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Case Report

Allergic fungal rhinosinusitis leading to catastrophic multiple ischemic strokes: a case report

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ABSTRACT

Allergic fungal rhinosinusitis (AFRS) is a non-invasive subtype of chronic rhinosinusitis. Although bone erosion and cerebrovascular involvement are rare, they may lead to severe complications. Standard management includes endoscopic sinus surgery and perioperative systemic corticosteroids. We describe a case of a 38-year-old immunocompetent man presenting with a 3-month history of nasal obstruction and progressive frontotemporal headache without fever or neurological deficits. Imaging revealed a large expansile sphenoid lesion with bone erosion and parasellar extension. Despite surgery and broad medical therapy, he developed multifocal ischemic events. Histopathology confirmed AFRS, and Aspergillus fumigatus was isolated; other microorganisms were excluded. His condition deteriorated, and he died 37 days after presentation. This case illustrates the rare but life-threatening potential of AFRS, particularly with sphenoid involvement. Although limited by its single-case design, it highlights the need for early recognition and intervention, as progression to fulminant forms may occur.

Keywords: Allergic fungal rhinosinusitis, Chronic rhinosinusitis, Endoscopic sinus surgery, Perioperative systemic steroids, Ischemic stroke, Case report

INTRODUCTION

Allergic fungal rhinosinusitis (AFRS) is a non-invasive subtype of chronic rhinosinusitis, typically characterized by benign clinical behavior. Nevertheless, bone erosion and cerebrovascular involvement can substantially elevate the risk of severe complications, despite the overall rarity of major neurological complications in AFRS.

Current management relies primarily on surgical intervention, along with the use of perioperative systemic corticosteroids. We aimed to describe a rare case of AFRS complicated by multiple ischemic cerebrovascular events in an immunocompetent patient.

CASE REPORT

A 38-year-old immunocompetent male with a history of asthma and allergic rhinitis presented to the Otorhinolaryngology Emergency Department with a 3-month history of nasal obstruction and progressively worse left frontotemporal headache, associated with nausea and vomiting, but no fever or alteration in neurological status. The patient denied visual disturbances or prior nasal surgery. Anterior rhinoscopy and nasal endoscopy revealed purulent rhinorrhea, nasal polyps in the left nasal cavity and posterior ethmoidal edema on the right. Ophthalmological examination was unremarkable. Computed Tomography (CT) showed a well-defined, expansile, hyperdense lesion (5.5×6 cm), centered in the sphenoid sinus, with transcompartmental

extension into the parasellar region, bilateral cavernous sinuses, sella turcica, and prepontine cistern, along with regional bone remodeling and erosion; laboratory analysis showed no increase in inflammatory markers. The patient was admitted for investigation and empiric treatment with antibiotics and systemic corticosteroids. During hospitalization, he experienced analgesic-refractory headache, nausea, vomiting, and new-onset intermittent blurred vision and diplopia. Magnetic Resonance Imaging (MRI) (Figure 1) revealed a space-occupying lesion in the sphenoid sinus (5.9×5.6×4.1cm), with central T1/T2 signal void, peripheral enhancement, posterior displacement of both internal carotid arteries and cavernous sinuses, and invasion of the clivus and dura mater.

