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MR Features of Pachymeningitis Presenting with Sixth-Nerve Palsy Secondary to Sphenoid Sinusitis

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Summary: Pachymeningitis manifested by localized enhancing dural thickening adjacent to the clivus on MR images of two patients with sphenoid sinusitis and sixth-nerve palsy is presented. This is an unusual complication of sphenoid sinusitis. Prompt diagnosis and therapy may avoid significant intracranial complications.

Index terms: Pachymeningitis; Paranasal sinuses, inflammation; Nerves, abducens (VI)

Pachymeningitis is an unusual disease characterized by localized thickening of the dura from a variety of causes (1, 2). Two patients with pachymeningitis who presented with sixth-cranial-nerve palsy and sphenoid sinusitis are discussed.

Case Reports

Case 1

A 17-year-old boy was in good health until 1 week before admission when fever, headache, nausea, and vomiting developed that were attributed to a viral syndrome. On the day of admission to another hospital, purulent drainage from the left ear and meningismus occurred. Cerebrospinal fluid examination revealed a white blood cell count of 1600/mm^3 with 99% polymorphonuclear leukocytes; glucose was 39 mg/dL and protein was 13 mg/dL. He was treated with cefotaxime and ampicillin for meningitis presumed secondary to otitis media. Over the next 2 days, bilateral sixth-nerve palsies and rapid deterioration in vision developed, with only light perception in the left eye and blindness in the right. He continued to spike fevers to 40.6°C and was transferred to the University of California Davis Medical Center. Magnetic resonance (MR) imaging of the brain showed sphenoid sinusitis, bilateral otomastoiditis, and an enhancing dural mass along the clivus (Fig 1A–C). A computed tomographic scan (Fig 1D), however, showed the petrous apex to be normal and unpneumatized. A left simple mastoidectomy, endoscopic ethmoidectomies, and a sphenoidotomy were performed, revealing pus under pressure in the sphenoid sinus. Cultures were negative for organisms. Rapid defervescence and return to normal vision occurred immediately after surgery, but a left hemiparesis and persistent bilateral sixth-nerve palsies were present. Dramatic improvement in the left hemiparesis occurred within 1 week. Two months after surgery, only a left foot drop remained. Follow-up MR 1 month after surgery showed absence of flow void in the cavernous portion of the left internal carotid artery compatible with occlusion (Fig 1E).

Case 2

A 68-year-old man living in the Philippines had a 1-month history of headaches and several episodes of transient diplopia, thought to be caused by a right sixth-nerve palsy. The diplopia disappeared and the headaches decreased in severity after treatment with antibiotics, vitamins, and calcium channel blockers. An MR examination of the brain performed in the Philippines was interpreted as consistent with a mass in the sphenoid sinus with bone destruction. He was transferred to the University of California Davis Medical Center.

Physical examination on admission was unremarkable. MR showed an air-fluid level in the sphenoid sinus with enhancing mucosa and enhancing soft tissue cloaking the posterior aspect of the clivus (Fig 2A–C). At endoscopic ethmoidectomy and sphenoidotomy, there was markedly thickened mucosa in the sphenoid sinus and purulent drainage. On removing the mucosa, the posterior bony wall of the sphenoid sinus was thin and violated. A cerebrospinal fluid leak was identified and repaired with a nasoseptal mucoperichondrial flap and abdominal fat graft. Cultures grew *staphylococci,* coagulase negative. Biopsy revealed only chronic inflammation. The patient was treated with antibiotics, became asymptomatic, and was discharged on the fourth hospital day. A follow-up MR study 9 months later showed resolution of the dural thickening adjacent to the clivus and residual soft tissue in the sphenoid sinus (Fig 2D and E) from the surgical repair.

