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Sphenoid sinus mucocele mimicking pituitary apoplexy

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Abstract

Isolated sphenoid sinus mucoceles are rare entities but when large can present with cranial neuropathies and visual loss. We present a case of an otherwise healthy male presenting with acute-onset headache, progressive visual loss, and oculomotor palsy concerning for pituitary apoplexy but found to have an isolated sphenoid sinus mucocele without intradural extension. His symptoms markedly improved following sphenoidotomy and highlight the importance of considering paranasal sinus disease as a potential etiology for a pituitary apoplexy-like presentation and should be on the neurosurgeon's differential diagnosis.

Keywords

Sphenoid sinus mucocele; Pituitary apoplexy; Cranial neuropathy; Sphenoidotomy; Endoscopic endonasal approach; Case report.

Introduction

Pituitary apoplexy is a heterogeneous but rare clinical syndrome usually caused by hemorrhage into a pituitary adenoma or acute ischemia of a large macroadenoma [1]. Rarely, sphenoid sinus mucoceles may present with isolated cranial nerve palsies and mimic cavernous sinus or optic nerve compressive pathology [2-4]. However, the chronicity of these presentations can range from acute to more chronic and progressive[4]. Here we present a unique case of a pituitary apoplexy-like syndrome including multiple cranial neuropathies with severe headache caused by a sphenoid sinus mucocele.

Case Presentation

The patient was a 65 year-old, right-handed, black male with chronic tobacco use who presented to the emergency department with 2 weeks of progressive right eye (OD) visual loss, diplopia, and severe headache. His symptoms began with acute-onset, bifrontal headache with near-immediate right eyelid ptosis and diplopia. He subsequently noted worsening blurred vision. His neurologic exam was notable

for anisocoria with a sluggishly reactive, 4 mm right pupil that was deviated inferolaterally with impaired extraocular movements (EOM). He was only able to count fingers on visual acuity (VA) testing, and he had a complete temporal field cut. His left visual exam was normal: pupil 2 mm, briskly reactive, VA 20/30 (uncorrected) with full EOM. His presentation was concerning for pituitary apoplexy, aneurysmal subarachnoid hemorrhage, or an inflammatory cavernous sinus syndrome. His labs were notable for primary hypothyroidism with thyroid stimulating hormone level 0.43 mInt-units/mL and free T4 level 0.57 ng/dL. Prolactin level was low at 0.9 ng/mL. Computed tomography (CT) imaging demonstrated an expansile sphenoid sinus mass with erosion of the skull base and optic canal (Figures 1 and 2). Magnetic resonance imaging (MRI) demonstrated a large, T2 hyperintense, non-enhancing lesion filling the sphenoid

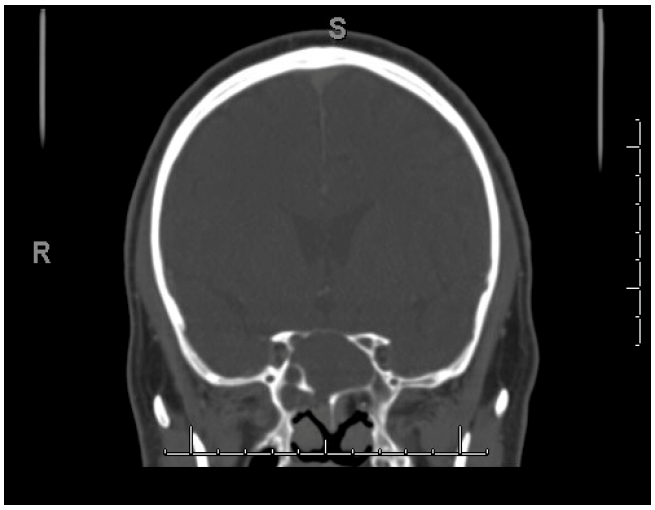

Figure 1: Coronal CT image with expansile sphenoid sinus mass eroding into right optic canal, displacement of right cavernous sinus, and marked thinning of sellar floor.

