Successful Treatment of Rhinocerebral Mucormycosis with Wide Local Debridement & Nanosomal Amphotericin

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Article ID: WMC003927
Article Type: Case Report
Submitted on: 01-Jan-2013, 09:20:59 AM GMT Published on: 02-Jan-2013, 04:08:42 PM GMT
Article URL: http://www.webmedcentral.com/article_view/3927
Subject Categories: OTORHINOLARYNGOLOGY
Keywords: Mucormycosis, Amphotericin-B, Exenteration, Ketoacidosis

How to cite the article: Daterao-karodpati N, Gundecha V, Bafna P, Bhape R, Gandhi R. Successful Treatment of Rhinocerebral Mucormycosis with Wide Local Debridement & Nanosomal Amphotericin. WebmedCentral OTORHINOLARYNGOLOGY 2013;4(1):WMC003927

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Source(s) of Funding:
NIL

Competing Interests:
NIL
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Abstract

Rhinocerebral mucormycosis is a frequent complication of diabetes mellitus. Advanced age predisposes to mucormycosis, however severe ketoacidosis predisposes to local spread of infection irrespective of age. Our case study describes a young patient with rhinocerebral mucormycosis. The patient was not a known diabetic but was diagnosed to have severe hyperglycaemia on admission. Aggressive treatment includes early surgical intervention along with optimum doses of amphotericin B which are pivotal in controlling the spread of mucormycosis thereby preventing the dreadful complications. The surgical treatment includes wide & thorough debridement of the involved tissues. This means an early enucleation of the eyeball to stop the spread to other eye via optic chiasma.

Introduction

Mucormycosis refers to different diseases caused by infection with fungi in the order of Mucorales. Rhizopus species are the most common causative organism. In descending order, the other genera with mucormycosis-causing species include Mucor, Cunninghamamellia, Apophysomyces, Absidia, Saksenaea, Rhizomucor, etc.(1)

Most mucormycosis infections are life-threatening. The risk factors, such as diabetic ketoacidosis and neutropenia, are present in most cases. Severe infection of the facial sinuses, which may extend into the brain, is the most common presentation. Pulmonary, cutaneous, and gastrointestinal (GI) infections are also recognized.

Successful mucormycosis treatment requires correction of the underlying risk factors, antifungal therapy with amphotericin B, and aggressive surgery.

Mucoraceae are ubiquitous fungi that are commonly found in soil and in decaying matter. Rhizopus can be found in moldy bread. Given the ubiquitous nature of these fungi, most humans are exposed to these organisms on a daily or weekly basis. Nonetheless, they rarely cause disease because of the low virulence of the organisms; instead, they mainly affect individuals with immunocompromising conditions. Immunocompromised hosts with poorly controlled diabetes mellitus (especially with ketoacidosis), those who are receiving glucocorticosteroids, have neutropenia in the setting of hematologic or solid malignancy, have undergone transplantation, have iron overload, and who have burns are at risk for contracting the disease.

The major route of infection is via inhalation of conidia, other routes include ingestion and traumatic inoculation. Ingestion leads to GI disease and occurs primarily among malnourished patients, but it can also occur after ingesting nonnutritional substances (pica). Additionally, natural disasters (after hurricanes or tsunamis) may be associated with wound infections due to mucormycosis and should be considered in the setting of a necrotic wound or poor response to antibiotic treatment.(2,3,4)

When spores are deposited in the nasal turbinates, rhinocerebral disease develops. When spores are inhaled into the lungs, pulmonary disease develops, when ingested GI disease ensues. When the agents are introduced through abraded skin, cutaneous disease develops.

Mucoraceae are molds in the environment that become hyphal forms in tissues. Once the spores begin to grow, fungal hyphae invade blood vessels, producing thrombosis, tissue infarction and necrosis. Neutrophils are the key host defense against these fungi. Few cases of mucormycosis have been reported in patients with acquired immunodeficiency syndrome (AIDS), suggesting that the host defense against this infection is not primarily mediated by cellular immunity.

Rhinocerebral disease may manifest as unilateral, retro-orbital headache, facial pain, numbness, fever, hyposmia, and nasal stuffiness, which progresses to black discharge. Initially, mucormycosis may mimic sinusitis.(5)

Late symptoms that indicate invasion of the orbital nerves and vessels include diplopia and visual loss. These late symptoms indicate a poor prognosis and are usually followed by reduced consciousness. Most patients with rhinocerebral disease have diabetes
(especially with ketoacidosis) or have malignancies in combination with neutropenia and may be receiving broad-spectrum antibiotics.

