Nasal foreign body presenting as pansinusitis with superior orbital fissure syndrome

***Niharika Shetty[1], Nireeksha Shetty[2], Praveen Nayak K[1]**

Sri Lanka Journal of Child Health, 2024; **53**(2): 168-171
DOI: http://doi.org/10.4038/sljch.v53i2.10683
(Key words: Pansinusitis, Ptosis, Nasal foreign body)

Introduction

Pansinusitis is inflammation of the mucosa of all four paranasal sinuses[1]. Common symptoms of sinusitis include nasal congestion, nasal discharge, facial pain, and decreased perception of smell[2]. In children, if timely management of sinusitis is not accomplished, complications can occur involving the orbit and the intracranial structures[3]. 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 fissure[5]. It can affect an individual of any age, from newborns to the elderly[5]. 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 cellulitis[5]. Pansinusitis as a cause of ptosis is extremely rare. There are only a few cases reported that attribute pansinusitis as a cause for ptosis[6]. 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).

[1]*Department of Paediatrics,* [2]*Department of Radiodiagnosis, KS Hegde Medical Academy, Nitte (Deemed to be University), Mangalore, India*
**Correspondence:* drniharika.shetty@nitte.edu.in
https://orcid.org/0000-0002-3288-3595

(Received on 20 July 2023: Accepted after revision on 18 August 2023)
The authors declare that there are no conflicts of interest
Personal funding was used for the project.

Figure 1: *Bilateral ptosis with bilateral medial deviation of eyeball*
Permission given by parents to publish photograph

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).

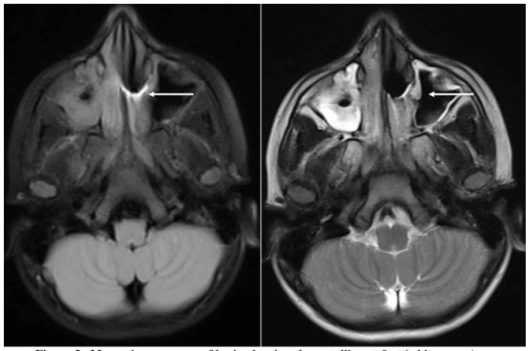

Figure 2: *Magnetic resonance of brain showing the metallic artefact (white arrow)*

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.

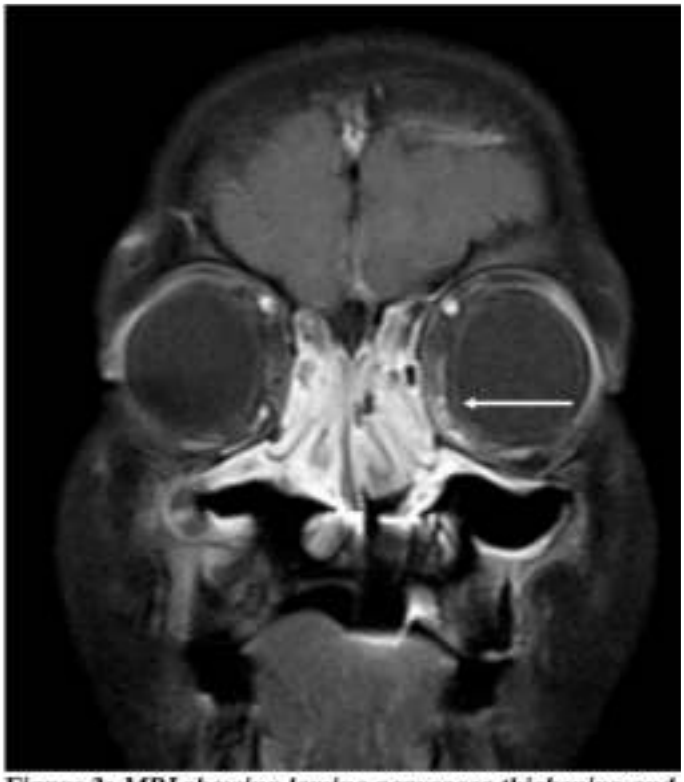

Figure 3: ***MRI showing lamina papyracea thickening and periorbital inflammation on left side (white arrow)***

