



Bilateral sixth nerve paralysis as a sequelae of cavernous sinus thrombosis in a child
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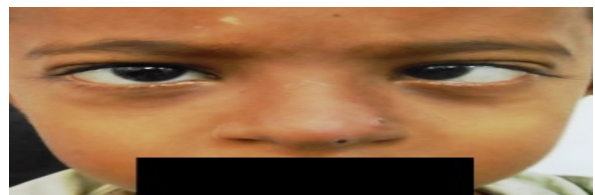
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Abstract : A five year old male child presented with complaint of inward deviation of the left eye for the past two days. There was history of fever with cellulitis and boil over the tip of nose for the past two weeks for which the child was treated outside. On general examination, there was cellulitis over the tip of nose. The child was febrile. On ocular examination, visual acuity was 6/6 in both eyes. Anterior segment examination and fundus were normal. Orthoptic assessment of the child revealed left convergent squint. There was limitation of abduction in both eyes right eye more than left eye. A clinical diagnosis of bilateral sixth nerve palsy probably due to cavernous sinus thrombosis due to spread of infection from dangerous zone of face was made. The child was advised emergency MRI brain with contrast and systemic antibiotics and steroids. The child was started on parenteral cloxacillin, amikacin, metrogyl, and cefotaxime. Inj dexamethasone 2 mg was given intravenously for three times a day in paediatric hospital. MRI brain was normal. After one week there was improvement in abduction of both eyes with residual abduction limitation in left eye. With the clinical features of bilateral sixth nerve palsy with infection in the dangerous area of face suggestive of cavernous sinus thrombosis as a possibility, the child was treated with parenteral broad spectrum antibiotics and steroids. Clinically the child improved. This is to emphasize the importance of starting earlier broad spectrum antibiotics and steroids in case of suspicious facial infections to prevent the complications of cavernous sinus thrombosis.

Keyword : Cavernous sinus thrombosis, Dangerous area of face

Case history and examination:

A five year old male child presented with complaint of inward deviation of the left eye for the past two days. There was history of fever with cellulitis and boil over the tip of nose for the past two weeks for which the child was treated outside. There was no history of trauma. There was no history of headache and vomiting. There was no other systemic complaints.

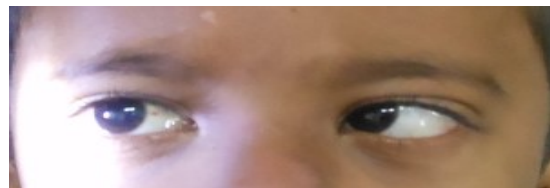


Profile of the child showing boil over the tip of the nose with left convergent squint

On general examination, there was cellulitis over the tip of nose. The child was febrile. Pulse rate was 100/minute. Examination of central nervous system, cardiovascular system, abdomen and respiratory system were normal.

On ocular examination, visual acuity was 6/6 in both eyes. Anterior segment examination was normal with brisk pupillary reaction. Posterior segment examination of both eyes revealed clear media with normal disc and vessels. Macula was normal.

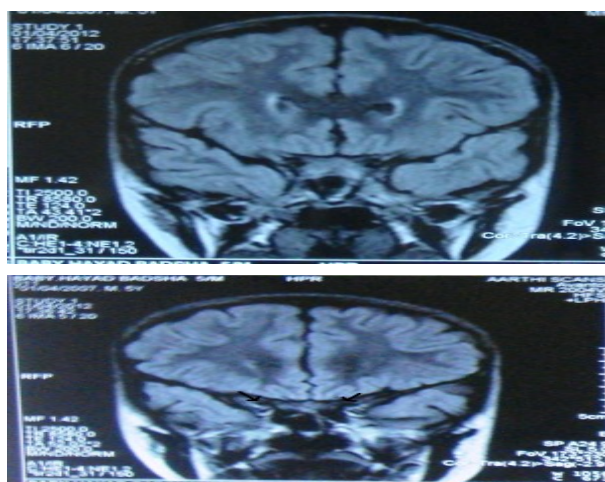
Orthoptic assessment of the child revealed left convergent squint. There was limitation of abduction in both eyes right eye more than left eye. Forced duction test was negative in both eyes. The child was not co-operative for diplopia charting, fields and colour vision. Tension was normal in both eyes.



