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Cavernous sinus thrombosis: Departmental guidelines

Anatomy- cavernous sinus

- 2cm in length, height of 1cm
- Paired venous sinus, on either side of body of sphenoid.
- Traversed by numerous trabeculae, dividing it into a several caverns (spaces), hence cavernous sinus
- Relations:
  - Medial – pituitary above, sphenoidal air cell below
  - Lateral – temporal lobe, uncus
  - Anterior - superior orbital fissure
  - Posterior - petrous apex
  - Superior – optic chiasm
- Tributaries:
  - Superior and inferior opthalmic veins
  - Sphenoparietal sinus
  - Inferior cerebral veins
  - Superficial middle cerebral veins
  - Central vein of retina
- Drainage:
  - Superior petrosal sinus → transverse sinus
  - Inferior petrosal sinus → internal jugular vein
- Communication:
  - Intercavernous sinuses – communication between the 2
  - Pterygoid plexus – via emissary veins passing through foramen ovale, emissary sphenoidal foramen and foramen lacerum.
  - Pharyngeal plexus – via a vein passing through carotid canal.
  - Facial vein – via superior opthalmic vein.
- Contents of cavernous sinus
  - carotid artery
  - CN 3
  - CN 4
  - CN 5 (1^{st} and 2^{nd} divisions)
  - CN 6

C.S.T- pathophysiology

* Includes cases of phlebitis, thrombo-phlebitis and aseptic thrombosis
* Septic type (most common) - coagulase positive *staphylococcus*
* Aseptic types may follow trauma, local stasis or a failing circulation.

* **Sources:**
* Ear **40%**
* Orbit- Face **35%**
* Mouth – Teeth **13%**
* Nose – Paranasal **9%**
* Other – tonsil, soft palate, pharynx, posterior portions of the superior and inferior alveolar arches **3%**
* Afferent and efferent tributaries are valveless, hence easy spread of infection.

* **Path:**
  - VENOUS OBSTRUCTION
  - INVOLVEMENT OF CRANIAL NERVES
  - SEPSIS

* **Venous obstruction:**
  - Proptosis (first before oedema & chemosis)
  - Oedema of eyelids and bridge of nose
  - Chemosis
  - Dilatation and tortuosity of retinal veins
  - Retinal hemorrhages
  - Involvement of the contralateral eye – (48 hours)
  - (anatomic communications between the two cavernous sinuses)
  - When pterygoid plexus is occluded along with sinus, - oedema of the pharynx or tonsil

Involvement of cranial nerves
* Ptosis - paralysis of oculomotor nerve (and edema)
* Dilatation of pupil- third nerve and stimulation of sympathetic plexus
* Decreased abduction (paralysis of abducens nerve)
* Ophthalmoplegia - CN 3,4,6 (and oedema)
* Loss of vision
* Pain in region supplied by ophthalmic branch of 5^{th} cranial nerve.
* Bulb may also be fixed from orbital swelling
* Retro-orbital pain and supra-orbital headache → Vi

Sepsis

- Pyrexia
- Rapid, weak, thready pulse
- Chills and sweats
- Delirium - meningitis supervenes terminally
- Septic emboli to (1) lungs (2) kidney (3) spleen (4) liver and various other parts of body.

Diagnosis

- **Proptosis** followed by edema and chemosis on the same side of body as infection near afferent or efferent venous connections with cavernous sinus
- Second eye involved within forty-eight hours.
- If unilateral throughout Dx is open to a some question. They occur only rarely, as pure unilateral type.

Differential diagnosis

- Orbital cellulitis - unilateral (hardest to differentiate)
- Cellulitis of cheek and face - accompanies by edema of the eyelids
- Infections of accessory sinuses.
- Exophthalmis goiter (particularly malignant type of exopthalmus)
- Tumors of orbit / optic nerve / lacrimal gland.
- Fractures and trauma to head with sterile thrombi in cavernous sinus.

