale ochre to medium brown. In the untanned white skin, the affected areas are darker than normal, but fail to respond to light exposure. In some subjects, the abnormal skin is commonly pale, as it is usually in black people that residual depigmentation may remain for many months without any scaling.[59]

Another rare disease which may be caused by M.furfur is confluent and reticulate papillomatosis. This condition is more common in puberty. The lesions consist of greyish and brown papules located mainly in intermammary, interscapular areas and axillae where they tend to be confluent and become reticulated towards the periphery. The lesions bear a clinical resemblance to pityriasis versicolor.[2,60]

The material is obtained by scraping the skin lesion with the blunt end of scalpel and is then smeared onto a glass slide and examined after application of a 10% potassium hydroxide solution with or without dimethyl sulfoxide (DMSO). If DMSO is used, gentle heating is not necessary. One usually looks for round budding cells and hyphae often referred to as a pattern of "spaghetti and meatball". Staining with 1% methylene blue, chlorazol black E, or paragon multiple stain may facilitate the diagnosis. Albert solution has been reported to be as effective as KOH mount,

although culture is usually not necessary, scales can be inoculated into glucose neopeptone-yeast extract medium.[4]

Various clinical types presented by different authors on pityriasis versicolor as reported in literature are as follows:

- Multiple hypopigmented macules and patches of various sizes and shapes with furfuracious scaling.
- Multiple dark brown to fawn coloured macules, patches and plaques of various sizes and shapes.
- Erythematous follicular papules with or without other types of manifestations.
- Acneiform pityriasis folliculitis, authors claimed it as a more severe form of follicular variety.
- Greyish brown coloured confluent and reticulate papillomatosis is commonly seen in intermammary and interscapular areas.
- In dark coloured infants the infection starts in the diaper areas, described as 'Pityriasis versicolor alba' or 'Achromia parasitica'.
- Intertrigo like pityriasis versicolor named as 'Dermatophytoid tinea versicolor'.
- Rarely some patients are seen only with dandruff of scalp skin. A condition known as pityriasis capitis.

Rarely organism can colonize in the lacrimal sac, causing enlargement and obstruction of the sac, dacrolithiasis often develops leading to inflammation and interference with normal tearing process. Condition is called obstructive dacrocystitis.

