A CLINICO-MYCOLOGICAL STUDY OF 100 CASES OF PITYRIASIS VERSICOLOR

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CERTIFICATE

Certified that this dissertation entitled ***A CLINICO-MYCOLOGICAL STUDY OF 100 CASES OF PITYRIASIS VERSICOLOR"*** is a bonafide work done by **Dr.K.P. SARADHA,** Post Graduate Student of the Department of Dermatology, Venereology and Leprosy, Madras Medical College, Chennai – 600 003, during the academic year 2005 – 2008. This work has not previously formed the basis for the award of any degree.

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CONTENTS

PITYRIASIS VERSICOLORCLINICO MYCOLOGICAL STUDY

INTRODUCTION

Pityriasis versicolor is a common, chronic, mild usually asymptomatic infection of the skin caused by malassezia species.

The organism is a lipophilic yeast. It has been found plenty in the androgen dependent, sebaceous secretory areas of the scalp, face, trunk, upper chest, back, shoulder, and upper arm.

The sebum dependent nature of this lipophilic yeast has been well brought out by the presence of lesion around the hair follicles not only in the trunk and scalp but also on the dorsal hairy regions of the phalanges of finger and toes and the beard areas of face and neck.

It has also been associated with other dermatological conditions like psoriasis, seborrheic dermatitis atopic dermatitis and confluent reticulate papillomatosis.

In recent years, they have been associated with serious systemic infections, in low birth weight infants and other debilitated individuals.

REVIEW OF LITERATURE

The genus malassezia has been placed among the Basidiomycota in the family cryptococcaceae (Yarrow and Ahearn 1984)[2].

The normal flora of the skin includes a number of morphologically distinct lipophilic yeasts[1]. It was thought a single polymorphic yeast pityrosporum ovale or two species P.ovale and P.orbiculare were present but it is now recognized that this genus name was invalid and these yeasts were re-classified.[2]

In the genus malassezia spp. M.furfur recognized in 1846 by Eichstedt[2].

Robin 1853 named the agent as microsporum furfur [18]

1874 Malassez gave the name Malassezia furfur[25]

In 1873 Rivolta described double contoured budding cells in a patient of Psoriasis. Baillon 1889 later used the name malassezia furfur in his text in 1889[25]. 1913 Castellani & Chalmers for the first time isolated lipophilic yeasts from normal skin & seborrheic dermatitis and coined the name pityrosporum ovale[44]. 1907 Acton & Panja considered it to be a synonym of malassezia in 1927. In 1935

C.W. Dodge described M. pachydermatis[2]. Rhoda Benham 1939 discovered the lipophilic nature of the genus malassezia[25]. 1951 Morris Gordon isolated authenticated a round double contoured budding yeast that produced spherical to oval buds in scales of pityriasis versicolor as well as on the normal skin[2]. 1990 M. sympodialis recognized by Simmons & Gueho [25,46].

In addition to above species molecular techniques described further species. There are more than 100 isolates[46].

Some are named they are

M. globosa

M. slooffiae

M. restricta

M. obtusa

M. dermatitis

M. nanum

M. equinum

M. japonicum

The current consensus from these studies in that M. globosa in the species most frequently associated with pityriasis versicolor[3,12.] M sympodialis is that found most commonly on normal skin - [3]

SYNONYMS[3,18]

- Tinea versicolor
- Dermatomycosis furfuracia
- Chromophytosis
- Tinea flava
- Liver spots [3]

EPIDEMIOLOGY

Tinea versicolor is a common benign superficial cutaneous fungal infection, Characterized by scaly hypo or hyper pigmented macules and patches that are located over the seborrheic areas, patient occasionally complaints of pruritus otherwise it is asymptomatic and it is not contagious[13,35].

The organism is a dimorphic lipophilic yeast that is cultured only in media enriched with C12 or C14 sized fatty acids.[4,35]

Race:

It is usually more apparent in darker skinned individuals. The incidence of tinea versicolor appears to be same in all races.[4,35]

Sex Distribution

Females and males are equally affected.[1, 4]

Age

In temperate zones, tinea versicolor is rare in children. When children develop tinea versicolor facial involvement is more common than adults.[4] It is more common in young adults 19-24 years .[4,7] In tropical climates it is common in all age groups but most common in age 10-19 years [4] Incidence decreases in later life[1] presumably as the sebaceous glands become non functional with old age[12]

Seasonal Variation

In tropical countries it is more common in warmer months, spring & summer and it is extensive due to increased temperature and humidity[4]. In colder climates the prevalence is less than 1%.[3,14] . In tropics the patients likely to have disease through out the year[4] with more extensive and persistent lesions[4]

Genetic Factors

A positive family history among the blood relations found more often than chance would suggest that in tinea versicolor whether this is caused by genetically determined host susceptible factor or the great opportunity for heavy colonisation at present is undetermined [3,5,50].

Conjugal cases also occur and it is possible that in some instances infection doesn't arise from the individuals own, autochthonous flora but by transmission from other individuals [3,5,50].

Pityriasis versicolor has been claimed to be more common in various disease states [18].

Cushing syndrome[18,] malnutrition, pregnancy, diabetes mellitus, tuberculosis, oralcontraceptivepill users, immunocompromised, those who are on immunosuppressive drugs, renal transplant recipients [18].

The organism is universally present as a member of normal flora and causes disease under special conditions when over growth of organisms occurs. The factors responsible for the over growth of these organisms in certain people is not known.[7]Malassezia species more readily become mycelial and have perhaps a slightly greater pathogenic potential.[3] Human peptide cathelicidin LL37 plays a role in skin defence against this organism[6].

MORPHOLOGY

Morphology of malassezia species

The appearance of the organism in scales from pityriasis versicolor shows spherical yeasts 2-8μ diameter arranged in clusters and associated with hyphae 10-25μ long 2-5μ wide aseptate angular.

They are aligned end to end or separated. [2] In tropical countries the appearance of the organism in the skin may reveal yeasts which are ovoid or cylindrical in shapes with filaments.[2] The method of cell division (i.e.) monopolar budding and the resulting formation of the scar or collarette at the base of the bud. There is considerable variation in the cell shape spherical, ovoid or elongated. Several forms may colonize a single site or groups of yeasts may be observed showing a unique morphology.[2]

IN VITRO MORPHOLOGY

Colonial and microscopic morphology of the cultures of malassezia species are based on cultures grown on a medium [2].

Modified form of Dixon formula Von Abbe 1964

- 3.6% malt extract
- 0.6% Mycological peptone
- 2% Ox bile
- 1% Tween 40
- 0.2% glycol
- 0.2% Oleic acid
- 1.2% Agar

The medium was always used with in a week and incubation was at 32^0C. The growth was assessed after 10 days.[2,25] Colonies of Malassezia species have a characteristic brittle texture.[2,25] The common features of cells of all species are the method of blastoconidia formation and the possession of a multi layered wall.[2]

The ultrastructure of Malassezia species by electron microscopy revealed the characteristic cell wall the innermost layer exhibits invagination which form spiral bands around the cell[2]. (Barfatanil, Munn, and Schejude 1964)[2] In light microscope double contour to the edge of the yeast indicates the thickness of cell wall and the appearance of diagonal striation across the cell surface represent the ridges in the inner layer.[2,42]

COLONY MORPHOLOGY

M. furfur

Thick convex or umbonate colonies surface is smooth, rough variants occur [2]. The texture is soft; colour is cream [2,25]

M.sympodialis (Simmon and Gueho 1990)

M.sympodialis forms flat or slightly convex colonies cream to buff in colour with smooth shiny surface sympodial budding has also been observed.[2]

M. pachydermatis

Thick pale, convex colonies with a smooth surface. It is not lipid dependent.[1]

M. globosa

Slow growing rough colonies deeply folded surface cream to buff in colour [2] and a texture that is very brittle and particularly difficult to emulsify spherical cells 6 - 8 µm in diameter with buds formed on a narrow base[2]

M. slooffiae

Fine folded, cream to buff in colour, brittle texture.[2] The cells are small cylindrical 1.5 – 3.5 µm long[2]

Growth Requirements

With the exception of M.pachydermatis malassezia species have an absolute requirement for lipids which can be supplied by the inclusion of medium of long chain fatty acids in the culture medium.[4]

Most frequent being olive oil, Its principal ingredient is oleic acid other growth factors were investigated by Nazzaro Porro et al 1976 who found that although isolates by Malassezia spp. were stimulated

by presence of glucose and asparagine in the medium. They were not essential. Glycine induces hyphal formation [4]

Tween Assimilation test

Briefly malassezia yeast suspension was mixed with Saborauds agar and mixtures were plated. Four holes were made in the agar by mean of 3 mm diameter punch and filled with 5 µl each of Tween 20, 40, 60, 80 respectively[20]. The agar plates were cultivated at 32°C for 1 week[20]

The growth of M. sympodialis is inhibited by high concentration of Tween 20.

M. furfur exhibits similar growth with both Tween 20 & 80.

M. sloofiae grows better with Tween 20 than with Tween 80.

M. globosa, M. obtusa, M. restricta are unable to utilise any of this four Tween compound.

M. restricta is the only lipid dependant species lacking catalase[19].

Although most Malassezia spp are able to grow at 37^0C this temperature is not recommended the optimum temperature is between 32^0C – 35^0C.[1,2] For good growth freshly prepared medium and a humid atmosphere, growth also occur under micro aerophilic conditions.[1,7]

Flow Chart Showing Identification Scheme of Malassezia Isolates

Skin Scarpings

KOH mount, Parker Quink's Stain

Spaghetti and meat ball appearance

Modified Dixon's agar and SDA with Olive oil

If growth present

Urease test

If positive

Subculture on SDA without Olive oil

+ **-**

Non - Lipid Dependent species

M. Pachydermatis

Test **-**

Lipid Dependent species

+ Catalase

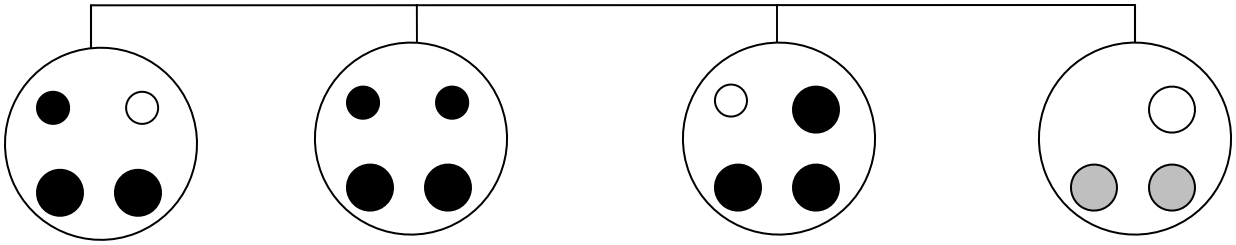
+ Growth at 41°C **-**

M. restricta

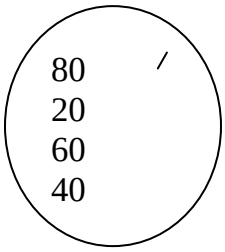
M. Sympodialis
M.furfur
M.sloofiae

M.globosa
M.obtusa

Tween Assimilation Test



M.sympodialis **M. furfur** **M. sloofiae** **M. globosa,**
M. obtusa



Gram's Stain

Globose cells with narrow based budding based **M.globosa**

Cylindrical cells with broad budding **M. obtusa**

PHYSIOLOGICAL AND BIOCHEMICAL TESTS[34]

Species	Catalase test	Urease test	Growth at 41°C	Tween				Lipid Dependence	Gram's Stain
				20	40	60	80		Type of budding
M. furfur	+	+	+	+	+	+	+	+	Broad based
M. sympodialis	+	+	+	-	+	+	+	+	Sympodial
M. globosa	+	+	-	-	+	+	-	+	Narrow based
M. restricta	-	+	-	-	-	-	-	+	Narrow based
M. obtusa	+	+	-	-	+	+	-	+	Broad based
M. sloofiae	+	+	+	+	+	+	-	+	Broad based
M. pachydermatis	+	+	+	+	+	+	+	-	Broad based

PATHOGENESIS & IMMUNOLOGY

The initial views that patients with pityriasis versicolor have a cell mediated Immune deficiency specific to Malassezia species or depletion of specifically reactive T-cells from the blood [16, 17] T cell inhibition by a lipid component of the Malassezia cell wall has recently been reported [17]. Serum antibodies against M. furfur are present in the sera from healthy adults; the titer is of no value in diagnostic and prognostic consequences[24,25].

