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High risk and low prevalence diseases: Cavernous sinus thrombosis

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ABSTRACT

Introduction: Cavernous sinus thrombosis (CST) is a serious condition that carries with it a high rate of morbidity and mortality.
Objective: This review highlights the pearls and pitfalls of CST, including presentation, diagnosis, and management in the emergency department (ED) based on current evidence.
Discussion: CST is a potentially deadly thrombophlebitic disease involving the cavernous sinuses. The most common underlying etiology is sinusitis or other facial infection several days prior to development of CST, though other causes include maxillofacial trauma or surgery, thrombophilia, dehydration, or medications. *Staphylococcus aureus*, streptococcal species, oral anaerobic species, and gram-negative bacilli are the most frequent bacterial etiologies. The most prevalent presenting signs and symptoms are fever, headache, and ocular manifestations (chemosis, periorbital edema, ptosis, ophthalmoplegia, vision changes). Cranial nerve (CN) VI is the most commonly affected CN, resulting in lateral rectus palsy. Other CNs that may be affected include III, IV, and V. The disease may also affect the pulmonary and central nervous systems. Laboratory testing typically reveals elevated inflammatory markers, and blood cultures are positive in up to 70% of cases. Computed tomography of the head and orbits with intravenous contrast delayed phase imaging is recommended in the ED setting, though magnetic resonance venography demonstrates the highest sensitivity. Management includes resuscitation, antibiotics, and anticoagulation with specialist consultation.
Conclusion: An understanding of CST can assist emergency clinicians in diagnosing and managing this potentially deadly disease.

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1. Introduction

This article series addresses high risk and low prevalence diseases that are encountered in the emergency department (ED). Much of the primary literature evaluating these conditions is not emergency medicine focused. By their very nature, many of these disease states and clinical presentations have little useful evidence available to guide the emergency physician (EP) in diagnosis and management. The format of each article defines the disease or clinical presentation to be reviewed, provides an overview of the extent of what we currently understand, and finally discusses pearls and pitfalls using a question and answer format. This article will discuss cavernous sinus thrombosis (CST), a rare condition that portends significant morbidity and mortality making it a high risk and low prevalence disease. This condition's low prevalence but high morbidity and mortality, as well as its variable atypical patient presentations and challenging diagnosis, makes it a high risk and low prevalence disease.

1.1. Definition

CST is a rare but potentially deadly thrombophlebitic disease resulting in clot formation within the cavernous sinuses [1-6]. The condition most commonly begins with a sinus or facial infection, which may spread to the cavernous sinus and lead to formation of a thrombus [1,4,6]. However, it may also be aseptic. The thrombus within the cavernous sinus obstructs venous drainage and may result in cranial nerve compression, sepsis, and even death [1,4-6].

1.2. Epidemiology

Overall CST is uncommon, with an annual incidence of 2–13 per million [2,4,7]. CST appears to occur more frequently in adults between 20 and 50 years (mean 22 years) [2,5,8-11]. <10% of cases occur in adults over age 65 years, and there is a slight male predominance [5,7-10].

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Before antibiotics, mortality rates reached 80–100% [12,13]. In the current era with antibiotics, mortality has significantly decreased but remains between 11 and 30% [4,5,10,12]. Unfortunately, 50–75% of patients experience neurologic complications with CST, with the most common sequelae including vision loss, cranial nerve (CN) palsies, pituitary dysfunction, hemiparesis, facial disfigurement, seizures, and cortical vein thrombosis [4-8,14,15].

