

ORBITAL CELLULITIS AND CAVERNOUS SINUS THROMBOSIS

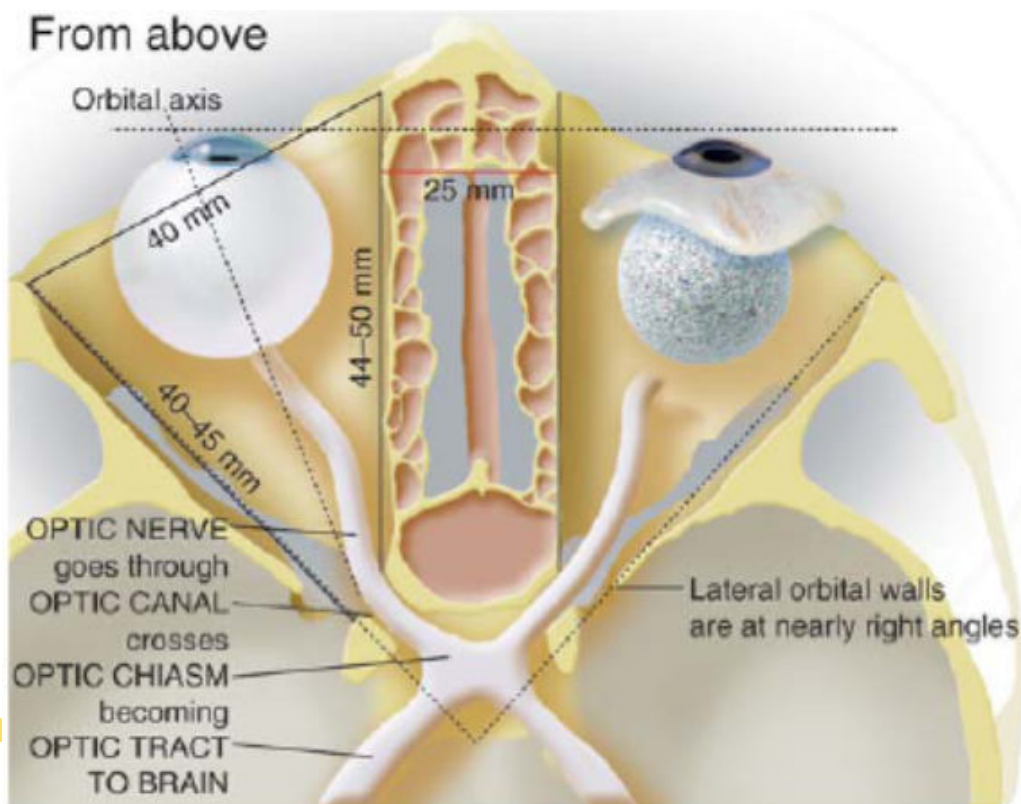
