An Isolated Abducens Nerve Palsy Disclosing Pachymeningitis Secondary To Sphenoid Sinusitis

N Panda, P Singh, A Jain, C Reddy

Citation

Abstract
We investigated a rare case of isolated abducens palsy for its causes and found pachymeningitis secondary to sphenoiditis as primary cause for this. Further treatment of sphenoid sinusitis led to relief in headache and diplopia. We discuss the differential diagnosis of isolated abducens palsy and radiological features of pachymeningitis.

INTRODUCTION
Isolated sphenoid sinus disease is very uncommon and is most commonly inflammatory in origin. Isolated sphenoid sinusitis accounts for 1-2% of all sinus infections. Sphenoid sinusitis at onset may be asymptomatic but headache, localized to vertex, is the most common symptom. Sphenoid sinus is closely related to 13 vital structures resulting in wide range of signs and symptoms due to their involvement. A delay in diagnosis and treatment can lead to serious intracranial and orbital complications. We present a case of sphenoid sinusitis presenting as isolated abducens nerve palsy and also discuss the radiological features of pachymeningitis.

CASE REPORT
A 65-year-old-female presented with a 3-day history of headache. The headache was severe in intensity, localized to the vertex, disturbing her sleep, used to get aggravated by head movements and poorly relieved by analgesics. Only significant medical history was that she was a known hypertensive on medications for last 20 years. She presented to the emergency where neurological examination was found to be normal and computed tomography of brain ruled out subarachnoid hemorrhage. Two weeks later, she developed left sided lateral rectus palsy and headache persisted therefore MRI and CT angiography studies were done.

MRI revealed sheath like dural thickening along left cavernous sinus and superior orbital fissure that appeared isointense on T1 & T2 Weighted images. This was most marked along postero superior edges of cavernous sinuses. Dural enhancement was also seen along left tentorial edge of petroclival dura and the dura over basifrontal area (Fig.1). No definite enhancement of cisternal portion of 5th, 7th and 8th cranial nerve was seen. However 6th nerve was not visualized. It also showed heterogeneous soft tissue in left sphenoid and ethmoid sinus that was hypointense on T1 weighted and isointense to hypointense on T2 weighted images with areas of relative hypointensity (Fig. 2a,b). CT Angiography showed evidence of dolicoectatic basilar artery. There was no evidence of aneurysm within circle of Willis.

Figure 1
Figure 1: T1 Post contrast gradient echo axial section shows dural thickening along the left cavernous sinus and petroclival dura.
Figure 2
Figure 2: T1 and T2 fast spin echo images show a soft tissue in the left sphenoid sinus which appears hypointense on T1 weighted images (Fig.2a) and iso to hyperintense on T2 weighted images (Fig.2b).

Her leukocyte counts and biochemical profile were normal but ESR was 18 mm at the end of first hour. Biochemical analysis of CSF revealed 28 mg% protein, 61 mg% glucose and 8 units/litre of adenosine deaminase. Cell counts of CSF showed 250-leukocytes/ ml, 90% of which were polymorphs.

A diagnostic nasal endoscopy revealed purulent discharge in left sphenoid recess. Staphylococcus aureus sensitive to amoxycillin and cefotaxime was isolated from the pus. She was given intravenous amoxy clavulanic acid antibiotic along with metronidazole for one week after which she was switched over to oral amoxyclavulanic acid antibiotic. She was also given steam inhalation and nasal decongestant drops under strict blood pressure monitoring. With this therapy her headache and diplopia disappeared. Her nasal symptoms subsided markedly.

DISCUSSION
Headache followed by visual dysfunction are the commonest presentation of sphenoid sinusitis. Visual dysfunction secondary to adjacent posterior ethmoidal or sphenoidal sinusitis rarely occurs without associated orbital or cavernous sinus inflammation signs such as chemosis, proptosis, multiple cranial nerve palsies etc.

An abducens (sixth cranial nerve) nerve palsy, which results in a lateral rectus muscle paresis is the most common type of ocular nerve palsy. Among myriad of causes of abducens nerve palsy (Table 1), sinusitis as a cause of isolated abducens palsy is rarely thought of especially in absence of associated symptoms as in this case. To understand how sphenoid sinusitis can cause cranial nerve palsies, anatomical facts are necessary to be recapitulated.