Hyperkeratosis , parakeratosis and slight acanthosis[3] with a mild inflammatory infiltrate. The infiltrate revealed a dominance of memory T Cells. macrophages and lack of B cells [3]. In various studies, defects in lymphokine production and natural killer T cells were found. Phytohemagglutinin and concanavalin (A) stimulation is decreased. IL_2 IL_{10} and IFN δ production by lymphocytes is decreased in affected patients [4,25].

SEROVARS

1990 using the medium formulated by Leeming and Notman Cunningham et al observed and differentiated 3 distinct forms of M. furfur. They were different morphologically, genetically,

physiologically. They possessed 3 distinct cell surfaces antigens. Three forms are serovars A, B and C[20] . Serovar A was the predominant isolate over both chest and back. Whereas on the forehead and cheek no serovar was predominant. The difference is due to the physiological requirement. Serovar A is nutritionally less fastidious[19,45]. Microscopically serovars A & B had round, whereas serovar C had oval blastospores[25], serovars A & B are described as P.orbiculare and serovar C as P.ovale. Serovar B occurs over the trunk & scalp. Serovar C occur over the scalp of normal individual[25]

ANIMAL STUDIES:

The disease has been reported in domestic wild and laboratory animals[2] M.pachydermatis associated with otitis externa of dogs[2,47].

Yeasts of Malassezia species are also known to colonize the skin of bears, monkeys, pigs, elephants, rhino's and birds[2,18,47].

A higher prevalence of among a group of water birds compared with tropical birds would be explained by the adaptation of the birds to their habitat those in an aquatic environment have an increased supply of lipids to the feathers[2,18,47].

MOLECULAR STUDIES:

A number of molecular methods have been applied to the malassezia genes. The states of species was confirmed in 1990 by Simmons & Gueho by estimation of percentage of G + C content of DNA and by the reassociation of DNA between different isolates[2,4,6].

Recently karyotyping using pulse field electrophoresis and sequencing of large subunit of RNA have added more information. In addition the application of molecular probes and the use of polymerase chain reaction (PCR) methods to discriminate strains even further are now possible [2,8,25].

Restriction fragment length polymorphism, Randomly amplified polymorphic DNA analysis[25], PCR–Finger printing, multilocus enzyme[25] Electrophoresis are the newer technique in the diagnosis of Malassezia spp[25].

CLINICAL FEATURES

All lesions of tinea versicolor are clear cut with well defined borders, in various shapes [4]. The colour various according to the normal pigmentation of the patient, exposure of the area to the sunlight, and severity of the disease [18.]

Colour may be hypopigmented or hyperpigmented varies from individual to individual, but each individual lesion is of approximately the same colour. Some may present with both hyper and hypopigmented lesions[3].

The lesions may be macules, patches, follicular or perifollicular hypopigmented or hyperpigmented. Papular lesions are some times seen and are usually perifollicular[18], some are erythematous, the lesions may be large enough to cover most of the trunk[6]. Usually asymptomatic but there may be mild pruritis[6].

Chief complaint is cosmetic one, an inverse form of tinea versicolor also exists in which the condition has an entirely different distribution, affecting the flexural regions, face, or isolated areas of the extremities.[6] This form of tinea more commonly seen in

immunocompromised. Follicular tinea versicolor occurs around the hair follicle. An atrophic variant is also described [13]

PSEUDO ACHROMIA PARASITICA

Residual hypopigmentation without overlying scale may remain for many months following effective therapy [3,5].

In dark skinned infants particularly in tropics the infection starts in the diaper covered areas and spreads rapidly causing marked depigmentation. This is called the pityriasis versicolor alba or achromia parasitica [18]

DISTRIBUTION OF THE LESION

Upper trunk is most commonly affected but the lesion often spread to upper arms, antecubital fossa, neck, abdomen, popliteal fossa.[2]

Facial lesions have been reported in tropical countries also has the involvement of the lower abdomen and limbs[12]. Axillae, groin, thigh, genitalia less commonly involved. Penile involvement has been reported[12] Facial, scalp and palmar region involved in tropics, but rare in temperate zones. Sometimes pityriasis rosea like lesion can occur [15].

COUP D' ONGLE SIGN OF BESNIER

A normal coloured or hypo pigmented underlying skin in visualized when the pigmented scales are removed enmasse as a sheet by scraping with a glass slide or scotch tape stripping of the palpable lesions. This is called Coup d' Ongle Sign.[3]

UNUSUAL PRESENTATIONS OF MALASSEZIA

1) Pityrosporum Folliculitis (Rausch and Jacabs 1984)

It resembles steroid acne, involves the hair follicle. Localized to the back, chest and extremities. It presents as perifollicular itchy erythematous papules or pustules common in HIV patients. It represent a delayed hyper sensitivity response to Malassezia species[3,13] treatment with selenium sulphide wash is helpful. Ketoconazole orally 200mg daily for 4 weeks or Fluconazole 150mg weekly for 2 – 4 weeks or Itraconazole 200mg daily for 2 weeks are useful systemic agents.

2) Confluent Reticulate Papillomatosis

This is a relatively rare disease, consisting of grey brown pigmented confluent papules on the neck, interscapular region under the breast and on the abdomen. Some have regarded it as a

genodermatosis others have demonstrated M. furfur in the scales. Many patients have been treated successfully with antifungal preparation (Yesudian et all 1973)[11.] Onset after puberty[11] it is due to abnormal host response Electron microscope also shows two major findings[13,37].

(i) A marked alteration of the cornified cell structure showing snake coil like or triangle like stacks [21,39].

(ii) Increase in the number of lamellar granules as Pityrosporum organisms are lipophilic and could play some part in production of those finding[21,39].

3) Seborrheic Dermatitis

It is due to altered cell mediated immune response to Malassezia spp. It is a chronic dermatitis with greasy scales. The cell wall of malassezia activates the alternate pathway leads to inflammation[24,40].

Fungemia

In premature infants on total parenteral nutrition with intravenous lipid supplementation has been reported to have had an extensive vasculitis of small pulmonary arteries as well as pneumonia[3]. Colonization of the catheter occurs probably by

proliferation of growth from the patient's skin and subsequent dissemination [18,2,43].

Malassezia species has been cultured from the peritoneal dialysate and blood[7,22]. Malassezia causes fungemia in immunocompromised host [7]

Dacryocystitis; Malassezia furfur may colonize the lacrimal sac causing enlargement of lacrimal sac, and obstruction. In such case dacryoliths develop leading to inflammation[8,22].

Transient acantholytic dermatosis has been reported with Malassezia [4]

Psoriasis

Malassezia also said to excite an immunological reaction considered to play a role in the pathogenesis of psoriasis[19].

Atopic dermatitis

In patients with atopic dermatitis, M. furfur was isolated from the itchy lesions of face and head. In individual with atopic phenotype eczema may be induced by malassezia yeast[13] IgE antibodies against M. furfur commonly found in atopic patients with head and neck dermatitis[19].

Infantile cephalic pustulosis[3] in New Born

It appears during the first month of life in both male and females with erythematous pustules and papules on the head and neck. It is thought to be an inflammatory reaction to Malassezia spp[3,43,48].

Onychomycosis and endocarditis have also been associated with Malassezia spp[18].

Malassezia has been recovered from the nasal passages and nasopharynx in a patient with maxillary sinus, osteitis[22].

CAUSES FOR PIGMENTARY CHANGES

Hypopigmentation

1. Inhibition of tyrosinase by Azelaic acid produced by the organism.[3]
2. Abnormally packaged melanosomes which are not transferred to keratinocytes.[3]
3. Abnormal structure of stage I melanosomes, with increased space between melanocytes and keratinocytes[3].
4. Increased number of melanosomes in the melanocytes but a decrease number with their irregular distributions of melanosomes[3,18].

5. Defective transfer of melanin pigment by increased epidermal turnover and sun screening effect of scales are the proposed causes of Hypopigmentation[3].

Hyper pigmentation is due to

1. Thickened keratin layer [3]
2. Inflammatory infiltrate causing stimulation of melanocytes [3]
3. Large melanosomes [3,18]

Differential Diagnosis for Hypopigmented Lesions

Seborrheic Dermatitis

It is a chronic dermatitis with a distinctive morphology characterized by erythematous sharply marginated lesions with greasy scales,[3]. It is due to altered cell mediated response to malassezia species [3,40].

Pityriasis Rosea

It occurs with herald patch with distinctive morphology. Erythematous patch, oval or circular with collarette of scales in a typical Christmas tree distribution[3].

Indeterminate Hansen

It occurs as minimally scaly hypopigmented patch vaguely defined without any loss of sensation over the face and gluteal

region. It may clear spontaneously or progress to one of the spectrum[51] of leprosy.

Naevus Achromicus

Occurs from birth with serrated borders, non scaly[3].

Naevus Anaemicus

Occurs from birth other wise called Pharmacological Naevus. On diascopy it merges with the normal skin. Wood's lamp does not accentuate the lesions[3].

Macular Syphilide

Faint pink lesions less than 1cm diameter round or oval distributed over the nape of neck, sides of the trunk and flexor aspects of the extremities. Serology may be positive[10].

Ash leaf macules of tuberous sclerosis

Characterized by hypopigmented macules which are accentuated by Wood's lamp examination.[3]

It occurs from birth or early infancy [8]. They are polygonal or ash leaf in shape 1 – 3 cm size 1 -100 in number [8]

Early Vitiligo

It presents as non scaly lesions. It is a depigmenting disorder due to loss of epidermal melanocytes. Lesions of vitiligo are milky

white with a well demarcated border. Sometimes the borders are hyper pigmented and rarely erythematous[3].

Post kala azar dermal leishmaniasis

Occurs as macules or patches after treatment of visceral Leishmaniasis[3]. The lesions begin as pinpoint hypopigmented macules and gradually enlarge to a centimeter or so in diameter. Later the lesions become raised.

Pityriasis Alba

It occurs over the face. It is an endogenous eczema with mild scaling with indistinct borders.[3] Associated in atopic individuals and nutritional deficiency states[3].

Lichen Sclerosus et atrophicus

It occurs as porcelain white macules and plaques[3].

Resolving Miliaria Rubra

Hypopigmented scaly macules around eccrine orifices[3].

Resolving Psoriasis

Post inflammatory hypopigmentation of psoriasis following application of PUVA , topical steroids. It is secondary to vascular rather than melanin related changes[11].

FOR HYPER PIGMENTED LESIONS

Melasma

It occurs as reticular, blotchy macular pigmentation over the face especially in women, non scaly[9].

Erythrasma

Well circumcised maculopapular lesions. Scales may be greasy or furfuraceous. Wood's lamp examination shows coral red fluorescence. Caused by Corynebacterium minutissimum, affecting the flexural areas [3] like groin, inframammary region axillae, perianal regions[3].

Lentigenes

Macular areas of hyper pigmentation. It can occur any where in the body, non scaly[11]. This is generally caused by increased melanin production by existing melanocytes[11].

Junctional Naevus

An acquired melanocytic naevus, non scaly, uniformly pigmented macules or patches[11].

Cafe au lait macules

Brown macules of hyperpigmentation of varying size well defined irregular borders non scaly, 2 - 20 μm[12]. They appear at or soon after birth and tend to disappear with age[11].

Acanthosis Nigricans

Flexural areas of hyperpigmentation which are verrucous to see velvety to touch and they are non scaly [3,43].

Melanoderma

The increased pigmentation in melanoderma is due to prolonged sun exposure[11]

Vagabonds Disease

It is associated with pediculosis[11.] It is actually a leucomelanoderma found in older ill kempt men and women with a history of poor hygiene.

DIAGNOSIS OF PITYRIASIS VERSICOLOR

The varied clinical types described associated with scaling help in the diagnosis.

Examination of the scales in 10% potassium hydroxide reveals the etiologic agent. [5]

The agent appear as spherical spores, often as grouped spores occurring in clusters resembling bunches of grapes, blastospores surrounded by short straight or angulated non septate hyaline hyphae [5].

This combination referred to as spaghetti and meat ball appearance,[3,18]

These hyphae and spores may show a distinct capsular outline. The spores are 3μm– 5μm diameter and hyphae 3-4μm in diameter, [2]

Liquid blue ink, methylene blue or Shwartz – Medrik stain can be added to the potassium hydroxide for better visualization of the agents[4].

