A RARE PRESENTATION OF CANDIDIASIS: CONGENITAL
CUTANEOUS CANDIDIASIS IN A NEONATE BORN TO AN
ASYMPTOMATIC MOTHER

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ABSTRACT
Neonatal candidiasis can either be acquired at birth by ascending infection through birth canal, or in
postnatal period via direct contact. Congenital cutaneous candidiasis (CCC) is a rare disorder that presents
within the first six days of life in neonates exposed in utero. It is usually seen in infants with maternal
candidal vulvovaginitis. We report a case of a full term neonate, presenting with generalized maculo
papular skin eruptions three days after birth, born to an asymptomatic mother. The manifestation of CCC
ranges from diffuse skin eruptions without any systemic symptoms to severe systemic manifestations.
Clinical features, direct microscopic examination of specimen, and appropriate cultures are useful in
differentiating the lesions from other more common dermatoses of the neonatal period. Although, this
disorder is usually benign and self-limiting in full term infants with normal birth weight, it can be life-
threatening in premature and low birth weight neonates.

Keywords: Congenital Cutaneous Candidiasis, Neonate

INTRODUCTION
Candida species are part of the lower genital tract flora in 20-50 % of healthy asymptomatic women
especially during pregnancy (Pradeep et al., 1998). Neonatal candidiasis can either be acquired at birth,
by ascending infection through birth canal, or in postnatal period via direct contact with nursing staff
(Lane, 1995). Neonates exposed to Candida species in-utero can present with Congenital Cutaneous
Candidiasis (CCC), a rare neonatal fungal infection, within six days of birth. It is usually seen in infants
with maternal vulvovaginal candidiasis (Darmstadt et al., 2000). Less than 100 cases of CCC have been
reported worldwide in the literature and a few from India (Torres-Alvarez et al., 2013; Srinivas
and Bhardwaj, 2014). We hereby report a case of CCC in a full term neonate born to an asymptomatic
mother without any other associated risk factors.

CASES
A full term female baby weighing 2.75 kg was born to a 30-year old third gravida mother by elective
lower segment caesarean section (LSCS) with an indication of previous caesarean. Mother did not have
any history of vaginal discharge or itching during pregnancy. Prior to onset of labor there was no per
vaginal bleed, or premature rupture of membrane (PROM). There was no history of intrauterine devices
being used in the past and per vaginal examination confirmed the same. Mother was seronegative for
Venereal Disease Research Laboratory (VDRL) test and Human Immunodeficiency Virus (HIV), there
were no rashes on her body neither she had any history of contact with chicken pox during perinatal
period. At birth baby had mild respiratory distress and kept in neonatal intensive care unit for monitoring.
Baby improved within few hours and a diagnosis of transient tachypnea was made. She was accepting
breast feed & was not on any medication. On 3rd day of life baby developed generalized, erythematous
macular eruptions over body especially on trunk, back and extremities, sparing face, palms and soles, few
lesions had fine scaling (figure 1). The eruption soon became confluent at some sites and progressed to
vesicles and pustules; no other systemic manifestations were observed. Skin scraping and vesicle fluid
were collected and subjected to direct (KOH) mount microscopic examination and Gram’s stain. Budding yeast cells with pseudohyphae were demonstrated. Sample was also inoculated into Blood agar (BA) and Saboraud’s dextrose agar (SDA) with antibiotics and incubated at 37º C and 25ºC, respectively. After 24 hrs of incubation white creamy pasty colonies were observed on BA and SDA. The isolate was identified as Candida albicans on the basis of gram’s staining, positive germ tube test, demonstration of chlamydospores on corn meal agar and sugar assimilation and fermentation tests. Blood and urine culture for bacteria and fungi obtained on admission to intensive care unit were negative. Total leucocyte counts and differential leucocyte counts were within normal limits. Mother was tested seronegative for Toxoplasma, Rubella, Cytomegalovirus, Herpes simplex (TORCH), test. A high vaginal swab collected from mother demonstrated the presence of C. albicans on culture. The diagnosis of CCC was made on the basis of clinical picture, positive KOH mount and culture of the skin lesions. Clotrimazole (1%) cream was applied on the lesions which resulted in a dramatic improvement and lesions healed with superficial desquamation of the involved skin after approximately four days, local ointment was discontinued and mother and child were discharged from the hospital.