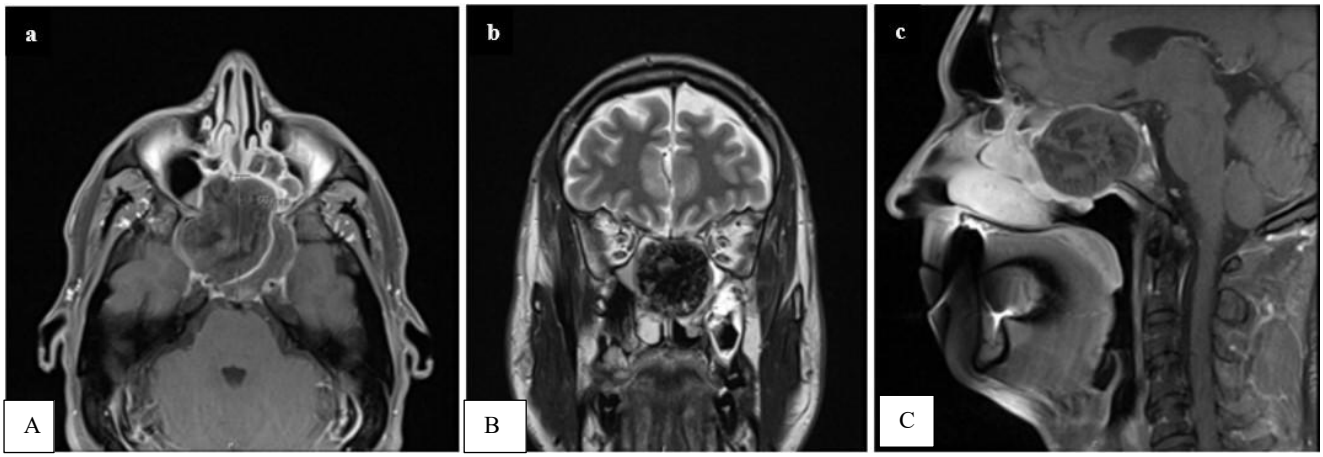

Figure 1: Preoperative magnetic resonance imaging with contrast revealing a space-occupying lesion in the sphenoid sinus, with central T1/T2 signal void and peripheral contrast enhancement: (A) axial plane, (B) coronal plane and (C) sagittal plane.

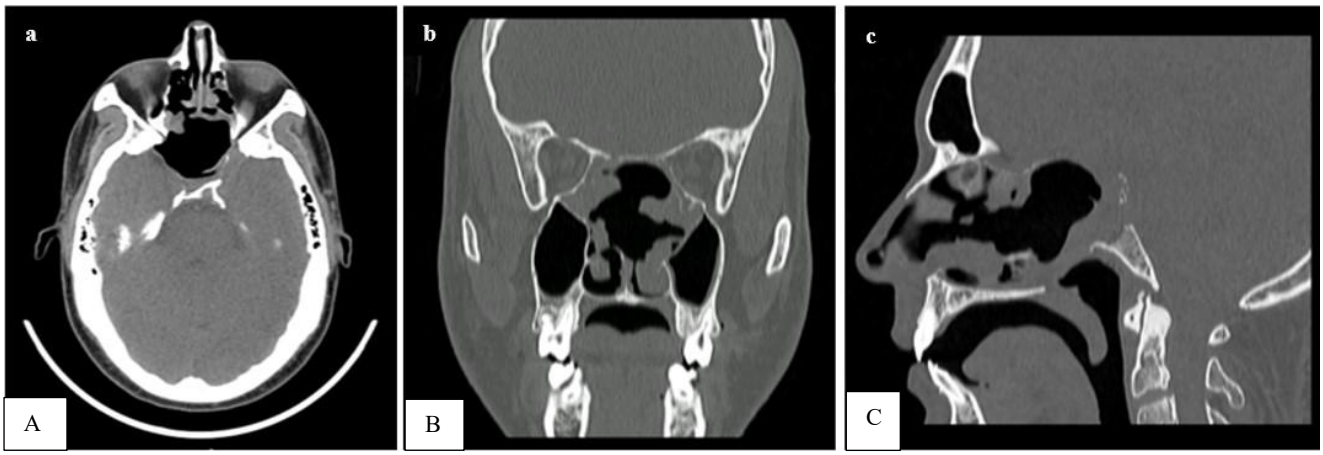

Figure 2: Postoperative computed tomography at 72 hours confirming subtotal lesion resection: (A) axial plane, (B) coronal plane and (C) sagittal plane.

Endoscopic sinus surgery (ESS) was performed. Intraoperatively, a polypoid lesion was identified in the right sphenoethmoidal recess, with bilateral posterior septal bulging. A septal incision revealed a cavity containing purulent material, which was aspirated and sent for histopathology and microbiology, along with fungal debris and necrotic tissue. At the end of surgery, the sphenoidal cavity was clear, with intact mucosa. Bone erosion was present, and although the dura mater was exposed, it remained intact. In the immediate postoperative period, the patient was feeling well, without headache. Follow-up CT at 72 hours (Figure 2) confirmed subtotal lesion resection without local or intracranial complications. On postoperative day 5, however, the patient developed confusion, slowed speech, prostration, and vomiting. Contrast-enhanced CT angiography revealed recent ischemic lesions in multiple vascular territories. Lumbar puncture demonstrated pleocytosis with mononuclear predominance, elevated protein levels, and preserved glucose levels. Inflammatory reactive vasculitis secondary to meningeal involvement by the sphenoid lesion was suspected. The patient was admitted to the Intensive Care Unit under multidisciplinary care. Histopathology confirmed AFRS with eosinophilic mucin and Charcot-Leyden crystals, without visible fungal structures. Aspergillus fumigatus

was isolated from tissue culture. No signs of malignancy or invasive fungal disease were identified. Laboratory results revealed elevated total IgE (1803 IKU/l), Aspergillus fumigatus-specific IgE (17.8 UK/l), and specific IgG (116 mg/l), and other microorganisms were excluded by laboratory testing. Despite high-dose antibiotics, systemic and topical corticosteroids, and voriconazole, the ischemic lesions progressed, with new infarcts. The patient died 37 days after initial presentation.