Scales may also be removed using clear adhesive tape. They are then directly examined. The tape must be clear and is pressed several times over involved areas of skin. A small drop of methylene blue or other appropriate stain is placed at the edge and allowed to run between the tapes and the glass slide spores and mycelium are easily seen [4].

COLOURS

Spore and mycelia

- Are Hyaline in KOH
- Blue in methylene blue or lactophenol cotton blue and 5% Parker's blue black ink[6]
- Purple in PAS (Periodic Acid Schiff)[3]
- Black in GMS (Gomori's Methenamine Silver)[6]

CULTURE

It is lipophilic organism which is cultured only in media enriched with C12-C14 sized fatty acid [4]. In Sabourauds dextrose agar with chloramphenicol and actidione, Tween 80 and layered with olive oil the growth occurs at 5-7 days.[8] The colonies are yeast like buff coloured on the surface and reverse, dome shaped colonies. The culture mounts under microscope reveal oval elongated blastospores and short hyphae. Germ tubes are produced when the culture is incubated at 70% CO^2 in atmospheric air.

Other culture methods are;-

Dixon agar provides substantial growth and gives features of colonies which help in identification of species. Alternative culture media like Gyp – S agar can also be used which can provide growth and quantitation of colonies with in 3 – 4 days. It contains glucose yeast extract peptone agar plus olive oil, Tween 80 glycerol mono stearate.[25]

A new minimal medium for malassezia furfur consisting of only L tryptophan and a lipid source induces formation of brown pigmentation which diffuses into the agar synthesis of pigments and

fluorochromes by yeasts of genus malassezia might help to explain several clinical characteristic of pityriasis versiolor[25].

WOOD'S LAMP EXAMINATION

Wood's lamp is a mercury vapour lamp with barium silicate and 0.9% Nickel Oxide as filter; it emits UVA in the wave length 365 nm.

The skin lesions of tinea versicolor when examined in dark room give a Golden Yellow fluorescence under Wood's lamp. [7]

HISTOPATHOLOGY

Histopathology of sections of tinea versicolor lesions in Haematoxylin & Eosin section shows.

Hyperkeratosis with or without basket weave stratum corneum [3] mild acanthosis – normal or increased pigment basal layer. The hyper pigmented lesions shows increased pigment not only in the basal layer, but also in the outer layer of epidermis [3].

Vacuolated cells are seen not only in the epidermis but also seen in the external root sheath[18], subepidermal hyalinization [23] is also seen. Absence of granular layer was observed in sections, where the filaments were seen in close approximation to the stratum malphigii[23] Hyphae can be visualized in the follicular plugging and in the vicinity of acrosyringium [38]. The presence of mycelia without

spores in the stratum corneum in the vicinity of sweat duct shows the role played by the sweat which is capable of inducing yeast to mycelial shift [23,39]. Fungal elements do not invade hair, the nucleated cells, or in the viable layers of epidermis or dermis [3,23.] Spherical spores, blastospores and short aseptate hyphae of malassezia are seen as blue elements in the outer two third of stratum corneum[5]. In atrophic variant, epidermal colonization with hyphae and spores with effacement of rete ridges, sub epidermal fibroplasia, pigment incontinence and elastolysis is seen[14] The upper dermis shows perivascular round or mononuclear cell collections. Acanthosis nigricans like lesions in more papular lesions. Dilated blood vessels are seen in erythematous lesions [23].

MANAGEMENT OF TINEA VERSICOLOR

There are a number of options available for the treatment of pityriasis versicolor both topical and oral medications have been shown to be effective. Relapse is common. Patients should be cautioned that even though pityriasis versicolor can be treated effectively with in 2 weeks or less of therapy, the pigmentary changes seen in this disorder may take some time to resolve[6].

TOPICAL TREATMENT

Earlier treatment options involved the use of non specific agents that did not have direct activity against malassezia, but they physically or chemically remove the infected stratum corneum [1,49].

The treatment include

White field ointment, (3% Salicylic acid, 6% Benzoic acid with 24% coconut oil in a paraffin base) Sulphur with salicylic acid as ointment, 2.5% Selenium sulphide as shampoo, 1% Tolnaftate ointment, 20% Sodium Hypochloride lotion, 20% Sodium Thiosulphate lotion, 1% Zinc Pyrithione as shampoo and also Haloprogin, 2.5% Benzoyl peroxide, 50:50 propylene glycol in water[18]

Topical Therapies has certain disadvantages.

Topical therapy requires regular applications, Some are cosmetically objectionable, some have unpleasant odour topical therapy is considered for localized lesions[5]. It is associated with high recurrence rate [1]

Topical Antifungal agents used are

Topical Imidazoles

2% Clotrimazole 1% Econazole 1% Miconazole

1% Sulconazole 1% Oxiconazole 2% Fenticonazole

1% Bifonazole

Topical Triazoles

1% Fluconazole 2% Ketoconazole

Topical Allylamines

1% Terbinafine 1% Naftifine

Topical Benzylamines:

1% Butenafine

Hydroxy Piridone

1% Ciclopiroxolamine

The treatment consists of application for 1-4 weeks twice or once daily contact period 10 – 15 minutes some preparations require overnight application. They are available as cream or shampoo or lotion formulation.

Systemic Therapy

Indications for systemic therapy include extensive lesion, recurrent, chronic, scalp infection, unusual presentation with special types of lesions attributed to Malassezia spp.

Systemic therapy include

T. Ketoconazole 200mg once daily, 15 days[5]

or

T. Fluconazole 50mg once daily, 15 days[5]

or

T. Itraconazole 200mg once daily, 7 days[5]

Pulse Therapy

T. Fluconazole 400mg single dose, repeated after a week

or

Ketoconazole 400mg, 2 consecutive days / week, for 2 weeks

or

Itraconzole 600mg stat [5].

To prevent Recurrence

T. Ketoconazole 200mg twice /day for 3-5 days / month for 5 months.

Though various treatment modalities are available the relapse rate is high because of the colonization of pityrosporum orbiculare in the follicular structure that contributes to the recurrence. The lack of host resistance also has been attributed to the difficulty in elimination[6].

AIMS OF THE STUDY

The aims of the study are

1. To study the age and sex distribution of the patients.
2. To study the predisposing factors.
3. To study the association of pityriasis versicolor with other dermatological condition.
4. Systemic diseases associated with pityriasis versicolor.
5. Pathological correlation of clinical lesions.
6. To isolate the agent.

MATERIALS AND METHODS

100 cases of pityriasis versicolor were collected from the outpatients attending mycology section. Department of dermatology Government General Hospital, Chennai over period of 2 years from September 2005 to September 2007 for the study.

Those who had pityriasis versicolor lesions for more than 3 years and involvement of more than 10% of the body surface area were selected for the study. Informed consent of the patients was obtained.

Subsequently careful history with particular reference to age and sex. Symptoms, duration of the disease, occupation, marital status, family history, history of exposure to sexually transmitted disease in those with high risk behavior were obtained.

A detailed systemic and dermatological examination was done.

Routine analysis of hemoglobin, total count, differential count, blood sugar, serum cholesterol, blood grouping and typing were done in all patients.

In appropriate cases ELISA for HIV, Blood VDRL were done.

The skin lesions of pityriasis versicolor were subjected to clinical examination.

The scales of the pityriasis versicolor lesions are collected by scraping and wet mount preparation was done with 10% potassium hydroxide and visualized under light microscope.

Culture

In 50 patients culture was done. The site of the lesion was thoroughly cleaned and scraping was done. the collected scales were inoculated using a sterile platinum loop in to Sabouraud's dextrose peptone agar with chloramphenicol, actidione and Tween 80 with olive oil overlaid and kept at 37°C in an incubator for 48-72 hrs. The colonies were again examined with KOH mount with 5% Parker's blue ink under light microscope. For species isolation Tween assimilation test was done from the culture isolates from the Sabouraud's dextrose peptone agar

Biopsy

In 20 selected patients the pityriasis versicolor lesions were biopsied with 4mm punch under aseptic precautions and Haematoxylin & Eosin section of the biopsy slides and Periodic Acid Schiff – staining were done and various histopathological findings were observed.

OBSERVATION

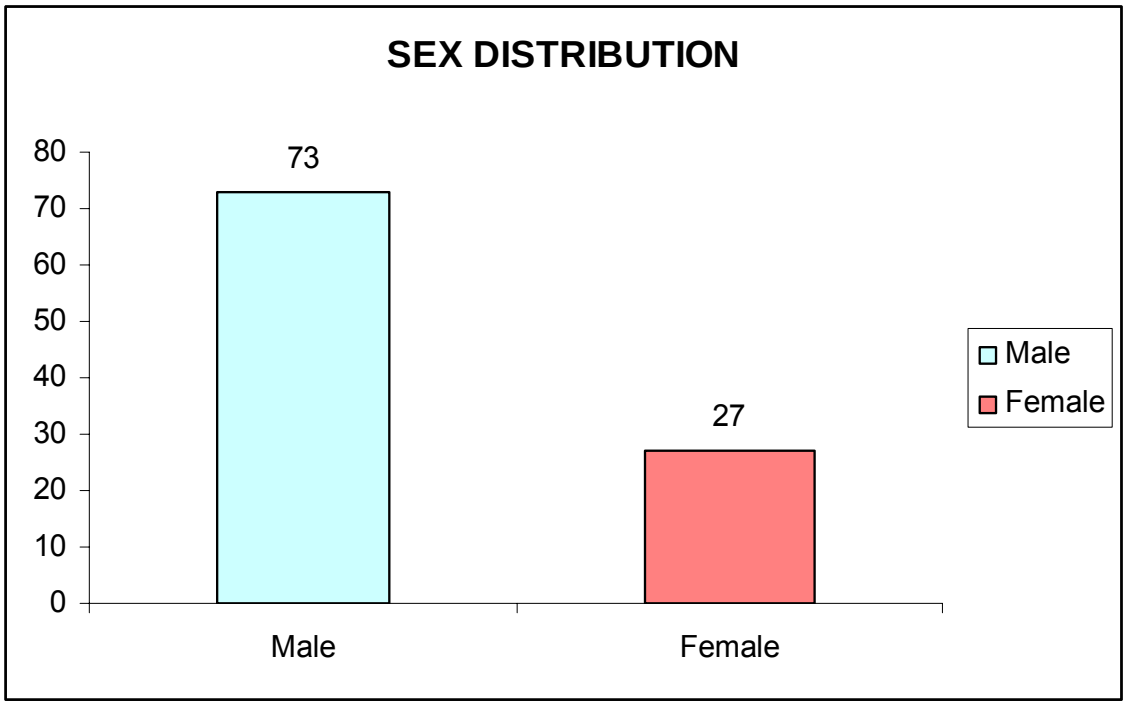
Sex Distribution

Among 100 cases pityriasis versicolor

No. of Males 73

No. of Females 27

Following bar diagram shows sex distribution



AGE & SEX DISTRIBUTION

Age in Years	No. of Cases Male	No. of Cases Female	Percentage
0-10	-Nil-	-Nil-	0
11-20	19	6	25
21-30	37	10	47
31-40	10	8	18
41-50	6	3	9
51-60	1	0	1
Total	73	27	100

72% of the cases in study were between 10-30 years

28% between 30-60 years

Age distribution in the study varied from 14 years to 57 years.

Youngest age in this study was 14 years, Oldest age was 57

Type of Skin Lesions	No. of Cases	Percentage
Achromic	57	57
Chromic	6	6
Perifollicular	14	14
Achromic and Chromic	15	15
Plaque type	6	6
Erythematous	2	2
Total	100	100

Distribution of lesions and percentage of body surface area involvement

In 56 patients the surface area involvement was more than 10%. In 40 patients the surface area involvement was more than 30% and in 4 patients the surface area involvement was more than 50%. In all hundred patients the lesions were present over the seborrhoeic area (i.e.) over the face, posterior auricular area, nape and sides of the neck, shoulders, axillae, front of chest, back of the chest, arms. In forty- four patients, the lesions were also present over the forearm, abdomen, lower back, genitalia thighs and legs in addition to the

seborrheic area. Sixty patients were asymptomatic and the remaining forty patients had cosmetic problem because of hypo and hyper pigmented lesions.

Duration of the diseases

In this study the duration of the disease among the patients was 3-15 years with remissions and relapses in 60% of the patients.

The relapses occurred during summer months as a result of increase in temperature and sweating.

Presence of the disease among the family members

In 46% of patients suffering from tinea versicolor the family members, either the parents or the siblings had lesions of tinea versicolor. There was no lesion of tinea versicolor reported in the spouse.