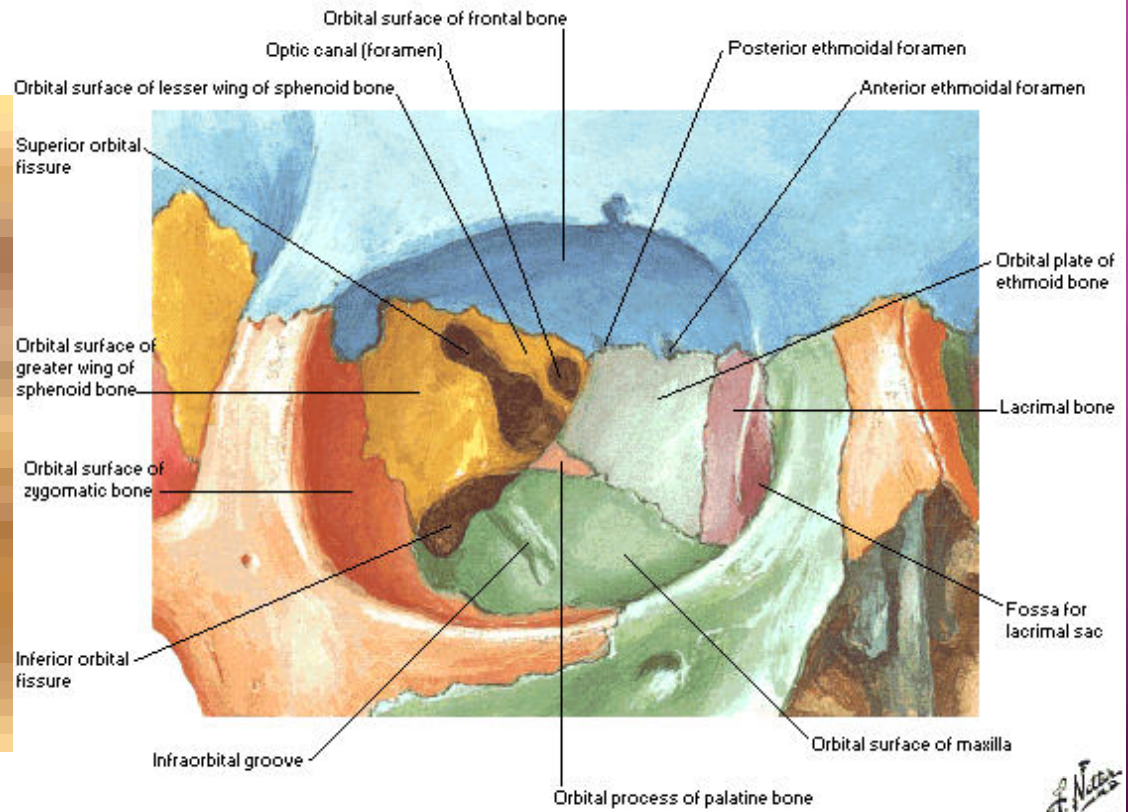
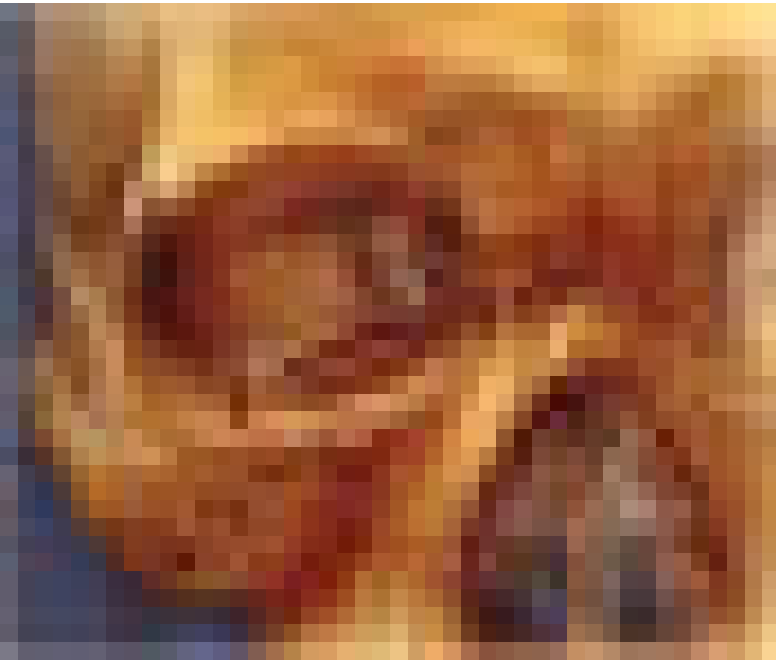
ORBIT

