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Fungal Infections in Children - Superficial Mycoses

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SUMMARY

Superficial fungal infections in childhood are a frequent presentation in the paediatric out patient clinic. Three groups of fungi are generally pathogenic in man. In the dermatophytes, children suffer mostly from scalp infections i.e. tinea capitis, which if not treated can lead to permanent alopecia. In the second group, tinea versicolor is a spreading infection of the trunk resulting in macules of various shapes and sizes. Candidiasis in the third group constitutes by far the largest group of infections in children from the neonatal age to adolescence. The spectrum varies from the rare chronic mucocutaneous candidiasis to diaper candidiasis, one of the most common fungal infections in toddlers. This article, deals with the superficial infections in children, their epidemiology, clinical features, diagnostic tests and treatment modalities, with special reference to drug regimens and laboratory tests in Pakistan.

INTRODUCTION

Bacteria and viruses constitute a large majority of pathogenic organisms that invade the human tissue. Fungi constitute a smaller group that are less common, however, no less pathogenic and invasive than bacteria.

Fungi constitute a large group of eukaryotic organisms which depend for their nutrients on previously elaborated organic materials on which they live as saprophytes or parasites. They show considerable diversity in size and morphology. Only some 180 of the very large family of fungi are capable of causing disease in man. The disease caused by fungi can be divided into 3 main groups;

A) Superficial mycoses affecting the keratinous tissues of the skin, hair, and nail.
B) Subcutaneous mycoses which involve the skin, subcutaneous tissue and bone.
C) Systemic mycoses usually initiating in the lung and sometimes becoming widely disseminated.

In children, fungi from all 3 groups are pathogenic. In superficial fungi, Dermatophytic infection, Tinea versicolor and candida infections are 3 major pathogenic groups. Trichophyton, epidermophyton and microsporum are the 3 genera of dermatophytes that infect man. Tinea versicolor is caused by Malassezia furfur and candidiasis by C.albicans. Subcutaneous mycotic infection in children is caused by C. albicans presenting as chronic mucocutaneous candidiasis. Systemic mycoses include histoplasmosis, aspergillosis, blastomycosis and mucormycosis amongst others.

Various fungal dermatological presentations will be discussed in this article with an emphasis on updated therapy.

A) SUPERFICIAL MYCOSES

I. DERMATOPHYTOSES

This group of fungal infection commonly reffered to as ring worm or Tinea infection includes cutaneous infection caused by 3 genera of fungi, Trichophyton, Epidermophyton and Microsporum[10]. These fungi may be arthropophilic (person to person contact e.g. T. Tonsuram) zoophilic (animal contact e.g. M Canis) or geophilic (soil contact e.g. M. gypseum).

1. Tinea Capitis

Tinea capitis is the most common cutaneous

fungal infection in children. Incidence in children is universally high in all parts of the world. Data from Pakistan suggests >90% infection in children <15 yrs of age[30,31], co-relating with data from the U.S[66]. Prevelance is the same in both sexes in Pakistan[30,31]. In the U.S. greater than 90% of the infections are caused by T. tonsurans[7,38,49,83], whilst in Pakistan studies conducted show greater than 82% infections by T. volacieum[30,49]. Microsporum canis also causes tinea capitis but in the very young[10]. These two predominant organisms show a geographic distribution that generally reimains constant but may vary with time[52,62,67].

FUNGI CAUSING INFECTION IN CHILDREN

A. Superficial Mycoses	**Infection Causes**
I. Dermatophytes	
a) Epidermophyton sp	Tinea capitis
b) Trichophyton sp	Tinea corporis
c) Microsporum sp	Tinea unguium
	Tinea Pedis
II. Tinea Versicolor	
a) Malassezia furfur	Multiple macules, various shapes and sizes over trunk.
III. Candidiasis	
a) Candida Albicans	Oral candidiasis
Diaper candidiasis	
Angular chielitis	
Systemic candidiasis	
Congenital candidiasis	
B. Subcutaneous Mycoses	
I. Chronic. Mucocutaneous Candidiasis	
a) Candida Albicans	Chronic candidal lesions present on the skin and the mucus membranes.
C. Systemic Mycoses	
1. H. Capsulatum	Histoplasmosis
2. C. Immitis	Coccidioidomycosis
3. A. flavus	Aspergillosis
4. C. Neoformans	Cryptococcus
5. S. Schenki	Sporotrichiosis

Clinical Features: Tinea capitis can generally be of 2 types; Inflammatory or Non-inflammatory. Non inflammatory is characterized by the presence of broken-off hair, 1-3 mm above the scalp and partial alopecia. The infected areas are round to oval. Individual patches generally measure 1-6 cm in diameter. Multiple patches are common[77]. T. tonsurans[66] traditionally has been known to cause such an infection. M. audouini causes a single circumscribed non inflammatory patch in children, though rarely seen[66].