1.3. Pathophysiology

The cavernous sinuses consist of the left and right sinus located superior to the sphenoid sinuses at the base of the skull lateral to the sella turcica (Fig. 1). They extend from the petrous portion of the temporal bone to the superior orbital fissure [1,4,6,15-17]. The sinuses are formed by the separation of the periosteal and meningeal layers of the dura mater, which are irregularly shaped and contain trabeculae [5,15-17]. The sinuses are connected by two intercavernous sinuses passing anteriorly and posteriorly to the sella turcica, which contains the pituitary gland. CN III (oculomotor), IV (trochlear), and the ophthalmic and maxillary branches of the trigeminal nerve (V1 and V2) pass along the lateral wall of each sinus, while the internal carotid artery, sympathetic nerve plexus, and CN VI (abducens) traverse the body of the sinus [1,15,18]. The sinuses receive blood from the cerebral veins, superior and inferior ophthalmic veins, and sphenoparietal and sphenoid sinuses, while blood drains from the sinuses via emissary veins that empty into the pterygoid venous plexus and petrosal sinuses. These in turn connect with the internal jugular vein and sigmoid sinus [1,5,8,15,16,19,20].

There are two forms of CST: septic (most common) and aseptic. CST most commonly develops from a local bacterial infection such as sinusitis or a facial infection, which can spread as a contiguous phlebitis, thrombophlebitis, or septic emboli [3-6]. As the connections within the cavernous sinus do not have valves, bidirectional spread of infection and thrombi can occur. Thus, infection may travel through the ophthalmic veins in an anterograde or retrograde route along the emissary veins that are connected to the pterygoid venous plexus [7,19,20]. Infection can also spread from adjacent tissues into the cavernous sinuses through soft tissue or bone defects. Indeed, the cavernous and sphenoid sinuses are only separated by a thin bone, allowing for spread of infection between the cavities [7,19,20]. The infection may also spread from the ethmoid sinus when bacteria travel through the lamina papyracea and the ophthalmic veins. Once present within the cavernous sinus, bacteria become entrapped in the trabeculations and further stimulate thrombosis, resulting in disease progression [21,22]. The most commonly involved bacteria include *Staphylococcus aureus*, accounting for up to 70% of cases. This is followed by *Streptococcus pneumoniae* (20%), oral anaerobic species (<10%), and gram-negative bacilli (5%) [3-9]. Fungi and viral species may also result in infection and CST, as can thrombophilia, surgeries, trauma, dehydration, and medications [8,9,23-26].

2. Discussion

2.1. Presentation

Early symptoms of CST are nonspecific. Patients typically have sinusitis or some other midface infection for 5–10 days prior to development of CST [4-8]. Patients with CST most commonly present with headache, which is followed by systemic symptoms (e.g., chills, fevers, rigors) and periorbital edema [1,4-6,8]. The headache is generally unilateral and retrobulbar or frontotemporal in location. Fever is present in the majority of patients [1,4-8,27,28]. Patients can display evidence of sepsis and systemic symptoms including tachycardia, nausea, vomiting, chills, and rigors [4-6,22]. Almost all patients will experience ocular symptoms, including eyelid swelling and pain, photophobia, difficulty with or pain with eye movement, and vision changes including complete loss of vision [4-6,22,29-32]. Changes in mental status including confusion and even coma can rapidly follow eye complaints, as well as seizures and neurologic deficits [1,4-8].

2.2. ED evaluation

The diagnosis of CST can be made clinically, though radiographic studies are typically utilized. These may include computed tomography (CT) of the head and orbits with intravenous (IV) contrast with delayed phase imaging or magnetic resonance venography (MRV) [4,6,27,33-35]. Laboratory testing may demonstrate elevated inflammatory markers (leukocytosis, elevated c-reactive protein [CRP], erythrocyte

