Research Paper Medical Science

Candida tropicalis colonization in a rare case of Acrodermatitis enteropathica

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ABSTRACT

Acrodermatitis enteropathica (AE) is a rare hereditary disorder caused by impaired absorption of zinc from the gastrointestinal tract. It is characterized by acral and periorificial dermatitis, alopecia, and diarrhea1. Here we report a case of Acrodermatitis enteropathica with superadded Candidiasis in a 4 months old male infant who presented with a typical inflammatory rash all over body, along with diarrhea and intermittent fever for last one month. His serum zinc level was low (52ug/dl) and direct KOH microscopy and growth on SDA from skin scraping material revealed Candida tropicalis after morphological and biochemical confirmation. Patient lesions started to heal with oral zinc therapy and application of local cotrimazole ointment within 5 days .Patient was asymptomatic on last follow-up.

KEYWORDS Acrodermatitis enteropathica, *Candida tropicalis*, Oral zinc, Oral Fluconazole

Introduction:

Acrodermatitis enteropathica is a rare genetic disorder characterized by diarrhea, hair loss and an inflammatory rash[1]. It is due to malabsorption of zinc through the intestinal cells but the precise cause is not known. It occurs in one of two forms: an inborn form and an acquired form [2]. The phenotypic triad of acral and periorificial dermatitis, alopecia and diarrhea is virtually pathognomonic cutaneous marker for zinc deficiency[3]. Lesions may become secondarily infected with *Staphylococcus aureus* or *Candida albicans*2.We report a case of Acrodermatitis enteropathica secondarily infected with *Candida tropicalis* in an infant presented with typical rash and diarrhea.

Case Report:

A four months old male infant of poor socioeconomic status from Malda district of West Bengal attended the Pediatrics OPD of a tertiary care hospital, Kolkata with the complaint of intermittent fever and swelling of the abdomen for the last two months. Skin was apparently normal at the time of presentation.

Baby born at term by normal delivery. Birth weight was 3kg. His developmental milestones were within normal limits. The child had been exclusively breast fed since birth. His immunization history was incomplete having received no vaccination since birth. Examination revealed a febrile, alert but irritable often inconsolable child weighing 4.6kg, length 60 cm, head circumference-40cm. Systemic examination revealed an enlarged liver and signs of malnourishment.

The day following admission the baby developed a generalized, pruritic, eczematous eruption especially on the extremities, buttock, and front of the trunk and periorificial areas. The skin rashes started as vesicles and then dried to form red patches (figure 1). Along with the rashes, he developed persistent diarrhea not responding to treatment. A few white plaques were seen on the tongue which was suggestive of *Candidiasis.*

Investigations including complete hemogram, renal function tests, urine and stool examination reports were normal. Serum albumin was 3.9gm % and serum globulin 4.2gm % with a reversal of the albumin: globulin ratio. Chest X-ray was normal. Ultrasonography of abdomen revealed moderate hepatomegaly. Alkaline phosphatase enzyme was 76 IU/L. Plasma zinc level was low 52ug/dL (60 – 120ug/dL).The baby was referred to us with differential diagnosis of candida dermatitis for microbiological investigation. Skin scrapings were taken from the periphery of the lesions and 10%potassium hydroxide preparation showed few budding yeast cells. Scraping material was inoculated on Sabouraud dextrose chloramphenicol agar (SDCA) and incubated at room temperature. On the third day, dry, white, flat and spreading colonies developed on SDCA (figure 2a). Germ tube test was negative. Blastoconidia in small groups along with pseudohypae developed on corn meal agar but chlamydospores were not observed (Figure 2b). The yeast isolate was identified with Hichrome differenciation agar(figure2c) and API 32C yeast identification kit (BioMerieux). Depending on the morphology, cultural and biochemical characteristics the isolate was identified as *Candida tropicalis*. On the basis of clinico-investigational findings, we arrived at a diagnosis of Acrodermatitis enteropathica with superadded *Candida tropicalis* infection. Antifungal susceptibility test was done according to CLSI guidelines which was sensitive to fluconazole and amphotericin B. The patient was treated with oral zinc sulphate supplements 2mg/kg/day, vitamin supplements, topical cotrimazole ointment and oral fluconazole 50 mg daily for one week.