Associated Dermatological Conditions with Tinea Versicolor

Infective Conditions

In this study dermatological conditions associated with tinea versicolor are 7 cases of dermatophyte infection of the skin and nail, 2 cases of Trichomycosis axillaris, 2 cases of wart, 1 case of Candidal Balanoposthitis and 1case of Scabies these are some infecting condition

OTHER DERMATOLOGICAL CONDITIONS ASSOCIATED WITH TINEA VERSICOLOR

Sl No.	Other Dermatological Conditions	No. of patients
1	Acne vulgaris	13
2	Seborrheic Dermatitis	7
3	Becker's Naevus	2
4	Pityriasis Rosea	1
5	Psoriasis	2
6	Lichenoid eruptions	1
7	Hand Eczema	1
8	Keloid	1
9	Melasma	4
10	Vitiligo	1
11	Hirsutism	1
12	Bullous Pemphigoid	1

SYSTEMIC DISEASES ASSOCIATED WITH TINEA VERSICOLOR

	Associated Systemic Disorders	Number of Patients
1).	Renal transplant patients	10
2).	Diabetes mellitus	9
3).	T.B. Chest	1
4).	T.B. Genitourinary Tract	1
5).	Bronchial asthma with steroid therapy	2
6).	Sub acute cutaneous Lupus erythematosus	1
7).	Left Atrial myxoma	1
8).	Hypothyroidism with Cushing's diseases	1
9).	Psoriatic arthritis on methotrexate	1

Investigations

Complete hemogram

Among hundred cases anemia was found in 26 cases.

Blood Sugar: Showed hyperglycemia in 9 cases among them 3 were known diabetic on injection insulin.

Serum Cholesterol was elevated in 13 patients

Blood grouping and typing:

Most common blood group was B group in 52 patients followed by O group in 38 patients, A group in 8 patients, AB group in 2 patients.

ELISA for HIV

Among hundred cases none of them showed positivity for Human immunodeficiency virus disease.

VDRL for Syphilis also non reactive in all the patients

Examination of scales under 10% KOH

Skin scraping was done for all patients. All skin scrapings under 10% KOH showed, short, straight, aseptate angular hyaline hyphae with blastospores and grouped spores with spaghetti meat ball appearance.

Fungal culture

The culture is done to isolate the causative agent in tinea versicolor infections. It is not a routine clinical practice because it is cumbersome to grow this organism. As malassezia is a lipophilic fungus, lipids are incorporated into culture media which include oleic acid, olive oil, glycerol monostearate and Tween. The skin scrapings from tinea versicolor lesions are inoculated in to Sabouraud's dextrose agar with chloramphenicol, actidione, Tween and a film of sterile olive oil in concentration of 10ml per litre. Small 3 – 6 mm cream colored yellowish colonies slightly raised with irregular edges develop within 5-7 days at 32 – 35°C on the surface and on the reverse. 5% Parker's blue ink mounts of colonies shows. 2 – 7 µm yeast with many small 2 – 3 µm blastospores and hyphae. Among 50 cultures, 25 cultures showed positivity for malassezia. The remaining 25 cultures were contaminated. Among the 25 positive cultures Tween assimilation test was done. In 14 patients (56%), M.globosa was isolated and M.sympodialis in 11(44%). other species were not isolated in this study.

Skin Biopsy

Skin Biopsy was taken for 20 patients under strict aseptic precaution. Haematoxylin and Eosin section of the biopsy specimen and Periodic Acid Schiff staining were done.

Skin biopsy was done for 20 patients among whom 15 were male and 5 female.

The type of skin lesions biopsied are six cases of achromic, five cases of perifollicular, two cases of chromic, two cases of plaque type, a case of erythematous type and two cases of mixed type, i.e. chromic and achromic.

In Haematoxylin and Eosin section of the biopsy the following features were observed. Majority of the patients (15) showed Hyperkeratosis (83.33%), Basket weave stratum corneum was observed in 7 patients (38.88%), 6 patients showed keratotic plugging (33.33%). Irregular acanthosis was noted in 16 patients(88.88). Psoriasiform acanthosis was noted in 2 patients(11.11%). Vaculoated cells in the epidermis was noted in 4 patients (22.22%).11 cases showed increased pigment basal layer 61.11%). The upper dermis showed pigment incontinence in 4 patients(22.22%). Hyphae and spores were demonstrated in the

stratum corneum in 11 patients (61-11%). Inflammatory infiltrate comprising mononuclear cells was found in the upper dermis in all of them (100%). In four patients infiltrates were present around the hair follicles and sebaceous glands. Dilated blood vessels were seen in 6 patients (33.33%). In PAS stained section fungal spores, blasto spores and hyphae were visualized in the stratum corneum in fourteen patients. Hyphae were also visualized in the follicular plugs in 5 slides (27.77%). Absence of granular layer was observed in 6 sections where the filaments were seen in close approximation to the stratum malphigii.

Treatment

All the patients were given topical 2% clotrimazole cream for external applications. Tablet Fluconazole 400mg stat per week was given for a period of 2 weeks in patients with extensive lesions and recurrence. Patients were followed up.

DISCUSSION

Sex Incidence

In literature tinea versicolor was reported to be equal in both sexes[3]. There was no sexual preponderance and equal sex incidence was reported by Dr. Mark Crowe MD AAD[6].

In our study male patients are affected more (73%) than the female (27%). Most of the affected males are catering students, sales personal, college and school students. This increased incidence may be due to excessive sweating, more out door activities, excessive sebum production, probably makes the patients more susceptible for infection corresponding to literature[6]. Synthetic clothing, occlusion from talcum powder result in increased Co_2 concentration and altered microflora predisposes to infection which corresponds to literature.

Age Distribution

The most common age group affected with tinea versicolor is between 11 – 30 years in this study. Youngest was 14 years and oldest was 57 years. This correlates with the literature[6]. In temperate countries most common age group is 19 – 24 years[6]. In tropical countries cases occur between 10 – 19 years[6]. The occurrence of

tinea versicolor in this age group may be due to the excessive sebum production[3]. Since malassezia is a normal commensal of the hair follicle and since it is lipid dependent most cases occur in this age group[2].

Symptoms

Almost 90% of patients were asymptomatic. 10% of patients were concerned about the cosmetic problem with hypo or hyper pigmentation which correlates with the literature. The patients who were concerned about the persistent hypopigmentation, i.e. pseudo achromia parasitica after complete course of treatment were about 40%[3]. This corresponds with literature.

Duration of the Disease

In our study all the patients had the occurrence of lesions for more than 3 years to 15 years with remissions and relapses which correlates to the literature. The relapses were attributed to the climatic changes [3,18] and persistent colonization of Malassezia in the hair follicle to the chronicity and recurrence[6].

Presence of Disease among the Family Members

In our study 46% of the patients showed tinea versicolor lesions among the family members either in the parents or in the siblings.

Among those spread of the disease from the parents to children was more 41% and 4% among the siblings. There was no conjugal spread of the disease. This correlates with the literature which indicates the role of a hereditary factor in the transmission of the disease [3,6].

Type of Skin Lesions

The most common type of lesion is achromic variety which occurred in 57% of the patients, followed by mixed type consisting of both chromic & achromic is15% of the patients.

Perifollicular type was seen in 14% of the patients, Chromic type in 6% of the patients, Plaque type in 6%of the patients and only 2 % of the patients had Erythematous type.

The dry atrophic variant encountered in the literature was not found in our study.

Distribution of the Lesions

The commonly affected areas were upper trunk, axillae, neck, back, arm, face in 56% of patients which corresponds to the literature. Involvement of the abdomen, thighs, forearm, hands occur in 44% of patients with extensive involvement of more than 30% of the body surface area. The occurrence of lesions over genital region

in one female and facial involvement is also common in this study. Coup d' Ongle sign of Besnier was positive in all the cases[3].

Other lesions like confluent reticulated papillomatosis, Pityrosporum folliculitis, fungemia, dacryocystitis, transient acantholytic dermatosis, infantile cephalic pustulosis, and onychomycosis were not reported in our study.

Associated Dermatological Conditions

7 patients with tinea versicolor had seborrheic dermatitis which corresponds to the literature. It is due to an altered immune response to the Malassezia species. The cell wall of the malassezia activates the alternate pathway leading to inflammation[7].

13 patients with tinea versicolor had acne vulgaris. It may be due to the excessive sebum production[3]. 2 patients were associated with Becker's nevus in this study which was incriminated to increased androgen sensitivity[3]. The superficial fungal infections associated with tinea versicolor in this study were dermatophytosis of skin and nail (7 cases), candidal balanoposthitis (one case), wart (2 cases), Trichomycosis axillaris (2 cases) and one case of scabies. Other skin

lesions like melasma vitiligo, psoriasis, lichenoid eruption, keloid, hand eczema and pityriasis rosea were incidental findings.

Associated Systemic Disorder

There was increased incidence of tinea versicolor in renal transplant patients(10) and also with diabetes mellitus (9) which corresponds to the literature[18] .

Tinea versicolor lesions were present in two patients receiving steroids and this corresponds with literature[18]. Two patients with tuberculosis had tinea versicolor which was attributed to night sweats. Four patients on immunosuppressive drugs also developed tinea versicolor in our study corresponds with the literature[18].

Investigations

Anemia was observed in 26 cases related to malnutrition which predisposes to tinea versicolor.[3] Hypercholesterolemia was observed in thirteen patients which suggests the possibility of increased cutaneous lipids promoting the growth of lipophilic yeasts[6].

As in general population in the study group the most common blood group was B group in 52 cases followed by O group in 38 cases, A group in 8 cases, AB group in 2 cases.

According to the literature, there is no increased incidence of tinea versicolor in patients with Acquired Immuno Deficiency Syndrome [51]. In this study also none of the patients were positive for ELISA for HIV.

Examination of the scales under 10% KOH showed tinea versicolor positive in all cases.

Culture

Culture was only positive for malassezia in 25 patients, M.globosa was the most common species isolated in this study(56%) followed by M. sympodialis(44%) correlated with the literature [3,12].

Biopsy

The presence of hyperkeratosis with basket weave stratum corneum, acanthosis vacuolation of the epidermal cells was the observations which were similar to other reports[38,39]. Increased pigment in the basal layer and its extension to the epidermis and pigment incontinence were similar to other studies[38,39]. However pigmentation in the basal layer in 5 achromic lesions enables one to consider whether the sun screening effect of scales could be a major factor involved in the hypopigmentation[23].

The presence of mononuclear cell infiltrate in the upper dermis shows that this innocuous agent is also capable of inducing a reaction in the host with its antigens. Dilated blood vessels were visualized in erythematous, chromic, achromic, and perfollicular lesions[36].

The observation of fungal elements in the stratum corneum is similar to other studies[40]. The presence of hyphae and spores in the hair follicle in 5 patients indicates the persistence of infection that contribute to the relapse and chronicity of the tinea versicolor infection[6].

CONCLUSION

1) The following conclusions were drawn from this study
2) More common among males
3) Mean age of occurrence 10 – 30 years.
4) Hypopigmentation and cosmetic problem are the main complaints.
5) Hypochromic macules and patches were the commonest presentation over the chest, neck, back, face, abdomen, axillae.
6) Disease was chronic with remission and relapses related to tropical climates.
7) Renal transplant, steroid therapy, diabetes mellitus, malnutrition associated with tinea versicolor.
8) Dermatophytosis, onychomycosis and candidal balanophosthitis were associated fungal infection.
9) Trichomycosis axillaris, wart and scabies were associated infective conditions.
10) Seborrheic dermatitis, Acne vulgaris, Becker's naevus, Melasma, Psoriasis, Vitiligo Pityriasis Rosea, Hand eczema, Keloid, Lichenoid eruptions were other associated conditions.
11) 10% KOH mount, culture, Biopsy were useful procedures to confirm the diagnosis of tinea versicolor.

