RHINO-CEREBRAL MUCORMYCOSIS IN AN IMMUNOCOMPETENT INDIVIDUAL

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ABSTRACT None

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Case report

An autonomous 72-year-old male with a previous medical history of hypertension with hypertension-mediated injury: nephropathy and left ventricular hypertrophy, dyslipidemia and traumatic brain injury at work fifteen years ago. Family members reported 15 days of prostration, with reduced interaction with them, episodes of loss of urinary sphincter continence and periods of absence. He was referred to the emergency department after a generalized tonic-clonic epileptic seizure. On admission, recurrent seizures, generalized tonic-clonic crises with response to 5mg of diazepam, doubt about the full recovery of the state of consciousness, cerebral computed tomography without evidence of acute injury but evidence of left frontal sequelae fractures with misalignment and bone defect in the posterior wall of the frontal sinus, as well as the roof and medial and lateral orbital walls of the left orbit. Analytical study with no increase in the inflammatory parameters, no worsening renal function, no ionic changes and normal glucose level. Lumbar puncture was performed with a clear cerebrospinal fluid containing 2 cells, normal protein and glucose levels. Chest X-ray without evidence of pneumonia and abdominal pelvic ultrasound without abscess lesions. Assuming seizures with probable infectious/metabolic complications, admission for surveillance. In the ward with recurrence of generalized tonic-clonic seizures, electroencephalogram showing nonspecific global dysfunction with epileptiform activity in the left frontotemporal region. He always remained apyretic and without evidence of an infectious process. Because of the maintenance of generalized tonic-clonic crises under levetiracetam and clonazepam and fluctuation in his state of consciousness, he underwent a cerebral magnetic resonance imaging (MRI - Figure 1) that revealed a left frontal cerebral abscess with a bone defect in the frontal sinus. He empirically started ceftriaxone and metronidazole and went to the Neurosurgery unit to drain the abscess. Isolation of Aspergillus fumigatus in brain tissue, and therapy for amphotericin B, penicillin and metronidazole was adjusted. The patient maintained an unfavorable clinical evolution, maintaining a Glasgow coma scale between 6-7, repeated EEG without evidence of epilepsy but with moderate to severe diffuse encephalopathy, and ended up dying on the tenth day of treatment.

Discussion/Conclusion

Mucormycosis is an infection caused by a group of moulds within the orders Mucorales and Entomophthorales presents on environmental [1,2]. Several conditions have been associated with the development of mucormycosis: poorly controlled Diabetes Mellitus; haematological malignancies with neutropenia; hematopoietic stem cell transplant receptor; solid organs transplant receptor; immunosuppression or chemotherapy; autoimmune or rheumatic disorders; human immunodeficiency virus infection; peritoneal dialysis; iron overload states; malnutrition; trauma; burns; and prior receipt of voriconazole (VORI) [2,3]. Rarely affects immunocompetent persons, but cutaneous, rhino-orbital, and (occasionally) disseminated infections have been reported following local cutaneous or soft tissue trauma. Diagnosis of mucormycosis relies upon histopathology and culture. Mortality associated with invasive mucormycosis is high (>30–50%), with 90% mortality associated with disseminated disease. Mucorales are resistant to many antifungal agents; the most active agents include Lipid formulations of amphotericin B (LFAB) and the newer triazoles, posaconazole (POSA) and isavuconazole (ISAV). By contrast, the echinocandins and VORI have poor activity against Mucorales. LFAB has been the mainstay of therapy for mucormycosis; POSA and ISA have been used primarily as salvage therapy for patients refractory to or intolerant LFAB. Early surgical debridement or excision plays an important adjunctive role [1-3].

This case exemplifies how, in a healthy individual, the history of traumatic brain injury constitutes a potential gateway for rhino-cerebral mucormycosis. Therefore, even in the absence of immunosuppressive disease, it is extremely important to remember this diagnosis to order a brain MRI and quickly initiate surgical and medical treatment. Despite the high mortality associated with this pathology, the sooner we think about this diagnosis, the sooner we act and potentially avoid this outcome.
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Conflict of interest
There are no conflicts of interest to declare by any of the authors of this study.

References


Figure 1a e 1b. Cerebral MRI: In the left anterior frontal planes, a lesion with peripheral annular enhancement is defined, with a rounded morphology (25 x 15.5 x 17.8 mm with the largest AP, CC and T axes, respectively) and that includes content that exhibits restriction of diffusion.

Figure 1c. Cerebral MRI: Bone defect on the posterior wall of the left frontal sinus and, inherently, to the tissue content that fills it (probably of an inflammatory/infectious nature).