DISCUSSION
Congenital candidiasis was first reported in 1960 (Sonnenschein et al., 1964). Prematurity and the presence of an intrauterine foreign body (for e.g intrauterine device, cervical sutures, tampons), PROM are reported to be associated with this condition, however cases have also been reported in healthy full term neonates, in the absence of associated risk factors (Pradeep et al., 1998; Darmstadt et al., 2000; Torres-Alvarez et al., 2013).

Exact pathogenesis of CCC is not known but many cases CCC have occurred in neonates born with clinically intact chorioamniotic membranes as observed in our case (Darmstadt et al., 2000). There is evidence that C. albicans can penetrate intact membranes. Once the membranes are penetrated, organisms are hypothesized to spread from the amniotic fluid to the skin and into the pulmonary and gastrointestinal tract, in case the fluid is aspirated or swallowed (Darmstadt et al., 2000; Jagtap et al., 2011). Acid proteinase secreted by C. albicans has keratolytic activity and is thought to facilitate initiation of cutaneous candidiasis and assist in invasion (Ray and Payne, 1990). Demonstration of pseudohyphae in direct skin sample also indicates towards the pathogenic potential of the C. albicans strain rather than a mere colonizer.

CCC can produce a spectrum of disease ranging from a diffuse skin eruption in the absence of systemic illness to severe systemic disease resulting in fetal demise or early neonatal death. However the most common presentation is an acute generalized eruption of erythematous macules, papules and or pustules with a benign outcome (Darmstadt et al., 2000). The risk of dissemination is increased if there is an evidence of respiratory distress or other signs of sepsis in immediate neonatal period, low birth weight (<1500 gm), administration of broad spectrum antibiotics, extensive instrumentation during delivery and a positive blood, urine, or cerebrospinal fluid culture (Johnson et al., 1981).

The differential diagnosis of maculopapular rashes in neonates are CCC, staphylococcal scalded skin syndrome, toxic shock syndrome, erythema toxicum, transient neonatal postural melanosis, drug eruptions, herpes simplex, infantile seborrhoeic dermatitis, atopic dermatitis, psoriasis, congenital syphilis, Listeria monocytogenes and varicella zoster virus infections (Table 1) and (Table 2) (Darmstadt et al., 2000; Jagtap et al., 2011). The vague clinical picture of CCC resembling other skin eruptive conditions, may lead to misdiagnosis of such cases. Although, this disorder is usually benign and self-limiting in full term infants with normal birth weight, it can be life-threatening in premature and low birth weight neonates (Johnson et al., 1981). So a high index of suspicion should be maintained by clinicians and clinical microbiologist, to prevent a benign treatable condition from taking a severe and protracted course, and avoid misdiagnosis of such cases.

Therefore, we highlight the importance of direct microscopic examination of a KOH wet mount and appropriate bacterial/fungal culture, of such skin lesions, to differentiate CCC from other causes of maculopapular eruptions, in neonates born to asymptomatic mothers.
**Case Report**

Table 1: Differential diagnosis of Congenital Cutaneous Candidiasis according to their clinical manifestations

