Radiology Quiz Case 1: Diagnosis

Diagnosis: Sigmoid sinus diverticulum

Vascular tinnitus may originate from arterial sources, including neoplasms (glomus tumors), atherosclerotic disease, carotid-cavernous fistulas, dural arteriovenous malformations, an aberrant internal carotid artery, and arteriovenous malformations. Venous bruits may originate from stenosis or tortuosity of venous structures or may be a manifestation of intracranial hypertension (pseudotumor).[1,2] Our examination of choice for vascular tinnitus of presumed venous origin is computed tomographic angiography (CTA), as for other authors.[1-3] In the present case, an axial image demonstrated a right distal transverse sinus venous diverticulum extending through a dehiscent sigmoid plate into the mastoid complex (Figure 1). Wider windows for bone detail confirmed the osseous defect and the diverticulum (Figure 2, arrow).

In patients with pulsatile tinnitus and normal findings on otoscopy, a CTA scan is very sensitive in the diagnosis of vascular abnormalities. Also, in this case, magnetic resonance imaging (MRI) alone was not able to detect the abnormality. The combination of MRI and magnetic resonance angiography/venography (MRA/MRV), however, appears to have a much higher sensitivity than MRI alone, but small bone defects with herniation of a distal transverse sinus into a defect may be missed by MRA/MRV. In the case of a sigmoid sinus diverticulum, it appears that CTA is still a powerful diagnostic tool, but CTA may miss small arteriovenous malformations or dural fistulas in some cases, and if the tinnitus is severe, especially if it is objective, conventional catheter angiography is indicated even if the findings of computed tomography, CTA, MRI, and MRA/MRV are normal.

REFERENCES

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Radiology Quiz Case 2: Diagnosis

Diagnosis: Allergic fungal rhinosinusitis (AFRS)

Allergic fungal rhinosinusitis was first reported in the early 1980s as an emerging form of chronic noninvasive fungal sinus disease. The pathogenesis of this disorder is thought to be IgE-mediated hypersensitivity to inhaled fungal antigens, resulting in a chronic, noninfectious, inflammatory process.[1] The disease is estimated to affect 5% to 10% of all patients with chronic rhinosinusitis who undergo sinus surgery.[1,2] It is the most common type of fungal sinusitis worldwide and is particularly seen in warm, humid climates.[1,3] Patients affected with AFRS tend to be younger (usually in the third decade of life), immunocompetent, and atopic, with an associated history of asthma, aspirin sensitivity, allergic rhinitis, or nasal polyposis.[1-4] They also tend to present with slowly progressive, typically unilateral symptoms of nasal congestion and obstruction, headaches, anosmia, and copious rhinorrhea or postnasal drainage.[1,3,4] Many patients report a long-standing history of sinus-related complaints that have persisted despite maximal medical therapy and previous sinus operations.[1,3,4] In severe cases, ophthalmic symptoms and signs, including visual deficits, may manifest.[5] The most commonly reported ophthalmic symptom is diplopia, while the most common sign is orbital proptosis.[3] Though still controversial, the most widely accepted diagnostic criteria for AFRS currently include the following: type 1, IgE-mediated hypersensitivity to fungi; nasal polyposis; characteristic CT (or MRI) findings; presence of eosinophils in nasal secretions; no evidence of invasion of the mucosal lining of the sinus by fungal elements; and a positive surgical sinus fungal culture.[2-4] Other studies that may help diagnose AFRS include determination of serum IgE and fungal-specific IgG levels, which can be elevated during acute flare-ups.[4,6]

On CT imaging, this disease process demonstrates mixed high- and low-attenuation signals, which can involve any of the sinuses.[7] Areas of hyperattenuation correspond to the fungal debris and mucinous material.[1,7] Polypoid mucosa and/or sinus mucocele formation may be present.[4] In extensive cases, AFRS can lead to erosion and remodeling of the bone surrounding the paranasal sinuses, including the lamina papyracea, walls of the frontal sinus, and/or anterior skull base, causing extrusion of the sinus concretions into the orbit or cranial contents.[3,4,8,9] The rate of bony erosion in AFRS has been reported to range from 45% to 56%, with 1 study suggesting rates as high as 93%.[4,8] However, in general, patients with AFRS are 10 times more likely to demonstrate bone remodeling compared with other causes of chronic sinusitis.[4] On MRI studies, T1-weighted images generally demonstrate mixed signal intensities, which are reflective of the variable composition of the diseased sinuses.[1,4] T2-weighted images are generally hypointense or demonstrate a signal void, which is indicative of both the high concentrations of metals found in the cells of the fungal organisms that can cause AFRS and the high-protein, low free-water content of the mucinous material.[1] T1-weighted images also demonstrate peripheral sinus mucosal enhancement after the administration of