Nasal foreign body presenting as pansinusitis with superior orbital fissure syndrome

*Niharika Shetty1, Nireeksha Shetty2, Praveen Nayak K1

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Introduction

Pansinusitis is inflammation of the mucosa of all four paranasal sinuses1. Common symptoms of sinusitis include nasal congestion, nasal discharge, facial pain, and decreased perception of smell2. In children, if timely management of sinusitis is not accomplished, complications can occur involving the orbit and the intracranial structures3. It can also lead to palsies of the cranial nerves (CN) III (oculomotor), IV (trochlear), V (trigeminal), and VI (abducens)4. Ptosis refers to a drooping or inferior displacement of the upper eyelid with associated narrowing of the vertical palpebral fissure5. It can affect an individual of any age, from newborns to the elderly5. The causes of ptosis in childhood include congenital ptosis, oculomotor nerve palsy, Horner's syndrome, myasthenia gravis, mitochondrial disorders, intracranial space-occupying lesions, and orbital cellulitis5. Pansinusitis as a cause of ptosis is extremely rare. There are only a few cases reported that attribute pansinusitis as a cause for ptosis6. We present a child with a nasal foreign body resulting in chronic pansinusitis and bilateral ptosis with ophthalmoplegia. An extensive literature search did not yield any similar cases.

Case report

A 6-year-old boy presented with complaints of progressive ptosis and diplopia for 2 weeks without diurnal variation. He also complained of a headache for 2 days. There was no history of fever, orbital pain, orbital swelling, or proptosis. On examination, the child was found to have bilateral ptosis with bilateral medial deviation of the eyeballs, suggestive of CN III and VI palsies (Figure 1).

His higher mental functions were normal. Visual acuity, vision fields, fundoscopy, direct and indirect pupillary reflexes were normal. The rest of the nervous examination was normal. His muscle tone, power and reflexes were normal. There were no signs of meningeal irritation. The differential diagnosis included intracranial space-occupying lesion (ICSOL), cavernous sinus thrombosis (CST), superior orbital fissure syndrome (SOFS) and orbital apex syndrome (OAS).

Full blood count showed that the haemoglobin level was 11.3 g/dl, total leucocyte count was 8700 cells/ cu mm (61% neutrophils, 33% lymphocytes, 6% eosinophils) and platelet count was 320,000 cells/ cu mm. The child was subjected to brain magnetic resonance imaging (MRI) rather than a computed tomography (CT) scan with orbital cuts as ICSOL was our first diagnosis. It revealed a metallic artefact lodged in the left nasal cavity (Figure 2). It also showed mild inflammatory changes in the left lamina papyracea and medial intra-orbital fat (Figure 3) and mucosal thickening of the bilateral maxillary, ethmoid, sphenoid, and frontal sinuses. (Figure 4a and Figure 4b). Figures 2 and 4 showed an opacity in the right maxillary cavity which was reported as thickening of the sinus mucosa suggestive of sinusitis. No other collection was reported.

Figure 1: Bilateral ptosis with bilateral medial deviation of eyelid

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https://orcid.org/0000-0002-3288-3595

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After reviewing the MRI of brain, the nasal foreign body was removed under direct visualization. The procedure was done without general anaesthesia. No endoscopy was performed. The foreign body was a metallic screw from a child’s toy (Figure 5). Child could not remember the exact duration of this foreign body. We diagnosed it as a case of a forgotten nasal foreign body that led to chronic pansinusitis, which in turn led to CN III and VI involvement. The child was started on antibiotics and steroid nasal spray.
The child was discharged. After 3 months, the child was found to have no sequelae. Ptosis and ophthalmoplegia had resolved completely (Figure 6).

Discussion

The paranasal sinuses are four air-filled paired structures located within the bones of the skull and face. The ethmoid and maxillary sinuses develop in the third month of gestation and are usually present at birth.10 They reach adult size at the age of ten. Sphenoid sinuses are seen on imaging before 3 years of age, become aerated by 5 years, and reach adult size by age of 12-14. The frontal sinus is aerated by 5-6 years of age and reaches adult size by 19 years of age.10 The maxillary, anterior ethmoid and frontal sinuses drain into the osteo-meatal complex in the middle meatus of the nasal cavity. The posterior ethmoid and the sphenoid sinus drain into the superior meatus. The mucosa of these sinuses is in continuity with that of the nasal mucosa; therefore, any process that affects the mucosa of the nose may also affect the sinus mucosa.