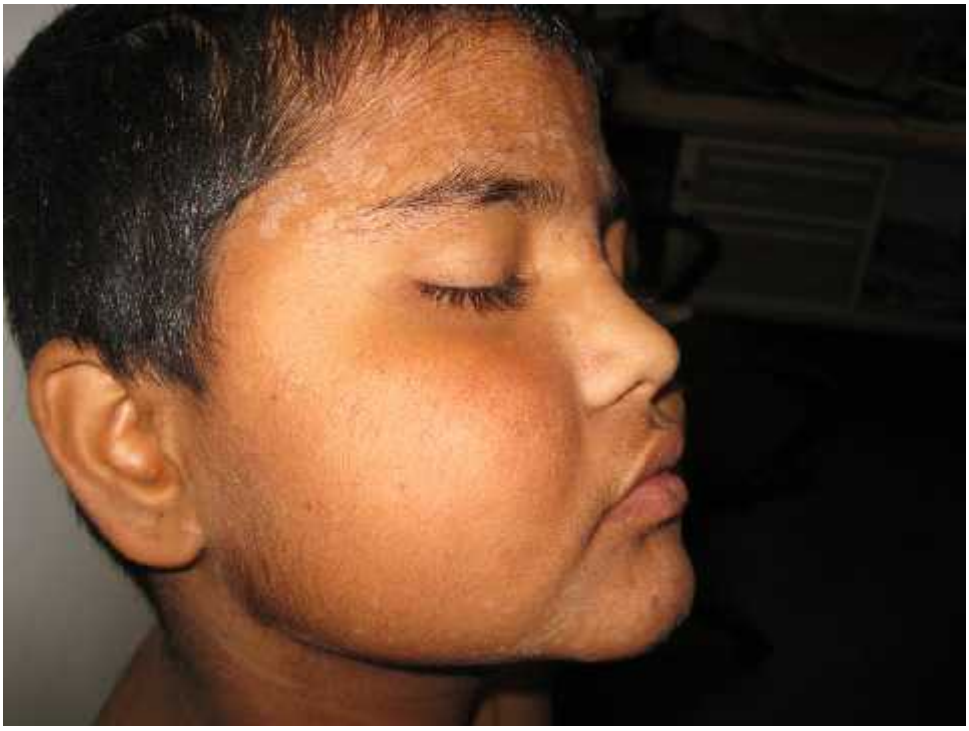


Figure 4 : Hypopigmented scaly macules over the forehead

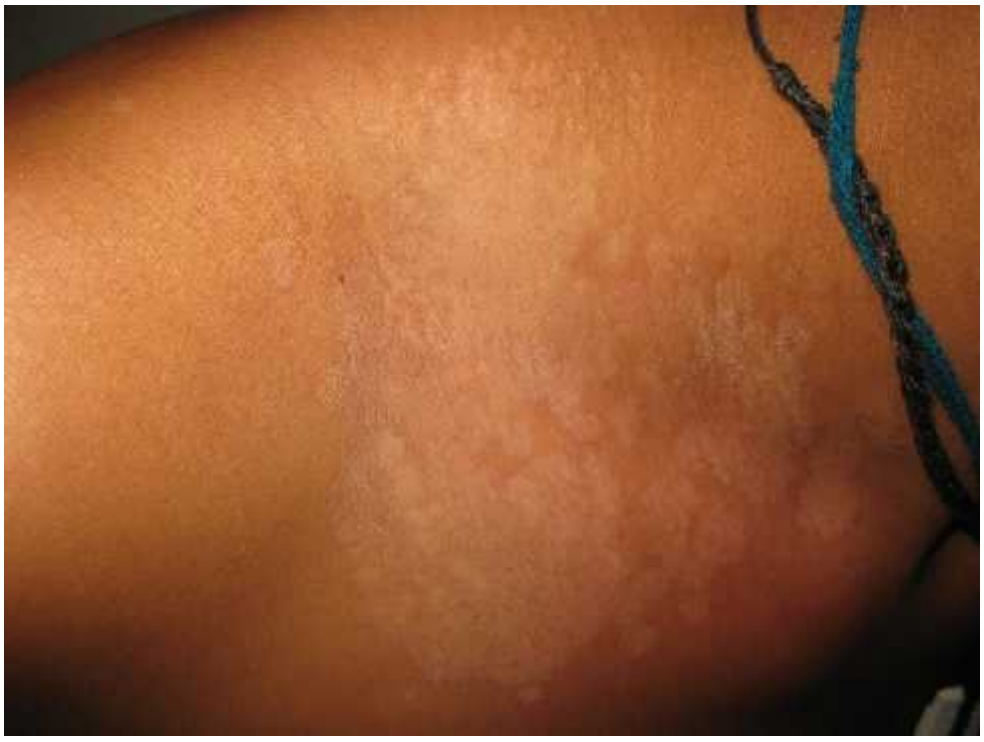


Figure 5 : Hypopigmented patch over the upper trunk

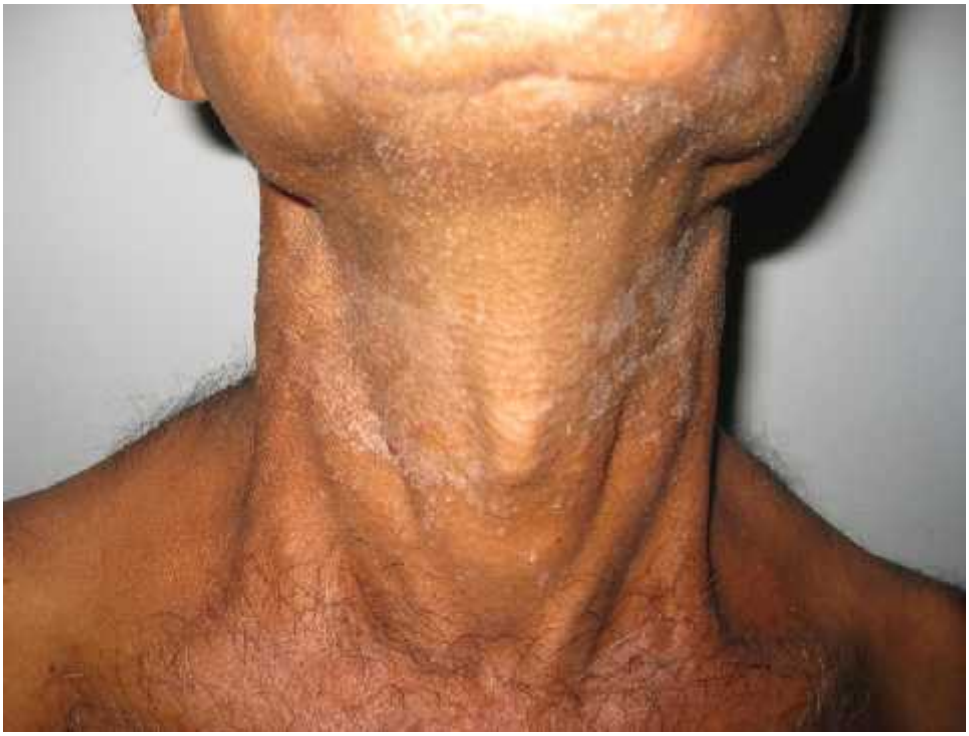


Figure 6 : Scaly patch over the neck

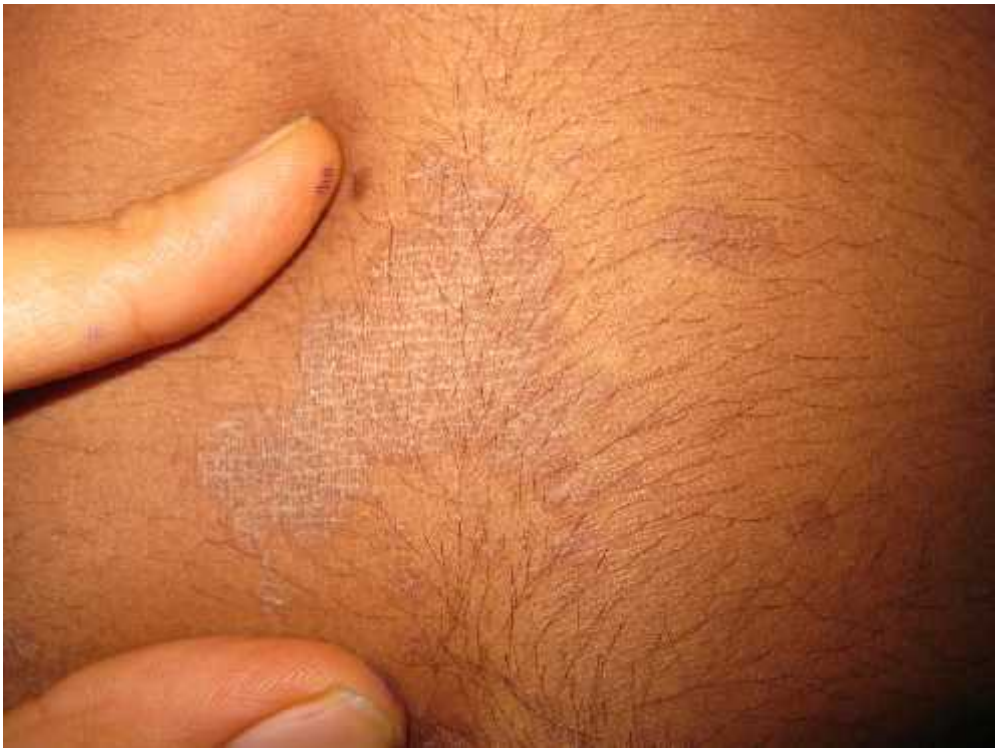


Figure 7 : Scales getting prominent after stretching of the skin

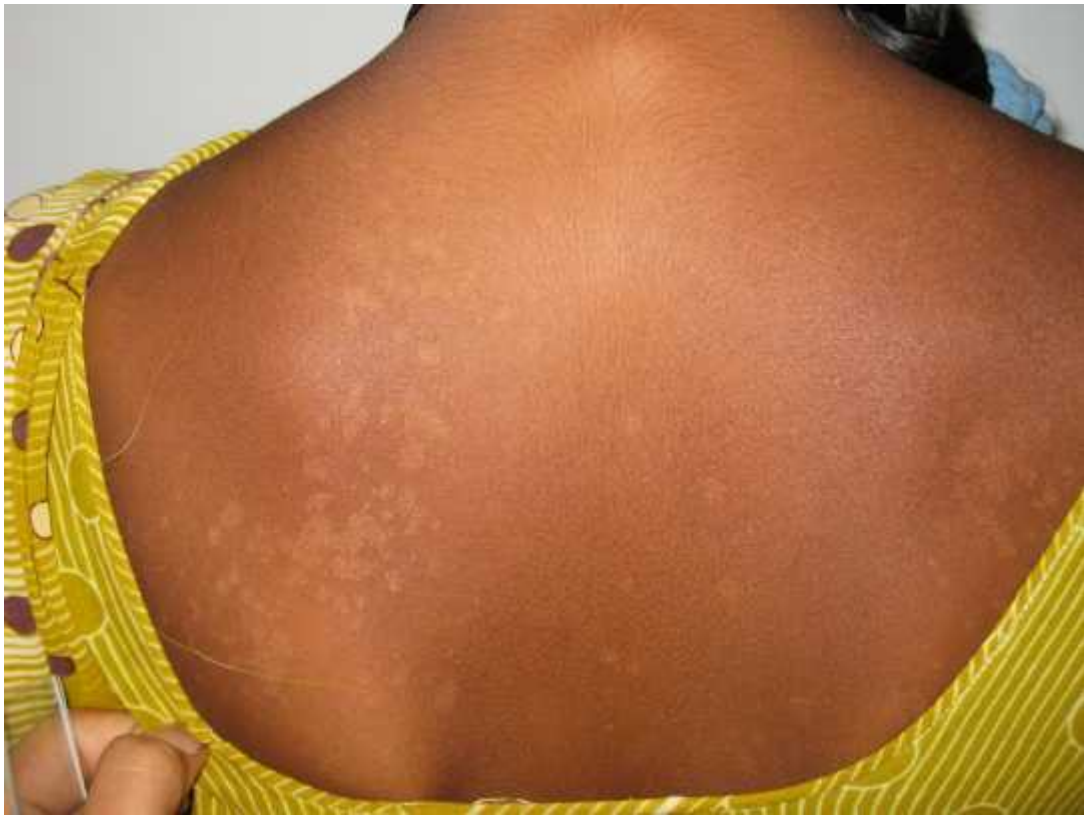


Figure 8 : Hypopigmented scaly macules over the back

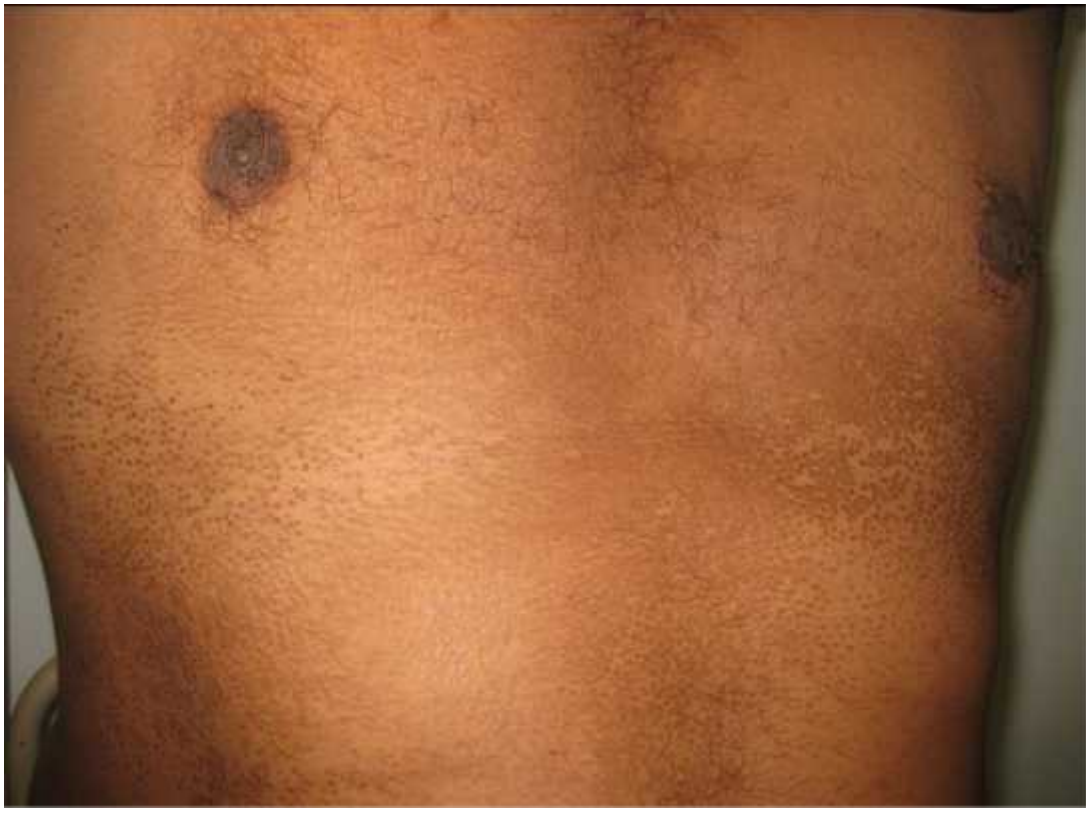


Figure 9 : Follicular variety of pityriasis versicolor

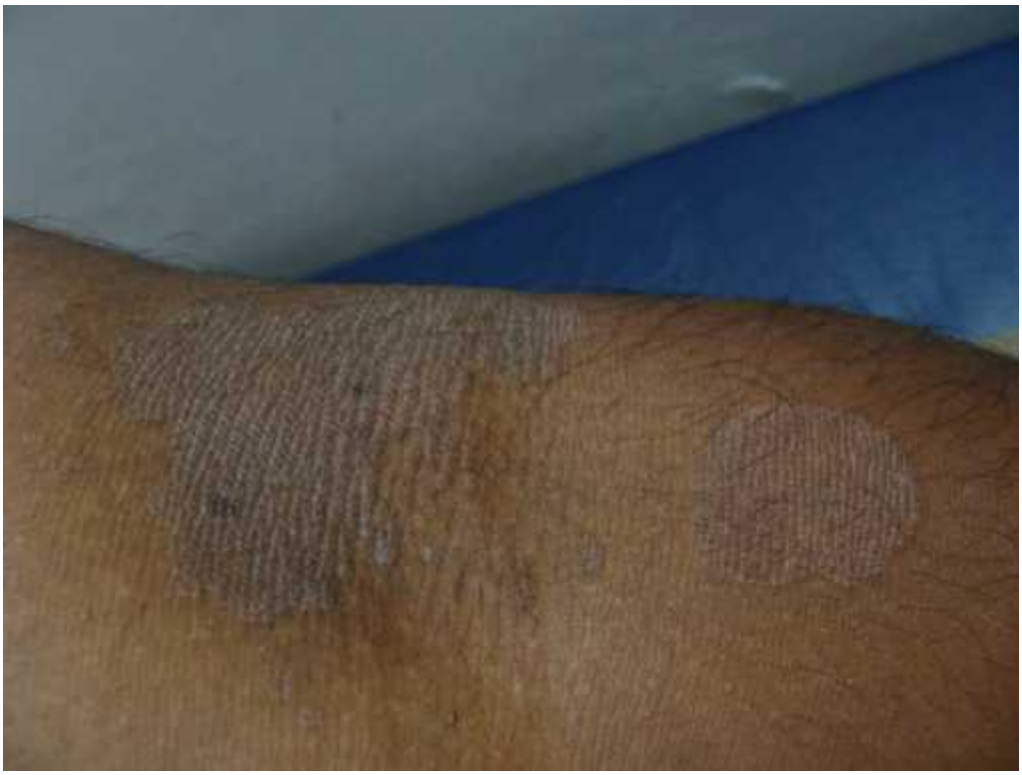


Figure 10 : Hyperpigmented variety of pityriasis versicolor

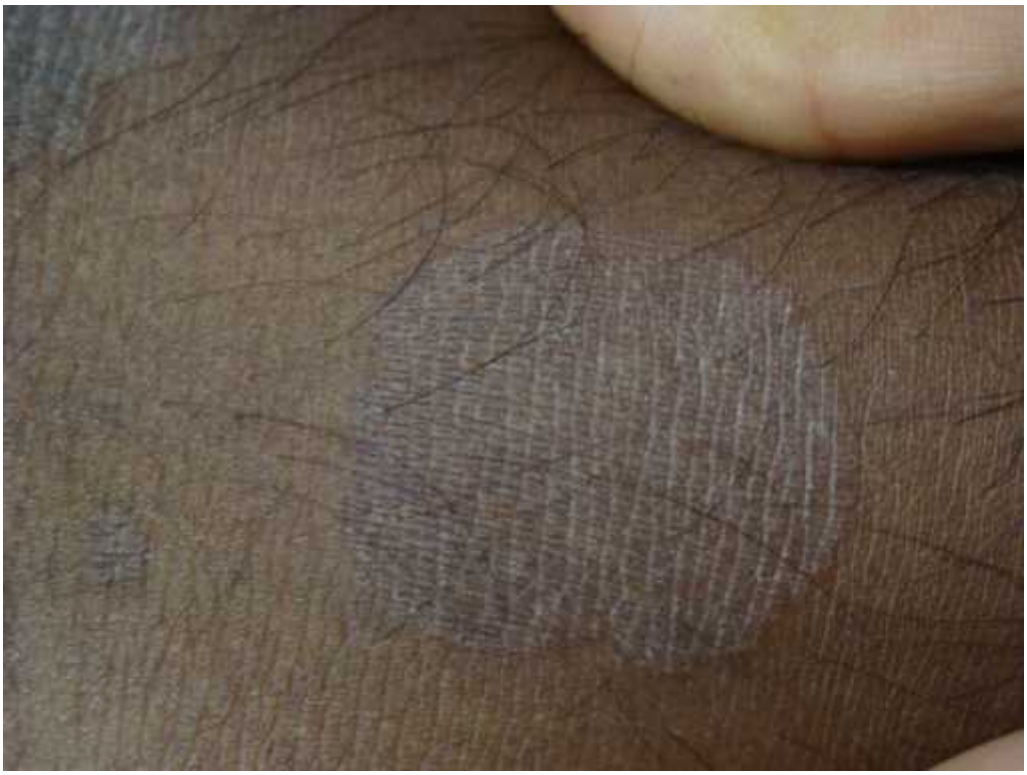


Figure 11 : Hyperpigmented variety of pityriasis versicolor

DIFFERENTIAL DIAGNOSIS

In most cases it is relatively easy to diagnose pityriasis versicolor, although the varied presentation of the lesions may be confusing to the inexperienced clinician.[61]

The differential diagnosis includes pigmentary disorders such as vitiligo and melasma. Other diseases to be excluded are pityriasis alba, pityriasis rosea, secondary syphilis and pinta.[10] In addition, seborrheic dermatitis and confluent and reticulated papillomatosis of Gougerot and Carteaud may also resemble pityriasis versicolor. These two latter diseases may also be caused by Malassezia species.[81,83]

Wood's light examination may help in the diagnosis of pityriasis versicolor. Under Wood's light, the lesion of pityriasis versicolor fluoresce a bright yellow or golden yellow.[10]

The distinguishing feature of pityriasis versicolor, however, is the transformation from the yeast to a mycelia form, resulting in a characteristic "spaghetti and meat balls" appearance of the yeast under the microscope.[61]

TREATMENT

At the beginning of 20th century the compound used for the treatment of superficial fungal infections had a non-specific action spectrum and minimal effectiveness, there were no systemic antifungal agents. In 1903 de Bergmann and Gougerot discussed the use of potassium iodide in sporotrichosis. In 1907 Whitfield compounded an ointment to treat superficial fungal infections. In 1940 sulfonamides were reported to have fungistatic properties with limited efficacy in paracoccidioidomycosis. Long treatment time was required and a high relapse rate was observed. In 1948 hydroxystilbamidine, an antiprotozoal agent, was also noted to have antifungal actions.

In 1944, benzimidazole was the first azole discovered to have antifungal activity. The azoles can be subdivided into the imidazoles and triazoles. In 1952 substituted benzimidazole compounds were also found to have antifungal properties.

Developed as a 5% cream in 1958, Chlorimidazole was beneficial in wide range of cutaneous mycoses.

In 1960s thiabendazole and mebendazole were reported to have antifungal properties.

In 1969 the imidazoles, clotrimazole and miconazole were introduced, followed by econazole in 1974. Ketoconazole, developed in 1977, has become the standard among the azoles and is currently the most successful and widely used antifungal agent in these days.

In the mid – 1980s two broad spectrum, orally available triazoles were introduced, itraconazole and fluconazole.

The allylacrines and morpholines are two other classes of antifungal agents that will have a significant impact on antifungal therapy for superficial dermatomycoses, including onychomycoses.[79]

Pityriasis versicolor responds well to a variety of topical antifungal agents because the conversion of Malassezia yeasts to the mycelia form is thought to be due to the endogenous host factors, recurrence is common and clinicians may consider prophylactic treatment for those who have propensity for repeated bouts of the disease.[10]

Contrary to this observation in adults with pityriasis versicolor refractory to treatment, the lesions in infants respond very well to treatment.[41]

TOPICAL TREATMENT

NON-SPECIFIC ANTIFUNGAL AGENTS

Majority of them do not have direct action on fungus. Generally, they act by physically / chemically removing infected dead tissue in the stratum corneum and / or affecting cell turnover rates.[10]

Selenium sulphide – available as a 2.5% lotion, cream or shampoo.[77,] It has been effective in treatment of pityriasis versicolor

It is a cheaper approach to treat pityriasis versicolor, available in detergent base. The liquid is pinkish yellow and is best applied at bedtime and should be washed off the next morning. In most of the cases, it is necessary to apply the material regularly (i.e. every other night over 2 weeks). In some patients however one or two applications may be sufficient. The principal advantage is the low cost and the convenience of application. On the other hand it is irritant if inadvertently applied to the face or genitalia, hence necessitating care in its application. It also stains clothes and bedding.[59]

Propylene glycol - A keratolytic agent that is often used as a base in other topical medications.[4] 50:50 propylene glycol in water, has been used intermittently as long term suppressive therapy to prevent relapse.[59]

Whitfield's ointment – contains benzoic acid (fungistatic action) and salicylic acid (has keratolytic action) in a ratio of 2:1 (12% :6%).[10]

Sulphur and salicylic acid – several studies have reported the effectiveness of this combination of agent in treatment of pityriasis versicolor.[72]

Sodium hyposulphite – 10-20% of aqueous sodium hyposulphide can be used, but is less effective.[24]

Other non–specific agents used to treat pityriasis versicolor. These include povidone -iodine paint, benzoyl peroxide and tretinoin cream.[10]

SPECIFIC TOPICAL ANTIFUNGAL AGENTS

Halprogin – It is effective in treatment for pityriasis versicolor.[73]

Zinc pyrithione – Efficacy of zinc pyrithione shampoo 1% versus its vehicle has been shown in two studies.[78]

Tolciclate – Like tolnaflate, tolciclate is a member of the thiocarbamate group of antifungal agents. The mechanism of action is to block the sterol biosynthesis in fungal cells by inhibiting squalene epoxidase.[42] Both cream and lotion formulations, each of 1% are effective against pityriasis versicolor.[74]

Ciclopirox-olamine – It is a hydroxypridone and has a broad spectrum anti-fungal action. 1% creams formulation has been shown to be more effective than both vehicle and clotrimazole 1% cream. The effectiveness of ciclopirox-olamine has been supported in number of studies.[10]

Main mode of action is interfering with the uptake and accumulation of products required for cell membrane synthesis. At higher concentrations alternation in

cell permeability and inhibition of respiratory activity may occur. The drug also has an anti-inflammatory activity and inhibits synthesis of prostaglandins and leukotrienes.[79]

Bifonazole – may produce irreversible changes in Malassezia yeast that can be seen using electron microscopy. Multiple treatment options have been reported, bifonazole cream 1%, spray 1%, solution 1%, gel 1%, powder 1% and shampoo 1%. In some patients a single application may be effective, although a three-day treatment schedule has been more effective. [75]

Clotrimazole - A broad-spectrum imidazole reported to be effective against pityriasis versicolor in both open and controlled, double-blind trials. Comparative studies have shown clotrimazole 1% cream to be as effective as Whitfield's ointment, but more acceptable to patients.[10]

It is mainly a broad–spectrum imidazole, used to treat superficial dermatomycoses and oropharyngeal and vaginal candidiasis. It is not indicated in systemic mycosis.[79]

Fluconazole – In a 2% shampoo, it is considered to be effective.[10]

Ketoconazole – The 2% cream formulation has been reported to be more effective than placebo[140] in a double – blind, randomized study. Ketoconazole 2% shampoo[141] can be effective with only one day of treatment, although longer treatment durations have been reported.

Lange et al, demonstrated use of ketoconazole 2% shampoo to be highly effective in relieving the signs and symptoms of pityriasis versicolor.[70] Results have showed that treatment of pityriasis versicolor with topical ketoconazole is as effective as treatment with oral ketaconazole.[10]

Autoradiographic results indicate that the penetration of tritium – labelled ketoconazole cream is restricted to the stratum corneum and to the boundary of the stratum corneum and stratum granulosum. Prolonged retention in the stratum corneum may be the key to the high efficacy of topical ketoconazole. Unlike miconazole, ketoconazole is only weakly inactivated by the presence of host proteins, so that a substantial part of the applied ketoconazole may be bioavailable.[140]

Topical treatment offers high effectiveness, easily applied, once-daily, cosmetically acceptable topical treatment that is therapeutically equal to if not better than systemic ketaconazole.[13]

Miconazole and Econazole – These two synthetic phenylethyl imidazole derivatives are closely related. Both have shown to be effective in treatment of pityriasis versicolor. Miconazole nitrate 2% cream is effective when applied twice daily for 2 weeks [66]. Econazole nitrate has been reported to be effective as a 1% foaming solution cream or shampoo,[67] when used once daily.

Sertaconazole – The 2% cream formulation has been shown to be effective in treatment of pityriasis versicolor.[10]

Sulconazole – Sulconazole 1% cream may be used to treat pityriasis versicolor successfully.[10]

Tioconazole – This is a 1 – substituted imidazole, both the 1% lotion and 1% solution have been shown to be effective in pityriasis versicolor.[65]

Terbinafine – As a 1% solution, terbinafine has been shown to be more effective than vehicle with 7 days of twice daily application treatment. 1% solution provides effective, safe, easy to use topical therapy. This is a significant advance in treatment of an infection that is extremely common in tropical climates.

This new 1% formulation is of particular value because it contains propylene glycol, a keratolytic that is effective in treatment of pityriasis versicolor. Systemic

(oral) terbinafine is poorly active against pityriasis versicolor, probably because of the superficial nature of this infection.[64]

The drug remains pharmacologically active for weeks after administration, most likely because it is highly lipophilic and keratophilic and thus remains in therapeutic concentration at the site of infection for some time after administration has ceased.[64]

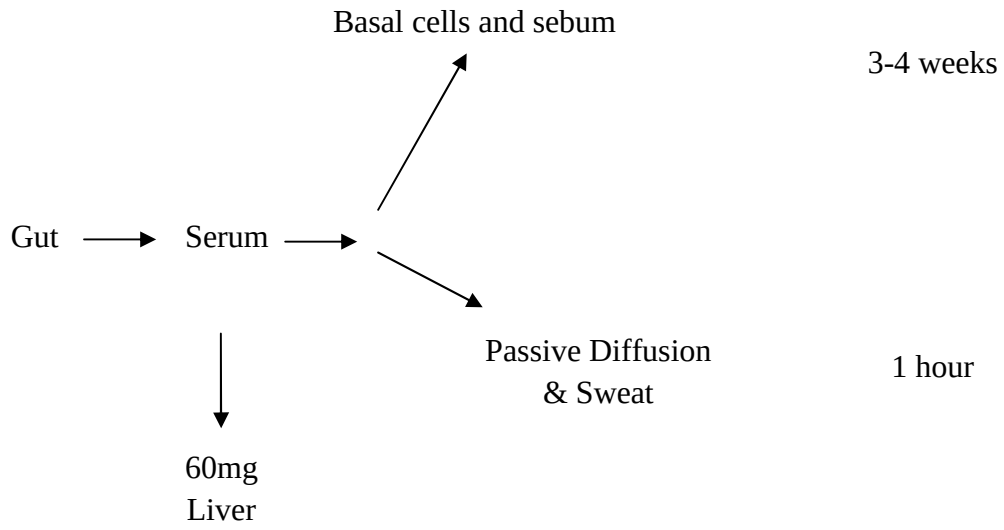
The advantages of oral medications are increased patient compliance, oral therapy can be more convenient and less time consuming for the patient. However, some physicians may prefer to reserve systemic treatment for cases in which the pityriasis versicolor involves a large area of the skin or when the disease is recurrent.

ORAL TREATMENT

Ketoconazole was the first significant broad-spectrum orally administered imidazole that was effective against many of the fungal infections. It is well absorbed in subjects with normal gastric acidity. The bioavailability may be reduced in situations in which there exists some degree of achlorhydria. Time to peak the serum concentration is 2 to 4 hours. Steady state plasma levels are achieved within 3 to 4 days. A major route by which ketoconazole rapidly reaches stratum corneum is the eccrine sweat glands. It has been detected in eccrine sweat less than 1 hour after a single dose of 400 mg. Slower routes of drug delivery to the skin are excretion into the sebum and incorporation into the epidermal basal layer.[79]

Ketoconazole inhibits the cytochrome P-450 enzyme lanosterol 14-demethylase, thereby inhibiting conversion of lanosterol to ergosterol. There is accumulation of C14 methylated sterols with depletion of membrane ergosterol. Within the fungal cell there is also an inhibition of triglyceride and phospholipid biosynthesis. Cell wall chitin synthesis is inhibited, hence membrane permeability, fluidity and synthesis are affected. The activity of other membrane – associated

enzymes may be altered. The oxidative and peroxidative enzyme system may be inhibited leading to an accumulation of toxic reactive peroxides within the cell. The drug is primarily fungistatic. At high concentration, generally not achieved therapeutically, the drug may also be fungicidal.[79]



Flow Chart 1 : Ketoconazole reaches the skin through both a slow and a rapid route. The eccrine apparatus allows ketoconazole to rapidly reach the human stratum corneum.

In early studies, the duration of therapy was 4 weeks, although later studies suggested that shorter courses of treatment were also effective.[39] The drug is effective in 200 mg doses.[40]

Itraconazole – It is a newer broad-spectrum azole dioxaline. Persists in the basal layer of the epidermis, maintaining drug levels for 3-4 weeks, after cessation of therapy. This is due to its extensive excretion in the sebum, as well as its lack of redistribution into the circulation after incorporation in to the epidermis, hair and nails. This has resulted in increased cure rates between discontinuation of therapy and follow-up visits (the reservoir effect).[4]

Drug absorption is enhanced when the medication is taken with food. It is effective at a dosage of 200 mg/day, when taken for either 5 or 7 days. The minimum cumulative dose for itraconazole is 1000 mg, although an improved efficacy is more likely to occur with the regimen of 200 mg/day for 7 days.[10]

Fluconazole - has favorable pharmacokinetics, especially its slow elimination from the skin, allowed its effective systemic administration in single weekly doses in a variety of superficial fungal infections.[38]

It is not as dependent on gastric pH for absorption, so it could be used with antacids or H2 blockers. It is excreted from kidney and is only 11% bound to serum proteins. It exhibits much more selectivity for fungal cytochrome P450 vs mammalian enzymes than other azoles at antifungal doses. There are very limited data on the use of fluconazole in children, however its excellent safety profile and an available liquid formulation may make it an important treatment is future.[4]

Pramiconazole – It is a broad–spectrum triazole antifungal with potential for oral treatment of pityriasis versicolor. It has prominent affinity to fungal cytochrome P450, which is involved in the biosynthesis of ergosterol from lanosterol and to its favorable pharmacokinetics.

It is a convenient treatment of short duration for pityriasis versicolor. It is well tolerated and shows beneficial effects in the treatment with dosing regimens of 200 or 400 mg taken once and 200 mg taken once daily for 2 or 3 days. It reduces the severity of erythma, desquamation and pruritus. In a study done, it was well tolerated at all dose strengths.[91]

Prophylactic treatment – While pityriasis versicolor is relatively easy to treat, the importance of endogenous host factors and uncontrollable environmental factors in the development of the disease mean that recurrence may be common especially in predisposed individuals. Ketoconazole has been shown to prevent relapse in patients when administered as a 400 mg dose, once per month or as 200 mg/day for three consecutive days. Once per month follow up for upto 11 months showed a low rate of recurrence in patients receiving prophylactic treatment. Itraconazole 400mg one dose (four capsules 100 mg each) given once a month for 6 months have been demonstrated to be significantly more effective than placebo.[10]

MATERIAL AND METHODS

The study was conducted in the O.P.D of Dermatology, Venereology and Leprosy and Department of Microbiology at K.L.E'S Dr. Prabhakar Kore Hospital and MRC attached to Jawaharlal Nehru Medical College, Belgaum. One hundred and fifty patients who attended O.P.D were selected and included in the study group irrespective of their age, sex, socio-economic status and occupation after taking the consent. A detailed history was obtained from the patients particularly regarding their disease, its evolution, involution, habits, occupation, onset, duration and symptoms.