REFERENCES

1) Gupta AK, Batra R, Bluhm R, et al: skin diseases associated with malassezia species. J Am Acad Dermatol 2004 Nov; 51 (5) 785 – 98 [Medline]

2) Topley Wilson Medical mycology; volume III, IVth Edition

3) Rook's Textbook of Dermatology, volume II, VIIth Edition Blackwell Published Co., Chapter 31, 2004: 5-15

4) Mark A, crowe MD: Tinea versicolor, American Academy of dermatology Jan 2007

5) Janaki.C, Senthamil Selvi .G, Janaki.V.R. Medical mycology colour atlas

6) Okuda C, Ito.M, Naka.W, et al: Pityriasis versicolar with a unique clinical appearance. Med Mycol 1998 Oct; 36(5):331-4 [Medline]

7) IADVL text book of dermatology volume I, IIIrd Edition

8) Fitzpatric's Dermatology in Gerneal Medicine, Volume II, VIIth edition

9) Gupta AK, Ryder JE, Nicol.K, Cooper.EA: Superficial fungal infections: an update on pityriasis versicolor, seborrheic dermatitis, tinea capitis, and onychomycosis. Clin Dermatol 2003 Sep – Oct; 21(5): 417-25 [Mediline].

10) Anderson –Text book of dermatology volume, IVth edition

11) Gordon MA, Kobayashi GS,Yeast infection, In: Dermatology in General Medicine, Editors, Fitzpatric TB, Elsen AZ, Wolff.K et al 4th edition Mc Graw - Hill New York 1993; 2462 – 2465

12) Moschella- Text of dermatology volume I, IVth Edition

13) Robert & Rich Clinical Immunology,Volume III, IVth Edition

14) Bailey & Scotts Diagnostic microbiology volume I IIIrd edition

15) Principle and practices of infections diseases volume III Edition VI

16) Malcolm D. Richard'sson, David W. Wornock; Fungal infections Diagnosis and management.

17) Dermatologic Therapeutics Tata Mc-Graw Hill Edition 2006

18) Johan Willard Rippon P.H.D;.Medical mycology

19) The carriage of M. furfur serovar ABC in patients with pityriasis versicolor, seborrheic Dermatitis and Atopic dermatic.

20) Nakabayashi A, Sei Y, Guillot J: Identification of Malassezia species isolated from patients with seborrheic dermatitis, atopic dermatitis, pityriasis versicolor and normal subjects. Med Mycol 2000 Oct: 38 (5): 337 – 4 [Mediline]

21) Peter J Sunenshine MD; Review Tinea versicolor IJD 1998 (37)

22) Eduardo Silva Lizame MD; IJD Tinea versicolor review 1995 Sep.

23) Janaki C, Senthamil Selvi G, Janaki V.R, Boopalraj JM: Unusual observation on the Histology of Pityriasis versicolor. Micropathologya 1997; 139 (2): 71- 4 [Medline]

24) Panikar and Ananda Subramaniam; Medical microbiology – Volume I, VIIth Edition

25) Dr. Jagadeesan; Medical microbiology volume I Edition IV

26) Crowson AN, Magro CM: Atrophying tinea versicolor: a clinical and histological study of 12 patients. Int J Dermatol 2003 Dec; 42 (12): 928 – 32 [Medline]

27) Di Silverio A, zeccara C, Serra F, et al: Pityriasis versicolor in a newborn. Mycoses 1995 May – Jun; 38 (5-6): 227-8 [Medline}

28) Gupta AK, Einarson TR, Summerbell RC, Shear NH: An overview of topical antifungal therapy in dermatomycoses. A North American perspective. Drugs 1998 May; 55 (5): 645-74 [Mediline]

29) Karakas M, Durdu M, Memisoglu HR: Oral fluconazole in the treatment of tinea versicolor J Dermatol 2005 Jan; 32 (1): 19 – 21 [Mediline].

30) Kaur I, Handa S, Kumar B: Tinea versicolor: involvement of unusual sites [letter]. Int J. Dermatol 1996 Aug; 35(8): 604 – 5 [Mediline]

31) Rajashekhar J. Lava R; Mycological study of tinea versicolor,

32) Panja G, The malassezia of the skin, their cultivation, morphology and species, Trans Far Eastern Assoc Trop Med, 1972;2:442 – 456.

33) Emmons CW, Binford CH, Utz JP et al. Pityriasis versicolor, in: Medical Mycology 3rd Edition Febiger, Philadelphia 1977;174 – 180.

34) Herbert A, Tinea versicolor in: Dermatology Clinics, Vol -2 / No.1. Editor Smith B.E, WB. Saunders, Philadelphia 1984;29 – 43.

35) Rao Gathe S, Kuruvilla Maria, Kumar Pramod, Vinod Vimala – Dept. of skin and STD Kasthuriba Medical college, Mangalore, Karnataka – 575 001, India. Clinico - epidemiological studies on Tinea versicolor

36) Kamalam A, Observation on the Gamut/Galaxy of the fungal infection in Tamil Nadu State, Kavaka 1984; 12:15 – 36.

37) Yesudian P, Kamalam S, Razack A. Confluent and recticulated papillomatosis (Gougerot Carteaud) an obnormal host response to Malassezia furfur. Acta Dermato vener (Stockhom) 1973; 53: 381 – 384.

38) Lever WE, Lever GS. Fungal diseases, Chapter -19, In: Histopathology of the Skin, VIIth edition. Philadelphia J.B Lippincott Company 1989; 364 –393

39) Graham JH, Superficial fungal infection Chapter 7 In: Dermalpathology Graham JH, Johnson, WC & Helwig, EB, eds., 1st Edn. Harper and Row publishers 1972; 137 – 253.

40) Van Abbe NJ, The investigation of Dandruff J, Soc cosmetic chem.15. 1964

41) Acton HW, Panja G, 1927, Seborrhoeic dermatitis or pityriasis capitis: a lesion caused by malassezia ovale, Indian Med. Gaz, 62: 603 – 41.

42) Barfatani M, Munn RJ, Schjeide OA, 1964, An ultrastructure study of Pityrosporum orbiculare, J Invest. Dermatol, 43: 231 – 3.

43) Belkum A van, Boekhout T, Bosboom R, 1994, Monitoring spread of Malassezia infections in a neonatal Intensive care unit by PCR-mediated genetic typing, J Clin Microbiology, 32: 2528 – 32.

44) Castellani A, Chalmers AJ, 1913, Manual of Tropical Medicine 2nd edn, Bailliere, Tindall and Cox, London, 1747.

45) Cunningham AC, Leeming JP et al., 1990, Differentiation of three serovars of Malassezia furfur, J Appl Bacteriol 68: 439 – 46.

46) Simmons RB, Gueho E. A new species of Malassezia. Mycol Res 1990; 94: 1146 – 9.

47) Smith JMB, 1968, The association of yeasts with chronic otitis externa in the dog, Aust Vet J, 44: 413 – 15.

48) Terragni L, Lasagni A et al. 1991, Pityriasis versicolor in the pediatric age, Pediatr Dermatol, 8: 9 – 12.

49) Elmets CA. Management of common superficial fungal infections in patients with AIDS. J Am Acad Dermatol 1994; 31: 560 – 563.

50) Hafez M et Shamy S, Genetic susceptibility in pityrasis versicolor J med. Mycol 1985.

51) Mathes BM., Douglas MC., seborrheic dermatitis in patients with acquired immuno deficiency syndrome J Am Acad Dermatol 1985 13; 947 - 51

KEY TO MASTER CHART

Sex

M - Male

F - Female

Marital status

M - Married

N - Unmarried

Predisposing Factor

SCLE - Sub acute cutaneous lupus erythematosos

Associated Dermatological Condition

T - Tinea

Sebderm - Seborrheic Dermatitis

Dermatological Exam

Colour

↓P - Hypo pigmented

↑P - Hyper pigmented

TC - Total Count

DC - Differential Count

Culture

G - Globosa

S Sympodialis

PROFORMA

Sl.No.

Name : OP No :

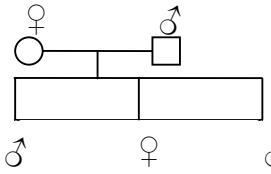
Age : Sex : M / F

Chief complaint :

Duration :

Past history : Previous episodes Single / Multiple

Family history :



♂ ♀ ♂ → Children

Martial history :

Predisposing factors : Pregnancy

Diabetes

TB

Immunosuppressive therapy

HIV

Transplant patients

Malnutrition

Endocrine diseases

Associated
dermatological disorders : Acne

Seborrheic Dermatitis

Psoriasis

Dermatophyte

Candidiasis

Acanthosis nigricans

Other dermatological Conditions

General examination : Anaemia

Obesity wt

Avitaminosis

Diet – nutritional status

Systemic : CVS

RS

Abdomen

CNS

Others

Dermatological examination

Site of involvement : Scalp / face / neck / shoulder/ front of chest / back of chest / abdomen / arm / fore arm / thigh / legs / nails / others / palm / soles / oral mucosa / hair / oral mucosa / genitalia

Percentage of surface area

Type of the lesion : Achromic / chromic / perifollicular / plaque / papular / erythematous / dry type / pityrosporum folliculitis / others

Investigations

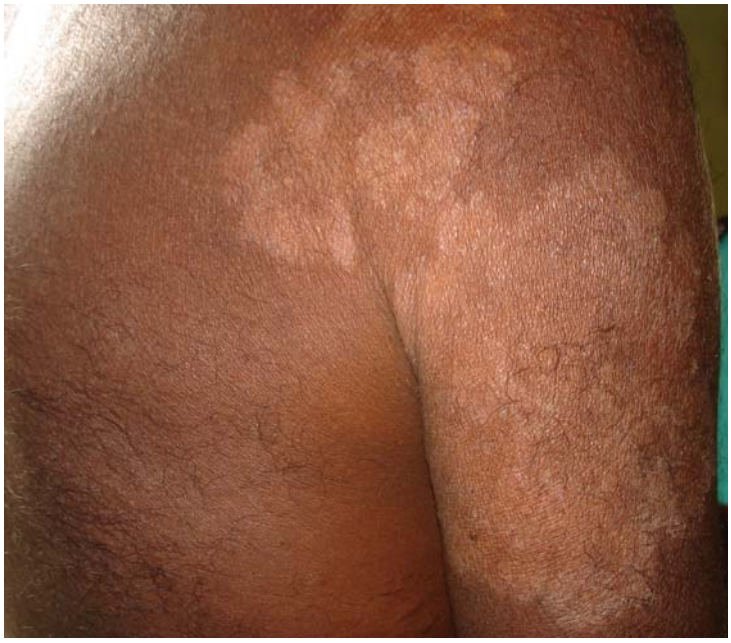
1. Skin scales – 10% KOH / Parkers Blue Black
2. Culture
3. Biopsy H&E, Special stains PAS
4. Blood grouping & typing
5. Blood VDRL
6. HIV ELISA
7. Blood sugar
8. Serum lipid profile
9. Others – Endocrine assessment