T. tonsurans produces inflammatory tinea capitis characterized by plaques of pustular dermatitis, suppuration, edematous nodules i.e. kerion formation[66]. Onset is acute and the lesion usually remains localized to one spot[77].

Rasmussen and Ahmed noted[65] that 16 of the 52 patients with proven T. tonsurans infection of the scalp had an inflammatory reaction i.e. kerion, to this infection. 15 of the 16 children had a positive skin test to trichophyton antigen indicating that the inflammation may be related to the development of cell mediated immunity against the organism.

Elaine Saltiel[76] described in January 1989 patchy alopecia in a young girl with symptoms suggestive of Discoid Lupus Erytheomatosis. This pattern of symptomatology, though rare, is seen in the follicular invasive phase in tinea capitis, where hairs are broken at the surface giving a characteristic black dot appearance.

T. tonsurans appears to exist in as much as 4% of the patients in an asymptomatic state as well[82]. This may be explained by recent evidence that suggests that the chemical appearance of tinea capitis may be determined by the patient's immunological response to the fungus[65].

Diagnosis: Wood's light examination: Tinea capitis due to infection with microsporum species may be diagnosed by observing a yellow green flourescence of thickened scalp hairs using wood's light examination. Trichophyton infections have a negative result in wood's light. Postassium Hydroxide examination may be done on broken hairs to reveal presence of spores on the shaft or inside the hair.

Culture should be performed routinely in all children with suspected tinea capitis. Specimen may be obtained by swabbing the infected area with a damp cotton tipped applicator. This produces a high yield of positive cultures specially in children[5,35].

Differential Diagnosis: Non inflammatory tinea capitis may be confused principally with sebborhoeic dermatitis, alopecia areata, trichotillomania, and traction alopecia.
Inflammatory tinea capitis may be misdiagnosed as impetigo, follicultitis or dissecting cellulitis.

Treatment: Systemic therapy with griseofulvin 12-20mg/kg for 6 weeks to 3 months is the treatment of choice[66]. For children unable to swallow tablets, griseofulvin suspension may be recommended or the grisactin capsules can be pulled apart and the powder dispensed in milk or other suitable vehicles[77].
Selenium sulphide lotion used as shampoo appears to be sporocidal and when prescribed in conjunction with griseofulvin, this results in earlier negative cultures from lesions of tinea capitis[37]. Because griseofulvin kills the hyphae of the dermatophytes, but not the spores, it has been postulated that the addition of twice weekly shampoo with selenuim sulphide may lessen the chances of spreading infectious spores to other suseptible individuals.
Recently in a clinical pilot study on the efficacy and safety of oral Terbinafine, the first oral allylamine, it was shown that terbinafine is effective and safe in the treatment of dry non-inflammatory Tinea capitis[32]. This is reported to be due to its superior penetration into the epidermis and the high concentration of the drug in sebum and hair due to its lipophilicity[88].
Haroon and colleagues[31] have shown similar successful therapeutic results with terbinafine in treating children with tinea capitis.
Several recent studies by Robert and colleagues[87] and Gan V.M et al.[22] have compared the efficacy of griseofulvin with ketoconazole in the treatment of tinea capitis in paediatric patients. In each of these studies ketoconazole was found to be effective and safe; although it was not found to be more efficacious than griseofulvin therapy.
Griseofulvin is recognised as relatively safe and effective in children provided that it is given to individuals without contraindications or sensitivity to the drug and provided that it is given in proper dosage[87,88]. Kerion is best treated with glucocorticoids to reduce inflammation[92]. Intra lesional steroids can be injected in lesions <3 cm in diameter. For larger areas of scalp involvements, prednisolone 1-2mg/kg/day for 3 weeks may be required.