History like precipitating factors, similar complaints in family, personal history and presence of any associated illness were also noted. A thorough clinical examination was done with reference to the location, colour, extent of the lesion and all the details were recorded.

The skin was also screened for associated conditions, and then blood investigations, urine examination, random blood sugar, KOH examination and Wood's lamp examination were done in all the patients.

Microscopic examination for fungal elements : Direct examination of the scraped material from all the 150 patients were mounted on the slide with 10% KOH and examined for the yeast. The patients were made to sit in natural light and the site from which the material would be collected was cleaned with spirit. The scales from the lesions were collected by scraping with a blunt scalpel blade and were placed on a clean glass slide. One to two drops of 10% potassium hydroxide was added with the coverslip placed over it and slightly warmed over a low flame or kept for 20 minutes. A coverslip was gently pressed and excess of KOH was blotted with the help of blotting paper and microscopic examination for characteristic fungal elements was done by screening both under low and high power objectives.

By this method filaments having tendency to break, into short segments of various sizes and grape like clusters of round cells, with occasional buds were seen.

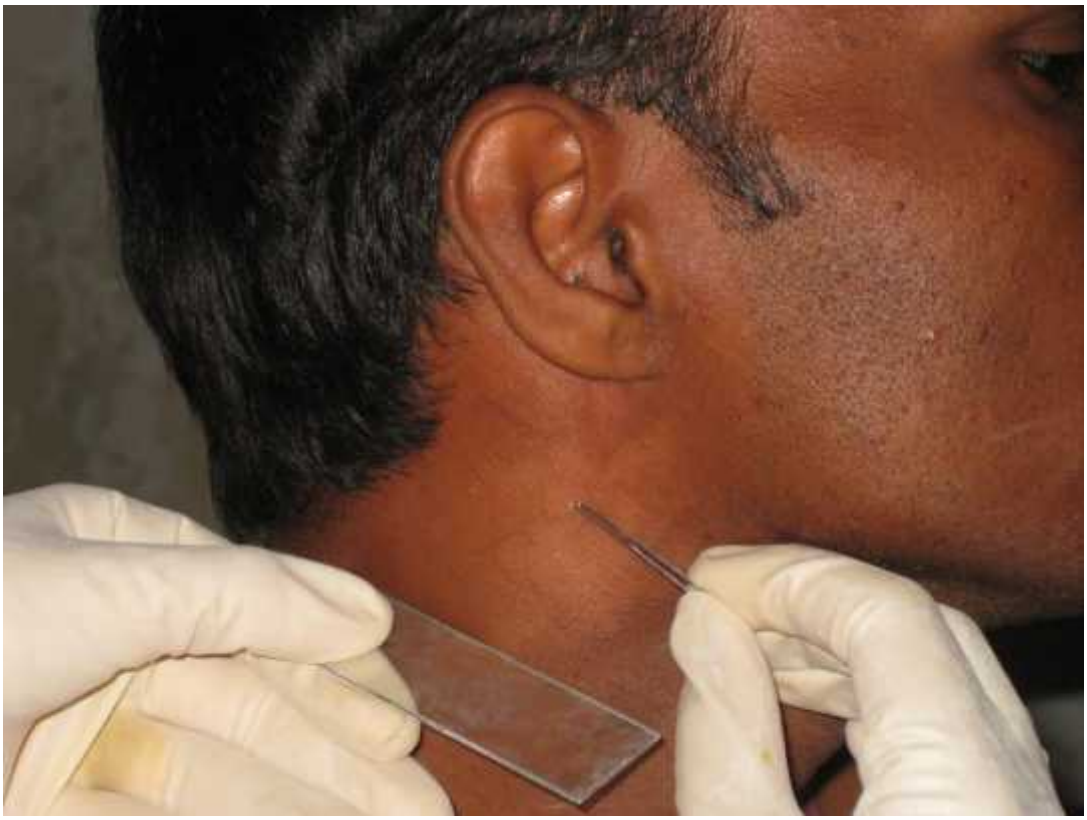


Figure 12 : Collection of sample from the lesion

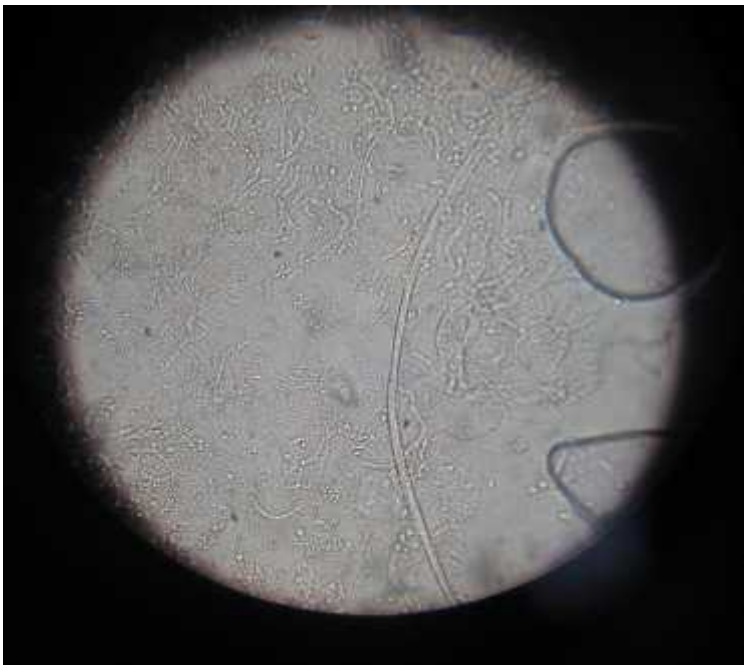


Figure 13 : Direct microscopy examination of fungal elements (typical spaghetti and meatball appearance) in KOH mount

Wood's lamp examination : All the cases were studied with Wood's lamp examination which is done in a dark room. The skin area to be examined was cleansed with spirit. The Wood's lamp was switched on and allowed to warm up for one minute to attain optimum intensity following which light source was made to fall on the lesional and perilesional skin by keeping it 4-5 inches away from the body. The affected areas were observed for fluorescence.

Routine examination of the blood and urine including random blood sugar levels were done.

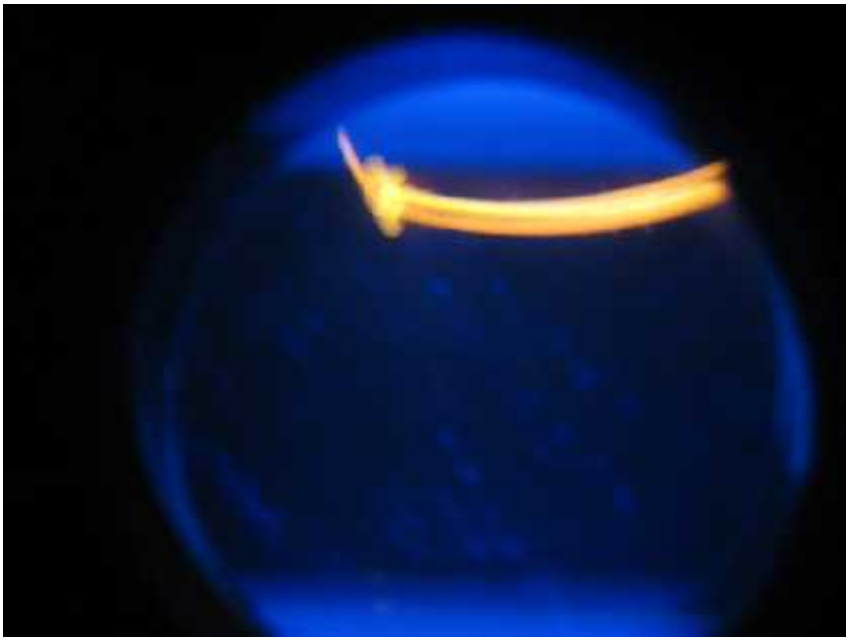


Figure 14 : Wood's lamp examination

Culture **:** Samples from all the 150 cases were collected with a blunt scalpel onto butter paper and thereafter transferred onto Sabouraud's Dextrose Agar (SDA) with chloramphenicol and Actidione (cycloheximide).

Two slopes were inoculated, one with olive oil and other without, were incubated at room temperature. Slopes were observed for growth after 3 to 4 days and everyday upto 10-15 days. If no visible growth seen after 15^{th} day, slopes were discarded.

Figure 15 : Growth of Malassezia on SDA with olive oil overlay

Positive growth for Malassezia was taken as creamy, moist, pasty growth on the slopes. To confirm, the smear from colonies was made and stained with saffranine.

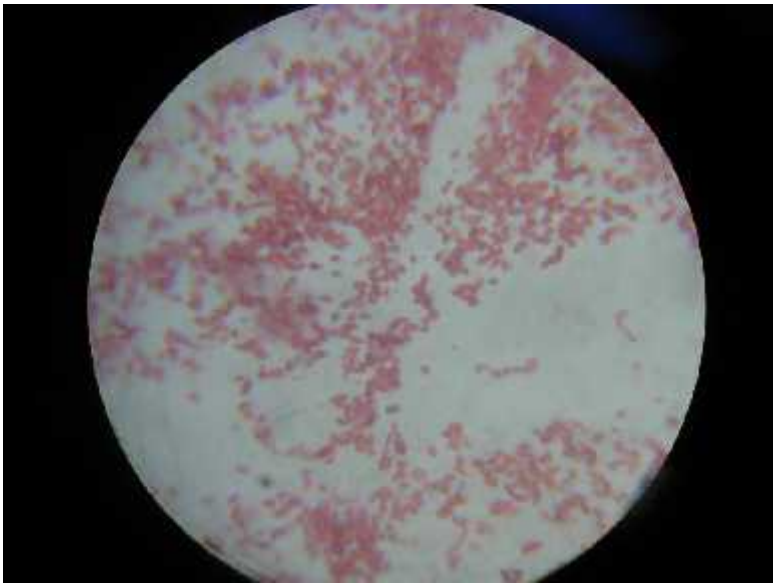


Figure 16 : Culture smear of Malassezia sympodialis

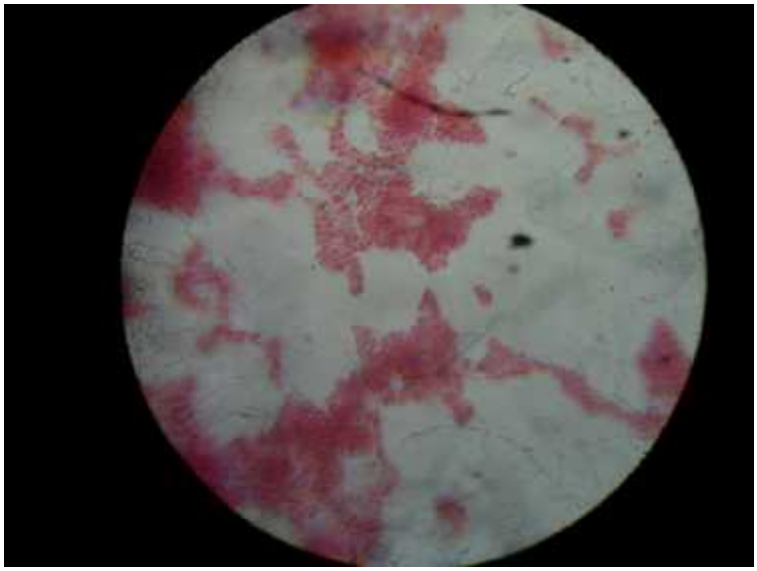
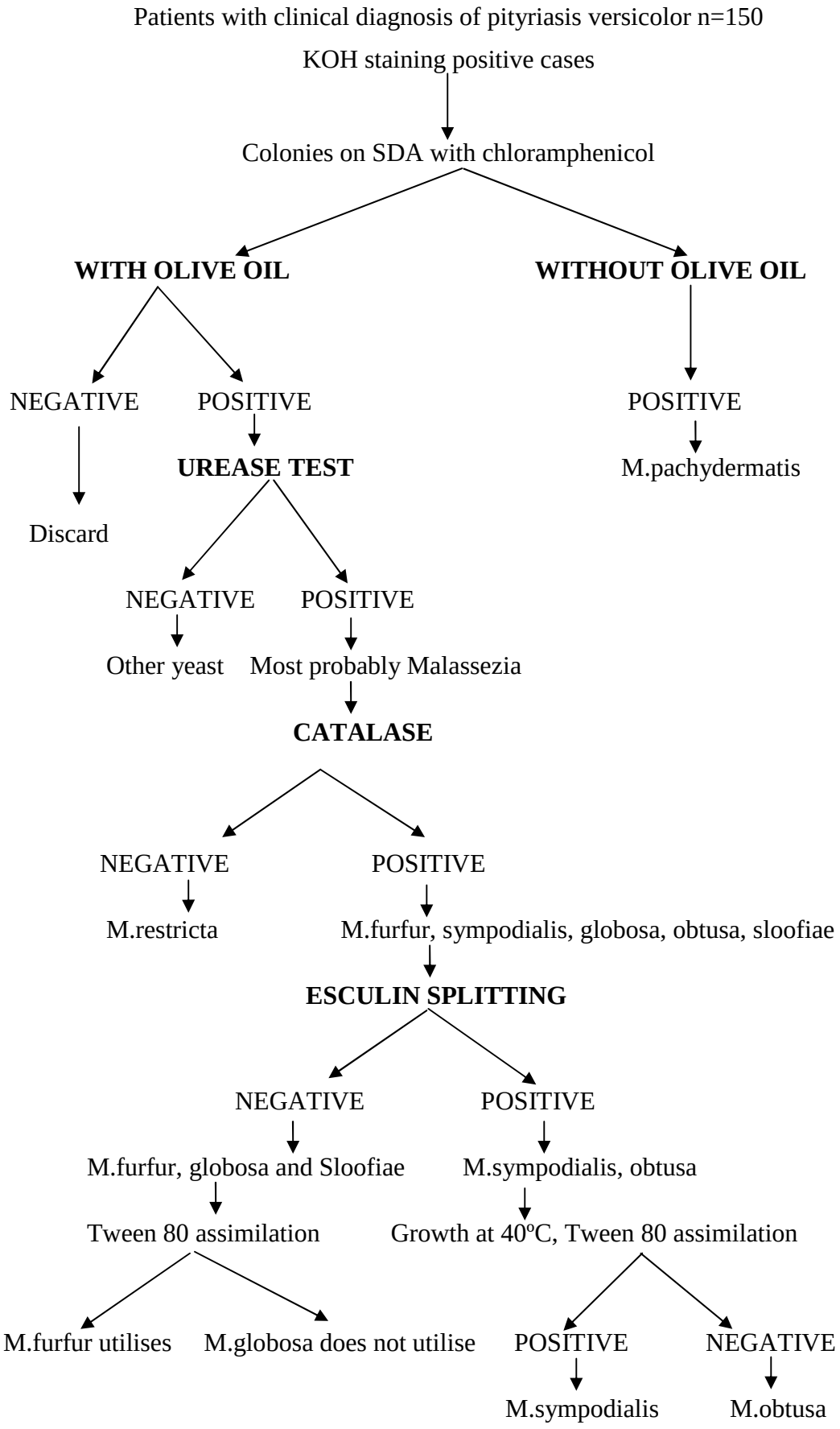


Figure 17 : Culture smear of Malassezia globosa

Yeast cells with broad based budding were taken as Malassezia species. Sometimes oval to spherical or different morphological forms may also be obtained like short hyphae. Further confirmation of the species was done by biotyping.



FLOW CHART 2 : METHODOLOGY OF SPECIES IDENTIFICATION

TABLE 4

Figure 18 : Tween 80 with positive growth in the center

Analysis method :- Percentage distribution of the patient with different condition and different species were computed.

Chi square test was applied to find the distribution of the species in hyperpigmented and hypopigmented lesion.

OBSERVATION AND RESULTS

The present study is a one–year cross–sectional descriptive study and included 150 patients who attended the O.P.D of Dermatology, Venereology and Leprosy at K.L.E'S Dr. Prabhakar Kore Hospital and M.R.C from November 2008 to October 2009.

