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Fusarial Sinusitis from Immunocompromised Child: A Rare Case Report from New Delhi, India

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Abstract

Fusarium causes a broad spectrum of human diseases which can be superficial, locally invasive or disseminated. In immuno-competent host, it causes allergic sinusitis or chronic non-invasive sinusitis while in immuno-compromised patients it causes invasive sinusitis. In this paper, the authors present a case of a 15-year-old boy who was admitted in the pediatric ward of PGIMER Dr. RML hospital, New Delhi, India with history of spontaneous erosion of dorsum of nose. Local examination showed complete erosion of dorsum of nose. The nasal debris was then sent to the Microbiology department for fungal culture and microscopy. On microscopic examination, branched septate hyphae were seen. The culture on Sabouraud's Dextrose Agar (SDA) showed cottony white growth initially after 1 week of

incubation and this growth later showed grayish pigmentation. The lactophenol cotton blue (LPCB) mount of this growth showed septate hyphae along with typical hyaline, multi-septate, curved sickle shaped macroconidia. A tentative diagnosis of fungal sinusitis by Fusarium Spp. was done in this case.

Key words: Fusarium, Sinusitis, Hyphae.

Introduction

Fusarium spp. is a common soil saprophyte and an important plant pathogen that causes various diseases like head blight, crown rot etc. (Nelson 1994) It causes a broad spectrum of infections in humans which includes not only superficial infections like onychomycosis and keratitis but also disseminated infections occurring exclusively in severely immuno-compromised patients. (Nucci 2002) Allergic diseases like sinusitis and mycotoxicosis following ingestion of toxins produced by fusarium are also known to occur. (Nelson 1994).

Among immuno-competent patients, tissue breakdown (as caused by trauma, severe burns or foreign body) is the risk factor for fusariosis. Risk factors for disseminated fusariosis include

severe immunosuppression (neutropenia, lymphopenia, graft-versus-host disease, corticosteroids), colonisation, tissue damage and receipt of a graft from an HLA-mismatched or unrelated donor. Clinical presentation includes refractory fever (> 90%), skin lesions and sino-pulmonary infections (approximately 75%).The most frequent cause of human infections is *F. solani,* although more than 50 species of fusarium have been identified, including plant and animal pathogens. (Dignani 2003) The portal of entry includes the Para-nasal sinuses (Anaissie 1988), lungs (Brint 1992) and skin (Dignani 2003). The organism is usually acquired in the community among normal hosts and also in the hospital setting in the patients severely immuno-compromised.

Case Report

A 15-year-old male patient was admitted in the pediatric ward of PGIMER Dr. RML hospital, New Delhi, India, with complains of high grade fever, vomiting and excessive irritability and decreased urine output for the past two weeks. The patient had a history of spontaneous erosion of dorsum of nasal septum over a period of two years. The patient also had past history of developmental delay due to birth asphyxia and loss of attained motor milestones like walking. There was no family history of any neurological abnormality, Diabetes mellitus, pulmonary tuberculosis, hypertension, or ischaemic heart disease.

On examination, the child was irritable, malnourished and febrile. He was 28 Kg weight for 149cm of height with body mass index

(BMI) of 12.61. He was febrile with temperature -102˚ F, pulse rate was 128 per minute and respiratory rate 28 per minute. His blood pressure was 70/38 mm of Hg. There was no history of edema, cyanosis, clubbing, lymphadenopathy and raised juglar venous pressure. Local examination showed complete erosion of dorsum of nose. The nasal debris was then sent to the Microbiology department for fungal culture and microscopy. Neurological examination showed progressive paralysis in both the limbs with grade 0 powers in both lower limbs and grade 2 in upper limbs and trunk. Speech was dysarthric and plantars were mute. Higher functions, cranial nerves and reflexes could not be elicited. There were no signs of meningeal irritation and spinal cord involvement. On abdomen examination, liver and kidneys were non- palpable whereas spleen was slightly enlarged.

Microscopy was performed on 20% KOH mount of the nasal debris which revealed branched septate hyphae 3-4μ wide (photograph 1). The specimen was inoculated on 2 slants of Sabouraud's Dextrose Agar (SDA) with and without antibiotics which were incubated at 25˚C and 37˚C aerobically. All the slants showed cottony white growth initially after 1 week of incubation and this growth later showed grayish pigmentation. The lactophenol cotton blue (LPCB) mount of this growth showed septate hyphae, 2-3 μ wide in diameter giving rise to short hyphal branches with foot cells at the base of macroconidium. Typical hyaline, multi-septate, curved sickle-shaped macroconidia were also visualized (photograph 2). Thus, the fungus was identified as *Fusarium spp.*

Other laboratory investigation showed a total leucocyte count (TLC) of 8700 cells/mm^3 with differential count (DLC) of polymorphs -72%, lymphocytes- 23%, eosinophils 3% and monocytes-2%. Liver function tests, Blood urea nitrogen, serum urea and serum creatinine were within normal limits. Platelet counts was 3.65 lacs and the peripheral blood smear was positive for plasmodium vivax which was responsible for current episode of high grade fever and the child was treated with artesunate (2.4 mg per kg/day) and clindamycin (15mg/kg/day) for 7 days. After 7 days of treatment the response rate was unsatisfactory and the patient was put on monocef, amikacin and Vancomycin. The blood culture was sterile throughout the stay in the hospital while the urine culture revealed candida spp. twice. Endotracheal suction catheters grew *Acinetobacter baumannii* in mixture which was sensitive to piperacillin tazobactum. The child

was non-reactive for HIV 1 & 2 antibodies and his CD4 count was only 291 cells/ µl by Facs calibur.