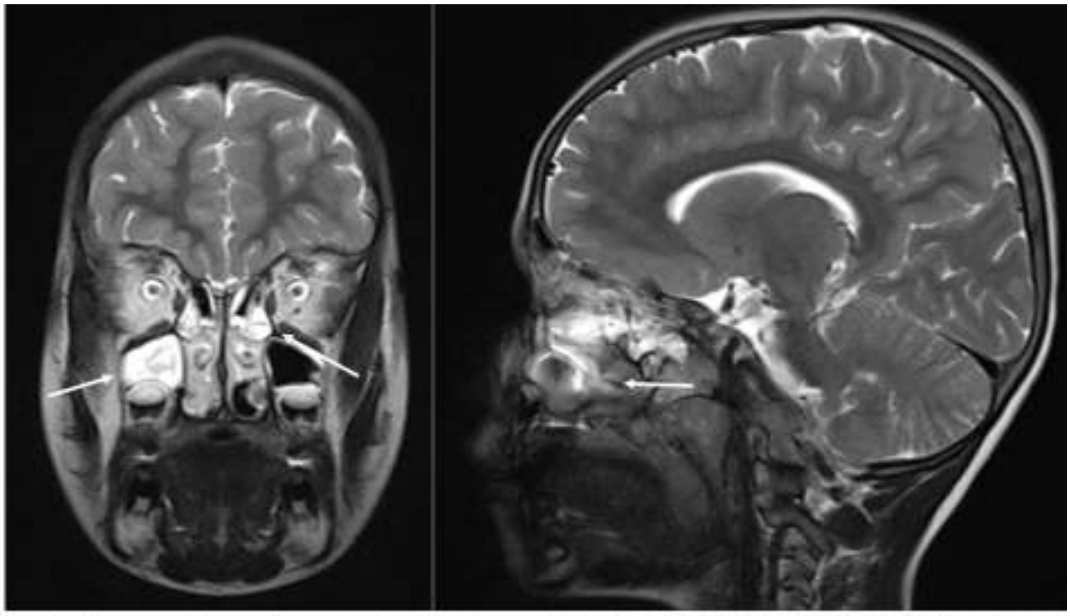

Figure 4: ***MRI showing mucosal thickening of bilateral maxillary, ethmoid, sphenoid and frontal sinuses (white arrow)***

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.

Figure 5: ***Image of the nasal foreign body- a screw of a children's toy***

The child was discharged. After 3 months, the child was found to have no sequelae. Ptosis and ophthalmoplegia had resolved completely (Figure 6).

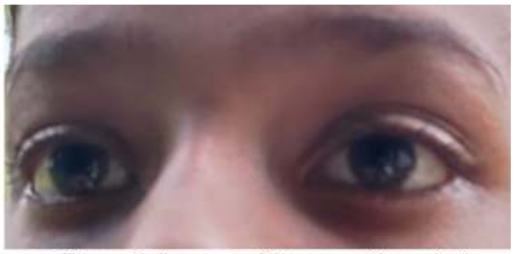

Figure 6: ***Image on follow up with no ptosis***
Permission given by parents to publish photograph

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[3,10]. They reach adult size at the age of ten[10]. 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[10]. 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[10]. The posterior ethmoid and the sphenoid sinus drain into the superior meatus[10]. 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[2].

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[2]. Foreign bodies cause mechanical obstruction of the ostium resulting in sinusitis[10]. 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[10].

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[13,14]. 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[15]. Both superior orbital fissure syndrome and orbital apex syndrome can have similar symptoms due to their overlapping anatomy[9].

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[16].

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[8].

Ptosis can be caused by the dysfunction of nerves or muscles regulating the elevation of the eyelid[5]. 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[5]. Ptosis can be acute or chronic, sporadic or familial, unilateral or bilateral, congenital or acquired, progressive or non-progressive[5]. 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[5]. Our patient did not manifest acute botulism symptoms, including bulbar paralysis and symmetrical descending flaccid paralysis[5]. Miller-Fisher syndrome, a variant of Guillain-Barre syndrome, was not considered as the child did not have ataxia or areflexia[5]. Myasthenia gravis presents with varying weakness of the extraocular muscles, bulbar muscles and limb muscles[17]. Patients with myasthenia gravis have weakness on exertion, which improves with rest[17]. Another differential for ptosis is Horner syndrome, caused by a disruption of the sympathetic nervous system pathway[5]. It affects the ipsilateral Müller's muscle and the

ipsilateral side of the face[5]. 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