<table>
<thead>
<tr>
<th>Diseases</th>
<th>Clinical Manifestations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Staphylococcal scalded skin syndrome</td>
<td>Skin tenderness, superficial blisters/bullas, positive Nikolsky sign.</td>
</tr>
<tr>
<td>Toxic shock syndrome</td>
<td>Skin tenderness, hypotension/shock</td>
</tr>
<tr>
<td>Erythema toxicum</td>
<td>Erythematous macules with overlying white or yellow papules or pustule, which resolves within 2 weeks, frequently individual lesions appear and disappear within minutes.</td>
</tr>
<tr>
<td>Transient neonatal pustular melanosis</td>
<td>Vesicles, superficial pustules, and pigmented macules are present at birth. They occur on the chin, neck, forehead, chest, buttocks, back, and, less often, on the palms and soles. The vesicles and pustules rupture easily and resolve within 48 hours. The brown macules may persist for several months.</td>
</tr>
<tr>
<td>Drug eruptions (ceftriaxone and vancomycin)</td>
<td>Generalised skin rashes.</td>
</tr>
<tr>
<td>Herpes simplex</td>
<td>Watery blisters on skin, oral mucosa and genitalia. Lesions heal with a scab formation.</td>
</tr>
<tr>
<td>Infantile seborrhoeic dermatitis</td>
<td>Cradle cap, accentuation in the skin folds of the neck, axillae, and nappy area.</td>
</tr>
<tr>
<td>Atopic dermatitis</td>
<td>Encrusted eczema on the scalp and face, generalised eczematous skin</td>
</tr>
<tr>
<td>Psoriasis</td>
<td>Erythematousquamous patches, can be pustular</td>
</tr>
<tr>
<td>Congenital syphilis</td>
<td>Maculopapular (coppery-brown) skin rash followed by desquamation, blistering and crusting, prominent on the palms and soles. Delays in development, seizures, fever, hepatosplenomegaly, anemia, jaundice and snuffles.</td>
</tr>
<tr>
<td>Listeria monocytogenes infection</td>
<td>Respiratory difficulty like cyanotic episodes, rapid breathing, and grunting.</td>
</tr>
<tr>
<td>Congenital Varicella</td>
<td>Skin lesions in dermatomal distribution, neurologic defects, eye diseases and skeletal anomalies</td>
</tr>
</tbody>
</table>
### Table 2: Diagnostic modalities used for differentiation of congenital cutaneous candidiasis from other causes of erythematous macules

<table>
<thead>
<tr>
<th>Diseases</th>
<th>Contributory evidence</th>
<th>Microbiological evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Staphylococcal scalded skin syndrome</td>
<td>History of preceding infection by <em>Staphylococcus aureus</em></td>
<td>Demonstration of toxin producing strain of <em>Staphylococcus aureus</em> from skin lesions.</td>
</tr>
<tr>
<td>Toxic shock syndrome</td>
<td>Concomitant maternal infection</td>
<td>From skin swab demonstration of toxin producing strains of <em>Staphylococcus aureus</em> or <em>Streptococcus pyogenes</em>.</td>
</tr>
<tr>
<td>Erythema toxicum</td>
<td>Fluid from lesions shows many eosinophils; High levels of circulating eosinophils</td>
<td>Nil</td>
</tr>
<tr>
<td>Transient neonatal pustular melanosis</td>
<td>Nil</td>
<td>Tzanck smear/gram’s stain from pustule reveals predominance of neutrophils, occasional eosinophils.</td>
</tr>
<tr>
<td>Drug eruptions (ceftriaxone and vancomycin)</td>
<td>History of administration of antibiotics</td>
<td>Nil</td>
</tr>
<tr>
<td>Herpes simplex</td>
<td>Nil</td>
<td>Positive direct fluorescent antibody test from skin scraping and fluid, Demonstration of multinucleated giant cells in Tzanck smears of skin lesions.</td>
</tr>
<tr>
<td>Infantile seborrhoeic dermatitis</td>
<td>Diagnosis mainly clinical</td>
<td>Nil</td>
</tr>
<tr>
<td>Atopic dermatitis</td>
<td>Family history of atopy, Raised IgE and eosinophilia</td>
<td>Nil</td>
</tr>
<tr>
<td>Psoriasis</td>
<td>Positive family history, Skin biopsy shows hyper and parakeratosis, microabscesses</td>
<td>Nil</td>
</tr>
<tr>
<td>Congenital syphilis</td>
<td>Nil</td>
<td>Skin lesions or nasal discharge (snuffles) examined for spirochetes by darkfield microscopy or by direct fluorescent antibody techniques.</td>
</tr>
<tr>
<td><em>Listeria monocytogenes</em> infection</td>
<td>Meconium-stained amniotic fluid</td>
<td>Isolation of bacteria from skin lesions, blood or cerebrospinal fluid culture.</td>
</tr>
<tr>
<td>Congenital Varicella</td>
<td>Maternal varicella infection</td>
<td><em>Varicella zoster</em> virus specific IgM detection, Molecular diagnosis and in situ hybridization.</td>
</tr>
</tbody>
</table>
Figure 1: Full-term neonate with congenital cutaneous candidiasis, presenting with generalized erythematous macules

REFERENCES