

BACKGROUND

A 23 year old man attends ED due to 2/7 of increasing headache. He describes a sharp retro-orbital pain and has noticed periorbital swelling, initially around the left eye but now affecting the right. He also complains of double vision affecting his left eye which is watery.

He has recently had a coryzal illness, with a blocked/ runny nose and pain in his upper teeth. He now feels quite 'flu- like'.

He has no significant PMHx or allergies and doesn't take regular medications.

He is a joiner, doesn't smoke or take illicit substances. He admits to occasional alcohol binges but doesn't drink regularly.

On examination his obs are normal apart from a mild tachycardia of 93. Respiratory and cardio examination is unremarkable.

He is tender over his maxillary sinuses.

Pupils are reactive but his left pupil is sluggish and he has a lateral gaze palsy affecting his left eye.

QUESTIONS

1. What is the diagnosis?
2. What are the features of the presentation & examination?
3. What is the management?

ANSWERS & DISCUSSION

1) Infective cavernous sinus thrombosis as a complication of acute bacterial sinusitis

Cavernous sinus thrombosis is usually a late complication of an infection of the central face or paranasal sinuses. The cavernous sinuses are trabeculated cavities at the base of the skull. They are part of a complex web of venous drainage of the face and head, there are no valves within this web so blood, and infection, can travel in any direction depending on the prevailing pressures. These sinuses are just lateral and superior to the sphenoid sinus and posterior to the optic chiasm. The internal carotid artery passes through the cavernous sinus, and CN III, IV, VI are attached to the lateral wall. V1 & V2 branches of the trigeminal nerve are embedded in the wall.

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.

The differential diagnosis includes Acute Angle-Closure Glaucoma, Subdural Haematoma, Subarachnoid Haemorrhage, Meningitis, Epidural Hematoma, Orbital & Periorbital Infections

2) Presentation: The clinical presentation is usually due to the venous obstruction as well as impairment of the cranial nerves that are near the cavernous sinus. Patients generally have sinusitis or a midface infection for 5-10 days.

Headache is the most common presentation symptom. The headache is usually sharp, increases progressively, and usually localizes to CN V1 & V2 regions. Some cases of CST may present with focal cranial nerve abnormalities possibly presenting similar to an ischemic stroke. As the infection tracts posteriorly, patients complain of orbital pain and fullness accompanied by periorbital oedema and visual disturbances. Without effective therapy, signs appear in the contralateral eye by spreading through the communicating veins to the contralateral cavernous sinus. 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/or sepsis.

Examination: Other than the findings associated with the primary infection, the following signs are typical for cavernous sinus thrombosis:

- **Periorbital oedema**- may be the earliest physical finding
- **Chemosis**- caused by occlusion of the ophthalmic veins
- **Lateral gaze palsy** (isolated cranial nerve VI)
- **Ptosis, mydriasis, and eye muscle weakness** from CN III dysfunction
- Signs of raised retrobulbar pressure e.g. **Exophthalmos, Ophthalmoplegia**
- Signs of raised intraocular pressure (IOP) e.g. **Sluggish pupillary responses, Reduced visual acuity**
- Paraesthesia over V1 & V2 branches of CN V.
- **Appearance of signs and symptoms in the contralateral eye is pathognomonic of CST**
- **Meningeal signs**, including nuchal rigidity and Kernig and Brudzinski signs
- Systemic signs indicative of sepsis are late findings

3) Treatment

- The patient will require early & aggressive **broad spectrum IV Abx** until a pathogen can be identified
- Anti- coagulation with **low molecular weight heparin**
- **Corticosteroids** may reduce associated oedema and inflammation