Table 5 : Prevalence (of pityriasis versicolor in the study hospital)

Total no. of patients (From November 2008 to October 2009)	**No. of patients with pityriasis versicolor**	**Percentage (%)**
23,424	150	0.64

Table 6 : Sex distribution

Sex	No. of Patients	Percentage (%)
Male	121	80.67
Female	29	19.4

Graph 1 : Sex distribution

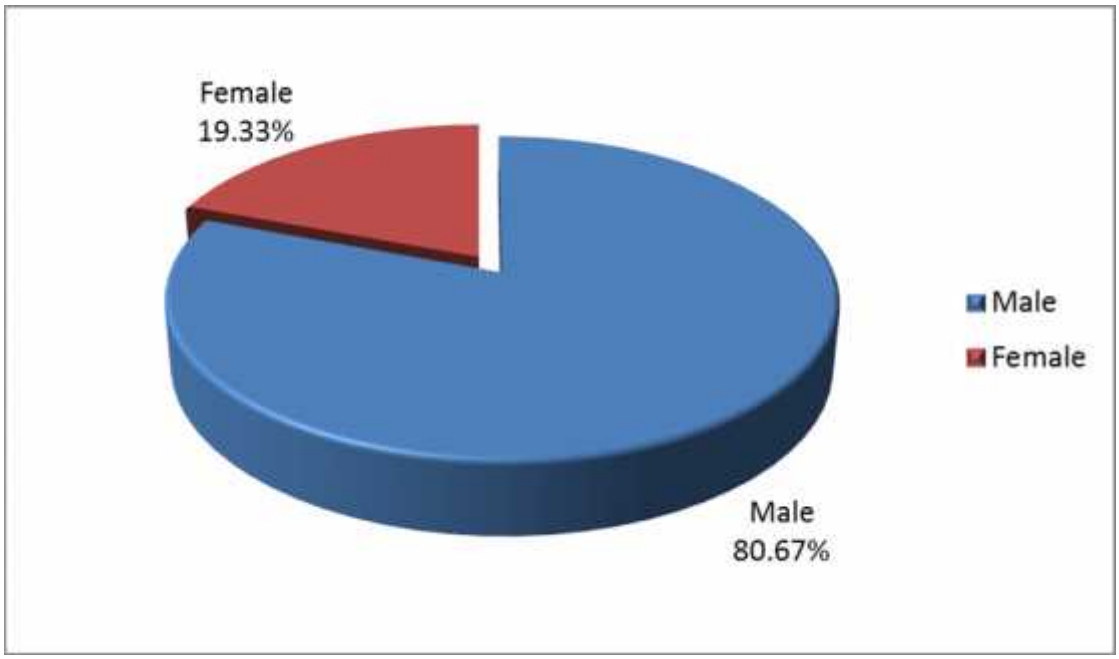


Table 7 : Age distribution

Age (In years)	No. of Patients	Percentage (%)
< 10	5	3.33
10 – 20	28	18.66
21 -30	59	39.33
31 – 40	28	18.66
41 – 50	13	8.66
> 50	17	11.33

Graph 2 : Age distribution

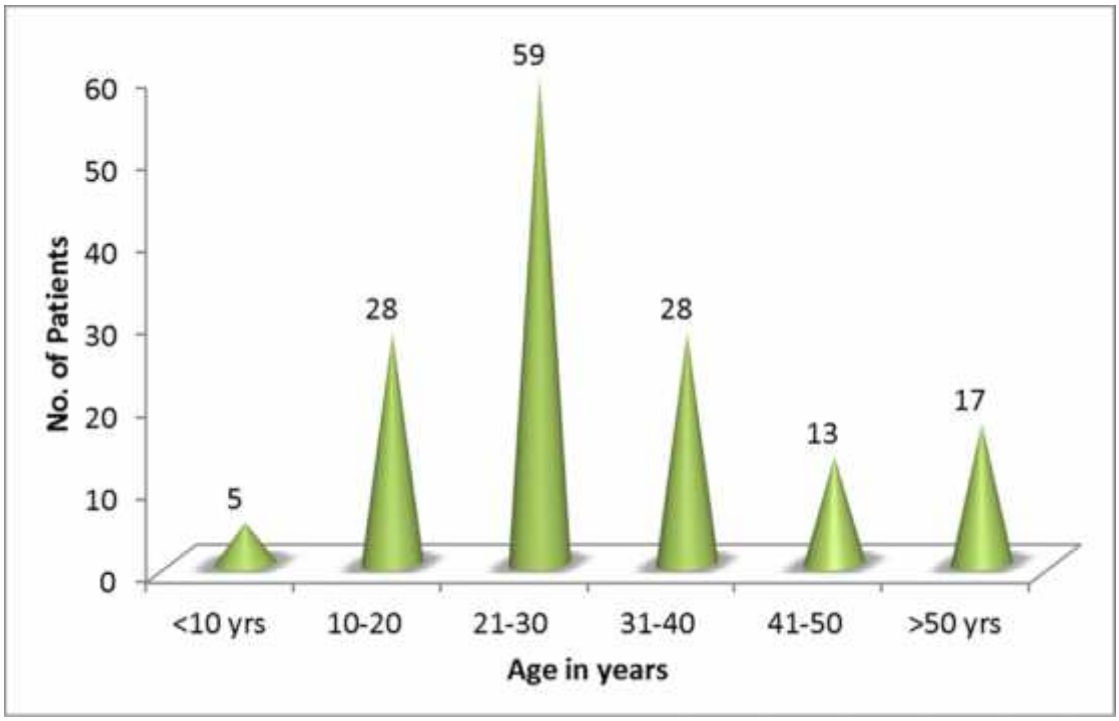


Table 8 : Occupation

Occupation	No. of Patients	Percentage (%)
Officer	46	30.66
Manual labourer	45	30
Housewife	20	13.33
Athlete	1	0.66
Student	38	25.33

Graph 3 : Occupation

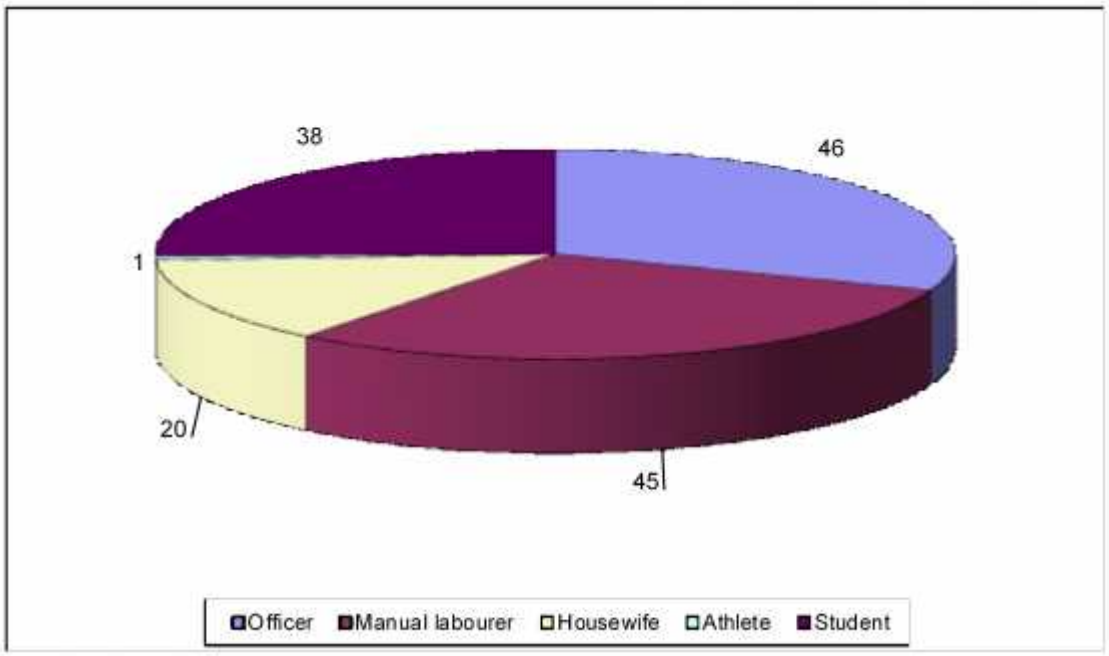


Table 9 : Chief complaints

Complaints	No. of Patients	Percentage (%)
Skin lesions	150	100
Symptomatic	60	40
Asymptomatic	90	60

Graph 4 : Chief Complaints

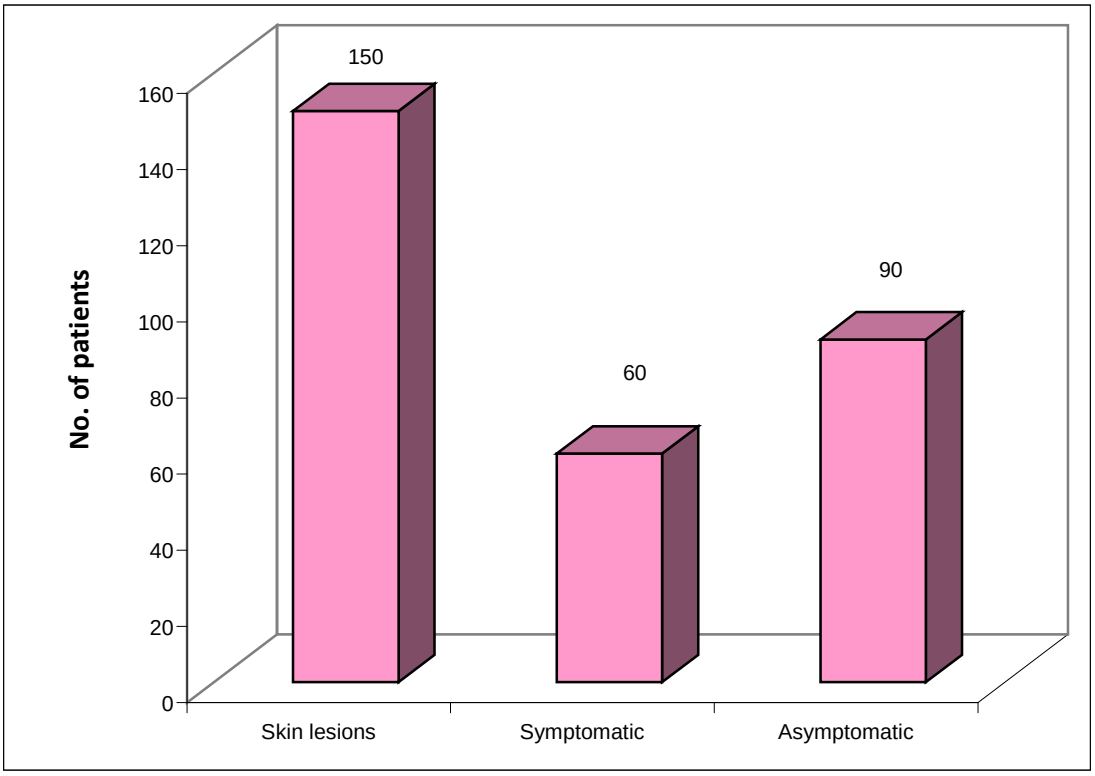


Table 10 : Skin type

Skin type	No. of patients	Percentage (%)
Type I	0	0
Type II	0	0
Type III	0	0
Type IV	117	78
Type V	33	22
Type VI	0	0

Table 11 : Type of pigmentation

Type of pigmentation	No. of Patients	Percentage (%)
Hyperpigmentation	24	16.0
Hypopigmentation	107	71.3
Mixed	19	12.7

Graph 5 : Type of Pigmentation

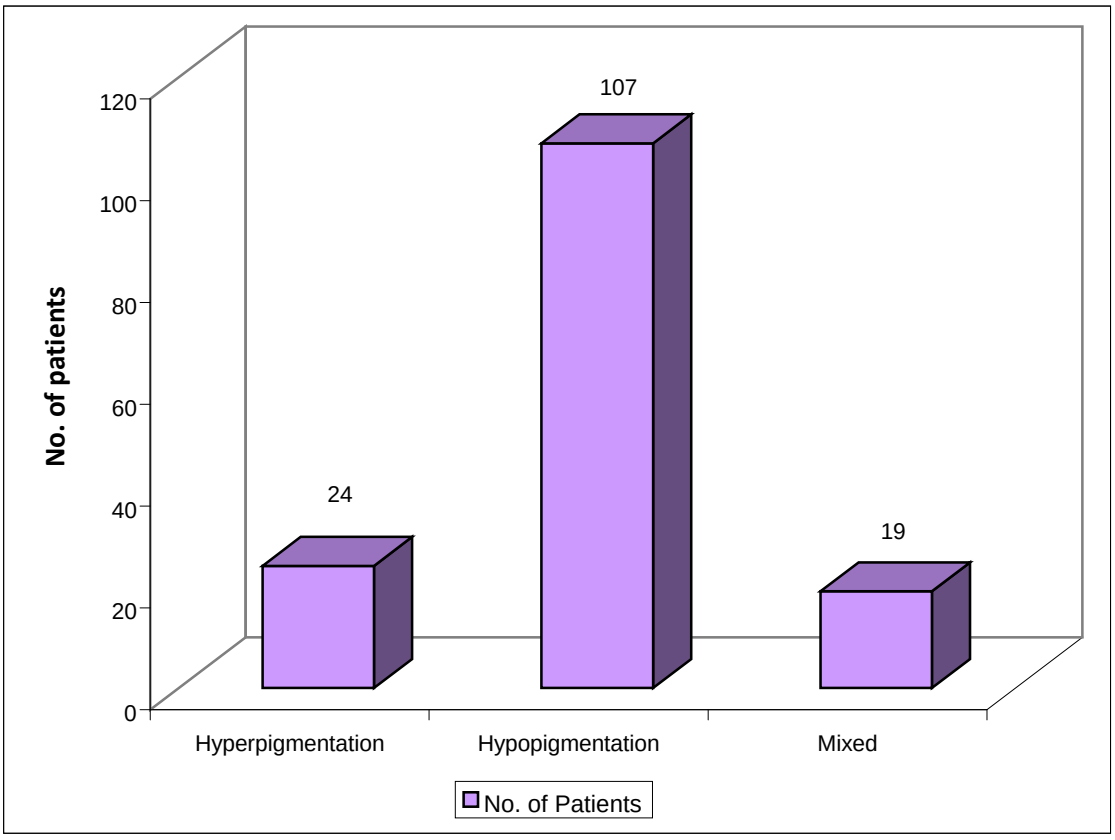


Table 12 : Site of lesions

SITE	No. of Patients	Percentage (%)
Neck	96	64
Back	69	46
Trunk	66	44
Face	20	13.33
Other	44	29.33

Graph 6 : Site of lesions

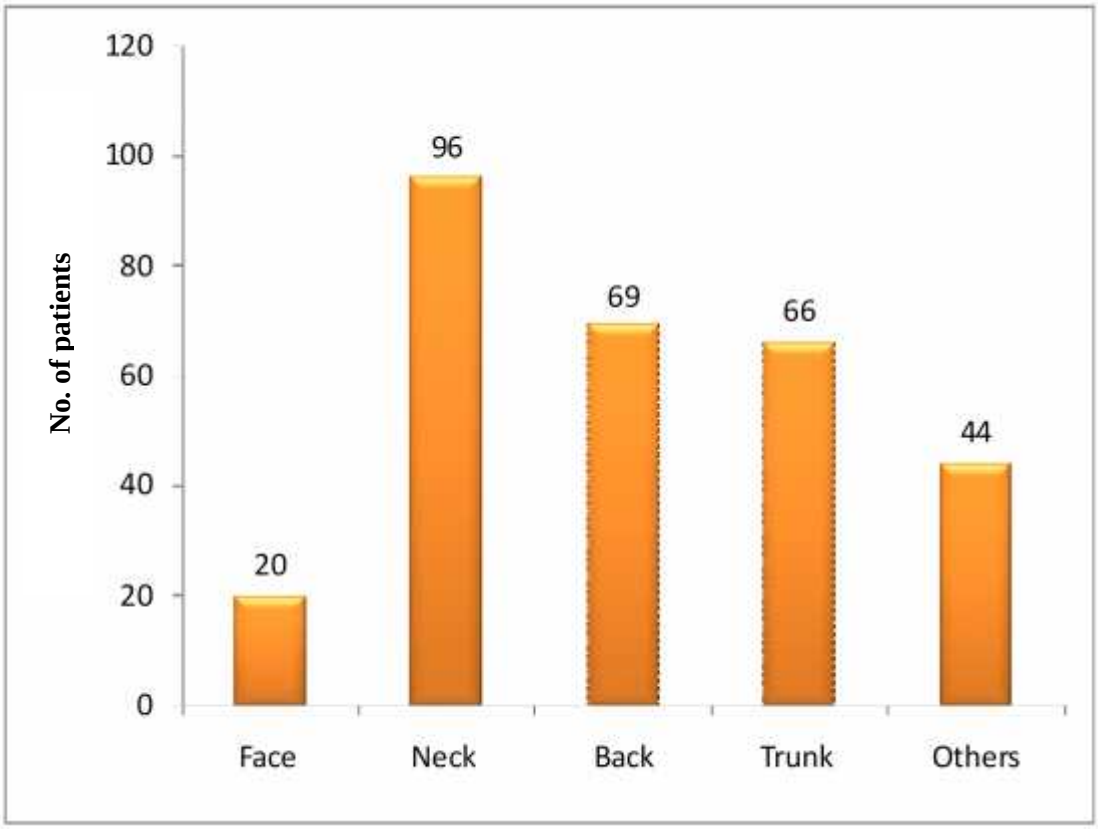


Table 13 : Type of lesions

SITE	No. of Patients	Percentage (%)
Macule	58	38.67
Patch	121	80.67
Follicular	21	14
Other	2	1.33

Graph 7 : Type of lesions

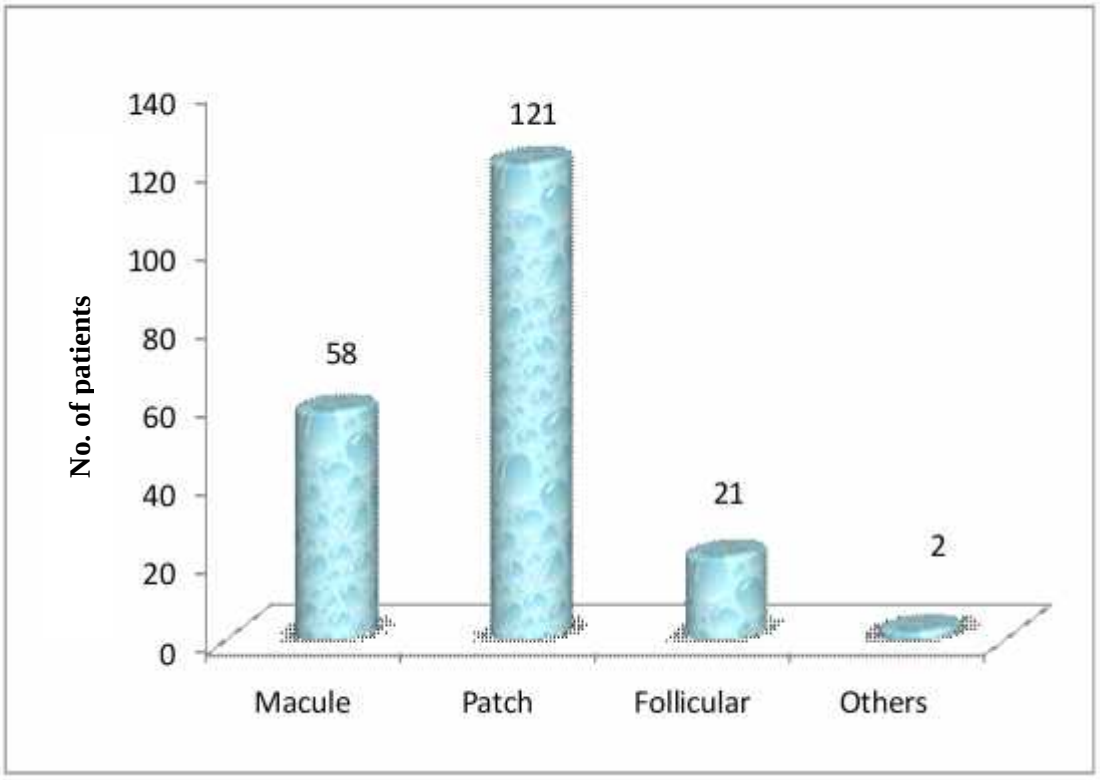


Table 14 : Family history

Family history	No. of Patients	Percentage (%)
Positive family history	8	5.33
Negative family history	142	94.66