- ◉ Bony house of eyeball
- ◉ Protects the eye
- ◉ Facilitates motility

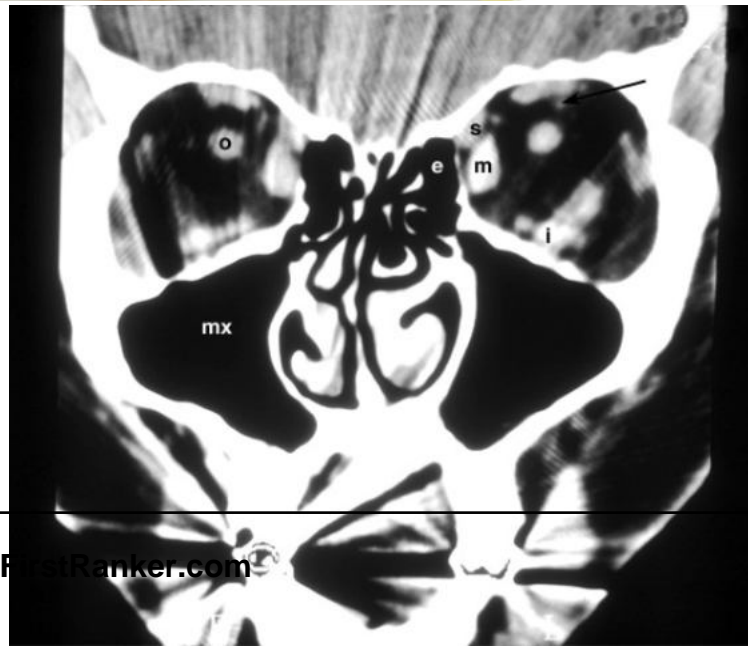
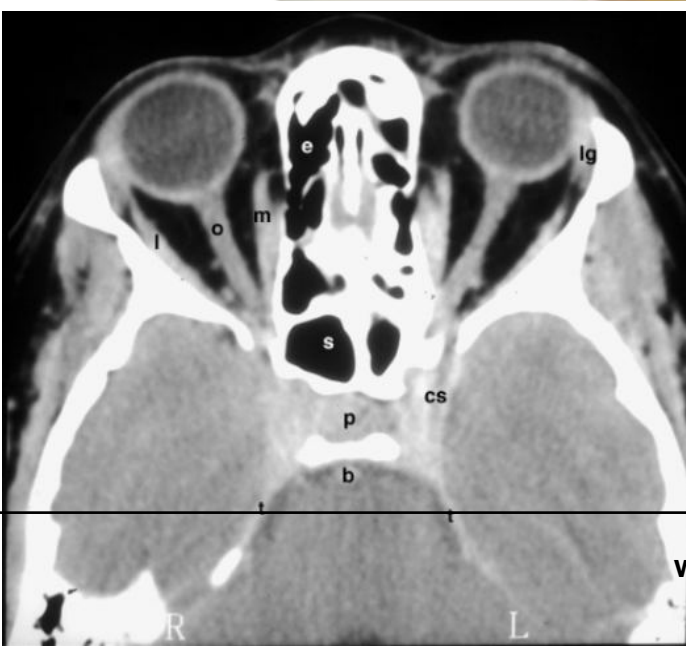
Contents:

- ◉ Eyeball
- ◉ Nerves - II, III, IV, VI and part of V nerve
- ◉ Blood vessels
- ◉ Lacrimal gland

- Pear shaped cavity
- Four walls tapering posteriorly
- Formed by Seven bones
- Volume - 30 ml
- Three walls are related to paranasal sinuses



Horizon



- ⦿ Preseptal cellulitis
- ⦿ Post Septal cellulitis/Orbital cellulitis

-Orbital cellulitis is purulent inflammation of eye tissues behind the orbital septum

ETIOLOGY

- ⦿ **Extension from neighbouring structures** : Paranasal sinuses, Teeth, Face, Lids, Intracranial cavity, Intraorbital structures
- ⦿ **Exogenous Infection** : Foreign body, Penetrating injury, Evisceration, Enucleation, Dacryocystectomy, Orbitotomy
- ⦿ **Endogenous infection** : Puerperal sepsis, Thrombophlebitis of leg, Septicemia, rarely as metastasis from Ca Breast

Predisposing factors like Diabetes mellitus and Immunocompromised state also increases risk of infection.

AGENTS

⦿ BACTERIA

- Childrens- Staph aureus, Strep pneumoniae and anaerobics
- Adults- Staph aureus, Strep pneumoniae, E.coli, mixed flora

⦿ FUNGUS

Diabetics and Immunocompromised
Aspergillus, Mucor species

⦿ PARASITE

- Ecchinococcus Granulosus •Taenia solium
- Trichinella spiralis •Toxoplasma gondii

PATHOLOGICAL FEATURES

- ⦿ Are similar to suppurative inflammations of the body in general, except that
 - **Due to the absence of a lymphatic system** the protective agents are limited to local phagocytic elements provided by the orbital reticular tissue
 - **Due to tight compartments**, the intraorbital pressure is raised which augments the virulence of infection causing early and extensive necrotic sloughing of the tissues
 - As in most cases the **infection spreads as thrombophlebitis** from the surrounding structures, a rapid spread with extensive necrosis is the rule

SYMPTOMS

- ◉ High Fever
- ◉ Painful swelling of upper and lower lids
- ◉ Eyelid appears shiny and is red or purple in color
- ◉ Infant or child is acutely ill or toxic
- ◉ Eyepain Especially with movement
- ◉ Decreased vision
- ◉ Eye bulging
- ◉ General malaise
- ◉ Restricted or painful eye movements