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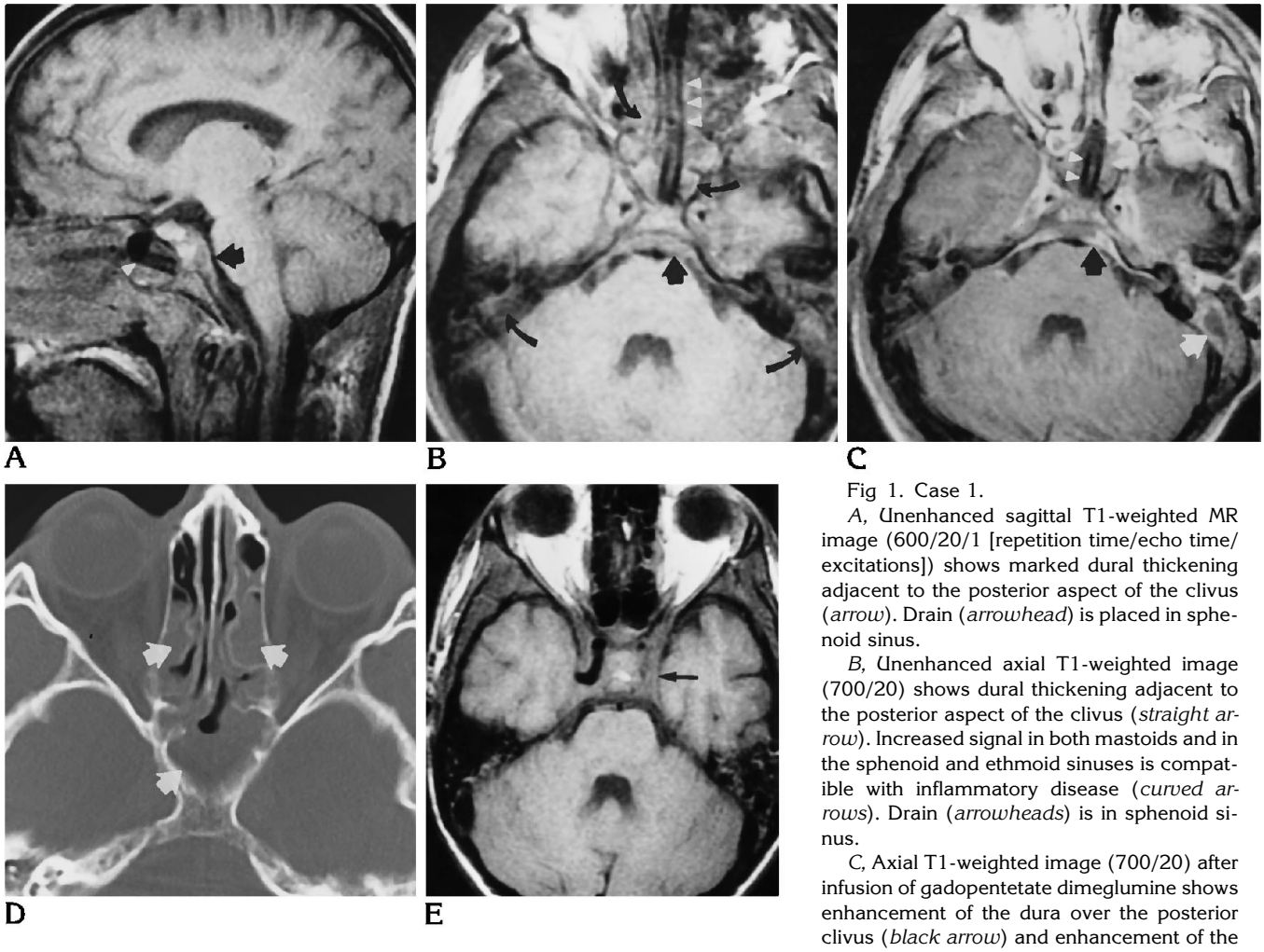

Fig 1. Case 1.

A, Unenhanced sagittal T1-weighted MR image (600/20/1 [repetition time/echo time/ excitations]) shows marked dural thickening adjacent to the posterior aspect of the clivus (*arrow*). Drain (*arrowhead*) is placed in sphenoid sinus.

B, Unenhanced axial T1-weighted image (700/20) shows dural thickening adjacent to the posterior aspect of the clivus (*straight arrow*). Increased signal in both mastoids and in the sphenoid and ethmoid sinuses is compatible with inflammatory disease (*curved arrows*). Drain (*arrowheads*) is in sphenoid sinus.

C, Axial T1-weighted image (700/20) after infusion of gadopentetate dimeglumine shows enhancement of the dura over the posterior clivus (*black arrow*) and enhancement of the inflammatory tissue in the left mastoid (*white arrow*). Drain (*arrowheads*) is in sphenoid sinus.

D, Axial computed tomogram shows inflammatory disease of the ethmoid and sphenoid sinuses (*arrows*). The petrous apices are normal and unpneumatized.

E, Unenhanced axial T1-weighted image (700/20) 1 month later shows absence of a flow void in the cavernous portion of the left internal carotid artery (*arrow*) compatible with carotid occlusion.

Discussion

Pachymeningitis is a localized inflammation of the dura that occurs adjacent to an inflammatory or suppurative focus (1), such as sinusitis. Other causes of dural thickness include epidural abscess, rheumatoid arthritis, sarcoidosis, dural carcinomatosis, menengioma, lymphoma, syphilis, and idiopathic primary pachymeningitis (2).

Paralysis of the sixth cranial nerve, associated with acute suppurative otitis media and severe facial pain in the distribution of the trigeminal nerve, was described by Gradenigo in 1907. Chole and Donald (3) attributed Gradenigo syndrome to suppuration in the aerated petrous apex (3). The fifth-nerve symptoms are caused by dural inflammation over the petrous apex or irritation of the gasserian ganglion in Meckel cave. The sixth nerve was thought to be involved in the suppurative process in Dorello canal. The triad was uncommon, because only two of eight patients with petrous apicitis had an abducens nerve palsy. The authors attributed the change in clinical findings to the use of antibiotics. The petrous apex was not pneumatized in our patients.