Graph 8 : Family history

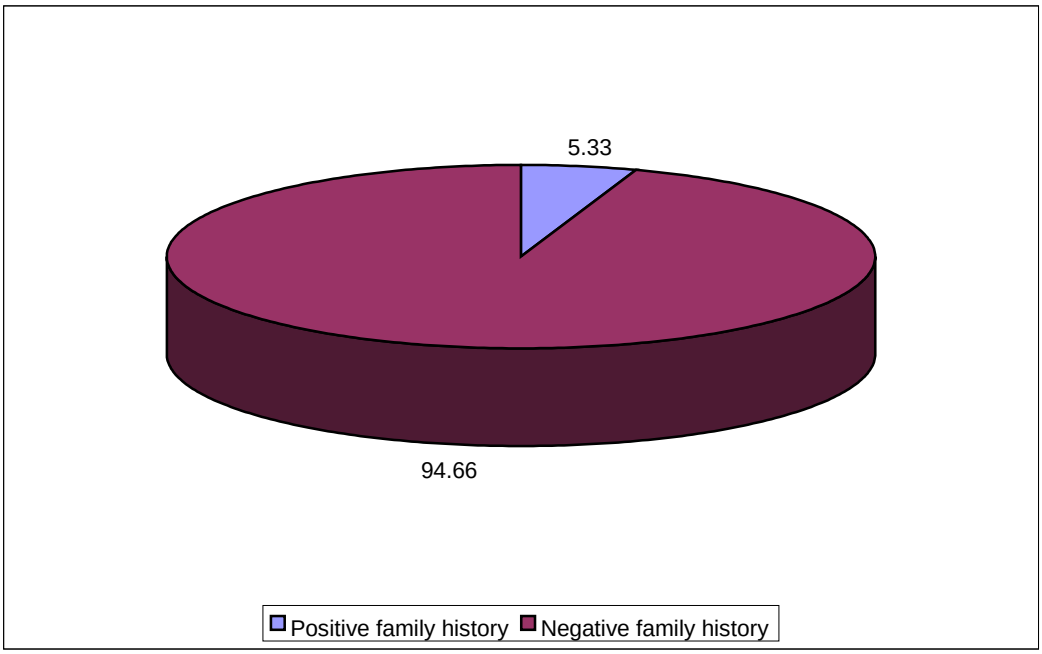


Table 15 : Personal history

History	No. of Patients	Percentage (%)
History of excessive sun exposure	103	52.82%
History of excessive sweating	92	47.18%

Graph 9 : Personal history

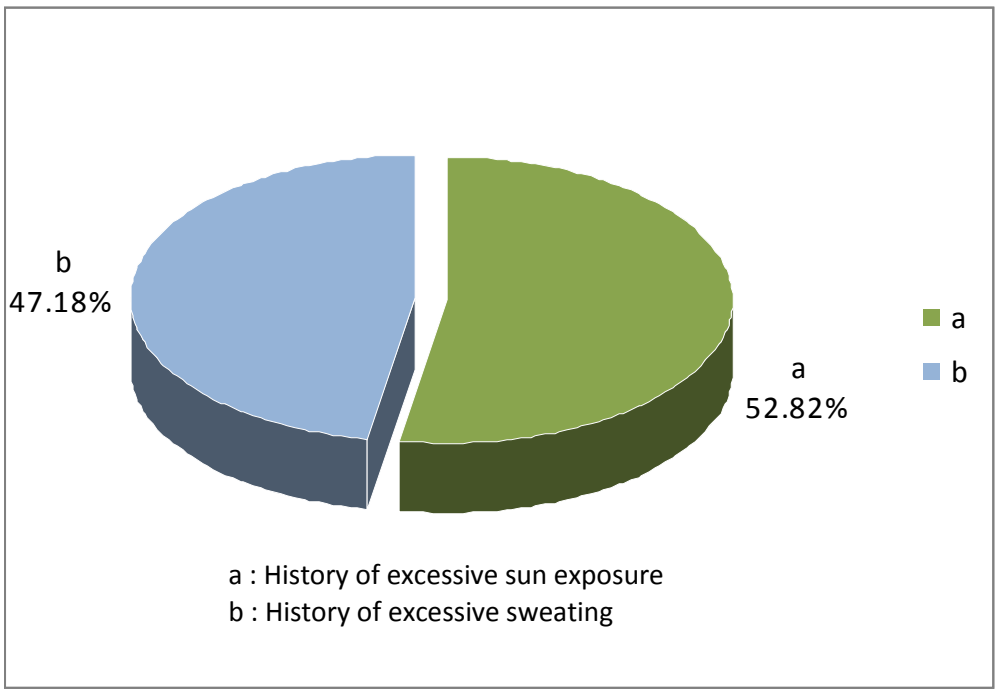


Table 16 : Comorbid conditions

	Diabetes	Other illness	Immunosuppressive therapy
No. of patients	17 (11.33%)	16 (10.67%)	7 (4.67%)

Graph 10 : Comorbid conditions

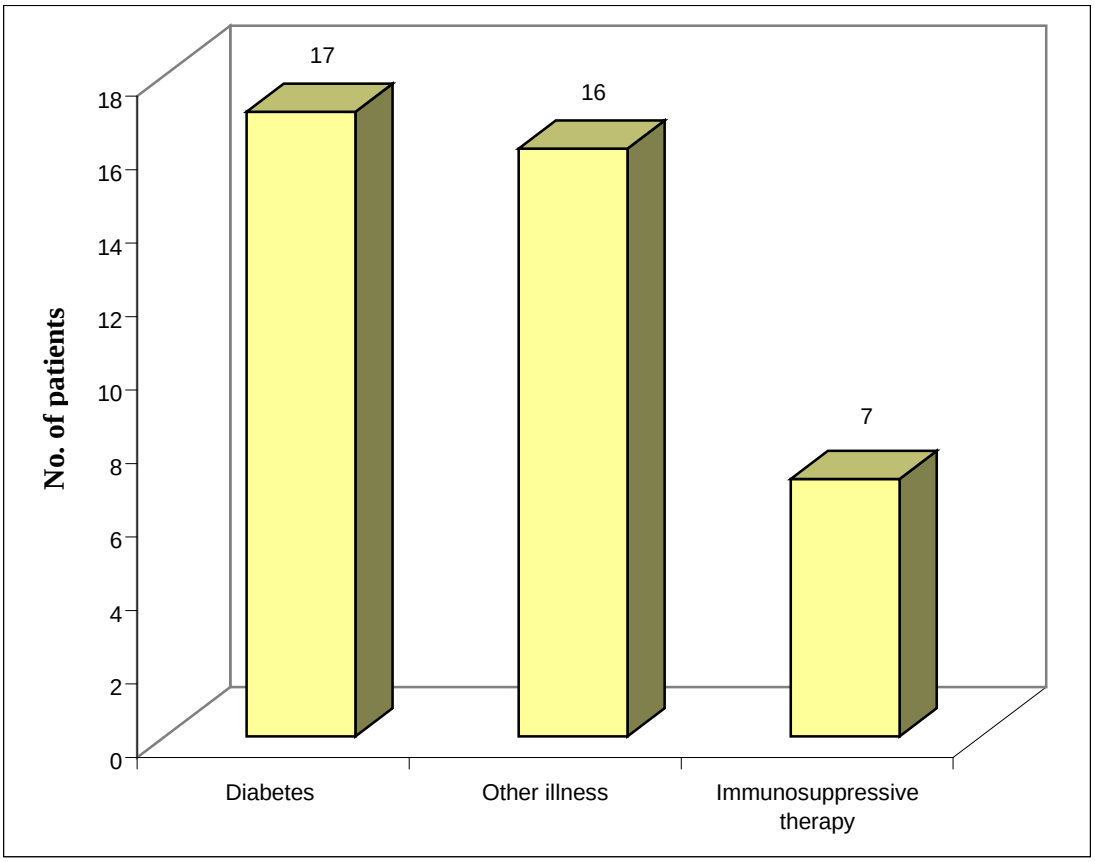


Table 17 : Associated dermatoses

Associated dermatoses	No. of Patients	Percentage (%)
Acne	13	8.66
Dermatophytic infections	3	2.00
Acrochordons	1	0.66
Molluscum contagiosum	2	1.33
Melasma	2	1.33
Warts	1	0.66
Schamberg's disease	1	0.66
Vitiligo	3	3.00
Fissure feet	2	1.33
Keloid	1	0.66
Folliculitis	1	0.66
Androgenetic alopecia	1	0.66
Nevus anaemicus	1	0.66
Pure neuritic leprosy	1	0.66
Oral candidiasis	1	0.66
Lichen planus	1	0.66

Table 18 : KOH examination

KOH	No. of Patients	Percentage (%)
Positive	145	96.7
Negative	5	3.3

Graph 11 : KOH examination

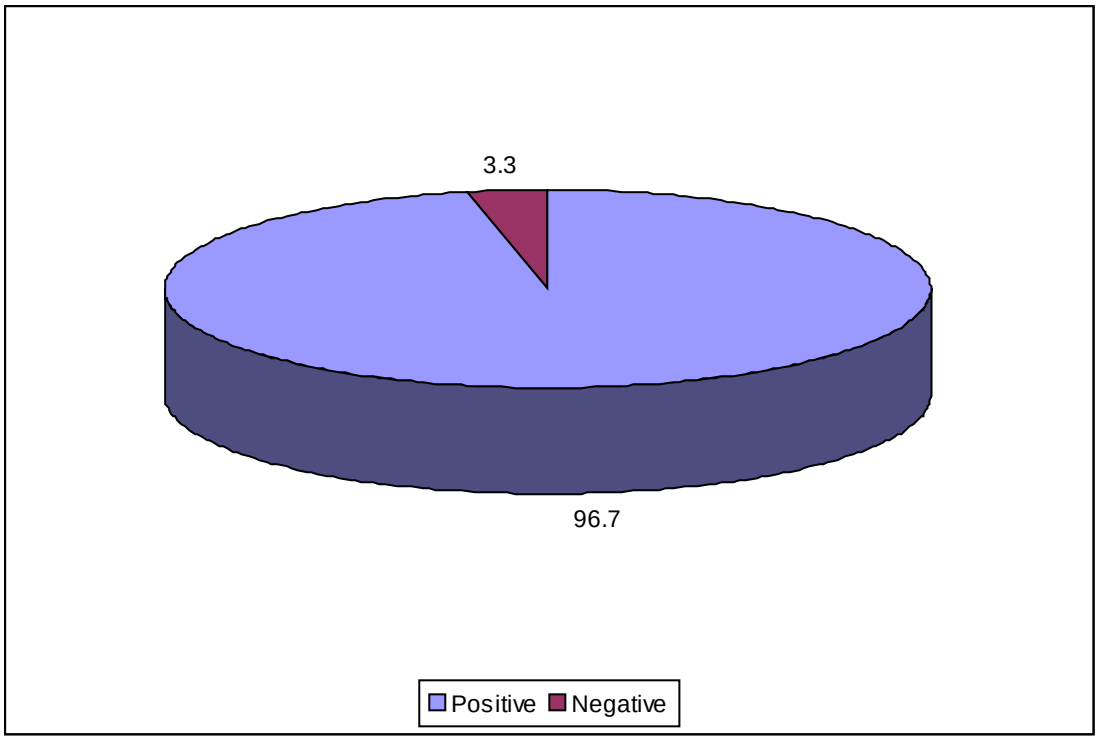


Table 19 : Wood's lamp

Wood's Lamp	No. of Patients	Percentage (%)
Positive	137	91.3
Negative	13	8.7

Graph 12 : Wood's lamp

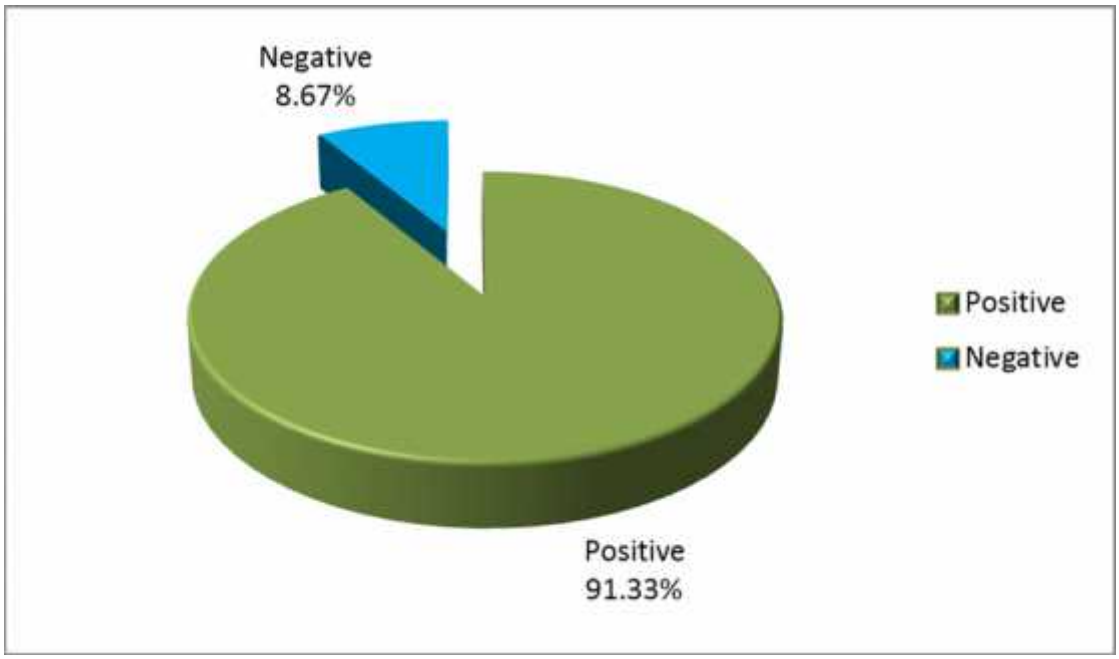


Table 20 : Culture

	No. of Patients	Percentage (%)
Positive for Malassezia species	82	54.67
Negative	60	40.00
Other yeast isolated	8	5.33

Graph 13 : Culture

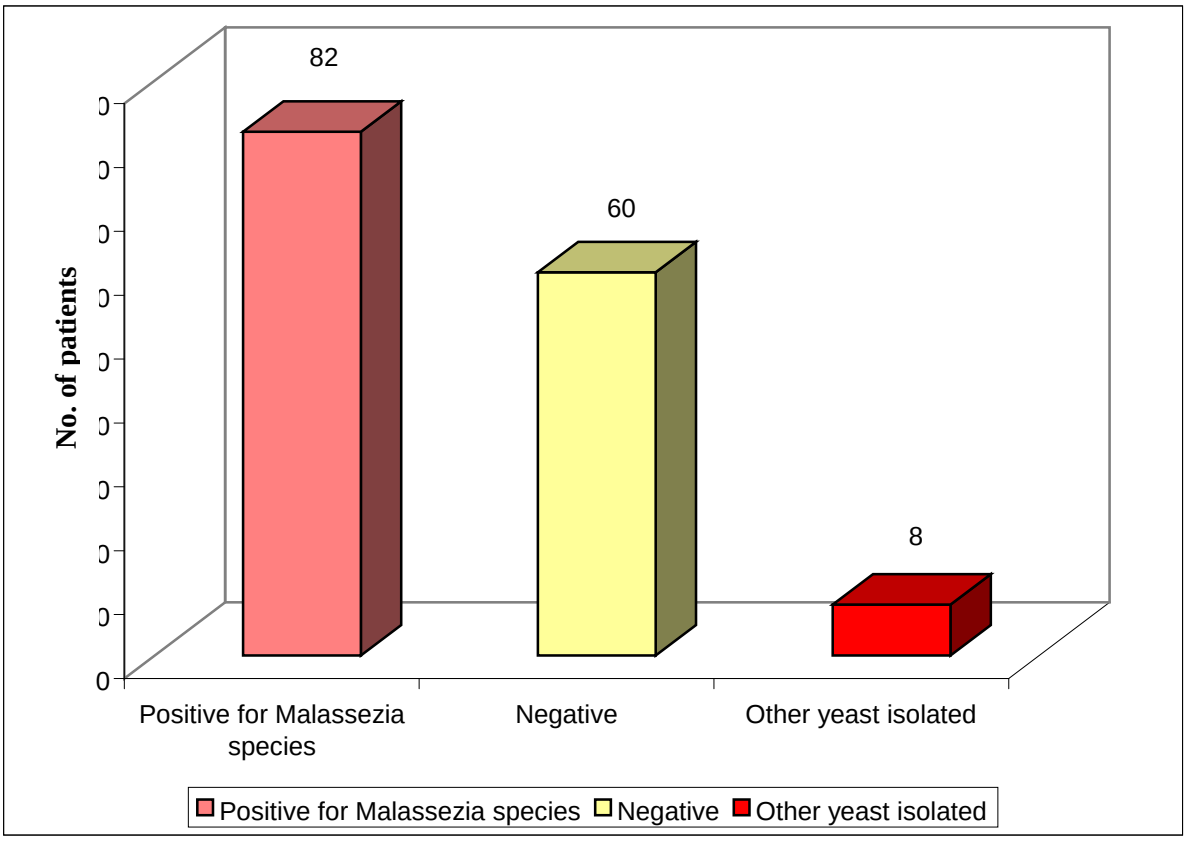


Table 21 : Species isolated

Species isolated	No. of Patients	Percentage (%)
M.sympodialis	36	43.9
M.furfur	21	25.61
M.obtusa	7	8.54
M.globosa	3	3.66
M.restricta	2	2.44
Malassezia species (untypable)	13	15.85

Graph 14 : Species isolated

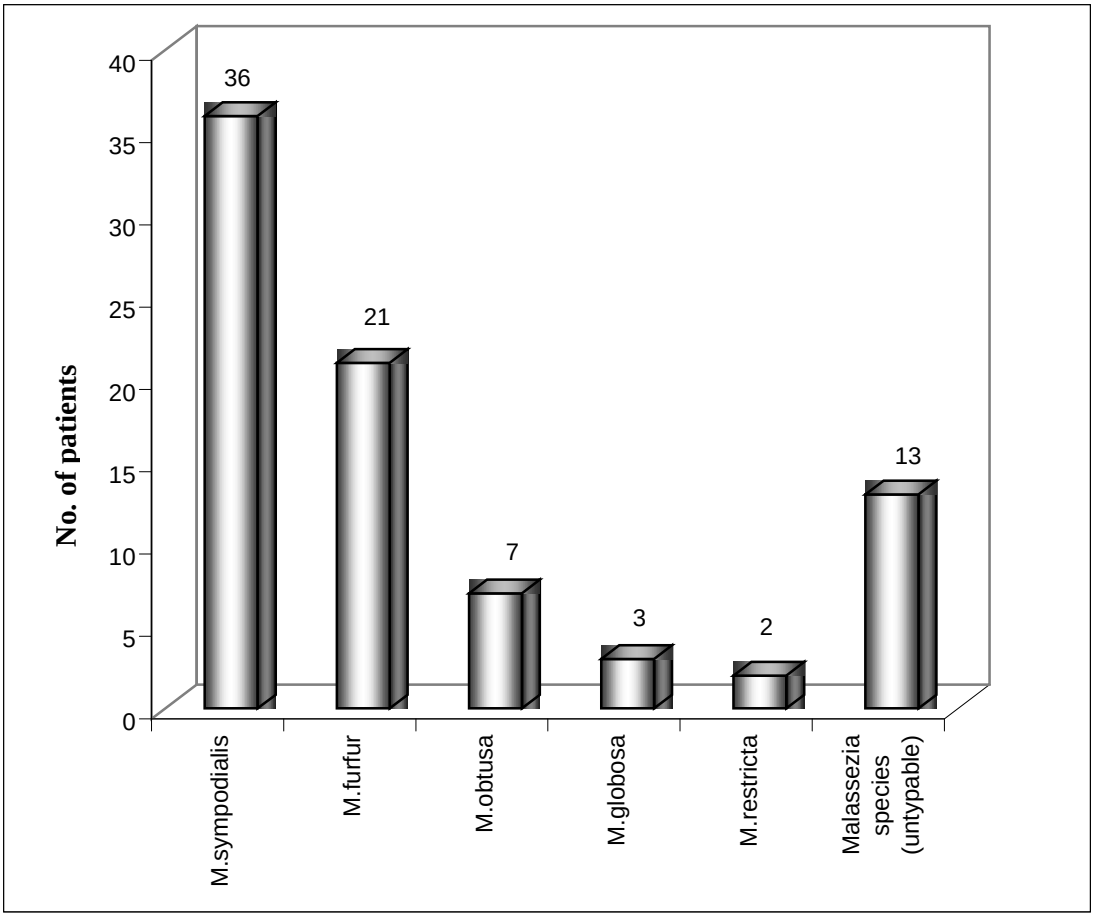


Table 22 : Species in specific lesion

Lesion	M.sympodialis (%)	M.furfur (%)	M.obtusa (%)	M.globosa (%)	M.restricta (%)
Hyperpigmentation	9 (13.04)	4 (5.80)	3 (4.35)	1 (1.45)	0
Hypopigmentation	23 (33.33)	13 (18.84)	4 (5.80)	3 (4.35)	2 (2.90)
Mixed	3 (4.35)	4 (5.80)	0	0	0

DISCUSSION

AGE INCIDENCE

In the present study most of the patients belonged to the age group of 21–30 years, 59 (39.33%) patients belonged to this group.