Table 1: Causes of abducens Nerve Palsy. (Source: Reference 3)

- Nuclear
  - Congenital, e.g. Mobius syndrome
  - Tumour
  - Infarction
  - Wernicke-Korsakoff syndrome

- Fascicular
  - Demyelination
  - Infarction, tumour

- Subarachnoid
  - Meningitis
  - Subarachnoid haemorrhage
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- Post-infections
- Clivus tumor
- Trauma
- Compression by aneurysm or ecstatic vessels
- Sarcoidosis

- Petrous
  - Mastoid or petrous bone tip infection
  - Fracture of petrous bone
  - Aneurysm
  - Thrombosis of inferior petrosal sinus
  - Downward displacement of brainstem by supratentorial mass (raised intracranial pressure)
  - Following lumbar puncture, epidural anæsthesia
  - Trigeminal schwannoma

- Cavernous sinus and superior orbital fissure
  - Aneurysm
  - Tumour (meningioma, naspharyngeal carcinoma, pituitary adenoma)
  - Carotid-cavernous fistula
  - Thrombosis
  - Dural arterio-venous malformation
  - Tolosa Hunt syndrome
  - Herpes Zoster
  - Sinusitis

- Orbital
  - Tumour
  - Localization uncertain
  - Infarction (often associated with hypertension or diabetes)
  - Migraine.

There are number of structures intimately involved with the sphenoid sinus. These are cranial nerves II through VI, internal carotid artery, cavernous sinus, pituitary gland, sphenopalatine artery, nerve and ganglion and dura. Some of these can indent the wall of the sinus, depending upon the degree of pneumatization. Most commonly internal carotid artery is seen posterolaterally in 65% of cases and optic nerve is seen superolaterally in 50%. Studies demonstrate a bony dehiscence in sphenoid sinus over the optic nerve in 4% of cadavers and between the sinus and the carotid artery in 8%. In addition 78% of cadavers had less than 0.5 mm thin bone between the sinus and the optic nerve. These variants show a lack of substantial barrier to the spread of infection from sphenoiditis, putting a patient at risk for a complication.\textsuperscript{4,5}

The internal carotid artery and sixth cranial nerve are within the cavernous sinus itself, closer to the sphenoid sinus, while abducen nerve lies lateral to the internal carotid artery and medial to the ophthalmic division of the trigeminal nerve, in close proximity to the oculomotor and trochlear nerves. Despite this, lesions in the sinus can lead to isolated abducens nerve palsy, probably because the nerve is not tethered to the dual wall or because of its medial location in sinus.\textsuperscript{1,3}

The resistance of the dura mater to infection usually limits the inflammatory process to epidural space. In occasional cases, spread of infection occurs within the layer of duramater causing pachymeningitis, which may subsequently spread into subdural space. The pachymeningitis can be very indolent and extensive and its etiology is not restrictive to pyogenic infection. Syphilis is classical example of pachymeningitis. Other causes are tuberculosis, sarcoidosis or autoimmune vasculitis. CSF examination is usually consistent with a parameningeal focus of infection and is characterized by elevated protein and increased leucocytes. The CSF WBC count generally does not exceed 150/mm\textsuperscript{3} unless there is coexistent meningitis and cells are usually mixed polymorphs and lymphocytes.\textsuperscript{6}

In this case, MRI revealed dural thickening along left cavernous sinus and superior orbital fissure that appeared isointense on T1W & T2W images. This was most marked along postero superior edges of cavernous sinuses with dural enhancement along left tentorial edge of petroclival dura.
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where abducens nerve enters through Dorello’s canal.

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 reported before. This is an unusual isolated complication of sphenoid sinusitis. Prompt diagnosis and therapy may avoid significant intracranial complications.

We treated our patient with intravenous antibiotics, topical decongestants and steam inhalation that brought relief from headache and diplopia. The other rare cause of abducens palsy that was considered in this case was dolicoectatic basilar artery that was detected on CT Angiography. The abducens nerve passes almost vertically in front of the clivus where it may be damaged by enlarged ectatic basilar artery or by tumors such as chordoma menenigioma or nasopharyngeal carcinoma. But improvement with conservative treatment in our case goes against this. Tolosa Hunt syndrome was also thought of but recovery without steroids is a point against this entity.

CONCLUSION

We investigated a rare case of isolated abducens palsy for its causes and found pachymeningitis secondary to sphenoiditis as primary cause for this. Further treatment of sphenoid sinusitis led to relief in headache and diplopia.

CORRESPONDENCE TO

Dr. Naresh Panda, FRCS, Ed. Additional Professor, Dept. of Otolaryngology and Head & Neck Surgery Postgraduate Institute of Medical Education and Research, Chandigarh – 160 012, INDIA. Fax: 91-172-2744401, 2745078 Phone: 0091-0172-2747586 to 94 Ext. 6759 Email: npanda@satyam.net.in

References

Author Information

Naresh Kumar Panda, MS, DNB, FRCS Ed
Additional Professor, Department of Otolaryngology and Head & Neck Surgery, Postgraduate Institute of Medical Education and Research

Paramjeet Singh, MD
Assistant Professor, Department of Radiodiagnosis and Imaging, Postgraduate Institute of Medical Education and Research

Ajay Jain, MS
Additional Professor, Department of Otolaryngology and Head & Neck Surgery, Postgraduate Institute of Medical Education and Research

C. Ekambar Eshwara Reddy, MS DNB
Senior Resident, Department of Otolaryngology and Head & Neck Surgery, Postgraduate Institute of Medical Education and Research