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Letter to Editor

Sphenoid Sinusitis and Stroke: How Perilous is the Relationship?

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DEAR EDITOR,

During the evaluation of acute stroke, do we look for sinuses or their anatomical variations? In individuals with diabetes or those in an immunocompromised state, sinus infections may sometimes play a crucial role in determining the etiology, management, and prognosis of a stroke.

A 64-year-old man with a history of uncontrolled diabetes underwent recent sinus surgery for frontal, ethmoidal, and sphenoid fungal (Aspergillus) sinusitis and presented in our emergency room in a drowsy state with septic shock requiring high vasopressor support. His vascular imaging revealed non-visualization of the left petrous and cavernous parts of the left internal carotid artery and severe stenosis of cavernous and supraclinoid segments of the right internal carotid artery. He was treated with antifungal, antibiotic, and other supportive measures for hemodynamic stabilization with a plan for revascularization. Unfortunately, shortly after his admission to our institute, he suffered a massive infarct in the right-sided ACA (anterior cerebral artery), MCA (middle cerebral artery), and left ACA territories, and he succumbed to it (Figure 1). It was noted that the patient had long-standing uncontrolled diabetes and sphenoid sinusitis. We probably missed the train, as we felt an early intervention could have been life-saving in his case.

Sphenoid sinus infections are rare (accounting for about 2.7–3% of all sinus cases) but potentially dangerous because of the sinus's proximity to the brain and its intimate relation to vital structures, including the internal carotid artery, optic nerve, cavernous sinus, and pituitary gland.

Sphenoid sinusitis is not accompanied by classical nasal symptoms of sinusitis, such as nasal obstruction, facial pain, or rhinorrhea. However, it presents with an atypical headache that is intense, deep, chronic, or stubborn and may be felt at the top of the head, behind the eyes, or in the back of the head; this headache sometimes does not respond to medication.

Sphenoid sinuses are often driven by their unique anatomical and biochemical environment. Fungal infections, particularly Aspergillus, are highly prevalent in immunocompromised patients. In a study of sinusitis, two out of 27 of the cases were chronic invasive fungal infections [1].

This phenomenon is primarily because the sphenoid sinus naturally maintains a microaerophilic (low oxygen) state, has a relatively poor blood supply, and features an isolated, static biochemical environment. The most common anaerobic bacteria involved include Peptostreptococcus species (spp.), Prevotella spp., Fusobacterium spp., and Clostridium perfringens. They are frequently mixed with aerobic bacteria (e.g., Staphylococcus aureus and Streptococcus). In cases of diabetes or a weakened immune system (like in our case), there is a higher risk of complications.

We need to recognize the key anatomical variations of sphenoid sinuses that promote most infections. The sellar-type pneumatization is the most common variation (found in 75–86% of people) and is frequently associated with inflammatory conditions like

chronic rhinosinusitis. Extensive pneumatization creates a larger surface area, increasing the likelihood of infection.

The other infection-prone variations are the presence of multiple or accessory septa within the sinus and septa that are deviated or attached to critical structures like the internal carotid artery or optic nerve, complicating drainage and potentially leading to trapped mucus and subsequent infection. Bony dehiscence over the carotid artery or optic nerve, protrusion of the optic nerve into the sinus cavity, presence of onodi cells (posterior ethmoid air cells that migrate into the sphenoid bone), and narrowing of the sphenoid ostium are other potentially dangerous variations we need to look for during stroke imaging in clinical practice.

Because the sphenoid sinus sits just millimeters away from these critical structures, an untreated or "silent" infection can cause a stroke secondary to vessel compression and spasm of the internal carotid artery, cavernous sinus thrombosis, and cerebral vasculitis. This causes local inflammation of the blood vessels (vasculitis), narrowing the artery or promoting the formation of blood clots. Early identification of sphenoid sinus infection, appropriate timing of endoscopic sinus surgery to remove the source of invasion, biopsy of the material, and the initiation of antibiotics/antifungals may prevent stroke or other vascular complications.

Endovascular therapy (EVT) is highly effective as a rescue treatment for infective vasculitis and related complications like mycotic aneurysms. However, its long-term durability depends on disease quiescence, pathogen eradication, and whether the intervention is aneurysmal or occlusive [2, 3]. Early recognition and timely endovascular procedure for the carotid artery could have saved our patient's life.

In fact, sphenoid sinusitis, because of its anatomical standpoint, can complicate an established stroke by igniting an inflammatory cascade. Similarly, untreated sphenoid sinusitis can complicate an intracranially or extra-cranially placed stent.

Hence, sphenoid sinusitis is considered a "silent bomb" and must be given special emphasis in an acute stroke setting. We need to address this issue with more research, as its incidence is fairly common in immunocompromised patients who present with stroke.

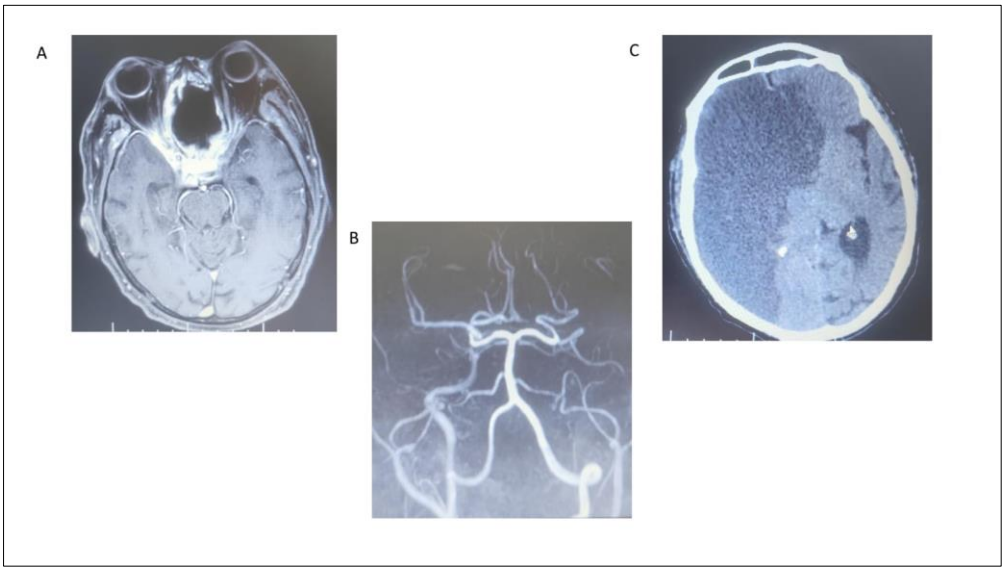

Figure 1: A: Post operative MRI showing expansile and coalescent T2W hypointense sinus area involving the ethmoid and sphenoid sinuses, with non-visualisation of bony sinus and nasal structures with heterogenous enhancement seen along the walls of sinuses and meninges. B: MR Angiogram with contrast revealed non-visualization of the left petrous and cavernous parts of left internal carotid artery and severe stenosis of cavernous and supraclinoid segments of right internal carotid artery. C: Final CT scan revealing massive infarct involving right frontal, parietal and temporal regions and left frontal region

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