There was an equal distribution of 28 patients in the age group of 10-20 years (18.66%). 13 patients (8.66%) belonging to age group 41-50 years, 17 (11.33%) patients were above 50years of age. Only 5 (3.33%) patients were below the age of 10 years.

According to Tarazooie et al, the peak age of incidence of pityriasis versicolor was in the age group of 20-30 yrs.[86]

Study by Amma showed 50% of the patients from the age group of 11-20 years, followed by 35% of patients from the age group of 21-30 years.[28]

Cullen[85], Michalowski et al,[47] Terragni et al[22] have observed that pityriasis versicolor is uncommon before puberty, possibly because of the changes in the skin sebum levels that occur at this time, although some cases have been reported in children, particularly in tropical countries.[85]

Michalowski et al[47] have observed that the incidence of pityriasis versicolor decreases in later life, it is less common in older, which may be related to alteration in sebum production which tends to decrease with age.

Thus the age incidence of the present study is concordant with the observation made by above authors; giving an impression that pityriasis versicolor is a disease of young adults, suggesting that the peak of the infection is coincided with ages when the sebum production is in the highest level.[86]

SEX INCIDENCE

In the present study, there were 121 (80.6%) patients whereas there were only 29 (19.4%) female patients.

Rao et al have observed an incidence of 73.3% in males and 26.6% in females[68].

Similarly Rasi et al[107] have observed an incidence of pityriasis versicolor of 62% in males and 38% in females.

Amma,[28] Singh et al[27] have observed higher incidence in males as compared to females.

Sex ratio of M:F in the study conducted by Kanta et al was approximately 2:1.[26]

Hence, our result correlates well with the above mentioned studies. The role of sex in propensity to develop pityriasis versicolor is still unclear. Some studies found it to be more common in men than women, while other indicated that the incidence of this infection is higher in women, which may be due to the extra attention by women to beauty and skin hygiene.[86]

OCCUPATION:

In our study, 46 (30.66%) patients were Officers, 45 (30%) patients were manual labourers, 38 (25.33%) patients were students and 20 (13.33%) patients were housewives. There was only one (0.66%) athlete patient.

According to the study by Rao et al,[68] maximum number of patients were students, followed by manual labourers.

Similarly, a study in Kerala showed 36% of the patients were manual labourers. This group included those exposed to direct sunlight, direct heat and others like weavers, carpenters.[28]

In a study conducted by Ghosh et al[21] maximum number of patients were students, as pityriasis versicolor is more common in young adults, whereas in our study 1/4th of the patients belonged to this profession.

Majority of our patients were labourers belonging to low socioeconomic class. Data particularly in Indian population is sparse in these aspects, but probably there is no direct association of pityriasis versicolor with poverty or personal hygienic condition.

FAMILY HISTORY

Our study did not correlate with others in this regard as only 5.33% of the patients gave a positive family history.

Rao et al, found a positive family history in 38.30% of patients, out of whom 10% gave history in spouses and 13.30% in parents.[68]

Terragni et al found little higher incidence of positive family history of pityriasis versicolor among family members, which was 43.8%.[22]

Ghosh et al, found 28% of cases having positive family history.[21]

Study by Amma showed, 60% of the patients gave a history of personal contact with other patients with pityriasis versicolor.[28]

SYMPTOMS

In the present study, 60 (40%) of the patients had symptoms in form of mild itching, whereas 90 (60%) had no symptoms. The itching was found to be more common in summer during exposure to hot and humid environment, when the patient had excessive sweating.

Gupta et al[61] opined that some patients may experience mild pruritus although in most patients, pityriasis versicolor is asymptomatic and chiefly a cosmetic concern.

Our results correlate with Rao et al[68] according to which 70% of patients were asymptomatic and mild itching was present in 30% of patients.

Majority of the patients reported by Ghosh et al[21] were asymptomatic.

Majority of the patients had cosmetic concern problem, hence, the above studies correlates to our findings.

COLOUR OF THE LESION

In the present study, 71.3% of cases had hypopigmented lesion, 16.0% has hyperpigmented lesions, whereas 12.7% had mixed type of lesions.

Ajaykrishna[20] had described majority of cases having hypopigmented lesions (84%), 9% of the patients had hyperpigmented and 6% of patients had mixed type of lesions. This study was done in South India and concluded that South Indian patients have predominance of hypopigmented variety that correlates well with our study.

Similarly Gupta et al[61] have described that, a single patient may have both hypopigmented (lesions particularly noticeable in dark skinned patients) and hyperpigmented type of lesions and the color of the hyperpigmented lesions varied from pink or tan to dark brown or black.

Assaf and Weil[18] have observed that lesions typically begin as reddish macules, later becoming depigmented. The hyperpigmentation that occurs occasionally may be due to individual differences in the inflammatory response. Thus the patients in the present study had lesions which were more of hypopigmented variety than hyperpigmented.

Lipoperoxidation process produced by pityrosporum accounts for the clinical hypopigmentation.

Proposed theory for hyperpigmentation is the increased thickness of the keratin layer and more pronounced inflammatory cell infiltrate in these individuals acting as a stimulus for melanocytes.[68]

SITE OF LESION

In the present study, 64% of the patients had lesion over the neck, 46% over the back, 44% over the trunk and 13.33% over the face, while 29.33% of the patients had lesions over other parts of the body like upper and lower extremities. Majority of the patients had involvement of more than one site.

Similarly, study by Amma showed 92% of the patients having lesion over the neck, followed by chest and back.

Gupta et al[61] found in their study that the lesions were more often present on lipid rich areas of the body like the face, neck and the trunk.

Rao et al[68] in his study found the disease was seen more commonly on the neck (71.60%) followed by back (70%), chest (58.30%) and face (38.3%).

Localization of the lesion reflects the distribution of the sebaceous glands, thus distribution of the lesions in the present study is similar to the observations made by other authors.

TYPE OF LESION

In our study, out of 150 patients, macules and patches were seen in 38.67% and 80.67% of patients respectively, follicular variety was seen in 27.33% of patients, while other varieties were observed only in 1.33% of the patients.

According to Assaf and Weil[18] patchy lesions are more commonly encountered.

A study conducted by Rao et al[68] found macular lesions maximum in the study, which was 86.60% and follicular variety was 6.60% of the patients.

Similarly, in a study, macular type of the lesion was encountered in majority of the patients.[28]

In the present study in contrast to above mentioned studies, both patchy and macular lesions were seen in majority, but follicular variety was also seen slightly more.

SCALES

In the present study, all the patients had lesions with very fine scales. Wherever scaling was minimal, it was elicited by scraping the skin.

In a study, scales were present in 95% of the cases.[28]

Above mentioned studies are in agreement with our study. Usually the primary lesion of pityriasis versicolor is sharply demarcated macule, sometimes slightly erythematous, but characterized by fine, branny scaling.

HISTORY OF EXCESSIVE SWEATING

In the present study, 103(68.67%) of the patients gave the history of exposure to excess of hot and humid environment and 92 (61.33%) had history of excessive sweating.

Rao et al[68] had found in their study, 35% of the patients observed the disease first in summer and probably increased sweating made the person more susceptible for infection.

Klenk et al[75] had observed pityriasis versicolor to be more frequent in humid, warm or tropical climates than in dry cold zones.

Similarly, Tarazooie et al[86] found in their study that hyperhydrosis can be considered as the endogenous factor in mediating the development of the infection.

Study by Amma showed, very high sweating tendency in 78% of the patients in the study.[28] It was found that hot season is favourable for the onset and progress of the disease.

The result of the present study concurs well with the above mentioned studies.

RELAPSE

In the present study, 31(20.67%) patients gave the history of similar condition in past.

According to Ingordo et al[16] a significant association was documented between active pityriasis versicolor and a previous clinical history of pityriasis versicolor.

This could confirm the hypothesis that constitutional factors i.e. seborrhea and chemical constitution of sebum may play a crucial role in climatic conditions, leading to relapse of pityriasis versicolor.

Whereas, according to Rao et al[68] only 1.60% of the patients relapsed after taking treatment.

Similarly, study by Gurmohan Singh demonstrated history of similar complaint in past in only 5% of the patients.[27]

Recurrence rate of pityriasis versicolor is very high, even though the yeast is a part of the normal flora, sometimes it resides deep in the hair follicles. Unless predisposing factors are removed after the completion of the treatment, its recurrence cannot be prevented.[68]

The present study was not in contrast to the above mentioned studies, as percentage of recurrence was higher.

DIABETES AND IMMUNOSUPPRESSIVE DRUGS

In our study, out of 150 patients, 17 (11.33%) patients had predisposing factor like diabetes. 7(4.67%) patients were on immunosuppressive therapy and 16(10.67%) patients had other systemic illnesses.

Rao et al[68] had found 18.30% patients suffering from systemic diseases like malignancy, tuberculosis and diabetes and 13.30% had history of taking immunosuppressive drugs.

Similarly Ghosh et al[21] found that, among the associated systemic conditions, diabetes mellitus and use of systemic steroids or immunosuppressive drugs were the commonest.

It is believed that the defect in production of lymphokines in patients with pityriasis versicolor may be one of the main endogenic predisposing factor. Other factors are genetic factors, hyperhidrosis, systemic corticosteroid treatment, immunosuppressive therapy and malnutrition.[43]

It is observed in association with systemic disease like malignancy, tuberculosis or diabetes, as it is believed that the disease flare up when the immunity goes down.[68]

KOH EXAMINATION

In our present study 96.7% of the cases were KOH positive.

Study by Tarazooie[86] demonstrated positive results in 98% of the patients, this was the same result reported by Erchiga et al.[9]

Our results are consistent with those previously published and confirm the significance of the yeast mycelium conversion in the pathogenesis of the infection.

Regarding high sensitivity and acceptable specificity of direct examination, diagnosis of pityriasis versicolor is based on observation of short hyphae and yeast in the scales giving characteristic "spaghetti and meat balls" appearance.[61,86]

WOOD'S LAMP

In our present study 91.3% gave positive on Wood's lamp examination.

Rao et al, found positive fluorescence in 78.03% of their patients.[68]

Whereas a higher percentage was obtained in studies conducted by Kristanty Ria et al[104] which was 82.6% that to some extent correlates with our findings.

Fluorescence on Wood's lamp is not obtained if the patient has applied some ointment or taken a recent bath. In our study, negative result may be due this reason.

CULTURE

In the present study, culture was positive in 54.67% and yeast other than Malassezia were isolated in 5.33% of total cases.

Our study correlates to the study conducted by Rajashekhar et al where the culture positivity was 60% on SDA.[71]

Whereas higher rate was obtained by Kindo et al[52] showing 68.75% growth on MDA.

Also Tarazooie et al[86] demonstrated 79.8% of the specimen yielding Malassezia, on culture.

In the study conducted by Kanta et al, the isolation rate increased to 17%, after addition of olive oil to SDA.[26]

Though it has been mentioned that the cultures can easily be made from infected scales,[71] we found it difficult to get positive cultures in all the cases. The

culture negativity in majority of the cases indicates that more precautions are necessary: during the procedure of collection of the skin scraping material, while incubating the material onto the media and also during the period of growth.

It is believed that because of hydrophobic characteristic of Malassezia yeast, preparing a homogenous suspension is very difficult to separate them by culture. Moreover, some Malassezia species may lose their viability after several subcultures.[16]

SPECIES ISOLATED

M.sympodialis was the most common species isolated (43.9%), followed by M.furfur (25.61%), M.obtusa (8.54%), M.globosa (3.66%) and then M.restricta (2.44%). In 13 cases (15.85%), only Malassezia yeast was identified and species isolation was not obtained.

It has been shown by Erchiga et al[9] unlike other dermatoses, that center of pityriasis versicolor lesions yields more viable materials for culture. Hence we scraped the center of the lesions instead of the borders to increase recovery rate and avoid isolation of surrounding commensal species.

Results obtained by Gupta et al[105] from Canada; M.sympodialis (59.5%), M.globosa (25.2%), M.furfur (10.8%), while M.restricta and M.obtusa were isolated only from single individuals. Leeming & Notman agar used by them enhances recovery of Malassezia species; MDA provides features of colonies.[14]

Similarly, a South Indian study, in Tamil Nadu by Kindo et al demonstrated M.sympodialis as the most common species, with only 1 (2%) case of globosa associated with pityriasis versicolor. However other species namely M.furfur, M.sloofiae and M.obtusa were not isolated.[52]

This was contrary to the observations made by Chaudhary et al[53] in Central India, Asplroz et al,[19] Nakabayashi et al[82] and Tarazooie et al[86] who isolated M.globosa at a frequency of 57.5%, 58.2%, 55%, and 53.3% respectively.

Also study done in North India demonstrated M.globosa isolation in most of the patients, followed by M.furfur.[51]

M.furfur is also responsible for pityriasis versicolor, particularly under tropical climate.[106] The finding of M.furfur predominance in the tropics might be explained by the recovery of pityriacytryn, an indole alkaloid produced by M.furfur. Pityriacytryn has the ability to protect fungus against ultraviolet exposure, which renders M.furfur more resistant to sun exposure.[104]

In the present study M.furfur was seen in 25.61% cases, higher than other studies.

Some studies have found higher prevalence of M.furfur, isolated as the most common agent.[104]

M.restricta was isolated in 3.4% of cases, by Chaudhary et al[53] and in 2.08% by Kindo et al[52] similar to our finding.

M.obtusa was isolated in 6.9% of patients in a study conducted by Chaudhary et al[53] similar to some of the earlier reports, that correlates to our study.

However, studies on isolation of M.globosa do not correlate with our findings. Our result is 3.66%, whereas higher percentage has been found in other studies.

No isolates of M.pachydermatis were demonstrated that correlate with our study, indicating that this species may not be considered the causative organism for pityriasis versicolor.

The above findings show that the prevalence of Malassezia species varies from place to place. This may be due to the difference in frequencies of Malassezia

species isolated from different culture media and perhaps to ethnic and geographic factors.

Our study indicated that, M.sympodialis is more common in South India.

The identification of Malassezia yeast to species level is of importance to determine which species are implicated in certain skin disease and whether there is variation in the distribution of the yeast with clinical data, body site, origin of population etc. further, the results of the in vitro susceptibility studies have shown variation in susceptibility of Malassezia species to various antifungal agents. Strains of M. furfur, M. globosa and M. obtusa have been found to be more tolerant to terbinafine than the remaining species, while M. sympodialis was highly susceptible. These results suggest that correct identification of Malassezia species may be important for selection of appropriate antifungal therapy.[109]

SPECIES IN SPECIFIC LESION

In the present study number of species isolated for M. sympodialis, M. furfur, M. obtusa, M. globosa and M. restricta in hyperpigmented lesions were 9, 4, 3, 1, 0 respectively, whereas in hypopigmented lesions they were 23, 13, 4, 3, 2 respectively.

Chi-square method was used to analyze this result of different species in different lesion. $\chi^{2} = 0.211$

Degrees of freedom (DF) = 2

p= 0.900

Hence no significant difference was present in distribution of species in hypopigmented lesion and hyperpigmented lesion.

Similarly, results were obtained from Chaudhary et al there was no conclusion drawn about the relationship of species and the pigmentary changes produced.[53]

CONCLUSIONS

The following conclusions can be drawn after making the observation in the present study :

- The incidence of pityriasis versicolor in our hospital was 0.64%. It was more common in males, probably due to their occupation. The people in 3rd decade of life were more affected by it, thus making it a disease of young adults. Most of the patients were asymptomatic, and the complaints were chiefly of cosmetic concern. Positive family history was present in very few patients.

- Most of the patients had history of excessive sweating. The neck was the most common site involved, patch was the commonest skin lesion encountered. Approximately 1/5th of the patients had similar disease in the past.

- KOH examination was positive in almost all the cases, whereas culture was obtained only in half of the cases, with M.sympodialis being the most common species isolated.

- It can be inferred that the disease is more common in people with outdoor activity, having history of excessive sweating. M.sympodialis being the most common species in this part of India, though many modalities available for the culture of pityriasis versicolor, none of them have shown accurate results due to easy contamination of the culture.

SUMMARY

The present study is "A one–year cross–sectional descriptive study that included 150 patients who attended the Out Patient Department of Dermatology, Venereology and Leprosy at K.L.E'S Dr. Prabhakar Kore Hospital and M.R.C, Belgaum from November 2008 to October 2009.