Key factors in the pathogenesis of sinusitis include ostial obstruction, ciliary dysfunction, and thickening of sinus secretions.10 Ostial obstruction results from mucosal swelling or a direct mechanical effect. Foreign bodies cause mechanical obstruction of the ostium resulting in sinusitis. In our case, the child had introduced a screw into the nasal cavity that acted as a foreign body, causing a mechanical obstruction that led to sinusitis.

Sinusitis can rarely, if left untreated, lead to complications. The proximity of the orbit to the paranasal sinus makes it more vulnerable to complications arising from sinusitis.10 There is a contiguous spread of infection/inflammation from the ethmoid sinus to the orbit through the lamina papyracea resulting in orbital complications.

Orbital complications include pre-septal cellulitis, orbital cellulitis, subperiosteal abscess, orbital abscess, superior orbital fissure syndrome, and orbital apex syndrome.11 The intracranial complications of sinusitis are meningitis, extradural abscess, subdural abscess, brain abscess and cavernous sinus thrombosis.11

The superior orbital fissure forms a passage for the superior ophthalmic vein (SOV), CN III, IV, VI and the V1 of the trigeminal nerve.12 The orbital apex is formed by the superior orbital fissure and the optic canal, which has the optic nerve and the ophthalmic artery. Since the superior orbital fissures and the orbital apex transmit multiple structures within a narrow conduit, even a minor injury/lesion can lead to significant symptoms and signs. Both superior orbital fissure syndrome and orbital apex syndrome can have similar symptoms due to their overlapping anatomy.

CN III, after emerging from the anterior end of the cavernous sinus, lies in close relation to the upper part of the lateral wall of the sphenoidal and ethmoidal sinuses.16 CN IV and VI, though more laterally placed in relation to the sphenoid sinus, may get involved by the extension of the infective process into the superior orbital fissure.

CN VI is in relation to the posterior wall of the sphenoid sinus in Dorello’s canal, which is bounded by the posterior clinoid process, the apex of the petrous bone and the petro-clinoid ligament.16 The possible explanation for the involvement of cranial nerves in sinusitis includes the spread of inflammation to involve the nerve sheath, mechanical compression of the nerve in the cavernous sinus and ischaemic infarction of the nerve due to cavernous sinus thrombosis. The involvement of the superior division of CN III in the disease process can lead to ptosis, and that of the inferior division of CN III, CN IV and VI leads to ophthalmoplegia.

Ptosis can be caused by the dysfunction of nerves or muscles regulating the elevation of the eyelid. Two muscles are involved in the elevation of eyelids viz. levator palpebrae superioris, innervated by the oculomotor nerve and Müller’s muscle innervated by the cervical sympathetic system. Ptosis can be acute or chronic, sporadic or familial, unilateral or bilateral, congenital or acquired, progressive or non-progressive. Our patient had acute-onset bilateral ptosis.

The causes for acute onset ptosis can be acute botulism, Miller-Fisher syndrome, or CN III palsy secondary to trauma, inflammation or neurotoxic events. Our patient did not manifest acute botulism symptoms, including bulbar paralysis and symmetrical descending flaccid paralysis. Miller-Fisher syndrome, a variant of Guillain-Barre syndrome, was not considered as the child did not have ataxia or areflexia. Myasthenia gravis presents with varying weakness of the extraocular muscles, bulbar muscles and limb muscles. Patients with myasthenia gravis have weakness on exertion, which improves with rest. Another differential for ptosis is Horner syndrome, caused by a disruption of the sympathetic nervous system pathway. It affects the ipsilateral Müller’s muscle and the
ipsilateral side of the face. Since our patient had bilateral ptosis, this diagnosis was not considered.

In our patient, forgotten nasal foreign body may have led to local sinus inflammation followed by pansinusitis and III and VI cranial nerve palsies.

References