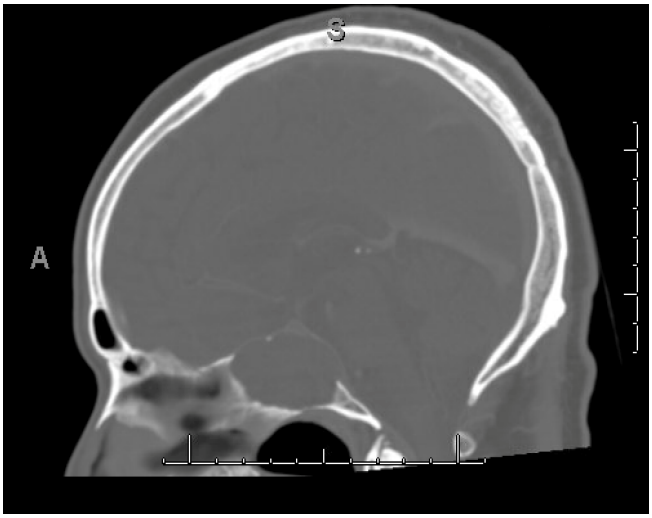

Figure 2: Midline sagittal CT re-demonstrating sphenoid sinus lesion with upward displacement of pituitary gland and significant thinning of anterior fossa floor, sella and clivus.

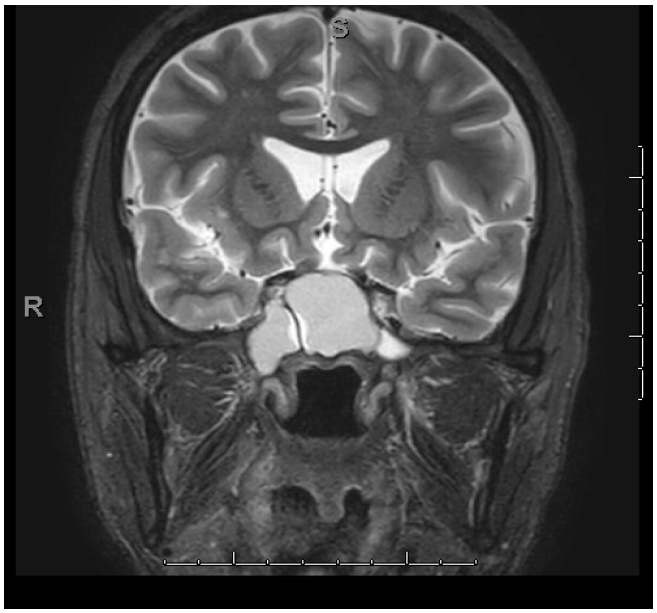

Figure 3: Coronal view through T2-weighted MR image demonstrating expansile sphenoid sinus mass with upward displacement of the sellar floor and compression of the right cavernous sinus and optic canal.

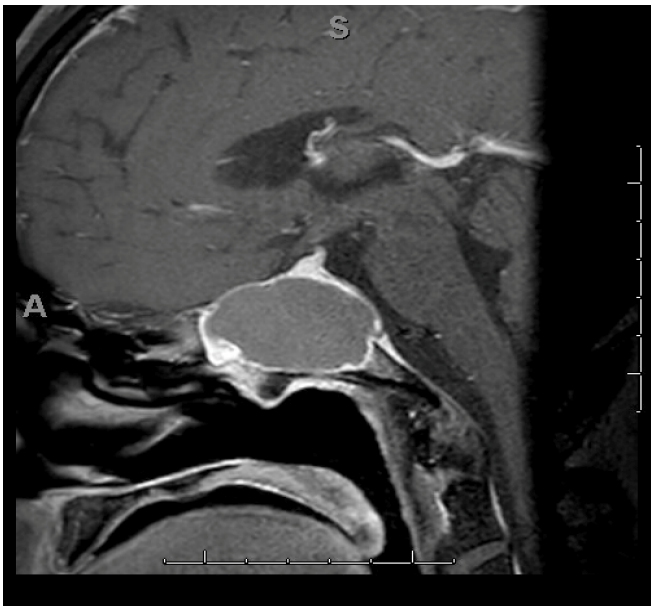

Figure 4: Midline sagittal T1 post-contrast MR image with a homogeneous, isointense, non-enhancing sphenoid sinus fluid collection with displacement of sellar floor. The normally enhancing pituitary gland can be seen draped over the posterosuperior margin of the lesion.