2. Tinea Corporis:
Superficial tinea infection of the non-hairy skin is termed tinea corporis. M. canis and T. rubrum are the dermatophytes most commonly responsible for tinea corporis[92].
Although the infection may involve people of all ages, the disorder is seen most commonly in children, or in those with systemic diseases such as diabetes, leukemia, or other debilitating illnesses. William J Barson [6] reported one such case where a patient with Systemic Lupus Erythematosis or immuno suppressive therapy developed tinea corporis with M. Cannis.
Contact with domestic animals, particularly young kittens and puppies is a common cause of affliction in young children.

Clinical Features: Clinically tinea corporis usually occurs as pruritic annular plaques, with advancing scaling borders and evidence of central clearing. Lesions may occur any where on the body. Occasionally in the immunosuppressed patient, the lesions may be wide spread, forming confluent plaques.
The presenting picture of tinea that results from the application of a topical steriod preparation to an underlying tinea corporis is called tinea incognito[40]. This alters the host immunologic response to the fungus resulting in large multicentric ring lesions with an active peripherally advancing border.
Diagnosis is confirmed by Potassium Hydroxide examination of skin scrapings from the border of the lesions. Culture may be required if Potassium Hydroxide examination is negative.
Differential Diagnosis: Any from of dermatitis particularly nummular eczema, may be confused with tinea corporis as may herald patch of pityriasis rosea and some form of psoriasis.

Treatment: Topical therapy is the treatment of choice for dermatophytic infection limited to the skin in children. Clotrimazole, Miconazole haloprogin and tolnaftate are still all efficacious against most dermatophytic species. They are applied as a cream or solution daily twice to the

entire lesion and an area of approximately 1 cm outside the lesion for 2-3 weeks or until clearing occurs.

E.N Macasaet[53] from Manila also showed in a preliminary study topical 1% solution of griseofulvin to be effective against tinea corporis with no adverse effects suffered by any of the 53 patients.

Recent American studies show terbinafine, an allylamine, as efficacious and well tolerated as griseofulvin for oral therapy of tinea corporis[13,89].

3. Tinea Ungum:

Onychomycosis caused by infection with a dermatophyte is rare before puberty though occasional cases have been reported in infants[18]. This has been attributed to faster linear nail growth with subsequent elimination of the fungus[48].

Clincal Features: This condition presents as thickening and yellowing of the distal nail plate which may progress to involve the entire nail plate[96]. One or several nails may be involved, toe nails more frequently than finger nails.

The gold standard of diagnosing onychomycosis is Potassium Hydroxide examination and culture[19]. A variety of other techniques have been introduced to decrease false negative results.

Scher and Ackerman suggested nail unit biopsy for identifying fungal hyphae in a patient suspected with Onychomycosis[78]. Jillson[13] and Puper, and Sagher[72] recommended histopathological evaluation of the nail plate. And as recent as 1991, Sylvia and David[86] re-emphasized histological evaluation as an adjunct to routine mycological analysis, to pick false negatives that filter through Potassium Hydroxide examination or have a negative culture.

Treatment: May be very difficult. Prolonged treatment with griseofulvin for upto 9-12 months has shown low success rate and a high rate of recurrence[17]. Avulsion of the nail plate either surgically or non-surgically along with systemic griseofulvin[29], ketoconazole or topical antifungal therapy[69] may result in a better cure rate.

In a review published by Kortig[17], high cure rates have been shown in a preliminary study with oral terbinafine for 12 months which cleared toe nail tinea in 15 of 17 patients[24,25].

4. Tinea Pedis:

The occurance of tinea pedis in childhood is often regarded as an infrequent disease in the paediatric and dermatologic literature[66]. A recent study by Kearse and Miller[45] did however document Tinea Pedis to be more common than previously reported. A dermatophyte was isolated in nearly 50% of the children evaluated for foot dermatitis. Michelle[11] reports a case of bullous tinea pedis in a 2 year old child, further reinforcing upcoming paediatric tinea pedis. As recent as 1992 Anne and Bernard[51] in their study again showed tinea pedis not to be a rare occurence.