SIGNS

- ◉ A marked swelling of the lids characterised by woody hardness and redness
- ◉ A marked chemosis of conjunctiva, which may protrude and become desiccated or necrotic
- ◉ The eyeball is proptosed axially
- ◉ Frequently, there is mild to severe restriction of the
- ◉ ocular movements
- ◉ Fundus examination may show congestion of retinal veins and signs of papillitis or papilloedem

DIFFERENTIAL DIAGNOSIS

- ⦿ Cavernous sinus thrombosis
- ⦿ Endocrine dysfunction
- ⦿ Orbital myositis
- ⦿ Orbital pseudotumor
- ⦿ Wegener granulomatosis

STAGES OF ORBITAL CELLULITIS

- ⦿ CHANDLER CLASSIFICATION
 - Group 1 - Pre-septal Cellulitis
 - Group 2 - Orbital Cellulitis
 - Group 3 - Subperiosteal abscess
 - Group 4 - Orbital abscess
 - Group 5 - Cavernous sinus thrombosis

INVESTIGATION

- ◉ Complete blood count
- ◉ Blood culture
- ◉ Urine culture
- ◉ B scan
- ◉ CT Scan
- ◉ MRI

TREATMENT

- ◉ IV Antibiotics - anti biotic therapy should be continued until patient is apyrexia for 4 days
- ◉ Antifungals
- ◉ Nasal decongestants
- ◉ Diuretics to reduce the IOP
- ◉ Lumbar puncture is done in meningeal or lumbar signs develop and It is useful to do the swinging light test to check for a Marcus Gunn pupil, which would indicate optic nerve damage
- ◉ Frequent ophthalmic assessment is mandatory in case of intra cranial abscess formation, neurosurgical drainage may be necessary

COMPLICATIONS

- ◉ Ocular – Exposure Keratitis , Raised IOP, CRAO, CRVO, Optic Atrophy
- ◉ Orbital – Subperiosteal abscess, Orbital abscess
- ◉ Cavernous sinus thrombosis
- ◉ Meningitis, Brain abscess
- ◉ Bacteremia

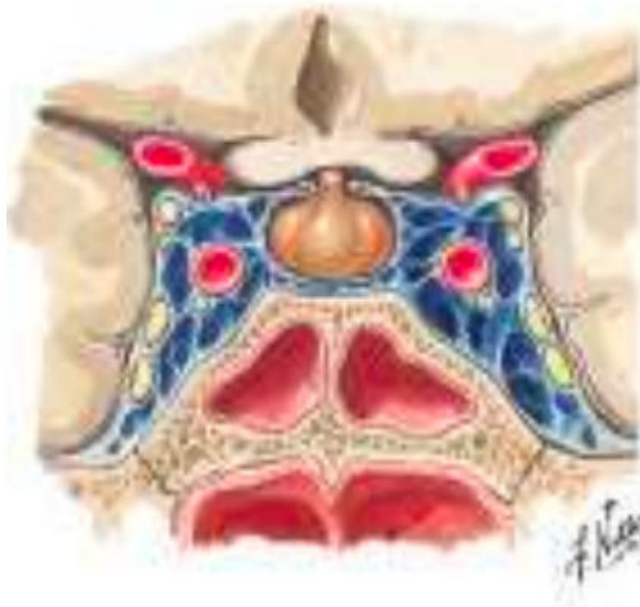
Cavernous sinus Anatomy

- Large venous space situated in the middle cranial fossa, on either side of body of the sphenoid bone.
- Each sinus is about 2 cm long and 1 cm wide.
- Interior is divided into a number of spaces or caverns by trabeculae.