There was an overall dramatic response to treatment in 7 days. The child became active and playful by the second day .The skin lesions started healing by the 5th day and the patient was discharged after 1 week on oral zinc supplement, and local cotrimazole ointment. On follow up after one month, the patient was asymptomatic and repeated culture showed no growth.

Discussion

Acrodermatitis enteropathica (AE) is a well recognized entity caused by an inherited defect in zinc absorption leading to hypozincemia [3]. Zinc has been recognized as a constituent of the human body. As it forms an integral part of carbonic anhydrase and many other highly purified enzymes, its importance in protein and carbohydrate metabolism is understandable[2]. Zinc deficiency is characterized by a triad of dermatitis, diarrhea, and alopecia. However, only 20% of patients present

with all three components at a given time[3]. In our case, the baby had an inflammatory dermatitis and chronic diarrhea, but significant alopecia was absent.

Zinc is an essential component of the diet and features of Acrodermatitis enteropathica start appearing after birth in infants bottle fed with bovine milk and in the first few months of life, on weaning from breast milk to formula or cereal; these have lower zinc bioavailability than breast milk[3] But our baby was four months old and according to the mother, he was on exclusive breastfeed.

Acrodermatitis enteropathica needs to be differentiated from other dermatological causes of anogenital rash such as diaper dermatitis, candidiasis, seborrheic dermatitis, psoriasis. Certain other disorders such as essential fatty acid, carboxylase and amino acid deficiencies may have similar cutaneous features and need to be excluded especially if the lesions persist despite zinc supplementation or relapse frequently[4].

Superadded colonization with some bacteria and fungi are also common. Among the fungi *Candida albicans* are most frequently isolated[2]. Due to the growing immunocompromised populations either due to certain primary diseases or modern aggressive therapies there has been a sharp increase in the incidence of infections due to yeasts[5].*Candida* species are recognized as the seventh most common nosocomial fungal infectious pathogen among the yeasts[5]. Over the past decade the species associated with infection has changed from *Candida albicans* to non- *C. albicans*; more infections with *C. tropicalis*, *C. krusei, C parapsilosis* and other *Candida* species is increasingly being reported[5].

Risk factors known to predispose *Candida* infections are parenteral nutrition, corticosteroids or immunosuppressive agents, drug abuse, extensive surgery, use of broad spectrum antibiotics and compromised host state, trauma of the skin and intravascular catheters.[6] This patient was suffering from grade II malnutrition together with Acrodermatitis enteropathica, a traumatized skin disease which predisposed to the *Candida* infection. In our case of Acrodermatitis enteropathica there was superadded infection with *Candida tropicalis* which was susceptible to fluconazole and amphotericin B. Our patient was put on both zinc supplement, oral fluconazole along with topical cotrimazole ointment and the disease was abated without any sequelae. Hence we have to deal such condition very cautiously as it requires early recognition and treatment to prevent long term morbidity and mortality.

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Legends to figures
Figure 1: Skin lesions in Acrodermatitis enteropathica.

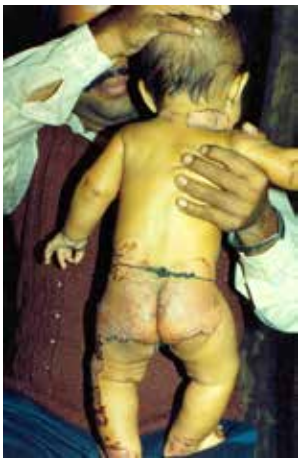
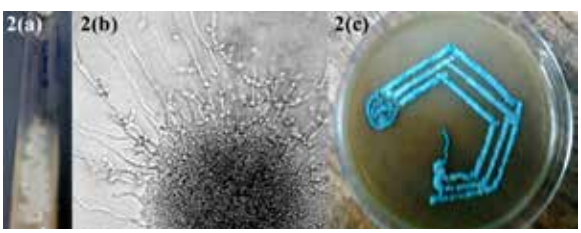


Figure 2: (a) Candida colonies on Sabouraud's Dextrose Agar, (b) morphological features of *Candida tropicalis* on CornMeal Agar, and (c) Deep blue colour *Candida tropicalis* colonies on Hichrome Differentiation Agar.



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