Treatment : Topical

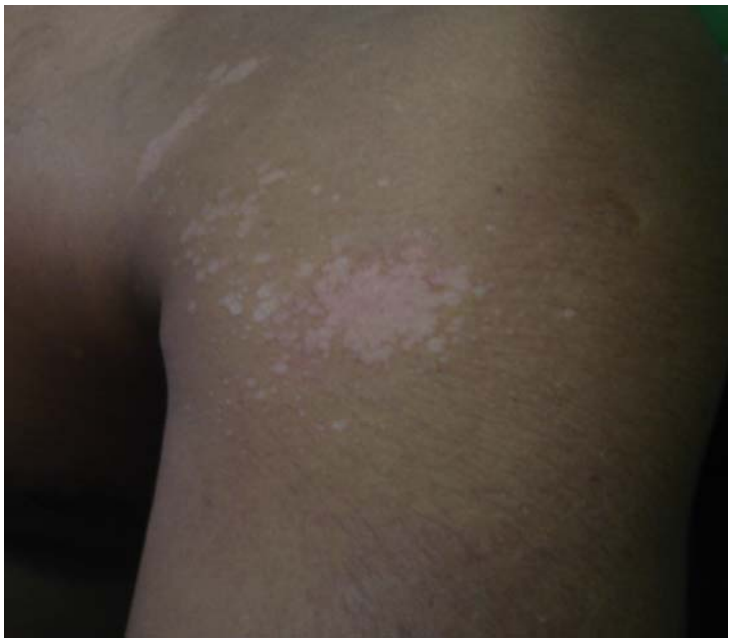
Systemic

Follow up

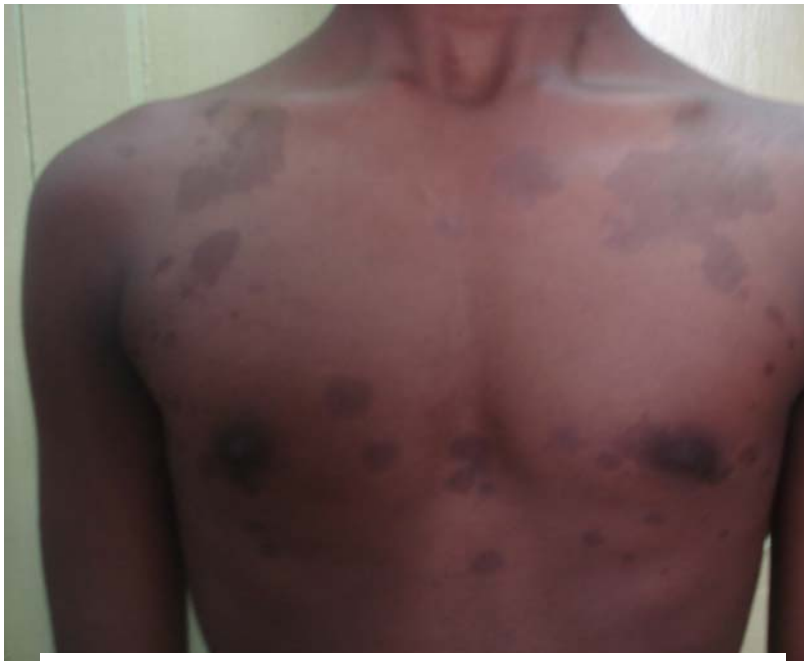
CLINICAL PICTURES



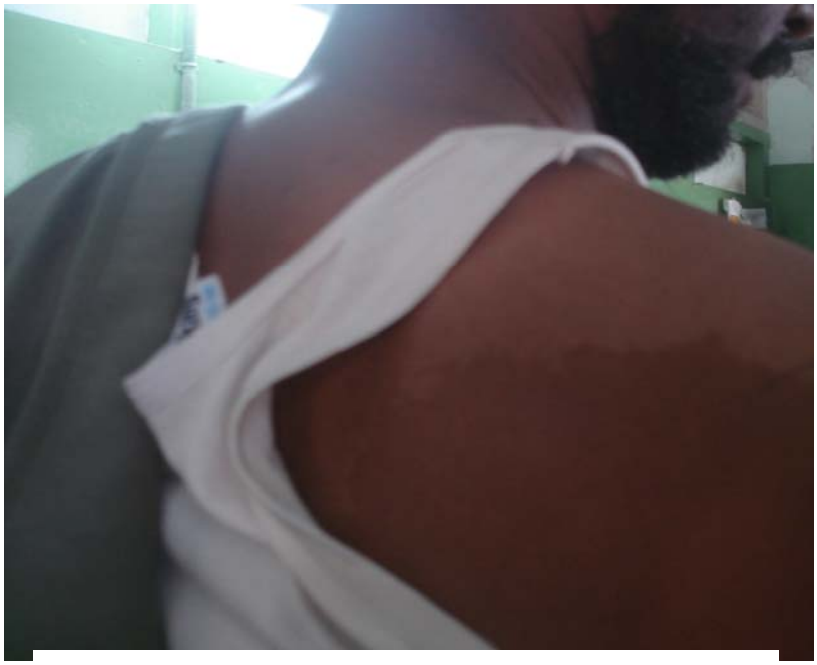
PLAQUE TYPE LESION RIGHT ARM AND RIGHT SHOULDER



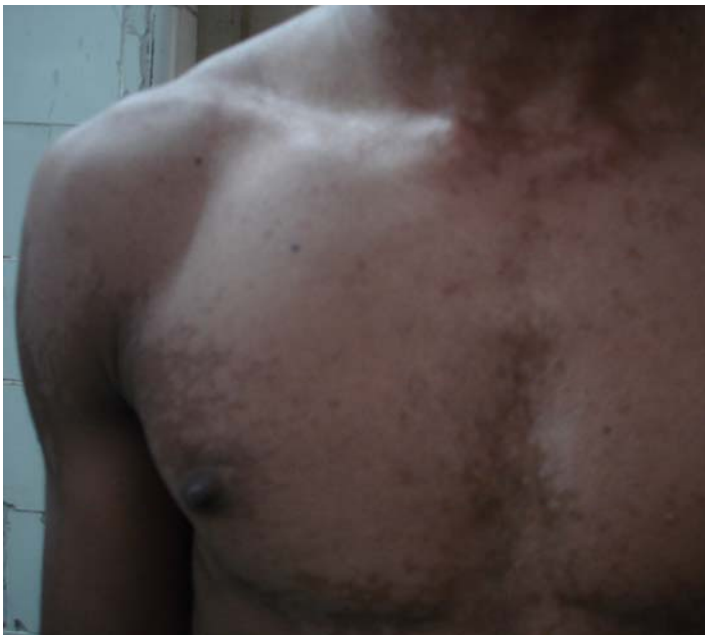
PERIFOLLICULAR AND ERYTHEMATOUS TYPE LEFT ARM



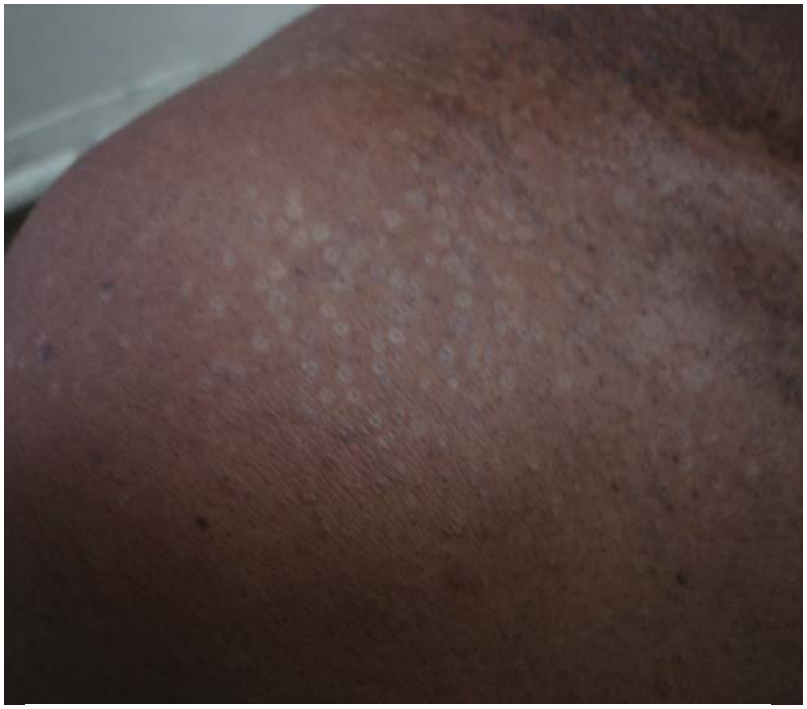
CHROMIC TYPE INVOLVOLVING CHEST AND ABDOMEN



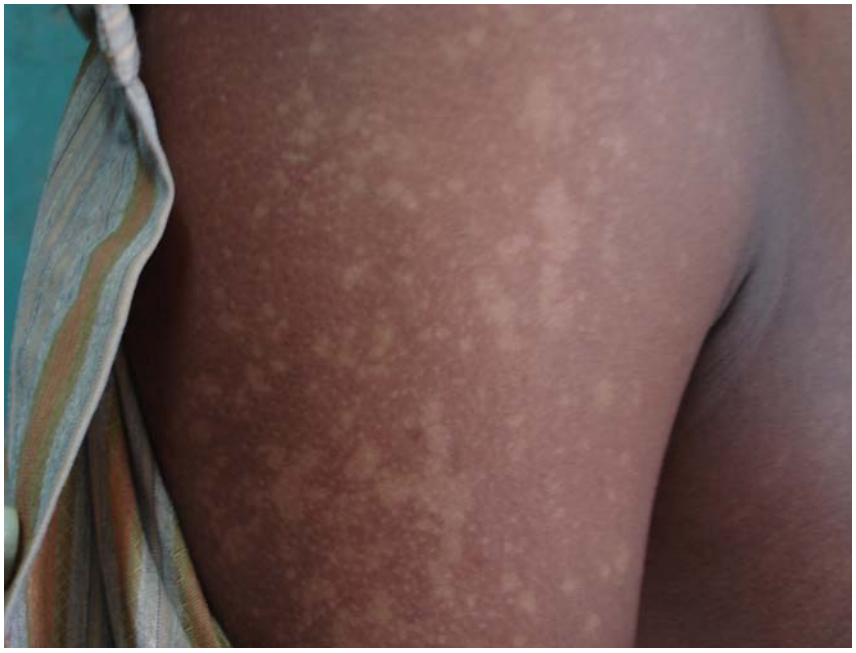
ACHROMIC PLAQUE TYPE INVOLVING RIGHT SHOULDER



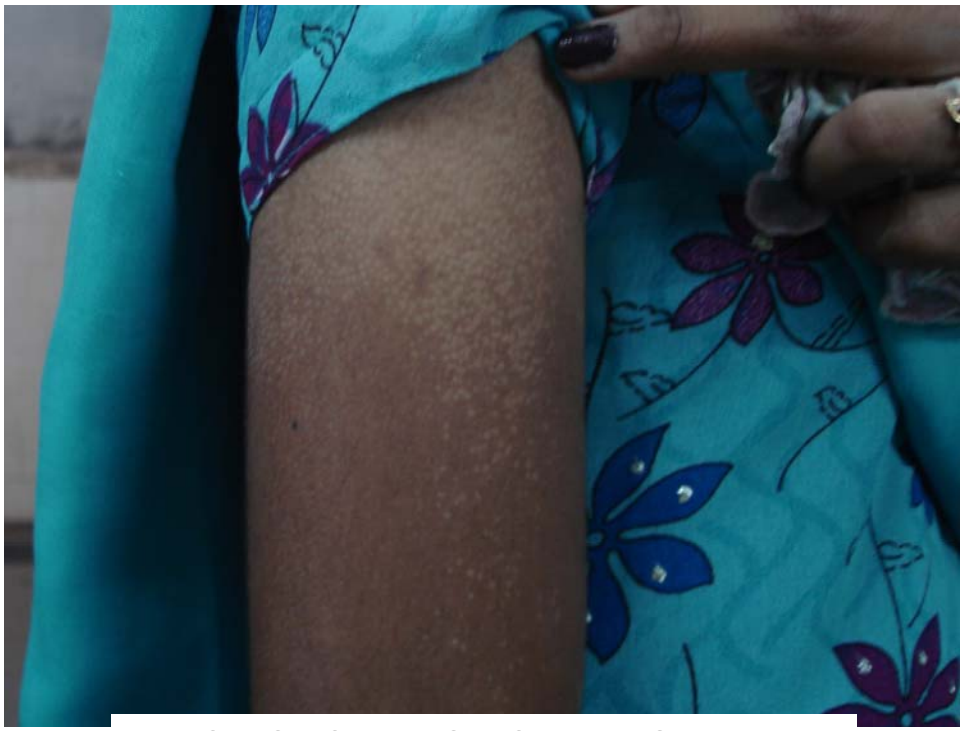
ACHROMIC PLAQUE TYPE INVOLVING ENTIRE CHEST



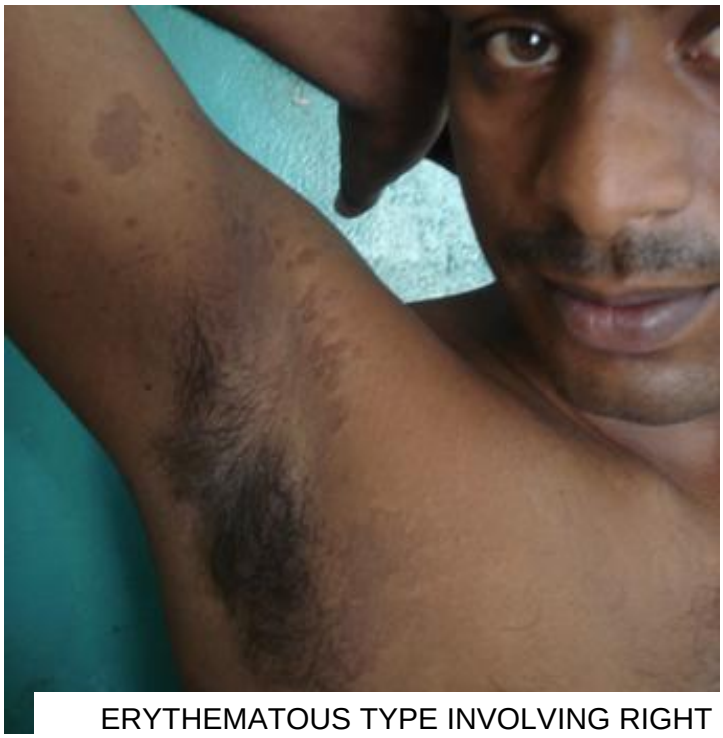
ACHROMIC AND CHROMIC PERI FOLLICULAR – RIGHT SHOULDER



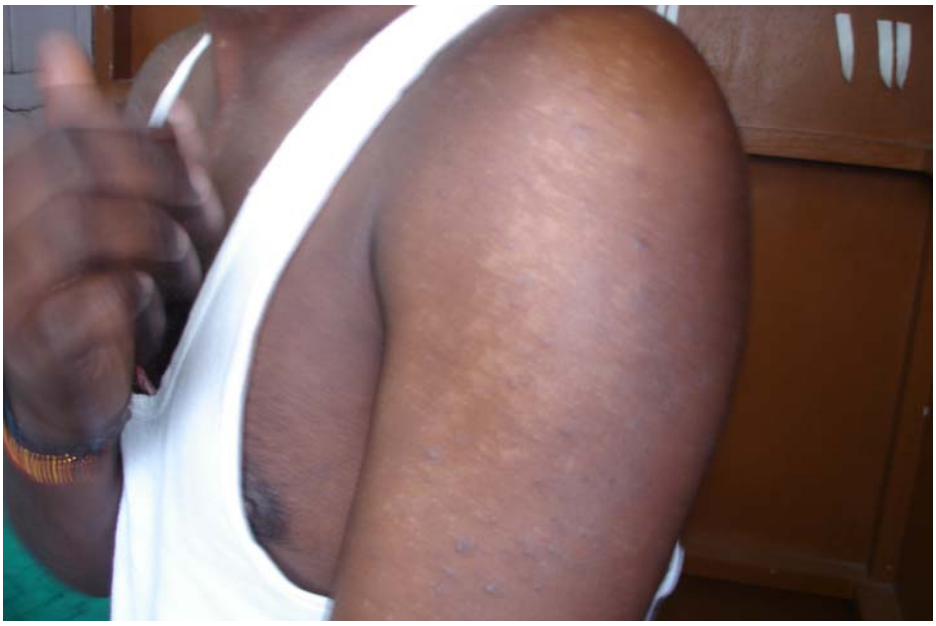
ACHROMIC AND PERI FOLLICULAR PATCH
RIGHT ARM



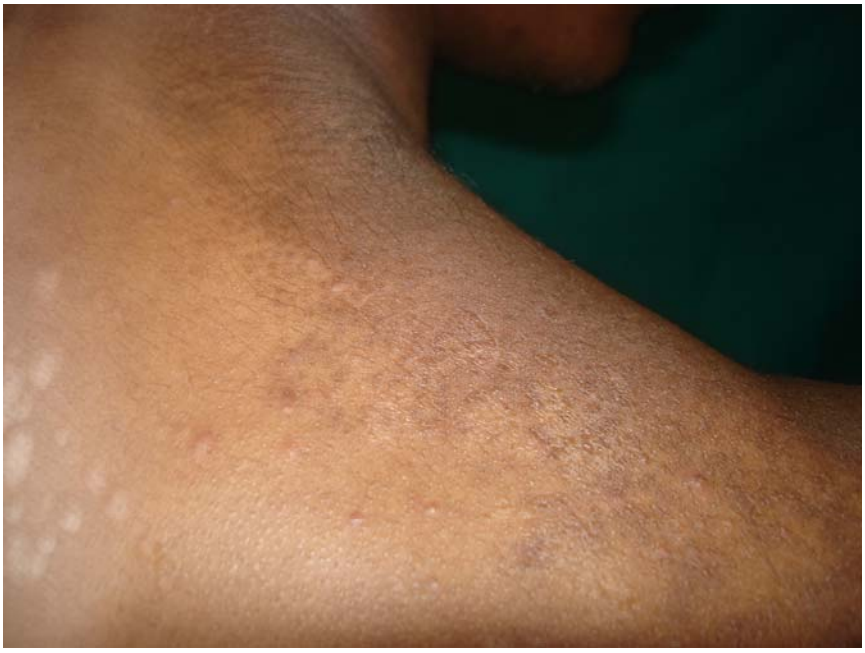
ACHROMIC PERIFOLLICULAR RIGHT ARM



ERYTHEMATOUS TYPE INVOLVING RIGHT AXILLA