CAVERNOUS SINUS THROMBOSIS

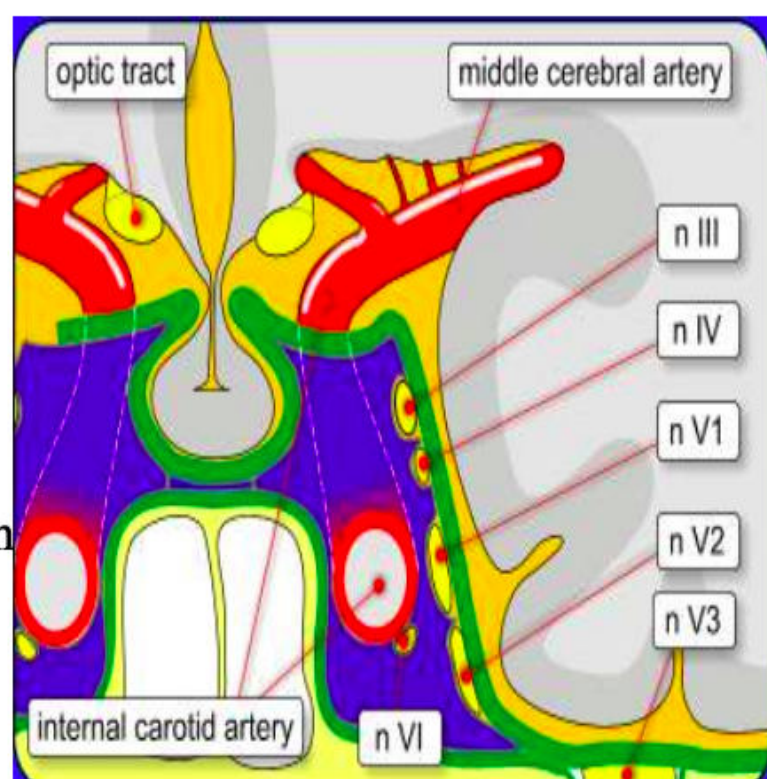
Cavernous sinus Anatomy Boundries

- **Anterior** - extends into medial end of superior orbital fissure.
- **Posterior** - upto apex of petrous temporal bone.
- **Medial** – Pitutary above and sphenoid below
- **Lateral** – temporal lobe and uncus
- **Superior** – optic chiasma
- **Inferior** - endosteal dura mater, greater wing of sphenoid



Contents

- **Superior to inferior (within the lateral wall of the sinus)**
 - **oculomotor nerve** (CN III)
 - **trochlear nerve** (CN IV)
 - **ophthalmic nerve**, the V₁ branch of the trigeminal nerve (CN V)
 - **maxillary nerve**, the V₂ branch of CN V