The most common complication of paranasal sinusitis is orbital cellulitis, followed by intracranial complications (4). The prevalence of intracranial complications in patients hospitalized

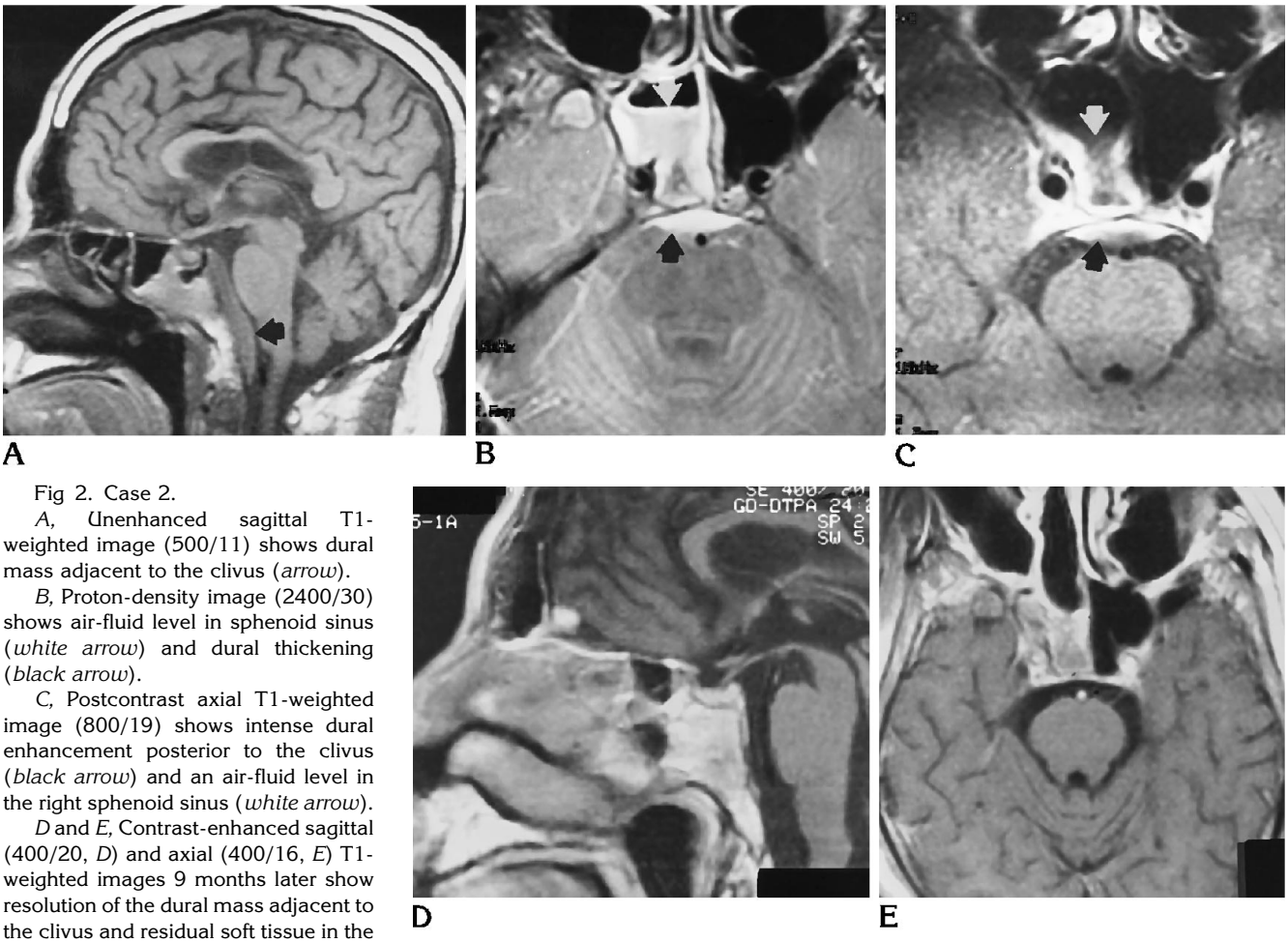

Fig 2. Case 2.

A, Unenhanced sagittal T1-weighted image (500/11) shows dural mass adjacent to the clivus (*arrow*).

B, Proton-density image (2400/30) shows air-fluid level in sphenoid sinus (*white arrow*) and dural thickening (*black arrow*).

C, Postcontrast axial T1-weighted image (800/19) shows intense dural enhancement posterior to the clivus (*black arrow*) and an air-fluid level in the right sphenoid sinus (*white arrow*).