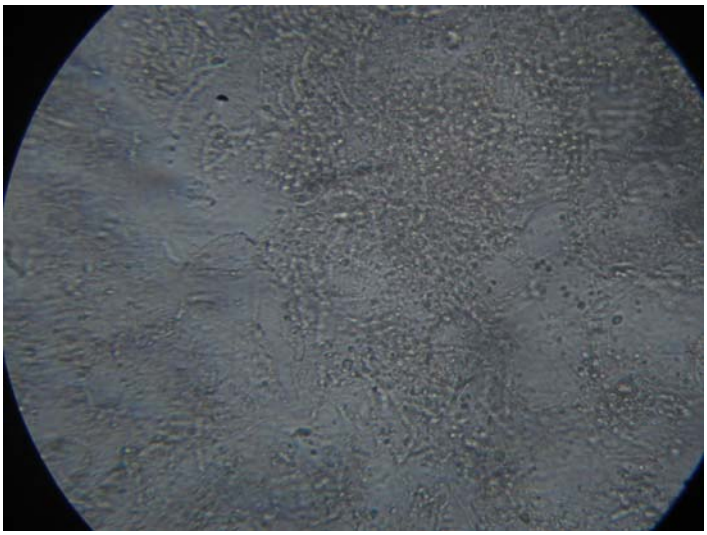
TINEA VERSICOLOR WITH ACNE VULGARIS LEFT ARM



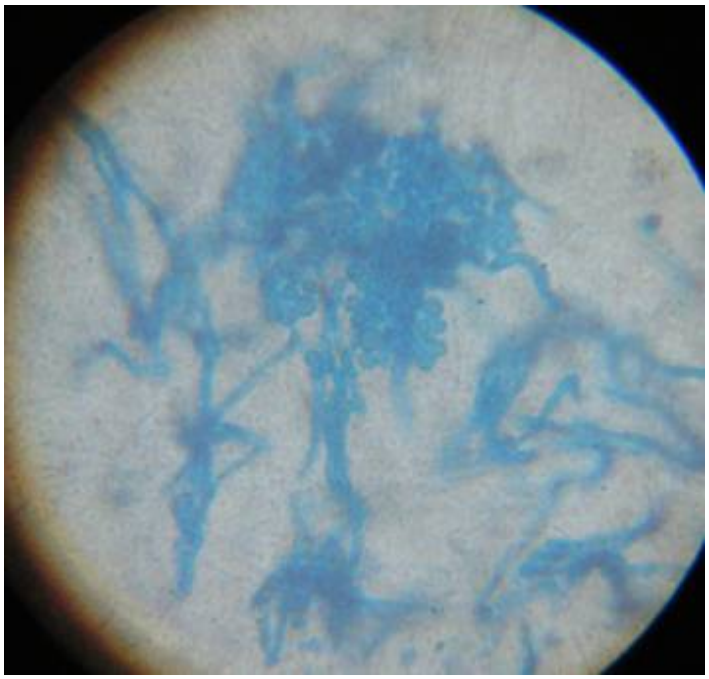
TINEA VERSICOLOR AND BECKER'S NAEVUS
RIGHT SHOULDER

ACHROMIC TYPE WITH FACIAL AND NASO
LABIAL INVOLVEMENT

MICROSCOPY



POTASSIUM HYDROXIDE MOUNT OF THE TINEA VERSICOLOR SCALES SHOWING THE HYPHAE AND SPORES



PARKER'S BLUE BLACK INK PREPARATION SHOWING HYPHAE AND SPORES

CULTURE

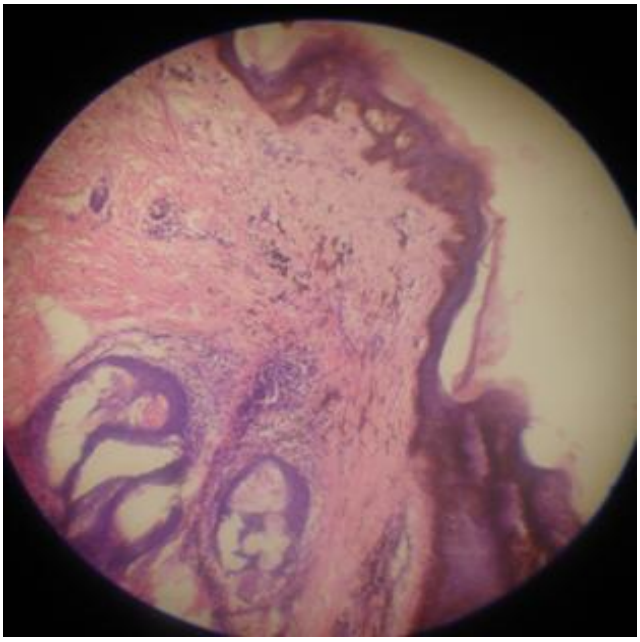
ON SDP AGAR WITH CHLORAMPHENICOL, ACTIDIONE TWEEN 80 WITH OLIVE OIL – CREAMY WHITE COLONIES

COLONIES ON THE REVERSE

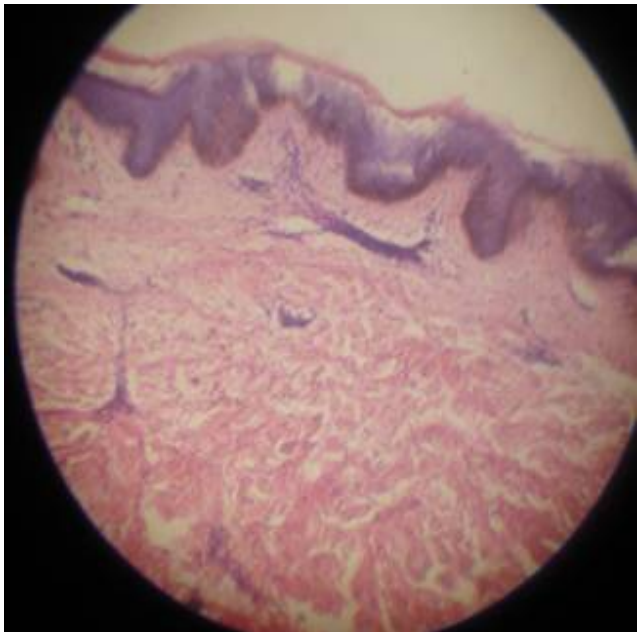
TWEEN ASSIMILATION TEST FOR
M. SYMPODIALIS

TWEEN ASSIMILATION TEST FOR M.GLOBOSA

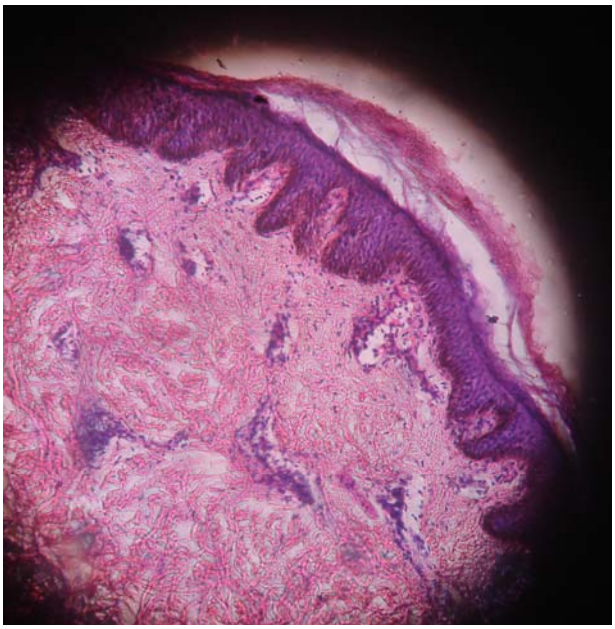
HISTOPATHOLOGY



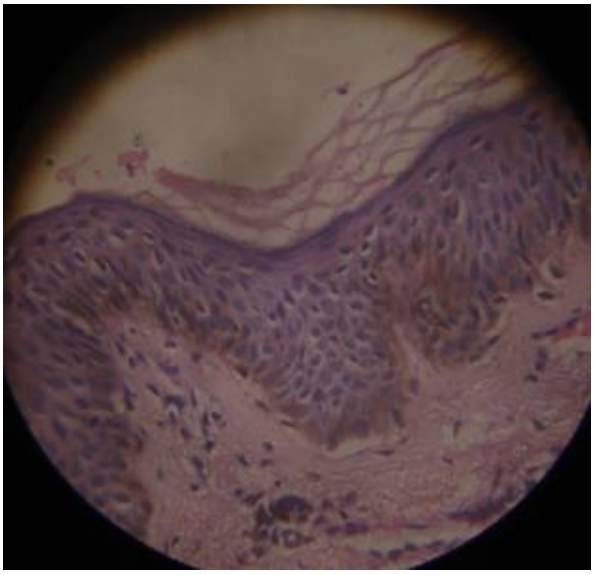
H AND E SECTION SHOWING INCREASE PIGMENT BASAL LAYER PIGMENT INCONTIENCE AND INFLAMMATORY INFILTRATE AROUND THE APPENDAGES