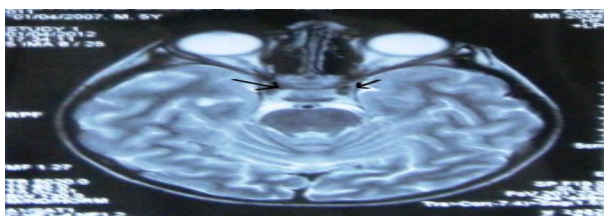


Picture showing abduction limitation of the right eye at presentation Picture showing abduction limitation of the left eye at presentation

A clinical diagnosis of bilateral sixth nerve palsy probably due to cavernous sinus thrombosis due to spread of infection from dangerous zone of face was made. The child was advised emergency MRI brain with contrast and systemic antibiotics and steroids.



Heavy T2 weighted FLAIR sequences showing normal cavernous sinus



T2 MRI - Axial section showing normal cavernous sinus

The child was started on parenteral cloxacillin, amikacin, metrogyl, and cefotaxime. Inj dexamethasone 2 mg was given intravenously for three times a day in paediatric hospital. MRI brain was normal. As the child's parents were not affordable MRI brain with contrast could not be done. After one week the child was reviewed in our OPD. There was improvement in abduction of both eyes with residual abduction limitation in left eye. Vision, anterior segment and fundus of both eyes were normal. The antibiotics were continued for three weeks.



Picture showing the abduction recovery in the right eye after treatment



Picture showing the abductin recovery in the left eye after treatment

Discussion:

Cavernous sinus thrombosis (CST) was initially described by Bright in 1831 as a complication of epidural and subdural infections.

Cavernous sinus thrombosis is usually a late complication of an infection of the central face or paranasal sinuses¹. Other causes include bacteremia, trauma, and infections of the ear or maxillary

teeth. Cavernous sinus thrombosis is generally a fulminant process with high rates of morbidity and mortality. Fortunately, the incidence of cavernous sinus thrombosis has been decreased greatly with the advent of effective antimicrobial agents.

The cavernous sinuses are irregularly shaped, trabeculated cavities located at the base of the skull². The cavernous sinuses are the most centrally located of the dural sinuses and lie on either side of the sella turcica. These sinuses are just lateral and superior to the sphenoid sinus and are immediately posterior to the optic chiasm.

The internal carotid artery with its surrounding sympathetic plexus passes through the cavernous sinus. The third, fourth, and sixth cranial nerves are attached to the lateral wall of the sinus. The ophthalmic and maxillary divisions of the fifth cranial nerve are embedded in the wall.

The cavernous sinuses receive venous blood from the facial veins (via the superior and inferior ophthalmic veins) as well as the sphenoid and middle cerebral veins. They, in turn, empty into the inferior petrosal sinuses, then into the internal jugular veins and the sigmoid sinuses via the superior petrosal sinuses³. This complex web of veins contains no valves; blood can flow in any direction depending on the prevailing pressure gradients. Since the cavernous sinuses receive blood via this distribution, infections of the face including the nose, tonsils, and orbits can spread easily by this route.

Staphylococcus aureus accounts for approximately 70% of all infections. *Streptococcus pneumoniae*, gram-negative bacilli, and anaerobes can also be seen. Fungi are a less common pathogen and may include *Aspergillus* and *Rhizopus* species.

Patients generally have sinusitis or a midface infection (most commonly a furuncle) for 5-10 days. The clinical presentation is usually due to the venous obstruction as well as impairment of the cranial nerves that are near the cavernous sinus. Headache is the most common presentation symptom and usually precedes fevers, periorbital edema, and cranial nerve signs. In some patients, periorbital findings do not develop early on, and the clinical picture is subtle. Some cases of CST may present with focal cranial nerve abnormalities.

As the infection tracts posteriorly, patients complain of orbital pain and fullness accompanied by periorbital edema and visual disturbances. Without effective therapy, signs appear in the contralateral eye by spreading through the communicating veins to the contralateral cavernous sinus⁴. Eye swelling begins as a unilateral process and spreads to the other eye within 24-48 hours via the intercavernous sinuses. This is pathognomonic for CST.

The patient rapidly develops mental status changes including confusion, drowsiness, and coma from CNS involvement and sepsis. Death follows shortly thereafter. **Signs:** Lateral gaze palsy (isolated cranial nerve VI) is usually seen first since CN VI lies freely within the sinus in contrast to CN III and IV, which lie within the lateral walls of the sinus.

Ptosis, mydriasis, and eye muscle weakness from cranial nerve III dysfunction
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Signs of increased intraocular pressure (IOP) may be observed.