Recent literature suggests functional abnormality of the stratum corneum as one of the local factors of the host affecting trichophyton infection, since trichophyton is parasitic only in the stratum corneum[81].

Clinical Features: The most common form of tinea pedis is intertriginous dermatitis characterized by peeling, maceration and fissuring of the lateral toe web spaces; "the toe web variant". The condition often spreads to involve the undersurface of the feet, "the moccassin variant". Fissures may develope leading to secondary bacterial infection particularly gram-ve organisms [54]. "the vesicular variant".

The infection is diagnosed by Postassium Hydroxide examination and fungal culture. Various studies have quoted fungal culture to be a definitive adjunct to wet mount examination to decrease the false negatives[60].

Treatment: In most cases, it consists of application of topical antifungal agents including clotrimazole econazole twice daily, until symptoms have cleared completely and a negative wet mount examination is obtained. Aluminuin chloride 30% is helpful in treating pruritis, maceration and malodour[51]. Terbinafine has also shown to be a safe and efficacious drug for treating tinea pedis[79].

SUPERFICIAL MYCOSES

ANTIFUNGAL AGENTS

Drugs	*Indication*	*Route*	*Paed dosage*	*Comment*
Amphotericin B	Systemic candidiasis	I/V	0.25-1 mg/kg qd over 2-4 hrs; may given 0.5-1 mg/kg qod after initial daily therapy	Test dose of 1 mg is given first, then gradually increase to 0.5-1 mg/kg daily (nephrotoxic.)
Clotrimazole	Oral candidiasis Topical dermatophytic infection	PO	10 mg tab 5x/day	
Econazole	Cutaneous candidiasis tinea, cruris, tinea pedis	Topical	Twice daily application	
Griseofulvin	Dermatophytic Infection (all)	PO	15 mg/kg/day	
Ketoconazole	Mucocutaneous Candida, thrush dermatophytic infection	PO	5-10 mg/kg/day q 12-24 hrs	Potential hepatotoxic
Nystatin	Oral, cutaneous candidiasis	Topical PO	200,000-600,000 u/dose qid.	

II. TINEA VERSICOLOR

Tinea versicolor is an extremely common superficial fungal disorder of the skin usually seen in the ages 15-30 years, and having a predilection for sebaceous glands[68]. It has also been found in senior citizens[5], prepubertal children and at times even in infants[57,85,94].

Clinical Features

It is characterized by multiple macules of various sizes and shapes which may vary in colour from white to brown hence the name versicolor. This infection most frequently involves the upper trunk, neck and shoulders, but it can extend over the abdomen lower back and proximal extremities.

Controversy exists about why hypopigmented lesions result. One theory states that the organism itself may inhibit melanin formation via a shielding effect[55]. Excessive perspiration and warm humid weather seem to be more favourable for proliferation of tinea versicolor. This may be related to the decreased epithelial cell turnover, induced by these factors[68].

Diagnosis is confirmed by scraping of active lesions of tinea versicolor. Scrapings show numerous short curved hyphae and circular spores on Potassium Hydroxide examination.

Differential Diagnosis

The lesion may be confused with vitiligo, tinea corporis and post-inflammatory hypopigmentation.

Treatment

In mild cases, application of tropical antifungal creams and lotions such as clotrimazole twice daily is usually sufficient. With widespread involvement selenium sulphide 2.5% lotion applied daily for 7 days and then once a week for the next 4 weeks may be more efficacious[73]. Earlier concern about systemic absorption of selemium sulphide have not been substantiated[15,74]. The patient should be advised that this is a benign, occasionally pruritic disorder and that the hypopigmentation will not return to normal until sun exposure allows re-melanization of the affected areas.

III. CANDIDIASIS

Candidiasis is an acute or chronic infection of the skin, mucus membranes and occasionally the internal organs caused by yeast-like fungi of the candida genus. Candida albicans is the most frequent cause of this disorder[77] in humans. It exists in the microflora of the oral cavity, the gastro intestinal tract, vagina and becomes a cutaneous pathogen when an alteration in the host defenses

allowes the organism to become invasive.

Newborns are physiologically suseptible to candidal infections which may be manifested as oral moniliasis or less commonly as a localized or generalized dermatitis. Candidiasis in the infant is invariably traceable to the mother who may be a vaginal or intestinal carrier[77,36].