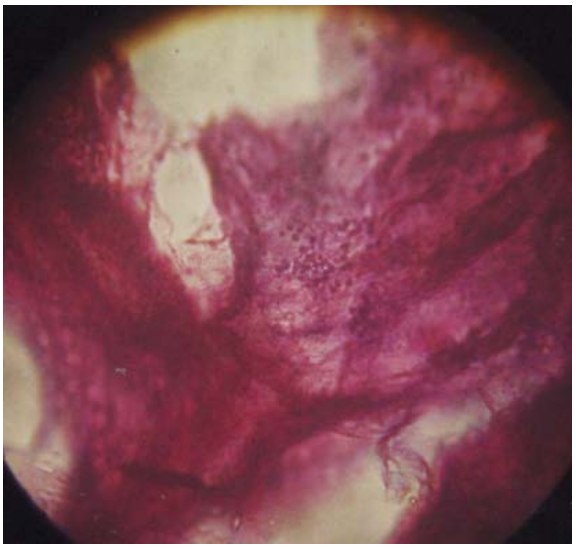
H AND E SECTION SHOWING EPIDERMAL SPONGIOSIS



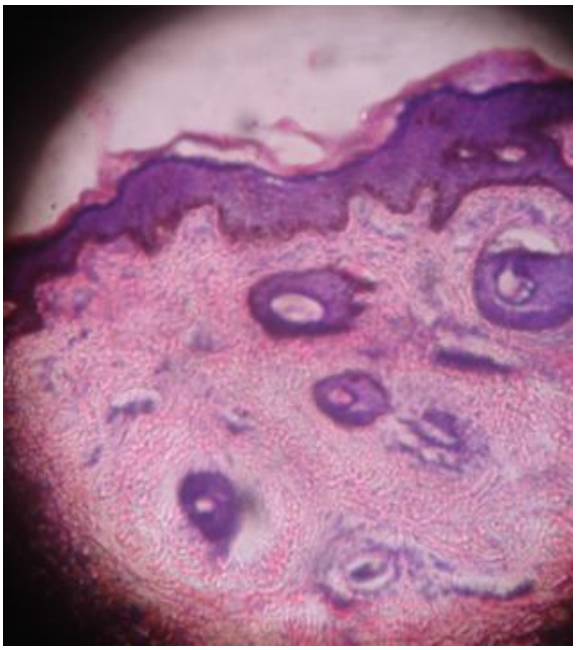
H AND E SECTRION SHOWING PSORIASIFORM ACANTHOSIS AND INFLAMMATORY INFILTRATE IN THE UPPER DERMIS



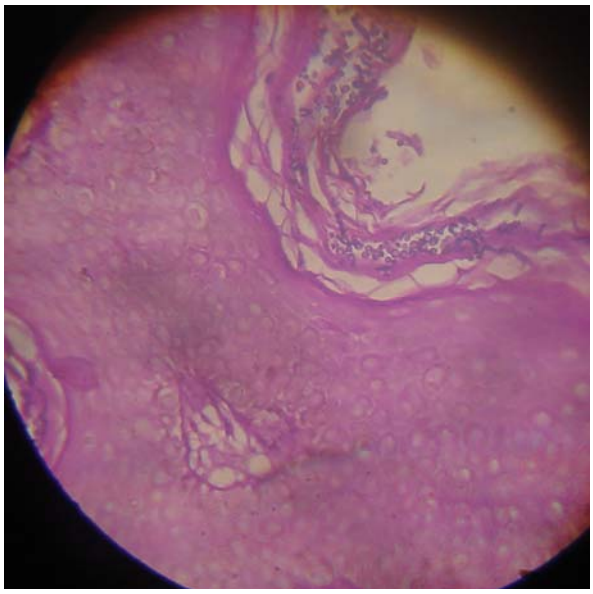
H AND E SECTRION SHOWING BASKET WAEVE STRATUM CORNEUM IRREGULAR ACANTHOSIS INCREASE D PIGMENT BASAL LAYER



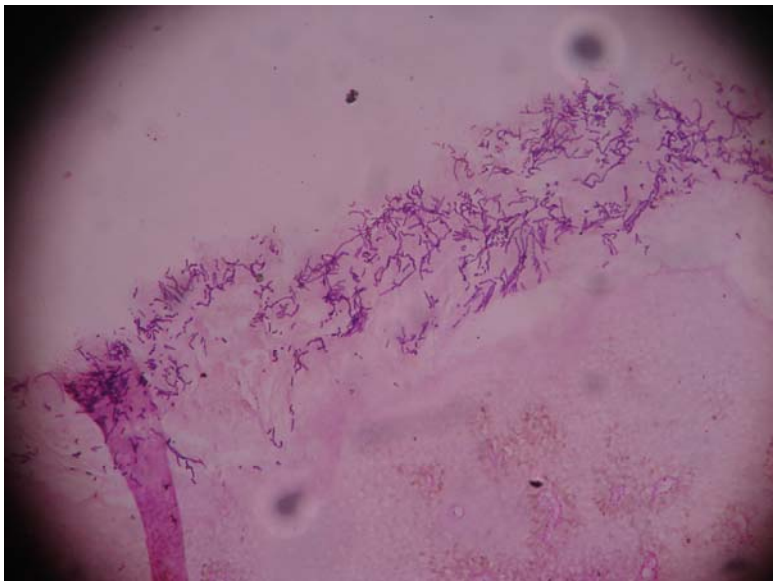
H AND E SECTRION SHOWING SPORES IN THE STRATUM CORNEUM



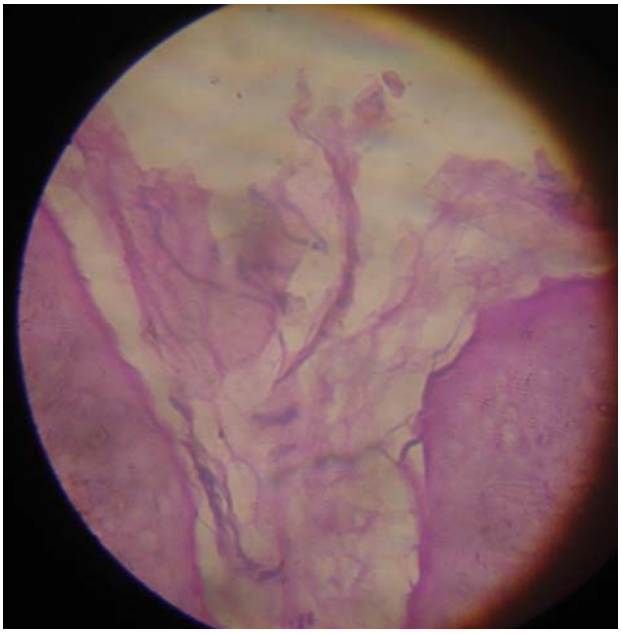
H AND E SECTION SHOWING DILATED BLOOD VESSELS INFLAMMATORY INFILTRATE IN THE UPPER DERMIS



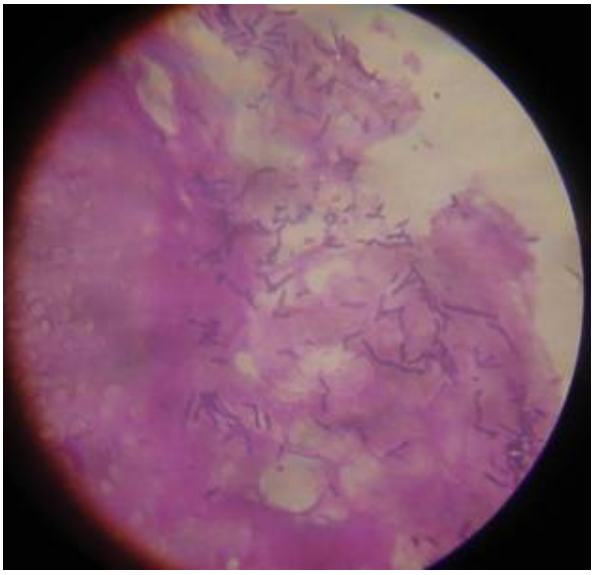
PAS – STAIN SHOWING – HYPERKERATOSIS, BASKET WAEVE STRATUM CORNEUM KERATOTIC PLUGGING VACULOATED CELLS IN THE EPIDERMIS SPORES AND MYCELIA IN THE KERATOTIC PLUG



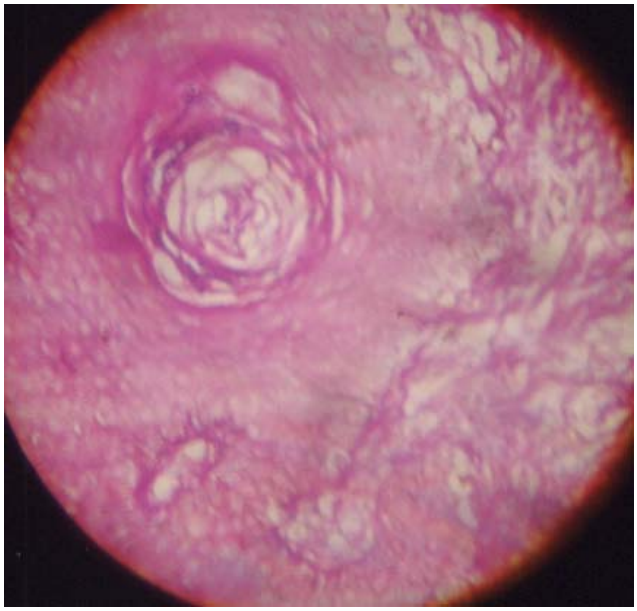
PAS – STAIN SHOWING – HYPHAE AND SPORES IN THE STRATUM CORNEUM



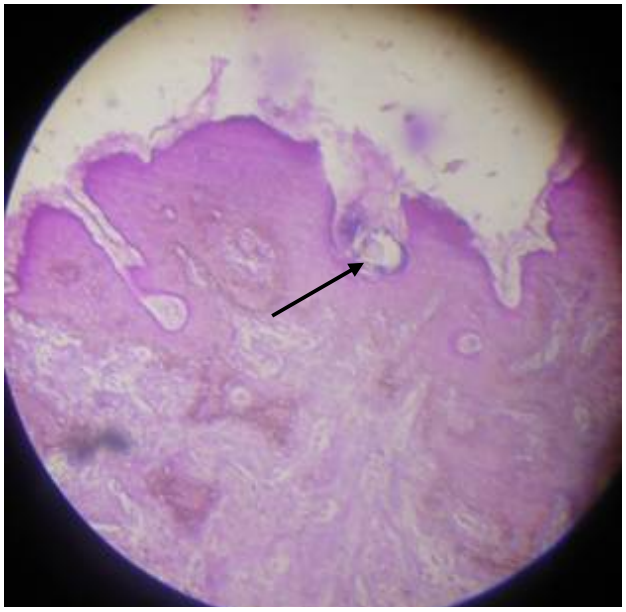
PAS – STAIN SHOWING – HYPHAE AND SPORES IN THE KERATOTIC PLUG



PAS – STAIN SHOWING – HYPHAE AND SPORES IN THE STRATUM CORNEUM



PAS – STAIN SHOWING THE HAIR FOLLICLE WITH HYPHAE AND SPORES.



PAS – STAIN SHOWING THE ABSENCE OF GRANULAR LAYER IN THE VICINITY OF SPORES