Pupillary responses are sluggish.
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Decreased visual acuity is common owing to increased IOP and traction on the optic nerve and

central retinal artery. Hypoesthesia or hyperesthesia in dermatomes supplied by the V1 and V2 branches of the fifth cranial nerve. Appearance of signs and symptoms in the contralateral eye is diagnostic of CST, although the process may remain confined to one eye. Meningeal signs, including nuchal rigidity and Kernig and Brudzinski signs, may be noted. Systemic signs indicative of sepsis are late findings. They include chills, fever, shock, delirium, and coma.

Imaging techniques:

In current practice, computed tomography (CT) scan or magnetic resonance imaging (MRI) with contrast is the modality of choice to confirm the diagnosis of CST. MRI with MR venogram (MRV) is the preferred imaging choice as the MRV will show the absence of venous flow in the affected cavernous sinus^{5,6}. On noncontrast CT, thrombosis of the cavernous sinus can be appreciated as increased density. The introduction of intravenous contrast can reveal filling defects within the cavernous sinus as well as thickening of the superior ophthalmic vein. This is the earliest sign of CST. Nevertheless, CT and MRI scan findings may be subtle, and a negative scan cannot rule out CST reliably when the clinical suspicion is high⁸.

Treatment:

The mainstay of therapy for cavernous sinus thrombosis is early and aggressive antibiotic administration. Although *S. aureus* is the usual cause, broad-spectrum coverage for gram-positive, gram-negative, and anaerobic organisms should be instituted pending the outcome of cultures.

Empiric antibiotic therapy should include a penicillinase-resistant penicillin plus a third- or fourth-generation cephalosporin. If dental infection or other anaerobic infection is suspected, an anaerobic coverage should also be added. IV antibiotics are recommended for a minimum of 3-4 weeks^{7,9}.

Corticosteroids may help to reduce inflammation and edema and should be considered as an adjunctive therapy. They should be instituted after antibiotic coverage. Dexamethasone or hydrocortisone should be considered¹⁰.

Surgery on the cavernous sinus is technically difficult and has never been shown to be helpful. The primary source of infection should be drained, if feasible (eg, sphenoid sinusitis, facial abscess). It is important to recognize the infected sphenoid sinus early and to prevent spread of the infection to the cavernous sinus.

Controversy exists on the use of anticoagulation for cavernous sinus thrombosis.

Conclusion:

In the case presented above, with the clinical features of bilateral sixth nerve palsy with infection in the dangerous area of face suggestive of cavernous sinus thrombosis as a possibility, the child was treated with parenteral broad spectrum antibiotics and steroids. Clinically the child improved. This is to emphasize the importance of starting earlier broad spectrum antibiotics and steroids in case of suspicious facial infections to prevent the complications of cavernous sinus thrombosis.

References:

1. Laupland KB. Vascular and parameningeal infections of the head and neck. *Infect Dis Clin North Am.* Jun 2007;21(2):577-90, viii
2. Seow VK, Chong CF, Wang TL, Lin CM, Lin IY. Cavernous sinus thrombophlebitis masquerading as ischaemic stroke: a catastrophic pitfall in any emergency department. *Emerg Med J.* Jun 2007;24(6):440.
3. Duong DK, Leo MM, Mitchell EL. Neuro-ophthalmology. *Emerg Med Clin North Am.* Feb 2008;26(1):137-80, vii.
4. Misra UK, Kalita J, Bansal V. D-dimer is useful in the diagnosis of cortical venous sinus thrombosis. *Neurol India.* Jan-Feb 2009;57(1):50-4.
5. Coutinho J, de Bruijn SF, Deveber G, Stam J. Anticoagulation for cerebral venous sinus thrombosis. *Cochrane Database Syst Rev.* Aug 10 2011;CD002005.
6. Naesens R, Ronsyn M, Druwé P, Denis O, Ieven M, Jeurissen A. Central nervous system invasion by community-acquired methicillin-resistant *Staphylococcus aureus*. *J Med Microbiol.* Sep 2009;58:1247-51.
7. Bhatia K, Jones NS. Septic cavernous sinus thrombosis secondary to sinusitis: are anticoagulants indicated? A review of the literature. *J Laryngol Otol.* Sep 2002;116(9):667-76.
8. Lessner A, Stern GA. Preseptal and orbital cellulitis. *Infect Dis Clin North Am.* Dec 1992;6(4):933-52.
9. Levine SR, Twyman RE, Gilman S. The role of anticoagulation in cavernous sinus thrombosis. *Neurology.* Apr 1988;38(4):517-22.
10. Peters KS. Secondary headache and head pain emergencies. *Prim Care.* Jun 2004;31(2):381-93, vii.