1. Rosenfeld RM, Piccirillo JF, Chandrasekhar SS, Brook I, Kumar KA, Kramper M, *et al.* Clinical Practice Guideline (Update): Adult sinusitis. *Otolaryngology- Head and Neck Surgery* 2015; **152** (2 Suppl): S1-S39. https://doi.org/10.1177/0194599815572097 PMid: 25832968
2. Battisti AS, Modi P, Pangia J. Sinusitis. [Updated 2023 Mar 2]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK470383/
3. DeMuri G, Wald ER. Acute bacterial sinusitis in children. *Pediatrics in Review* 2013; **34**(10): 429-37; quiz 437. https://doi.org/10.1542/pir.34-10-429 PMid: 24085791
4. Marmura MJ, Silberstein SD. Headaches caused by nasal and paranasal sinus disease. *Neurologic Clinics* 2014; **32**(2): 507–23.
5. Pavone P, Cho SY, Praticò AD, Falsaperla R, Ruggieri M, Jin DK. Ptosis in childhood: A clinical sign of several disorders: Case series reports and literature review. *Medicine (Baltimore)* 2018; **97**(36): e12124. https://doi.org/10.1097/MD.0000000000012124 PMid: 30200099 PMCid: PMC6133583
6. Wilbanks ND, Filutowski OR, Maldonado MD, Karcioglu ZA. Isolated left upper eyelid ptosis with pansinusitis and contralateral otitis media in a 9-year-old boy. *American Journal of Ophthalmology Case Reports* 2018; **11**: 6-9. https://doi.org/10.1016/j.ajoc.2018.04.023 PMid: 30051000 PMCid: PMC6058058
7. Allen CMC, Lueck CJ, Dennis M. Neurological disease. In: Colledge NR, Walker BR, Ralston SH, editors. Davidson's Principles and Practice of Medicine. 21st edition. China: Elsevier Limited; 2010.1215 https://doi.org/10.1016/B978-0-70203085-7.00026-2 PMid: 20061431 PMCid: PMC2841537
8. Plewa MC, Tadi P, Gupta M. Cavernous sinus thrombosis. [Updated 2022 Aug 7]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK448177/
9. Badakere A, Patil-Chhablani P. Orbital apex syndrome: A review. *Eye Brain* 2019; **11**: 63-72. https://doi.org/10.2147/EB.S180190 PMid: 31849556 PMCid: PMC6913296
10. DeMuri G, Wald ER. Clinical Practice. Acute bacterial sinusitis in children. *New England Journal of Medicine* 2012; **367**(12): 1128-34. https://doi.org/10.1056/NEJMcp1106638 PMid: 22992076
11. Chandler JR, Langenbrunner DJ, Stevens ER. The pathogenesis of orbital complications in acute sinusitis. *Laryngoscope* 1970; **80**: 1414-28. https://doi.org/10.1288/00005537197009000-00007 PMid: 5470225
12. Reymond J, Kwiatkowski J, Wysocki J. Clinical anatomy of the superior orbital fissure and the orbital apex. *Journal of Cranio-Maxillofacial Surgery* 2008;36(6): 346-53. https://doi.org/10.1016/j.jcms.2008.02.004 PMid: 18450458
13. Standring S (ed.) Gray's anatomy: The anatomical basis of clinical practice. 41st ed. London: Elsevier, 2015.
14. Harnsberger HR, Glastonbury CM, Michel MA, *et al.* Diagnostic imaging: Head and neck, 2nd ed. Philadelphia: Amirsys, 2011.
15. Chen CT, Chen YR. Traumatic superior orbital fissure syndrome: Current management. *Craniomaxillofacial Trauma and Reconstruction* 2010; **3**(1): 9-16. https://doi.org/10.1055/s-0030-1249369 PMid: 22110813 PMCid: PMC3052661
16. Dimsdale H, Phillips DG. Ocular palsies with nasal sinusitis. *Journal of Neurology, Neurosurgery and Psychiatry* 1950; **13**(3): 225-36. https://doi.org/10.1136/jnnp.13.3.225 PMid: 14774745 PMCid: PMC497158
17. Beloor Suresh A, Asuncion RMD. Myasthenia Gravis. [Updated 2022 Dec 7]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 Jan. Available from: https://www.ncbi.nlm.nih.gov/books/NBK559331/