However, child developed aspiration pneumonia and was shifted to pediatric intensive care unit where he was put on ventilator support. The child had developed pulmonary edema and hemorrhage. Colour Doppler showed bilateral juglar vein thrombosis. X-ray PA-view chest was done, which showed consolidation in superior segments of lower lobes, enlarged cardiac silhouette, prominent vascular markings in the upper lung zones, peri hilar alveolar infiltrate and costophrenic angle was obliterated. CT scan (Paranasal sinuses) was also performed which showed heterogenous opacities (photograph 3). Tazobactam and fluconazole were started and during the course of the patient's stay in intensive care unit for *Acinetobacter spp.* in

Endotracheal suction catheters and candida spp in urine respectively, where he developed generalized tonic clonic seizures. The child was also transfused with 1 unit of packed blood cells as hematological parameters got deranged due to pulmonary edema and hemorrhage.

A tentative diagnosis of neurodegenerative disease with birth asphyxia along with fungal sinusitis was made in this case and the treatment was continued for a further period of 1 week. A repeat nasal specimen was sent for confirmation to the microbiology lab where again there was growth of *fusarium spp.* the child was now put on Amphotericin B 15 mg OD over 4 hours (0.75mg/kg bodyweight/day). However, the child succumbed to his illness after four days of starting Amphotericin B.

Discussion

The Genus *Fusarium* causes a broad spectrum of human diseases including mycotoxins and infections which can be superficial, locally invasive or disseminated (Dignani 2003). The organism is usually acquired in the community among normal hosts and also in hospital setting in patients who are severely immuno-compromised. It has several virulence factors including the ability to produce trichothecene and other mycotoxins which suppress humoral and cell- mediated immunity and may also cause tissue breakdown. (Anaissie 1988) *Fusarium spp.* also has the ability to adhere to prosthetic materials like catheters and contact lens and to produce proteases and collagenases. (Kratka 1979).

In immuno-competent host, *fusarium spp* may cause allergic sinusitis (Anaissie 2002), chronic non-invasive or invasive sinusitis (Kurien 1992). By contrast, in immuno-compromised patients, sinusitis is always invasive. (Segal 1998) Amongst the reported cases of fusariosis, 18% had sinus involvement along with dissemination, suggesting that sinus serves as site for dissemination (Nucci 2003).

The clinical features of this case are indistinguishable from *Aspergillus* sinusitis and present as nasal obstruction. In the present case, it came in much advance stage where *fusarium* had already invaded through maxillary sinus. The fungus is known for its angio-invasive nature (Nucci 2003) and due to this; there must have been complete necrosis and erosion of dorsum of nose (Segal 1998). A similar case has been reported in the past where

fusarium caused abscess of the nose in an immuno-compromised child which led to partial destruction of cartilage of the nasal septum and the patient was treated with amphotericin B. Primary fusarial sinus involvement (Kurien 1992) rarely occurs in immuno competent patients, but 2 such cases have been reported in farmers.

Lung involvement is common in invasive fusariosis (39% cases) and it almost always occurs amongst immuno-compromised patients (Nucci 2003) and some must have proved fatal in the current case. Blood culture in the present case did not show growth of any bacteria or fungus as patient was already on broad spectrum of antibiotics and antifungals, which could have hampered the growth of any organism in blood culture.

The prognosis of fusariosis in immuno-compromised host is directly related to the immune status of patient, with high death rates in patients with persistent immunodeficiency. An analysis of 84 patients with hematological diseases revealed survival rates at 30 & 90 days after diagnosis of 50% and 21%, respectively. In the present case, the patient was already having multiple neurological manifestations due to birth asphyxia. Moreover, patient was malnourished and CD4 count of the child was also low (291cells/µl); hence the child was immuno-compromised because the child developed this fatal invasive fungal infection. The diagnosis of fusariosis depends on the clinical form of disease and culture report. The culture report in support of the infection is the isolation of several colonies from the same specimens, a positive direct examination of biological material and most importantly site of isolation and the host. Culture of the

sinus aspirate/ respiratory secretion in immuno-compromised host should always be considered diagnostic of fusarial infections, as opposed to isolation of the same from skin scraping of immuno-competent host. Thus, it confirms diagnosis since patient was already immuno- compromised and the fungus was isolated twice from nasal secretion and debris. Confirmatory diagnosis of *fusarium* requires histopathological examination in absence of microbial growth in culture. However, in the present case, culture was positive twice and sample for histopathological examination was not sent as the sample received was not adequate for histopathological examination. *Fusarium spp*. is known to grow easily and rapidly in most media without cycloheximide. In the present case also, the fungus grew rapidly within a week and was identified by production of hyaline, sickle-shaped, multi-cellular macroconidia with a foot cell at base.

Species identification could not be made as it requires molecular methods which are not readily available in routine laboratories. Recently, a commercially available PCR method was tested with 21 isolates of *fusarium spp*. and 5 ATCC isolates. Using sequencing identification as gold standard, seven of the nine different species were identified. (Healy 2005).

The prognosis of fusariosis in immuno-compromised host is directly related to the immune status of the patient, with high death rates in patients with immunodeficiency. The patient was not only immuno-compromised but was suffering neurological deficits due to a known history of birth asphyxia. The patient was treated with Amphotericin but the infection had already invaded through the sinus by the time the patient reported to the authors and succumbed to his illness. Infections in immuno-compromised

patients are generally fatal and successful outcome is determined largely by the degree and persistence of immuno-suppresion and extent of infection with virtually a 100% death rate for persistently neutropenic patients with disseminated disease. Thus, these infections may be clinically suspected on the basis of constellation of clinical and lab findings which should lead to prompt therapy.

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