OBJECTIVES OF STUDY WERE

i. To study the culture positivity rate in clinically diagnosed cases of pityriasis versicolor.

ii. To study the various clinical presentations in different skin colour people, risk factors and demographic characteristics of pityriasis versicolor.

1. In the study, 150 cases were clinically diagnosed to have pityriasis versicolor out of 23,424 patients who attended the O.P.D. The frequency of pityriasis versicolor comes out to be 0.64%.
2. In the present study most of the patients belonged to the age group of 21–30 years, i.e. 59 (39.33%) patients. Equal distribution was present in the age groups of 10-20 years (18.66%) and 31-40 years (18.66%), i.e. 28 patients in each. There were 13 patients (8.66%) belonging to age group 41-50 years. 17 (11.33%) patients were above 50 years of age. Only 5 (3.33%) patients were below the age of 10 years.
3. In the present study, males constituted 121 (80.6%) patients whereas there were only 29 (19.4%) female patients, giving the ratio of 4.17:1.
4. Most of our cases were manual labourers (30%) and Officers (30.66%) followed by students (25.33%).
5. 78% of the patients had Type IV skin type and 22% were of Type V skin type.

6. Most of the patients were asymptomatic (60%), 40% of the patients had mild pruritus as the chief complaint.
7. Very few patients gave history of similar complaints in family (5.33%).
8. Hypopigmented lesions (71.3%) were more common than hyperpigmented lesions (16%). Both type of pigmentations were observed in 19 patients.
9. 52.67% of the patients had more than one site of the body involvement. 47.33% of the patients had only one site of the body involved. 64% of the patients had lesion over the neck, 46% over the back, 44% over the trunk and 13.33% over the face. Other sites of the body like upper and lower extremities were involved in 44 patients.
10. 80.67% of the patients had lesion as patch, 38.67% of the patients had macule. Follicular variety was seen in 14% of the patients. Two patients had other variety of pityriasis versicolor lesion. 59 patients had more than one type of skin lesion.
11. History of excessive sweating was present in 61.33% of patients. Positive past history was present in few patients (20.67%). 11.33% of the patients were suffering from diabetes. There were seven patients on immunosuppressive therapy. Acne was the most common associated dermatoses.
12. 96.7% of the patients had positive finding on KOH examination.
13. Majority of patients showed positive result on Wood's lamp examination (91.33%).
14. More than half of the cultures gave positive growth for Malassezia (54.67%). M.sympodialis was the most common species isolated (43.9%). M.furfur was the second most common species isolated (25.61%). Seven cultures gave M.obtusa growth, whereas three had M.globosa growth. Only two cultures had growth for M.restricta. In 15.85% of culture, Malassezia was identified, but speciation was not obtained (unable to type). In eight cultures, yeasts other than Malassezia were isolated.

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ANNEXURE - I
PROFORMA

"A CROSS SECTIONAL STUDY OF PITYRIASIS VERSICOLOR WITH REFERENCE TO CULTURE POSITIVITY AT KLES DR. PRABHAKAR KORE HOSPITAL AND MRC, BELGAUM."

Case No. ☐☐ **OP/ IP No.** ☐☐☐☐☐☐☐

Name: First name ☐☐☐☐☐☐ **Middle name** ☐☐☐☐☐☐☐

Last name ☐☐☐☐☐☐

Age: ☐☐

Sex:

1. Male
2. Female

☐

Occupation:

1. Labourer
2. Housewife
3. Officer
4. Athlete
5. Student

☐

Address:

Presenting complaints and duration:

History of present illness:

Site of lesion:

Face

1. Present
2. Absent

☐

Neck

1. Present
2. Absent

☐

Back

1. Present
2. Absent

☐

Trunk

1. Present
2. Absent

☐

Other
1. Present
2. Absent

Onset:
1. Sudden
2. Gradual

Itching:
1. Present
2. Absent

History of excessive sun exposure
1. Present
2. Absent

Aggravating factors:
1. Present
2. Absent

History of excessive sweating:
1. Present
2. Absent

Any associated disease:
1. Present
2. Absent

Past History:

History of similar illness:
1. Present
2. Absent

History of Diabetes Mellitus:
1. Present
2. Absent

History of Hypertension:
1. Present
2. Absent

Family History:
1. Present
2. Absent

Other systemic illness
1. Present
2. Absent

Personal History:

Diet
1. Veg
2. Mixed

Appetite
1. Normal
2. Poor

Bowel/ Bladder
1. Normal
2. Altered

Sleep
1. Normal
2. Altered

Alcohol
1. Present
2. Absent

Smoking
1. Present
2. Absent

General Physical Examination:

Built
1. Poor
2. Moderate
3. Good

Vitals

Pulse / min

BP: Systolic

mmHg Diastolic

Temperature ^{0}F

Weight Kg

Pallor
1. Present
2. Absent

Icterus
1. Present
2. Absent

Cyanosis
1. Present
2. Absent

Clubbing
1. Present
2. Absent

Lymph nodes
1. Palpable
2. Non palpable

Edema
1. Pitting
2. Non Pitting
3. Absent

Mucocutaneous Examination:

Type of skin colour

Type of lesion:

Macules
1. Present
2. Absent

Patches
1. Present
2. Absent

Scales
1. Present
2. Absent

Site:
1. Face
2. Neck
3. Back
4. Trunk
5. Others

Type of pigmentation ☐

1. Hyperpigmentation
2. Hypopigmentation
3. Mixed
4. Others

Mucosal Examination:

Genital lesion:

1. Present
2. Absent ☐

Oral lesion:

1. Present
2. Absent ☐

Systemic Examination:

Cardiovascular system: Heart sounds

1. Normal
2. Abnormal ☐

Respiratory system: Breath sounds

1. Normal
2. Abnormal ☐

Per abdomen:

Spleenomegaly

1. Present
2. Absent ☐

Hepatomegaly

1. Present
2. Absent ☐

Any other

1. Present
2. Absent ☐

Central nervous system: Neurological examination

1. Normal
2. Abnormal ☐

Investigations: -

- Wood's Lamp examination: Green fluorescence ☐

1. Present
2. Absent

-Hb:

-TLC:

-DLC:

-RBS:

-Urine Routine:

- KOH:

- Fungal culture:
    1. Positive
    2. Negative

- Species isolated

Diagnosis:-

Signature:

Guide's Signature:

INFORMED CONSENT FORM

ID. NO. ☐ ☐ ☐

A CROSS SECTIONAL STUDY OF PITYRIASIS VERSICOLOR WITH REFERENCE TO CULTURE POSITIVITY

The study is conducted by Postgraduate student in M.D Dermatology, J N Medical College, Belgaum.

Respected Sir/Madam, we request you to enrol yourself to participate in our study as, you are eligible for participating in this study. During the study you will be asked some questions in detail regarding your presenting complaints.

Purpose of the study

The purpose of this study is to study the various clinical presentations of Pityriasis Versicolor (chibbu), common sites of involvement, risk factors and demographic characteristics. You are being asked to participate in this research because you have been clinically diagnosed as suffering from the disease, Pityriasis Versicolor. All patients attending the outpatient department, who are diagnosed to have this disease, will be requested to participate in this study during the period of one year.

Procedure and Treatment

Should you choose to participate, you will be asked to give a detailed history of your disease, undergo a physical examination, and consent to a few routine blood and urine investigations. You will undergo a KOH examination and wood's lamp examination. In addition to this, you will agree to undertake an RBS test.

Risks and Benefits

You may undergo slight amount of discomfort during the process of investigations, which may include a slight amount of pain and bleeding. However all necessary steps and precautions will be taken to ensure your safety. The result of you taking part in this research would help health care providers towards a better understanding of this disease, and thus we will be able to provide improved patient care

Alternatives

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

Privacy and confidentiality

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

Relations with the Institutional policy

The J N Medical College will provide, within the limitations of the laws of the State of Karnataka, facilities and medical attention to patients who suffer injuries as a result of participating in this projects. In the event of an emergency, you should contact KLES Dr. Prabhakar Kore Hospital and MRC.

Financial Incentives

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

Authorization to publish results

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

Voluntary participation

Your participation in this study is voluntary. Your decision whether or not to participate will neither affect the care of your current disease, nor your future relations with the doctor or the hospital. You are free to discontinue participation in this study at any time and for any reason. In case you need further information regarding your rights as a study participant, you may please contact J N Medical College, Belgaum.

STATEMENT OF CONSENT

I.D.NO: | | | |

I Mr/Ms/Mrs

_____

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

Participant's name:

Signature or left thumb print of participant:

Witness name:

Signature of witness:

Signature of Doctor:

Date:

If the participants are Minors (under 18), the parents sign the form, rather than the participants.

MASTER CHART

Case No.	OP/IP No.	Name	Age	Sex	Occupation	Complaints	Site of Lesion	Type of pigmentation	Past History	Skin Color	Type of Lesion	Scale	Wood's Lamp Examination	KOH Examination	Culture	Species isolated	Personal history	Associated condition	Address
1	993893	MVP	33	M	c	a	Others	a	-	IV	b	+	P	P	N	O	-		Belgaum
2	993327	RSH	24	M	c	a,b	Nck	b	a	IV	b	+	N	P	N	N	a,b		Belgaum
3	1022819	AAS	18	M	e	a,b	Nck, Bck, Tnk	a	-	IV	b	+	P	P	P	M.furfur	a,b		Bagalkot
4	998227	MBP	36	F	a	a	Nck, Bck, Tnk	b	-	IV	b	+	P	P	N	N	a,b		Belgaum
5	998472	PVP	35	F	a	a,b	Nck, Bck, Tnk, Others	b	a	IV	a	+	P	P	N	N	a,b		Belgaum
6	909274	VVS	40	F	b	a	Others	b	-	IV	b	+	P	P	P	N	a,b		Goa
7	999531	MKG	40	M	a	a,b	Nck, Bck, Tnk	a	a	V	a,b,c	+	N	P	N	N	a,b		Belgaum
8	999658	BCH	44	M	c	a,b	Nck, Bck, Tnk, Others	b	a,b,d	IV	b	+	P	P	P	Malasezzia	a,b	Daibetes	Saundatti
9	298696	SP	20	M	a	a	Fce, Nck, Tnk	b	-	V	a,b	+	P	P	N	N	a,b		Belgaum
10	1001090	RVM	22	M	c	a,b	Nck	b		IV	b	+	P	P	N	N	a,b		Raibag
11	998318	SVF	30	M	c	a,b	Nck, Bck, Tnk	c	-	IV	b	+	P	P	N	N	a,b		Belgaum
12	1004366	PMP	28	M	c	a	Nck, Bck, Tnk, Others	c	a	IV	a,b	+	P	P	N	N	a,b		Belgaum
13	298824	M	22	F	b	a,b	Fce, Nck, Bck, Tnk, Others	b	-	IV	b	+	P	P	O	O	a,b	AVI	Hindalga
14	1008776	MAI	31	M	a	a,b	Nck, Bck, Tnk, Others	n	-	V	b	+	P	P	N	N	a,b		Belgaum
15	1003768	KSK	30	F	b	a	Others	n	-	IV	b	+	P	P	N	N	-		Belgaum
16	1013111	SSY	42	M	c	a,b	Bck, Tnk	n	b,c,d	IV	b	+	P	P	N	N	a,b	Airochordos with mollusum contagious diabetes	Belgaum
17	1016330	SHS	30	M	a	a	Nck, Bck, Tnk, Others	c	b,c,d	V	b	+	P	P	N	N	a,b		Belgaum
18	1030222	G	27	F	b	a	Nck, Bck, Tnk	b	a	IV	b	+	P	P	N	N	-		Belgaum
19	953871	S	22	M	b	a	Fce, Nck, Bck, Tnk	c	-	IV	b	+	P	P	N	N	-	5 month Amenorrhea	Khanapur
20	1020431	SVY	34	M	a	a,b	Nck, Bck, Tnk	b	b,c,d	IV	b	+	P	P	N	N	a,b		Belgaum
21	1022532	RUN	23	M	c	a,b	Nck, Bck, Tnk	a	a	IV	a,b	+	P	P	P	M.obtusa	a,b		Goa

MASTER CHART

Case No.	OP/IP No.	Name	Age	Sex	Occupation	Complaints	Site of Lesion	Type of pigmentation	Past History	Skin Color	Type of Lesion	Scale	Wood's Lamp Examination	KOH Examination	Culture	Species isolated	Personal history	Associated condition	Address
22	1023977	BSY	15	M	e	a,b	Nck, Bck, Tnk, Others	b	a	IV	a,b	+	P	P	N	N	a,b		Telni
23	1026878	SIV	22	M	e	a	Bck, Tnk, Others	b	-	IV	a,b	+	P	P	P	M.obtusa	a,b		Belgaum
24	748053	DHM	20	M		a,b	Nck	c	-	IV	a,b	+	P	P	N	N	a,b	Rt CSOM	Belgaum
25	1005625	SGA	51	M	c	a	Nck, Bck, Tnk, Others	b	e,f	IV	a,b	+	P	P	N	N	-	Immunosuppresso therapy	Hubli
26	307740	B	43	M	a	a,b	Nck	b	c,d	V	a,b,c	+	P	P	P	M.restrica	a,b	Diabetes	Belgaum
27	1068949	BUN	37	F	b	a	Nck, Bck, Tnk, Others	b	a	IV	a	+	P	P	P	N	a,b	Melasma	Belgaum
28	1057047	IMB	10	M	e	a,b	Nck	a	-	IV	b	+	N	P	N	N	a		Belgaum
29	1048427	VVR	10	M	e	a,b	Fce, Nck, Tnk	b	e,f	IV	b	+	P	P	N	N	a,b	Chronic ITP	Belgaum
30	1055477	GSR	26	M	e	a,b	Tnk	b	d	IV	a	+	P	P	N	N	a,b	AVI DM	Sindudurg a
31	1062971	SYP	57	M	c	a,b	Nck, Tnk	b	d	IV	a	+	P	P	N	N	b	Pruritus diabetes investigation/	Goa
32	1005025	SMH	22	M	a	a	Tnk	b	-	IV	a,b	+	P	P	P	N	a,b		Bijapur
33	1062094	MAP	17	M	e	a	Tnk	c	-	IV	b	+	P	P	P	M.Sympodialis	a,b		Belgaum
34	1071020	SBI	27	M	e	a	Nck, Bck, Tnk	b	-	IV	b	+	P	P	P	M.Sympodialis	a,b		Belgaum
35	312664	CVK	20	M	b	a	Tnk	a	-	IV	a,b	+	P	P	P	M.Sympodialis	a	AVI	Belgaum
36	932652	PK	14	M	e	a	Tnk, Others	a	-	IV	a	+	P	P	P	M.Sympodialis	a,b		Belgaum
37	1077824	RSS	25	F	c	a,b	Nck	a	d	V	b	+	P	P	P	M.Sympodialis	a,b	Diabetes	Belgaum
38	1078977	BSC	26	M	c	a,b	Nck	a	-	IV	b	+	P	P	P	M.Sympodialis	a,b	Warts	Belgaum
39	1075715	SSK	18	M	c	a	Fce, Nck, Bck, Tnk	b	-	IV	b	+	P	P	P	M.Sympodialis	a,b	High grade fever AVI	Belgaum
40	842541	RJS	21	M	c	a	Nck	a	-	IV	b	+	P	P	P	M.obtusa	b		Samo
41	730975	BNC	28	M	b	a,b	Nck, Bck, Tnk	b	a	IV	a,b	+	P	P	P	M.Sympodialis	a,b	Relapse	Goa
42	315838	MM	32	M	b	a,b	Nck, Bck, Tnk	b	e,f	IV	a,b,c	+	P	P	P	M.Sympodialis	-	Breast Ca	Hatargi
43	1085359	KBP	33	M	c	a	Nck	b	-	IV	b	+	P	P	P	M.Sympodialis	a,b		Goa
44	902325	SSM	19	F	e	a	Fce, Nck, Bck, Tnk	b	-	IV	b	+	P	P	P	M.obtusa	a,b		Belgaum
45	106894	BUG	37	F	b	a	Nck, Bck, Tnk	a	-	V	a,b	+	P	P	O	N	a,b		Bagalkot
46	1090346	MBP	20	M	c	a	Nck, Bck	b	a	IV	b	+	P	P	P	M.restrica	a	Relapse	Kodogy
47	1096820	CMC	35	F	a	a,b	Bck	b	-	IV	c	+	P	P	P	M.Sympodialis	a,b		Belgaum

MASTER CHART

Case No.	OP/IP No.	Name	Age	Sex	Occupation	Complaints	Site of Lesion	Type of pigmentation	Past History	Skin Color	Type of Lesion	Scale	Wood's Lamp Examination	KOH Examination	Culture	Species isolated	Personal history	Associated condition	Address
48	1074590	NHM	5	F	e	a,b	Nck	b	-	IV	b	+	P	P	N	N	a,b		Raichur
49	1099455	LHM	22	M	e	a	Nck	b	a	IV	b	+	P	P	N	N	a,b	Relapse	Belgaum
50	585915	GKS	35	M	c	a	Bck, Tnk	b	c,d	IV	b	+	P	P	P	N	a,b	DM/HTN/	Dharwad
51	671021	JR	53	M	a	a	Others	c	-	V	b	+	P	P	N	N	a,b	Schambarg's dis	Goa
52	1101432	VDN	38	M	a	a,b	Others	a	-	IV	b	+	P	P	N	N	a,b		Bagalkot
53	1101388	AMW	38	M	a	a,b	Nck, Bck	b	-	IV	b	+	P	P	P	M.Sympodialis	a,b		Belgaum
54	1103464	AAR	20	F	e	a	Nck	b	e,f	IV	a	+	P	P	P	N	a,b	Vitiligo	Sawantwadi
55	1106758	SSP	21	M	e	a,b	Nck	b	-	IV	a,c	+	P	P	P	M.globosa	-	AVI	Belgaum
56	1108941	SS	14	M	d	a	Nck	b	-	IV	a,b	+	P	P	N	N	a		Belgaum
57	1107184	SHB	26	M	c	a	Bck	b	a	V	b	+	P	P	P	M.Sympodialis	a,b	Deviated Nasal Septum	Bagalkot
58	945387	RA	36	F	a	a	Bck, Tnk	b	a	IV	b	+	P	P	O	Rodotolla	a,b	Relaspse	Goa
59	1710311	RBH	25	M	c	a	Nck	a	-	IV	b	+	N	P	P	N	a,b		Belgaum
60	1139094	JR	20	M	a	a	Nck, Bck, Tnk	c	-	IV	b	+	P	P	P	M.furfur	a,b	Fissure feet	Belgaum
61	580629	SVB	56	M	e	a,b	Others	b	a,c,d,e	IV	b,c	+	P	P	P	N	-	Relaspse Rt sided paralysis (HTN, DM)/	Bagalkot
62	1113686	SS	21	M	e	a	Nck	c	-	IV	a,b,c	+	P	P	P	M.furfur	a,b		Belgaum
63	321821	C	50	F	a	a,b	Nck	a	c,d	IV	c	+	P	P	P	M.Sympodialis	a,b	Keloid, Folliculitis HTN, DM	Kanabaragi
64	1113722	SYM	23	M	c	a	Others	b	-	V	a	+	P	P	P	M.obtusa	a	AVI	Belgaum
65	1114270	VH	53	M	c	a	Nck, Bck	b	d	IV	b	+	P	P	O	Candida Species isoalted	-	DM	Goa
66	1114889	PK	35	F	b	a	Others	b	-	IV	b	+	N	P	N	N	a		Goa
67	321719	C	65	M	e	a	Nck, Bck, Tnk	b	c,e	IV	b	+	P	P	P	M.furfur	a,b	HTN, MI	Goa
68	1117317	SBI	42	M	a	a	Nck, Bck, Tnk	b	-	IV	a,b	+	P	P	P	M.Sympodialis	a	Contact dermatites	Belgaum
69	1119337	SGP	38	M	a	a,b	Bck	b	-	IV	b	+	P	P	P	M.Sympodialis	a,b	Relapse T.Cruris	Goa
70	1118911	SAC	32	M	a	a	Nck, Bck	b	a,b	IV	b	+	P	P	P	N	a,b	Family H/o	Belgaum
71	100424	MMC	2	M	e	a,b	Nck	b	b,e,f	IV	a,b	+	P	P	P	M.furfur	a,b	Hypotonus Ataxia, on steroid therapy	Goa