D and *E,* Contrast-enhanced sagittal (400/20, *D*) and axial (400/16, *E*) T1-weighted images 9 months later show resolution of the dural mass adjacent to the clivus and residual soft tissue in the sphenoid sinus, thought to be from surgical repair of the defect in the wall of the sphenoid sinus.

with a primary diagnosis of paranasal sinusitis is about 3.7% (4). In a combined institutional review of 649 patients, intracranial complications occurred in 24 cases, and included frontal lobe abscess (46%), meningitis (29%), subdural empyema (8%), cavernous sinus thrombosis (8%), and superior sagittal sinus thrombosis and osteomyelitis of the frontal bone (1 case each). Long-term morbidity occurred in 33% of patients, including hemiparesis or hypoesthesia in 17% and chronic seizure disorder in 12% (4).

In a review of 15 cases of acute sphenoid sinusitis, the diagnosis at initial presentation was correct in only 6 (5). Intracranial complications developed in 14 of the 15 patients, including 4 with aseptic meningitis and 2 with cavernous sinus thrombosis. Of 15 cases of chronic sphenoid sinusitis, only 9 were initially diagnosed correctly. The latter group of patients had symptoms such as headache varying in duration from 6 months to 3 years. Because presenting symptoms, such as headache and nasal discharge, are nonspecific, the diagnosis of sphenoid sinusitis can be delayed until intracranial complications occur. Alternatively, the intracranial manifestations are treated, but the source of infection is ignored (6). In our first case, the patient remained febrile and bilateral sixth-nerve palsies developed, even though the meningitis was treated. Follow-up MR also showed occlusion of the cavernous internal carotid artery.

The MR findings in our cases were sphenoid sinusitis with localized dural thickening and enhancement adjacent to the clivus. Intense dural enhancement after contrast administration has also been described in other causes of pachymeningitis (2). Many structures are in close proximity to the sphenoid sinus, including the second through the sixth cranial nerves; cavern-

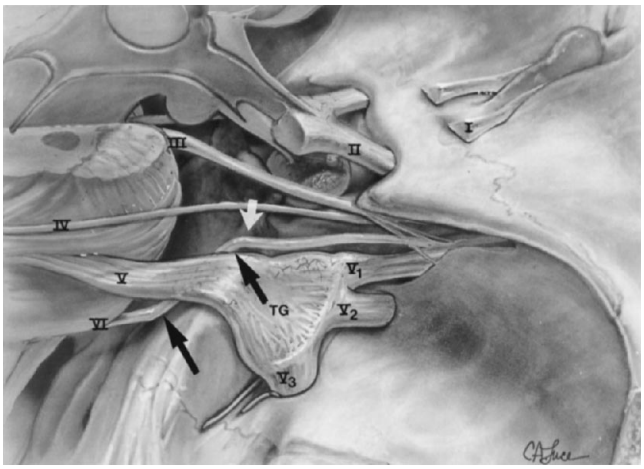

Fig 3. Diagram shows the long extradural course of the sixth cranial nerve (*black arrows*) on the posterior aspect of the clivus. The sixth nerve passes through Dorello canal at the level of the petrous apex (*white arrow*). *I* indicates olfactory nerve; *II*, optic nerve; *III*, oculomotor nerve; *IV*, trochlear nerve; *V*, trigeminal nerve; *VI*, abducens nerve; and *TG*, trigeminal nerve ganglion. (Reprinted with permission from Zide BM, Jelks GW. *Surgical Anatomy of the Orbit: Intracranial Dissection*. New York: Raven, 1985:68.)

ous sinus; internal carotid artery; pituitary gland; sphenopalatine artery, nerve, and ganglion; and dura (7, 8). Among the cranial nerves, the sixth cranial nerve has the longest course along the skull base (9). Axons of the abducens nerve exit the pontomedullary junction of the brain stem and ascend in the prepontine cistern to pierce the dura on the posterior aspect of the clivus. The abducens nerve follows a relatively long extradural course on the posterior clivus before entering the cavernous sinus beneath the petroclinoid ligament, at Dorello canal (Fig 3). Dorello canal is an osteofibrous conduit formed by the petrous apex and the petrosphenoidal ligament. The abducens nerve is particularly vulnerable to inflammation and compression along the posterior clivus and in Dorello canal, which may explain the sixth-cranial-nerve palsy in our patients. Neural deficits have been attributed to direct compression or compromise of vascular supply (2, 9). Infection can spread through venous channels, erosion or normal dehiscence of the sphenoid sinus wall (5, 8). Sphenoid sinusitis with adjacent pachymeningitis should be considered in patients presenting with sixth-cranial-nerve palsy. Prompt diagnosis and aggressive treatment of sphenoid sinusitis might prevent disasterous intracranial complications.

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