Since the advent of the antibiotic era, candida has become a significant neonatal pathogen. Incidence of all clinical forms of infection due to candida has risen abruptly[44]; and previously undocumented manifestations such as arthritis[1,46,63], endopthalmitis, endocarditis, meningitis and peritonitis have been reported.

1. Oral Candidiasis

In the newborn nursery, acute pseudomembraneous candidiasis is the most common form of candidal infection. Clinically scattered or confluent creamy white patches are detected on the buccal mucus membranes, palate, gums and tongue.

The pharynx is usually spared. The white patches which are almost pure colonies of candida are friable and when removed may leave an erythematous base.

Neonates initially acquire candida during passage throught the birth canal. Studies[36] have shown thrush to be 35 tines more common in infants of mothers with candidal vulvovaginitis, than in offsprings of non infected mothers. Candida was isolated from the vaginal tract of 25-50% of mothers whose infants developed oral candidiasis. The later in pregnancy the candidal infection occured the more likely it was to predispose the neonate to thrush[36].

In another study of 1442 mothers 18% had positive vaginal cultures of candida albicans in early labor. Oral cultures were positive in 20% of infants whose mothers had positive vaginal cultures and of these, 55% had clinical thrush.

Various factors have been studied in relation to oral thrush in neonates. It has still not become clear why C-Albicans is pathogenic in the mouths of neonates. Prematurity, antibiotics, air or droplet infection, and trauma all have failed to show any conclusive link to neonatal thrush[36].

Immunological factors may play a role. Transplacental antibodies have been suggested by many. Interestingly, it is seen in infants that serum transferrin is highly saturated with iron resulting in decreased iron binding capacity. Therefore more free iron is avaliable in infants to support the growth and proliferation of C. Albicans[11].

Presentation is generally the same in infants and children. It can be diagnosed by gently rubbing the affected area with a cotton applicator or tongue blade and identifying the organism in Potassium Hydroxide examination.

Treatment: In neonates, infants and children usually consists of nystatin suspension dropped directly on to the tongue four times per day for 2 weeks. In difficult cases, nystatin suppository can be inserted into the tip of a slit pacifier and the infant allowed to suck on one of these four times per day[36].

Clotrimazole troches can also be crushed in 2ml of nethylcellulose and applied with a soft brush every 4-6 hrs to the infants oral mucosa or to the thumb if he is a thumb sucker.

2. Diaper Candidiasis

The constant moisture and maceration in the diaper area causes candidiasis to be a frequent occurance in infants. It has been seen that candida has been recovered in upto 85% of patients if a diaper rash had been present for longer than 72 hrs[58,91]. Empirical treatment for candida should be instituted in such patients.

Clinical Features: Clinically a beefy red dermatitis is seen in the diaper area with involvement of the inguinal folds in contrast to the irritant dermatitis[3,58,59]. Scattered scaly satellite macules or pustules may also be seen. Diagnosis is made clinically and confirmed by Potassium Hydroxide examination.

Treatment: Consists of local application of clotrimazole or Nystatin four times per day. In addition certain general measures also help;

1) Change diapers frequently.
2) Discontinue use of occlusive plastic pants.
3) Leave diaper off for prolonged periods.

Occasionally low potency steriod creams may be used in adjunction with antifungals to speed the resolution of inflammation[91].

3. Angular Cheilitis

Also reffered to as candidial angular cheilitis, it is very common in children and adolescents who are undergoing orthodontic procedurres, and who wear braces for prolonged periods of time. This results in a change in the normal angle of the corners of the mouth with eventual maceration of the skin and subsequent colonization by C. Albicans. The diagnosis is confirmed by a positive Potassium Hydroxide scraping.

Treatment consist of application of topical anti fungal preparation as mentioned above.

4. Onychia and Paronychia

Previously candida had been implicated in nail desease as a primary invader in patients with chronic mucocutaneous candidiasis or secondary to paronychia[96] and rarely the primary cause of onychia. However recent studies[28] have shown candida to be a primary pathogen in onychomycosis or terminal nail plate erosion. In paronychias candida can often be recovered in young children.