MASTER CHART

Case No.	OP/IP No.	Name	Age	Sex	Occupation	Complaints	Site of Lesion	Type of pigmentation	Past History	Skin Color	Type of Lesion	Scale	Wood's Lamp Examination	KOH Examination	Culture	Species isolated	Personal history	Associated condition	Address
72	1119417	GPW	15	M	e	a	Nck	b	d	IV	b	+	P	P	P	M.Sympodialis	a	DM Post-inflammatory hyperpig	Belgaum
73	1119447	SY	28	M	a	a	Fce, Nck	b	e	V	a	+	P	P	P	M.furfur	a,b	Focal vitiligo	Belgaum
74	872343	BKC	60	M	a	a	Others	c	-	IV	b	+	P	P	P	M.Sympodialis	b	Fissure feet	Belgaum
75	1121310	RK	60	M	e	a	Others	b	-	IV	b,c	+	P	P	P	M.Sympodialis	-		Gokak
76	1659173	JAK	21	F	e	a	Nck	b	-	IV	a,b,c	+	P	P	P	M.Sympodialis	-	Pacba	Belgaum
77	899006	VSH	25	M	c	a	Nck	b	e,f	IV	b	+	P	P	P	M.furfur	b	Vitiligo Andmgenic alopecia	Belgaum
78	1127648	VPG	21	M	c	a	Nck, Bck	b	-	IV	b	+	P	P	P	M.Sympodialis	a,b		Belgaum
79	1122748	MSH	23	M	c	a	Nck, Tnk	b	-	IV	c	+	P	P	P	M.furfur	-		Belgaum
80	970283	CMC	24	M	c	a	Nck, Tnk	b	a	IV	b	+	P	P	O	Rodotolla	a	Relapse	Belgaum
81	1081216	VSS	34	M	c	a,b	Others	a	-	IV	a,b	+	P	P	P	M.Sympodialis	a,b		Belgaum
82	314115	AP	23	M	e	a,b	Nck	b	-	V	a,b	+	P	P	P	M.Sympodialis	b		Goa
83	989587	RSK	23	M	c	a,b	Bck, Tnk	b	-	IV	a,b	+	P	N	P	M.Sympodialis	b		Belgaum
84	1123181	SSM	22	F	c	a,b	Nck, Tnk	b	-	IV	b	+	P	P	N	-	b		Belgaum
85	1125111	SND	35	M	a	a,b	Fce, Nck, Bck, Tnk, Others	b	-	IV	b,d	+	P	P	N	N	a,b	Healed Herpes Simplex	Belgaum
86	617580	MDT	32	M	a	a,b	Bck	b	a	V	a,b	+	N	P	N	N	a,b	Relapse	Belgaum
87	323775	HBC	48	M	a	a	Fce, Bck, Others	a	-	V	b	+	P	P	P	Sympodialis	a	silicosis	Bagalkot
88	1128206	MAY	21	M	b	a	Nck, Bck, Tnk, Others	b	-	IV	b,c	+	P	P	P	Sympodialis	-	Primary	Goa
89	1129200	DST	12	F	e	a,b	Nck, Bck	c	c	IV	c	+	N	P	N	-	-	AVI	Belgaum
90	827789	PNN	18	M	e	a	Tnk	a	f	IV	b	+	P	P	P	Sympodialis	-	AVI with Ch, URTI	Belgaum
91	1135915	TTN	37	M	a	a,b	Others	c	-	V	b	+	P	P	P	M.furfur	a,b	Melasma	Goa
92	1129139	MSK	74	M	e	a	Fce, Nck	b	-	IV	a	+	P	N	P	M.furfur	-		Belgaum
93	986673	MMN	12	M	e	a	Fce, Nck, Bck,	b	d	V	a	+	N	P	N	N	-	Neuas Anaenisus DM	Belgaum
94	1136549	RG	29	M	e	a,b	Nck	b	-	V	b	+	P	P	N	N	a,b		Belgaum
95	1137980	MBB	31	F	a	a	Nck, Tnk, others	c	a	IV	b	+	P	P	N	N	a	Relapse	Kolhapur
96	1118795	NSN	20	M	e	a	Nck, Bck	a	-	IV	b	+	P	N	P	M.furfur	b		Belgaum

MASTER CHART

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97	578172	RHP	45	F	e	a	Nck others	b	-	IV	c	+	N	P	N	N	a,b	pure- neuritic Leprosy	Belgaum
98	328086	NJC	45	M	a	a	Nck, Bck, Tnk	b	e	IV	b	+	P	P	N	N	a,b	Bipolar disorder	Belgaum
99	1142583	GRB	75	M	a	a,b	Nck, Tnk	b	d	IV	a,c	+	P	P	P	N	a,b	Diabetes	Kolhapur
100	1143324	AYK	29	M	c	a	Bck	a	-	IV	b	+	P	P	P	N	-		Belgaum
101	1143387	SMS	16	M	c	a,b	Nck, Bck	c	-	IV	b	+	P	P	P	M.furfur	-	T.corporis	Belgaum
102	1139094	TKT	20	M	c	a	Nck	b	-	V	a,b	+	P	P	P	M.furfur	a	Polyarthritis	Kolhapur
103	1136604	MRJ	60	M	a	a	Nck	b	-	IV	b,c	+	P	P	N	N	a	Left eye meningions	Belgaum
104	845639	JCK	4	M	e	a	Fce, Bck, Tnk, Others	b	-	IV	a	+	P	P	P	M.furfur	-		Belgaum
105	1138385	DBG	25	M	b	a	Tnk	b	-	IV	b	+	P	P	P	M.Sympodialis	a,b	Acne	Goa
106	1141850	RNK	27	F	e	a	Others	b	-	IV	b	+	P	P	P	M.Sympodialis	a	Acne	Kolhapur
107	1141930	RSN	27	M	a	a	Fce, Nck, Tnk, Others	b	a	V	a,b	+	P	P	N	-	a,b	Relapse Acne	Goa
108	1145691	YSM	25	M	a	a	Nck	b	-	IV	b	+	P	P	N	-	a,b	Acne	Uchagaon
109	1145780	BAP	65	M	a	a,b	Bck, Others	c	-	IV	a,b	+	P	P	N	N	b		Belgaum
110	1146690	RBN	28	M	a	a	Nck	b	-	IV	b	+	P	N	N	N	a,b	Molluscum contagien	Gokak
111	634555	MB	38	M	c	a	Nck, Bck	b	-	V	b	+	P	P	N	N	b		Belgaum
112	1147222	MSC	43	M	c	a	Others	b	-	V	a,b	+	P	P	P	-	a,b		Bagalkot
113	330714	SMS	24	F	b	a	Nck, Bck	b	e	IV	b	+	P	P	N	M.furfur	b	Seve Deprassion	Belgaum
114	1137447	AKP	45	F	a	a	Fce	b	-	V	b	+	P	P	P	M.globosa	a,b		Sangali
115	1160500	MBM	29	M	a	a,b	Fce, Nck	c	-	V	a,b	+	P	P	O	O	a,b		Belgaum
116	1160933	GAS	69	M	c	a	Fce, Nck, Bck , Tnk	b	d	IV	b	+	P	P	P	M.Sympodialis	a	Diabetes renal calculus	Goa
117	1162741	DBK	42	M	b	a,b	Bck	c	d	IV	b	+	P	P	N	N	a,b	DM	Bagalkot
118	1022819	AAV	18	M	b	a	Nck	b	a	V	a,b	+	P	P	P	M.furfur	b	Relapse case	Belgaum
119	1166582	RRP	21	M	c	a	Others	b	a	V	b	+	P	P	P	M.Sympodialis	a	Relapse	Belgaum
120	1167869	ANP	27	M	c	a,b	Others	a	-	IV	a	+	P	P	P	M.furfur	a,b		Belgaum
121	597007	KPD	60	M	b	a,b	Nck, Bck	a	a	IV	a	+	P	P	N	0	-	Relapse	Goa
122	1168843	SBN	20	M	a	a,b	Nck	b	b	IV	a,b	+	P	P	N		a,b	Family H/o	Belgaum
123	1168751	NSK	52	M	a	a	Nck, Bck, Tnk	b	-	V	b	+	P	P	N	N	a	Post- herpetic neuralgia	Shimoga

MASTER CHART

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124	805974	PNN	20	M	c	a,b	Others	b	-	IV	c	+	P	P	P	M.furfur	-		Belgaum
125	1164472	KVB	60	M	a	a	Fce, Bck	b	d	IV	b	+	P	P	N	N	a	DM, Leukoplakia with oral candidiasis	Belgaum
126	1169864	SDC	22	M	c	a	Nck	a	-	IV	b	+	P	P	P	M.obtusa	a,b	Acne	Belgaum
127		BNN	45	M	b	a,b	Others	b	-	V		+	N	P	P	M.globosa	-		Belgaum
128	1060630	PBK	19	F	e	a	Bck, Others	b	b	IV	b	+	P	P	N	N	a	Parapsoriasis	Belgaum
129	333413	NGG	37	M	a	a	Bck, Tnk	b	c,e	IV	b	+	P	P	P	M.Sympodialis	-	RHD, HTN	Kanabargi
130	1161112	GSP	23	M	e	a	Nck, Bck, others	b	e	IV	a,c	+	P	P	O	O	-	Lichen planus	Belgaum
131	1173007	ASG	26	M	c	a	Tnk	b	-	IV	a	+	P	P	N	N	-	Modouosis	Belgaum
132	339351	LVP	48	M	a	a	Bck	b	a,e,c	IV	a,b	+	N	P	N	N	-	IHD, HTN	Goa
133	1176660	UBH	25	M	c	a	Bck, Tnk	a	b	IV	b	+	P	P	P	M.Sympodialis	-		Goa
134	1177895	SGU	23	M	c	a,b	Fce, Nck, Bck, Tnk, Others	b	a	IV	d	+	P	P	N	N	a		Gokak
135	1178818	AAA	42	M	c	a	Fce	b	c	IV	a,b	+	N	P	N	N	a	HTN	Belgaum
136	335219	ISS	33	M	a	a	Nck, Tnk	a	e	V	b	+	P	P	O	O	a	RVD, TB	Belgaum
137	335918	KVK	61	M	a	a	Bck, Tnk	b	d,e	V	b	+	P	P	N	N	-	DM/SC	Belgaum
138	335522	S	5 Months	M	e	a	Nck, Bck	b	e	IV	a	+	N	N	N	N	-	Acynotic heart disease	Belgaum
139	118405	CPG	25	F	c	a,b	Fce	b	a,e,f	IV	a	+	P	P	N	N	a	Rheumatoid steroid therapy	Goa
140	669511	AMT	22	M	e	a	Others	b	a	IV	a	+	P	P	N	N	-		Belgaum
141	1042729	WS	23	M	e	a,b	Others	b	-	IV	b	+	P	P	P	M.globosa	b		Belgaum
142	814111	SPK	21	M	c	a,b	Bck, Others	b	a	IV	a,b	+	P	P	P	M.obtusa	a,b		Belgaum
143	760191	KLB	21	F	a	a,b	Fce, Bck, Tnk	b	-	V	a,c	+	P	P	N	N	a,b	Multinodular goiter	Belgaum
144	1189639	ANP	23	M	c	a,b	Bck, Tnk, Others	b	a	IV	a,b	+	P	P	P	M.furfur	a,b		Belgaum
145	911161	VRH	16	F	e	a,b	Nck, Bck, Tnk, Others	c	b	IV	a,b	+	P	P	P	M.Sympodialis	a,b		Belgaum
146	1107113	YVG	24	M	a	a	Nck, Tnk	a	a,b	V	b	+	P	P	P	M.furfur	-		Belgaum
147	1193710	AI	30	M	c	a	Tnk	b	-	IV	a	+	P	P	N	N	-		Belgaum
148	1198364	SGD	9	F	e	a	Nck	b	-	IV	a,b	+	P	P	P	M.furfur	a,b		Goa
149	119786	DBP	25	M	b	a	Nck	b	-	V	a	+	P	P	P	M.Sympodialis	a		Belgaum
150	361018	KAD	35	M	b	a	Nck	b	a	V	b	+	P	P	N	N	-		Belgaum

KEY TO MASTER CHART

Column code	Column name		Description
1.	Serial Number		1,2,3…..
2.	OP/IO Number		Outpatient/ Inpatient number
3.	Name of the patient		Initials
4.	Age of the patient		In years
5.	Sex of the patient	M	Male
		F	Female
6.	Occupation	a	Labourer
		b	Housewife
		c	Officer
		d	Athlete
		e	Student
7.	Complaints	a	Skin lesion
		b	Itching
8.	Site of lesion	Nec	Neck
		Bck	Back
		Tnk	Trunk
		Others	Other site
9.	Type of pigmentation	a	Hyperpigmented
		b	Hypopigmented
		c	Mixed

Column code	Column name		Description
10.	Past history	a	Similar illness in past
		b	Family history
		c	Hypertension
		d	Diabetes
		e	Other illness
		f	On steroid therapy
11.	Skin colour	I	Skin type I
		II	Skin type II
		III	Skin type III
		IV	Skin type IV
		V	Skin type V
		VI	Skin type VI
12.	Type of lesion	a	Macule
		b	Patch
		c	Follicular
		d	Other
13.	Scales	+	Present
		-	Absent
14.	Wood's lamp examination	P	Positive
		N	Negative
15.	KOH examination	P	Positive
		N	Negative

Column code	Column name	Description	
16.	Culture	P	Positive
		N	Negative
17.	Species	O	Other species
		M	Malassezia
		N	No growth
18.	Personal history	a	H/o excessive sun exposure
		b	H/o excessive sweating
19.	Associated condition	AV	Acne vulgaris
		ITP	Idiopathic thrombocytic purpura
		DM	Diabetes mellitus
		HTN	Hypertension
		URTI	Upper respiratory tract infection

Table 3: Biological features and incidence of a typical phenotype of nine species of Malassezia

Species identified	No. of strains	Colony characteristic on CHROM (% of incidence)[a]			Growth characteristic (% of incidence)[b]				Catalase reaction
		Size	Colour/morphology	Precipitate	SDA	TE slant	EL slant	40ïC	
M. pachydermatis	43	Large (100)	Pale pink/smooth (100)	+(100)	Growth (95.3)[d]	Growth and produced a black zone (95.3)[d]	Growth (95.3)[d]	NT	+(100)
M.sympodialis	84	Large (100)	Pale pink/ smooth (100)	+ (100)	No growth (100)	Growth and produced a black zone (100)	No growth (100)	NT	+(100)
M.globosa	14	Small (100)	Purple/ smooth (100)	+(100)	No growth (100)	No growth and no change (100)	No growth (100)	NT	+(100)
M.dermatis	5	Large (100)	Pale pink to purple/ smooth (100)	+(100)	No growth (100)	Growth and no change (100)	No growth (100)	NT	+(100)
M.furfur	41	Large (100)	Pale pink/ wrinkled (100)	-(97.6)[c]	No growth (100)	Growth and produced a black zone (100)	Growth (85.4)	NT	+(100)
M.slooffiae	108	Small (100)	Pale pink/ smooth (100)	-(100)	No growth (100)	Growth and no change (65.7)[e]	No growth (98.1)	Growth (84.3)[e]	+(100)
M.obtusa	4	Medium (100)	Pink/ rough (100)	-(100)	No growth (100)	No growth but produced a black zone (100)	No growth (100)	NT	-(100)
M.restricta	71	Small (100)	Pink/ Smooth (100)	-(100)	No growth (100)	No growth and no change (100)	No growth (100)	NT	-(100)
M.japonica	2	Large (100)	Pink/ Smooth (100)	-(100)	No growth (100)	Growth and produced a black zone (100)	No growth (100)	No growth (100)	+(100)

a. Incubated at 32°C for 4 to 7 days.

b. Incubated at 32°C for 4 to 7 days. NT, not tested; +, positive; -, negative.

c. Only one strain of fresh clinical isolate produced a precipitate.

d. Only two strains of fresh clinical isolates did not grow on SDA and EL and did not produce a black zone on TE.

e. Thirty-seven strains of fresh clinical isolates produced a black zone on TE, and 17 strains did not grow on modified Leeming and Notman agar at 40°C for 4 days.

Table 4 : Different properties of Malassezia species [1]

Malassezia species	Lipid Dependence	Growth at > 37^0C	Catalase Reaction	Esculin Splitting	Cremophor (1-10%)	Tween 20 (High Conc.)	Tween 40 (0.1-10%)	Tween 80 (Low Conc.)
M.furfur	+	+	+	-	V	+	+	+
M.pachydermatis	-	+	V	V	V	+	+	+
M.sympodialis	+	+	+	+	-	-	+	+
M.globosa	+	-	+	_	-	-	-	-
M.obtusa	+	-	+	+	-	-	-	-
M.restricta	+	-	-	-	-	-	-	-
M.sloofiae	+	+	+	-	-	+	+	-

NOTE : (+) = Positive Reaction; (-) = Negative Reaction; (V)= Variable Reaction

Table showing important physiological and biochemical characteristics of clinically significant Malassezia species.