sinus and compression the right cavernous sinus and encroaching upon the right optic nerve (Figure 3). Upward displacement of the sellar floor with the normal pituitary gland draped posterosuperiorly were also noted (Figure 4). Surgical intervention via endoscopic approach was offered. Intraoperatively, upon sphenoidotomy, proteinaceous fluid and mucosal changes consistent with a mucocele were noted. No intradural extension was noted and the sellar floor appeared normal. No intradural intervention was undertaken. Post-operatively, he reported improvement in his headaches, diplopia, and ptosis. He was discharged home the day following surgery. He was seen in clinic for a 6 month visit and reports resolution of his diplopia, ptosis and markedly improved VA.

Discussion

Here, we have presented a unique case of a sphenoid sinus mucocele presenting as pituitary apoplexy with multiple cranial neuropathies and severe headache. Sphenoid sinus mucoceles are rare entities, comprising only 1-3% of all paranasal sinus mucoceles [4,5]. These lesions are usually asymptomatic, but can present as recurrent sinusitis, headaches, or with visual changes [2,6,7]. However, reports of a full syndrome including acute-onset headache with concurrent optic and oculomotor pareses are lacking. In this case, distinguishing between true pituitary apoplexy and a compressive mucocele was challenging and not fully confirmed until the pathology was identified intraoperatively. Additionally, recognizing that extra-cranial paranasal sinus diseases can result in sellar and cavernous sinus compression is important to avoid unnecessary surgical morbidity. The prompt and accurate recognition of a mucocele during endoscopic surgery precluded the team from opening the dura and exploring the sella, which may have predisposed to further morbidity such as infection, endocrinopathy, or cerebrospinal fluid leak.

Our case emphasizes the subtle imaging differences in sphenoid mucoceles and pituitary adenomas with apoplexy. In the event the mucocele is small and not compressing the sella or cavernous sinus, the diagnosis is more apparent from imaging alone. However, in cases such as the one presented, large mucoceles can be difficult to differentiate from sellar pathologies. Hemorrhagic pituitary apoplexy may demonstrate T1 isointensity and T2 hypointensity in the acute stages, while our mucocele demonstrated T2 hyperintensity. Both pathologies can exhibit peripheral enhancement after gadolinium administration. In the subacute setting, hemorrhagic apoplexy may show increased T1 and T2 signals while mucoceles usually exhibit overall stable MRI findings [8,9]. In ischemic or necrotic causes of apoplexy, the imaging findings are even more subtle. These ischemic apoplexy cases generally exhibit T1 hypo- to isointensity and T2 hyperintensity very similar to mucoceles. In these patients, the MRI intensities may be more heterogenous related to varying degrees of ischemia and necrosis within a pituitary adenoma which may be the only distinguishing feature from a mucocele [9,10].

While the imaging features of large sphenoid mucoceles may closely resemble those of pituitary apoplexy, it is important to recognize that patients with acute neurologic deficits warrant surgical intervention if possible. Our case and others demonstrate improvement in visual acuity and oculomotor function in patients with more urgent decompression [3,7,11]. However, it is critical to consider the possibility of extracranial compression from a mucocele as the cause of presenting deficits to prevent

unnecessary intradural exploration with the associated risks. However, our case highlights the difficulty in distinguishing large mucoceles from pituitary apoplexy, whether hemorrhagic or ischemic, based upon clinical presentation and imaging alone. It is important to keep these lesions in the differential diagnosis of pituitary apoplexy and to consider intraoperative findings at the time of intervention in the event the lesion is extra-cranial.

Conclusion

We have presented a case of a large, sphenoid sinus mucocele presenting with acute-onset, severe headache and concurrent optic and oculomotor neuropathies. These lesions can be very difficult to distinguish on imaging alone with many findings overlapping with either ischemic or hemorrhagic apoplexy. It is important to keep these lesions on the differential of pituitary apoplexy-like syndromes and to avoid unnecessary dural opening in the event of extra-cranial pathology.

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