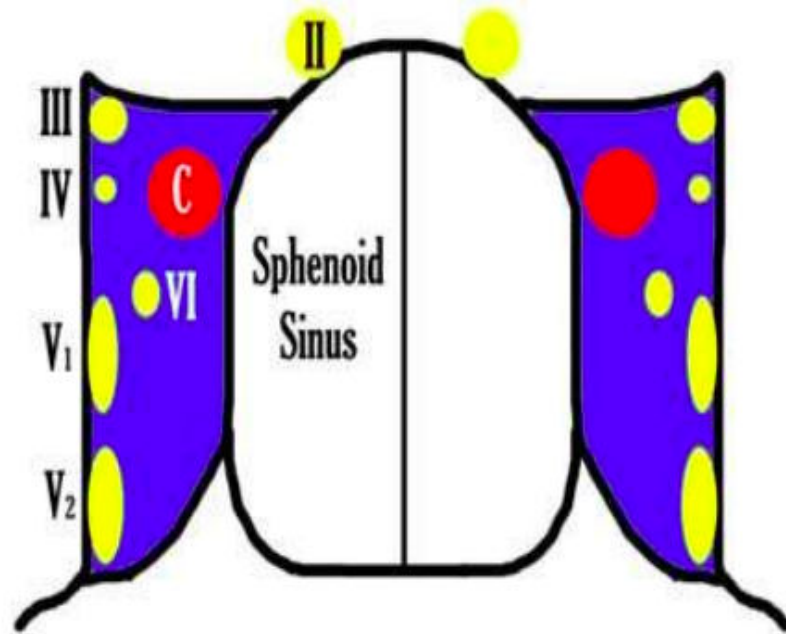


Contents

abducens nerve (CN VI) runs through

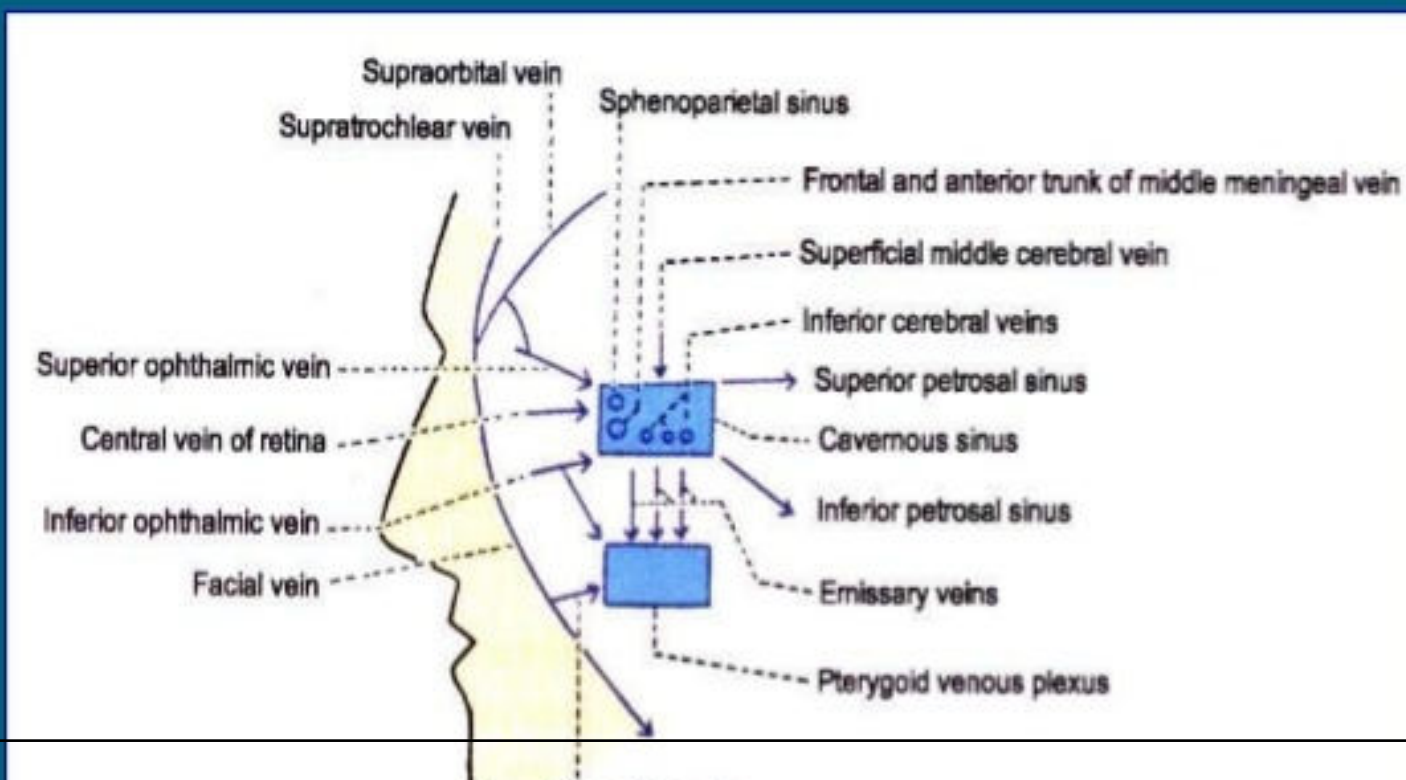
the middle of the sinus alongside the **internal carotid artery** (with sympathetic plexus)

These nerves, except the CN V₂, pass through the cavernous sinus to enter the orbital apex through the superior orbital fissure.



Communication with:

- a) Transverse sinus
- b) IJV
- c) Pterygoid venous plexus
- d) Facial vein
- e) Superior sagittal sinus
- f) Opposite cavernous sinus



Dangerous area of face

flow of blood in all tributaries & communication are reversible as they possess **no valve**

Spread of infection can lead to thrombosis of cavernous sinus

The cavernous communicate with **dangerous area of face** through 2 routes

- ✓ Superior orbital vein
- ✓ Deep facial veins , pterygoid plexus of vein , emissary vein.



Spread of infection to cavernous sinus

1. Infection of the upper lip, vestibule of the nose and eyelids → spread by way of the angular, supraorbital and supratrochlear veins to the ophthalmic veins. **Commonest route** of infection.
2. Intranasal operations on the septum, turbinates or ethmoid / sphenoid sinus infection → through the ethmoidal veins.