Clinical Features: The clinical picture most commonly seen is a long standing history of finger sucking which leads to the break down of the epithelial barrier and allows invasion of pathogens often candida. There is usually a long history of red fingers with proximal and lateral nail fold swelling. Occasionally acute painful exacerabations will occur with more swelling and exudate being present. At this time the patient will usually give up sucking the painful finger and seek medical attention. Occasionally onycholysis may also be seen[26].

Treatment: Clotrimazole or niconazole solutions or 3% thymol in alcohol used twice daily for several weeks may be helpful in clearing the infection.

5. Systemic Candidiasis

It is defined as the isolation of candida form or its histopathologic demonstration in a normally sterile body site. More than one clinical form exists and each requires individualized therapy.

Pathogenesis: To understand better the risk factors associated with the development of neonatal systemic candidiasis, its pathogenesis needs to be reviewed. Long term use of antibiotics and concommitant over growth of candida has long been noted. Antibiotics do not enhance the virulence of candida, nor do they suppress the immune response of the host[18], rather their role is related to directly facilitating candidal over-growth. Seeling[80] stated that although C. Albicans under usual circumstances possesses little pathogenicity for humans, this increases in the preseence of factors that impair host defenses. Once having penetrated the deeper tissues and lymphatics, it disseminates. The ensuing course depends both on the ability of the host to resist, localize, or eradicate the infection and on the degree of invasion.

TABLE

Risk Factor	*Relative Risk*
1. Very low birth weight	+ + +
2. Prolonged antibiotic therapoy	+ + +
3. Prolonged I/V catheterization	+ +
4. Hyperalimentation	+ +
5. GI surgery	+ +
6. Malnutrition	+ +
7. Prolonged endotracheal intubation	+
8. Use of steriods	+

Taken from: Candida: An increasingly important pathogen in the nursery. Paediatric Clinics. North America Vol 35, No3; June 88.

Intubation[20,81], I/V catherization, parentral nutrition have also been identified as important risk factors. As many as 2-3% of VLBW infants develop systemic candidiasis according to recent literature[4,11], due to invasive catheters, immature immune systems or broad spectrum antibiotic[36]. The fungi have been recovered from the tips of these catheters. Curry et al.[16] speculated that antibiotic creams and ointments applied to catheter sites would have inhibited bacterial proliferation and enhanced fungal colonization. This association between prolonged I/V catherter use and developement of fungemia has been substantiated by numerous reports[3,74,90].

Most babies with systemic candidiasis are symptomatic. Each of the infants reported by

Baley and collegues[3] and Johnson and co-workers[11] were symptomatic.

Clinical Features: The disorder may be suspected in patients with intermittent, spiking, therapy-resistant fever with cutaneous or unusual candidal lesions or cellulitis, at the site of an I/V catheter.
Sites of involvement in neonatal systemic candidiasis include a high reported incidence of CNS involvement[11,3,81], renal[3,41] and rare osteoarthritis[46]. Endopthalmitis and its associated invasive candidiasis in neonates is well documented[11,3]. Endocarditis may also be occasionally seen[95].
Lab investigantions sent should include a complete blood cell count with differential count; blood culture, gram stain, and culture of skin lesion, biopsy of the skin lesion with specific staining and a urine examination and culture.

Treatment: It is directed towards removal of iatrogenic factors, treatment of underlying illness and specific antifungal therapy. Amphotericin B remains the cornerstone of specific treatment. Several investigators advocate its use as a routine procedure[3,11]. However Butler and his collegues[8] have shown that meningeal infection in neonates treated only with Amphotericin B had an outcome equal to or better than in patients given Amphotericin B and flucytosine.
Amphotericin B may be administered in dosages of 0.25 mg/kg to 1mg/kg I/V daily or 1.5mg/kg every other day. The toxicity of amphotericin B however requires caution in the use of this preperation. In such cases oral clotrimazole flucytosine or I/V miconazole may be given[70].

6. Congenital Candidiasis:
In this rare form of cutaneous candidiasis the newborn has evidence of candidal infection at the time of birth or shortly afterwards[56]. This is in contrast to the usual candidal infection seen in the newborn which appears late in the first week, usually as oral thrush or perianal dermatitis.
Cutaneous candidiasis in the congenital form is believed to be due to inutero infection and is thought to occur by the ascent of the candidal organism; through the genital tract, since transplacental passage of candida has not been demonstrated[11].