Orbital swelling and facial cellulitis are progressive. Black pus discharges from the necrotic palatine or nasal eschars. Necrotic eschars can be noted in the nasal cavity, on the hard palate, or as facial lesions, although these lesions are suggestive of mucormycosis, their absence does not exclude the possibility of this disease.

Proptosis, ptosis, chemosis, and ophthalmoplegias indicate retro-orbital extension. Cranial nerves V and VII are the most commonly affected. Loss of vision can occur with retinal artery thrombosis.

A reduced conscious state denotes brain involvement. Rhinocerebral disease causes significant morbidity in patients who survive, because treatment usually requires extensive, and often disfiguring, facial surgery.

Surviving mucormycosis requires rapid diagnosis and aggressive coordinated medical and surgical therapy.

Mucormycosis carries a mortality rate of 50-85%. The mortality rate associated with rhinocerebral disease is 50-70%. The advent of novel antifungals, such as posaconazole, may offer improvement in these mortality rates. (6,7,8,9)

Case History

A 23 year old male was admitted in the ETU in our hospital in stuporous state with 3 days history of severe weakness, watery rhinorrhoea, mild swelling on right cheek. He was treated by the general practitioner as having sinusitis. He had headache, vomiting & irrelevant talk. He had history of polyuria & polydypsia during last few days. The patient had gained weight of 22kg in last 3-4 months.

O/E-

The vital signs were as under-

Pulse-140/min
BP-160/100mm of Hg
RR-36/min
Acidotic breath was present.

The central nervous system examination showed that he was in astuporous state and was opening eyes intermittently. He had no neck stiffness.

His tendon reflexes were elicitable normally ie, grade II & planters were withdrawal.

BSL on glucometer was high & confirmed to be 846 mg %. With these findings our provisional diagnosis was diabetic ketoacidosis with sinusitis.

In view with the swelling on the right cheek & stuporous state, a CT brain & paranasal sinuses was done.

Laboratory tests done urgently showed the following results -

BSL-846mg/dl
Blood urea-44mg/dl
S creatinine-1.6mg/dl
Na-132meq/dl
K -6.6meq/dl
S Ca-11.7mg/dl
ABG Ph-6.978
PCO2-11.3 mmHg
Bicarbonates -2.6 mEq/L
PO2-108.7 mm of Hg
O2 saturation 95%
Plasma acetone++++
Urine acetone ++++++

Patient was managed in ICU with all supportive treatment including mechanical ventilation, higher antibiotics, insulin infusion and IV fluids as per CVP monitoring.

After 12 hrs of admission proptosis on the right side increased.

Urgent MRI brain and CT of PNS showed haziness in right maxillary, ethmoidal and frontal sinuses with retro orbital extension. We advised urgent nasal swab for fungus. Laboratory test confirmed the diagnosis of mucormycosis.

The patient was immediately started with 100mg of nanosomal amphotericin B in IV infusion. The patient was taken for endoscopic evaluation in OT. On examination with sinuscope clear fluid discharge without any discoloration of the tissue was noted. All the involved sinuses were drained out on right side. The patient was toxic and did not show any improvement in spite of above treatment. The proptosis worsened and there was loss of vision in the right eye.

The patient was taken to OT for wide debridement of the right side as necrosis had started in the nasal turbinates. Exenteration could not be done due to lack of consent but after 24 hrs of intensive care and correction of metabolic factors exenteration was done in view to stop the spread to other side when the
consent was obtained.

The patient’s blood sugar was controlled with insulin infusion and amphotericin-B was continued as per renal dose adjustments. A total of 8 gm of nanosomal Amphotericin B was administered. The patient required intermittent haemodialysis. Despite his acute renal injury we managed to continue his Amphotericin B.

Endoscopic evaluation was done daily to check for further progress of the disease. There was progressive necrosis of the walls of maxilla which were debrided. Amphotericin B was also used in the local dressing material.

The disease slowly engulfed orbital floor and hard palate. By this time a total of 9 gms of nanosomal amphotericin B was administered.

When the nasalswab was negative for fungus & we could see the healthy bony margins the defect on the right side (fig 1) of the face (the orbit & maxilla) was covered with a lattisimus dorsi flap(fig 2).

Over the next 3 weeks the patient progressively improved. His renal parameters were normal. The patient was discharged and is awaiting for his orbital prosthesis.

Results

Rhinocerebral mucormycosis has a high morbidity & mortality rate. However with high index of suspicion and immediate medical management with aggressive surgical intervention patient can be revived. Our case describes a young male who could be saved with a co-ordinated team effort.

References

Illustrations

Illustration 1

Fig 1: Photograph after enucleation showing necrosis of the anterior wall, roof

Illustration 2

Fig 2: Latissimus dorsi flap in situ
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