Spread of infection to cavernous sinus

3. Operations on the tonsil, peritonsillar abscess, surgery or osteomyelitis of the maxilla, dental extraction and deep cervical abscess → spread by pterygoid plexus or by direct extension to the internal jugular vein.
4. Involvement of the middle ear and mastoid with lateral sinus phlebitis or thrombosis → retrograde spread through the petrosal sinuses to the cavernous sinus.

Etiology of CST

Septic CST

- **Infectious**

Aseptic CST

Trauma

Postsurgery

- **Rhinoplasty**
- **Cataract extraction**
- **Basal skull (including maxillary)**
- **Tooth extraction**

Hematologic

- **Polycythemia rubra vera**
- **Acute lymphocytic leukemia**

Malignancy

- **Nasopharyngeal tumor**

Other

- **Ulcerative colitis**
- **Dehydration**

Septic cavernous sinus thrombosis

- Most commonly results from contiguous spread of infection from the nose (50%), sphenoidal or ethmoidal sinuses (30%) and dental infections (10%).
- *Staphylococcus aureus* is the most common - found in 70% of the cases.
- *Streptococcus* is the second leading cause.
- Gram-negative rods and anaerobes may also lead to cavernous sinus thrombosis.
- Rarely *Aspergillus fumigatus* and mucormycosis.

Cavernous Sinus thrombosis

Characterized by multiple cranial neuropathies

Clinical feature -

- ✓ General feature of infection – fever , rigors , malaise, and severe frontal & periorbital pain.
- ✓ U/L exophthalmos & tender eye ball
- ✓ Oedema of eyelid & chemosis of conjunctiva

Oculomotor feature –

- ✓ External ophthalmoplegia
- ✓ Ptosis
- ✓ Slight exophthalmos
- ✓ dilated pupil with loss of accommodation reflex

Cavernous Sinus thrombosis

- Impairment of ocular motor nerves, Horner’s syndrome and sensory loss of the first or second divisions of the trigeminal nerve in various combination
- **The pupil may be involved or spared or may appear spared with concomitant oculosympathetic involvement.**

Ocular manifestation of cavernous sinus thrombosis

<i>SIGN</i>	<i>INVOLVED STRUCTURES</i>
Ptosis	Edema of upper eye lid Sympathetic plexus III cranial nerve
Chemosis	Thrombosis of superior and inferior ophthalmic vein
Proptosis	Venous engorgement
Sensory loss/ Periorbital pain	V cranial nerve
Corneal ulcers	Corneal exposure due to proptosis
Lateral rectus palsy	VI cranial nerve
Complete ophthalmoplegia	CN II, IV, VI
Decreased visual acuity or blindness	Central retinal artery/ vein occlusion secondary to ICA arteritis, septic emboli, ischemic optic neuropathy

Complication of Cavernous Sinus thrombosis

- Intracranial extension of infection may result in meningitis, encephalitis, brain abscess, pituitary infection, and epidural and subdural empyema.
- Cortical vein thrombosis can result in hemorrhagic infarction.
- Extension of the thrombus to other sinuses can occur.

TREATMENT OF CAVERNOUS SINUS THROMBOSIS

Septic cavernous sinus thrombosis –

- The mainstay of therapy 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 generation cephalosporin.
- Vancomycin may be added for MRSA.

-
- IV antibiotics are recommended for a minimum of 3-4 weeks.

TREATMENT OF CAVERNOUS SINUS THROMBOSIS

- A Cochrane review found 2 small trials involving 79 patients who were treated with anticoagulants.
- Limited evidence suggests anticoagulant drugs are probably safe and may be beneficial for people with sinus thrombosis.
- Anticoagulation carries a significant risk of hemorrhage if cortical venous infarction or necrosis of intracavernous portions of the carotid artery are present.
- Anticoagulant is contraindicated in the presence of intracerebral hemorrhage or other bleeding diathesis.

Prognosis

- 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 (50 %) have cranial nerve deficits.

Fungal infection

- Intracranial extension is the most dreaded complication of fungal sinusitis with high mortality rates.
- Aspergillus is the most common.
- Mucor, rhizopus, cladosporium, candida, cryptococcus are amongst the others.
- Mode of spread =
 - Hematogenous spread
 - Direct extension

Fungal infection - treatment

- Line of management-included debridement, clearing of disease from the sinuses and antifungal therapy with systemic Amphotericin B.
- In combined therapeutic modality, surgery + amphotericin B, the overall survival rate is 81%.
- It is 89% in diabetics with combined therapy and corrected ketoacidosis.