Foci of infection

- **Anterior foci:**
- Such infections result from suppurations of the upper lip, vestibule of the nose and eyelids, and spread by way of the angular, supraorbital and supratrochlear veins to the ophthalmic veins. This is the commonest route of infection.

- **Internal foci:**
- These infections occur as a result of intranasal operations on the septum, turbinates and sinuses; after the use of cautery in the nose

during acute infections, and from suppuration of the posterior ethmoid and sphenoid sinuses, rarely the antrums.

- The infection is spread directly through the ethmoidal veins or through the wall of the sphenoid sinus.

- **Inferior foci:**
- Such infections develop from peritonsillar abscess, operations on the tonsil, surgery or osteomyelitis of the superior maxilla, maxillary dental extractions and deep cervical abscess.
- They spread by way of the pterygoid plexus or by direct proximal (retrograde) extension of the internal jugular vein through the lateral sinus and the petrosal sinuses

- **Posterior foci:**
- These infections occur as a result of extensive involvement of the middle ear and mastoid with lateral sinus phlebitis or thrombosis and retrograde spread through the petrosal sinuses to the cavernous sinus.

- **<u>Mortality/Morbidity</u>:**
- 100% mortality prior to effective antimicrobials
- Typically, death is due to sepsis or central nervous system (CNS) infection.
- With aggressive management, the mortality rate is now less than 30%.
- Morbidity, however, remains high, and complete recovery is rare.
- Roughly one sixth of patients are left with some degree of visual impairment, and one half have cranial nerve deficits.

- **Race:** No predilection
- **Sex:** No predilection
- **Age:** All ages are affected

<u>Clinical</u>

- **History:**
  - Preceding sinusitis or midfacial infection
  - Headache, fever, malaise – precedes ocular manifestations.

- Followed by ocular dysfunction

* **Physical:**
  - Venous congestion
    * Chemosis
    * eyelid / periorbital oedema
  - Retrobulbar pressure increase
    * Proptosis
    * Opthalmoplegia
  - Increase in intraocular pressure
    * Sluggish pupillary response
    * Decreased visual acuity
  - Cranial nerve
    * VI palsy- 1st – IV,Vi,Vii
    * opthalmoplegia

Causes

* Most cases caused by:
  - Staph Aureus - common
  - Streptococci
  - pneumococci

Diagnosis:

* Good clinical evalluation, and a high index of suspicion
* Radiology

* MRI:
  - A sensitive, noninvasive method of imaging the internal structures. Can be combined with angiography to demonstrate lack of blood flow in the cavernous sinus.

Management:

* Therapy should include intravenous antibiotics and early surgical drainage of the primary pathology
* Immediate initiation of antibiotic therapy – broad spectrum , until microbiology received.

- Regular scanning – progression of disease process.

Controversies:

- **Anticoagulants:**
  - Mortality was lower among patients who received heparin treatment, 14% vs. 36%
    - Southwick FS, Richardson EP, Swartz MN.Septic thrombosis of the dural venous sinuses.Medicine 1986;65:82±106.
    - Levine SR, Twyman RE, Gilman S. The role of anticoagulation in cavernous sinus thrombosis.Neurology 1988;38:517±22.
  - Early administration of heparin may serve to prevent spread of thrombosis to the other cavernous sinus as well as to the inferior and superior petrosal sinuses. Intravenous heparin (maintaining the partial thromboplastin time or thrombin clot time at 1.5 to 2 times that of the control) must be continued until the patient is stable for at least several days. Empirically, warfarin (maintaining the pro-thrombin time at 1.3-1.5 times the control) could then be started and continued for 4 to 6 weeks to allow adequate collateral channels to develop.
    - Dinubile M. Septic thrombosis of the cavernous sinus. Arch Neurol 1988;45:567±72.

- **Steroids:**
- Steroid therapy use may partially prevent cranial nerve dysfunction caused by inflammation.
  - Yarrington CT. Cavernous sinus thrombosis revisited.Proc R Soc Med 1977;70:456±9.