Clinical features: This condition can be suspected at birth by evidence of chorioamnionitis which can be found on gross examination of the placenta and umbilical cord. Typically an affected umbilical cord has 0.5 to 0.4 mm, sharply circumcribed yellow white papules that are rounded, flat topped and protrude above the surface slightly[36].
The clinical course of these infants is variable. Whyte proposed in a review of his 18 cases of congenital candidiasis 3 possible outcomes;
The first group involves very immature infants which may be still born or die very early in the neonatal period. These infants will show wide-spread pneumonia[56] and sometimes other surface infections.
The second group generally will show diffuse cutaneous infection, that is usually present at birth but occasionally develops later in the first week of life. Characteristically a sparse or confluent papular and vesiculopustular eruption involving mainly the trunk associated with diffuse erythema is seen[6,12,23,93]. Desquamation is present late in the course. The face and the oral mucus membranes are spared. The nails may be involved and are characterized by dystrophy of the nail plates[36].
The third group may show evidence of chorioamnionitis on examination of the placenta but will have no evidence of infection and will be asymptomatic.

Treatment: Treatment of these infants can be successfully accomplished with topical antifungal treatment with Nystatin, Miconazole or clotrimazole. In addition to topical treatment, some authors suggest using oral nystatin to lessen the chance of dissemination of GI candida in the absence of obvious oral GI lesions[66].
Hematogenous spread is not a feature of congenital candidiasis. When sampled, blood and urine cultures are sterile and at autopsy candida infection is limited to the lungs, GI and skin.
Thus congenital candidiasis differs from

neonatal systemic candidiasis by age at onset (birth vs > 1 week) and extent of organ involvement. In systemic candidiasis multiple organ involvement is common, skin manifestations are rare and the prognosis without treatemnt is poor.

B. SUBCUTANEOUS MYCOSES

I. CHRONIC MUCOCUTANEOUS CANDIDIASIS

Chronic mucocutaneous candidiasis is a progressive candidal infection that involves a defect in cell mediated immunity leading to widespread candidiasis.

It may begin as an oral candidiasis, candidal diaper dermatitis, candidal intertrigo or paronychia that is persistent and resistent to treatment and usual modes of therapy. The disorder persists and spreads to involve the scalp, eyelids, nose, hands and feet, and may be characterized by intertrigo satellite pustules and scaly patches. Dystrophy of the nails, scarring, and loss of hair are common sequelae. Systemic candidiasis is uncommon. Though rarely seen, chronic mucocutaneous candidiasis masquerading as candidal umbilical granuloma has been reported[2].

Although an autosomal recessive inheritance is suggested in some families, most cases are sporadic[10,42]. Also associated with this condition are endocrine abnormalities, Myasthenia gravis, Iron deficiency anemia, and hypogammaglobulinemia[39].

Treatment

Chronic mucocutaneous candidiasis is refractory to topical antifungal treatment. Systemic ketoconazole 200mg per day has been shown to eradicate nail, cutaneous and mucus membrane lesions[27].

In recent years there have been reports of successful treatment of chronic mucocutaneous candidiasis with the newer antifungal agents- Clotrimazole 60-100 mg/kg/day taken in 3 divided doses or I/V miconazole 30-60 mg/kg daily eight intervals may be given[50]. Because the drug induces hepatic microsomal enzymes, it is recommended that it be given for 2 week courses followed by a 2 week rest period[50].

Recently, Sandra[75] and her collegues reported a successful treatment of a case of chronic mucocutaneous candidiasis with one of the newer triazole antifungals, Itraconazole whose lipophilic property is thought to contribute to the reported persistent therapeutic post treatment levels[15,75] and hence better efficacy.

SUPERFICIAL MYCOSES

DIAGNOSTIC MODALITIES

Superficial Mycoses		Lab. Tests
Dermatophytes	Tinea capitis Tinea corporis Tinea unguium Tinea Pedis	KOH examination Woods light examination Culture
Tinea Versicolor	Hypo- or hyper-pigmented macules	KOH examination culture
Candidiasis	Oral candidiasis Angular cheilitis Diaper candidiasis	KOH examination culture Gram stain
	Systemic candidiasis	Culture secretions CT Scan of vicera Tracheal secretion culture

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