DISCUSSION

AFRS is a non-invasive subtype of chronic rhinosinusitis with nasal polyps (CRSwNP), driven by IgE-mediated hypersensitivity to fungal antigens within the sinonasal tract. It typically affects young, immunocompetent, often atopic individuals.[1-5] Its pathophysiology is thought to involve mucosal barrier dysfunction, impaired innate immune signaling, and excessive fungal growth.[6]

Although peripheral eosinophilia may be absent, serum IgE levels are frequently elevated, often exceeding 1000 U/mL. Fungal-specific IgE and IgG are nonspecific, as they can also be present in CRSwNP without AFRS.[6] Histopathology reveals thick eosinophilic mucin, Charcot-Leyden crystals, desquamated epithelial cells, cellular debris, and sparse fungal hyphae; crucially, no fungal invasion of mucosa or bone is seen.[4,5]

Common symptoms include nasal obstruction, anterior/posterior rhinorrhea, and headache. CT imaging typically shows hyperattenuation within the sinuses, complete opacification, sinus expansion, bony thinning or remodeling, and adjacent soft tissue involvement without intracranial or intraorbital invasion. MRI often demonstrates central signal void on T2-weighted images, hypointense central signal on T1, and peripheral enhancement (especially on T1-weighted images), features considered highly specific for AFRS.[1]

Diagnosis relies on the Bent-Kuhn criteria: type I IgE-mediated hypersensitivity to fungi (supported by clinical history, skin testing, or serology), nasal polyposis, characteristic CT findings (serpiginous areas of hyperattenuation), eosinophilic mucin without fungal tissue invasion, and positive fungal staining of surgical specimens. Bone erosion on imaging does not indicate invasive disease and may occur in AFRS. Positive fungal culture, though helpful, is not mandatory. Unilateral predominance, asthma, Charcot-Leyden crystals, and peripheral eosinophilia support the diagnosis but are not consistently present. The disease often involves multiple sinuses, typically with unilateral dominance. Up to 30% of cases may exhibit extrasinus extension to the orbit or cranial cavity without true tissue invasion.[5-7] AFRS is often diagnosed at advanced stages, with extensive eosinophilic mucin difficult to evacuate. ESS is the mainstay of treatment, aiming for complete removal of fungal material and mucin. Preoperative systemic corticosteroids may minimize intraoperative bleeding. Maintenance therapy typically includes nasal saline irrigation and topical corticosteroids; systemic corticosteroids are also recommended postoperatively for early disease control. Despite surgery, recurrence is common. Adjunctive therapies such as immunotherapy, antifungals, or biologics may benefit the refractory cases.[1,4-7] Unlike AFRS, invasive fungal rhinosinusitis, more common in immunocompromised patients, involves vascular invasion with thrombosis, infarction, and necrosis.

Diagnosis is histological, and treatment involves aggressive surgical debridement and systemic antifungals.[4] Although AFRS often mimics CRSwNP, it differs in progression. Bony erosion is frequent, with up to 50% of cases exhibiting intracranial or intraorbital extension. Orbital involvement is rare but may present with proptosis, diplopia, or visual loss.[1,3,6] Optic neuropathy in AFRS usually results from chronic compressive, non-invasive mechanisms, leading to venous congestion, edema, and eventual infarction if untreated. Other cranial neuropathies may result from extrinsic compression and vascular compromise, rather than direct nerve invasion, and are potentially reversible with early intervention.[8] Intracranial complications are even more rarely described. Fungal extension into the cavernous sinus can cause carotid artery occlusion and subsequent infarction.[4] Additionally, eosinophilic meningitis may also occur through sinus wall erosion and mucin extrusion into venous sinuses or the subarachnoid space.[2]

CONCLUSION

In summary, AFRS is a chronic, severe inflammatory condition of the upper airway, associated with significant morbidity and high recurrence. Although usually benign, it can lead to catastrophic outcomes, particularly with sphenoid sinus involvement. Surgical debridement remains the cornerstone of treatment, with systemic corticosteroids recommended pre- and post-operatively. Although this report is limited by its single-case design, it underscores the potential for fatal cerebrovascular complications, highlights the critical importance of early diagnosis and timely intervention, and reinforces that this condition should be suspected in the appropriate clinical context, as it may progress to fulminant forms.

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