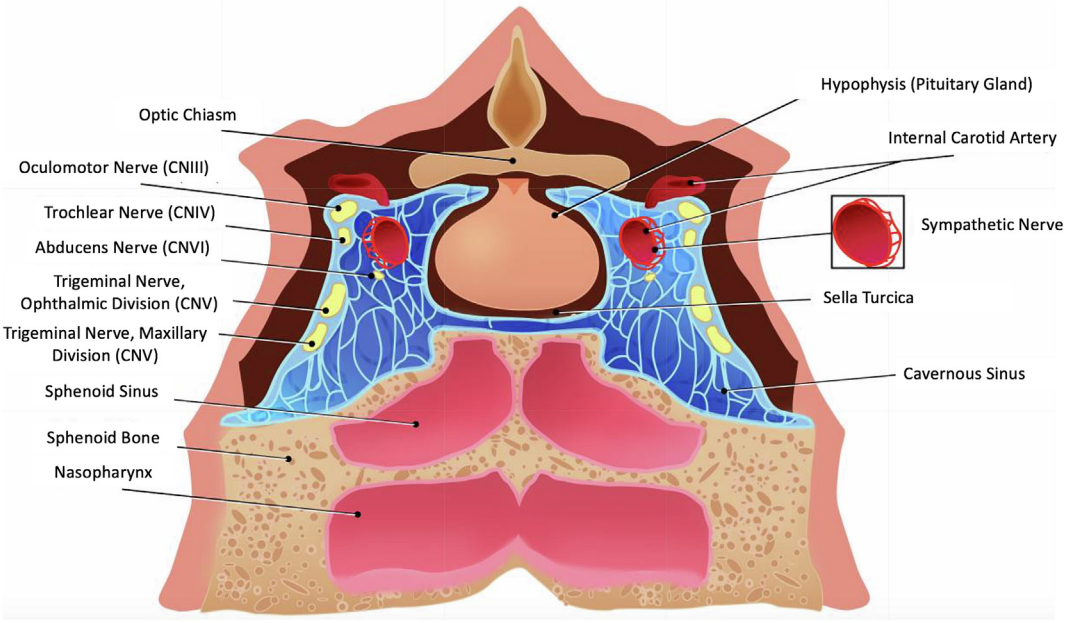

Fig. 1. Cavernous Sinus Anatomy. Modified from https://upload.wikimedia.org/wikipedia/commons/c/c4/Anatomy_of_the_cavernous_sinus.jpg.

sedimentation rate [ESR]) [4-6,22,36]. Blood cultures should be obtained. Lumbar puncture for cerebrospinal fluid (CSF) testing should be considered in those with evidence of meningitis or encephalitis [4].

2.3. ED management

Management includes resuscitation, broad-spectrum antibiotics, and anticoagulation [1,4-8]. Consultation with the otolaryngology specialist is recommended, though other specialty consultation may be necessary (e.g., neurosurgery consultation for intracranial complication, ophthalmology for ocular complications). Empiric antibiotics covering staphylococcal and streptococcal species, gram-negative bacteria, and anaerobic bacteria should be administered [4-8,37-39]. Antibiotics are typically required for three weeks at minimum. Anticoagulation with unfractionated heparin or low molecular weight heparin is typically utilized to prevent further thrombus extension, though there are limited guideline recommendations and data regarding anticoagulation [1,4,10,22,39,40]. Surgical drainage may be needed in certain patients, including those with sphenoid sinus infection. Steroids are controversial and not recommended at this time, unless adrenal or pituitary insufficiency is present [4,5,10,41,42].

3. Pearls / pitfalls

3.1. What are the common risk factors and etiologies for this disease?

The most common inciting event of CST is a local infection such as paranasal sinus, facial, odontogenic, otogenic, or pharyngeal infection in up to 80% of cases [1,4-8,18,43-47]. Ethmoid or sphenoid sinusitis is associated with over 50% of cases [1,4-8,18,43-47]. Middle ear infections account for 10% of cases, with oral and dental infections the underlying etiology in <10% of cases [1,4,5,44,48,49]. Orbital cellulitis or peritonsillar abscess may result in CST, though this is rare [34,44,50-55]. While staphylococcal and streptococcal species are the most common infectious agents associated with CST, fungi such as *Aspergillus* and *Rhizopus* may result in infection. However, these typically only occur in those with severe immunocompromise (e.g., uncontrolled diabetes, hematologic malignancy, bone marrow transplant recipients, those on immunocompromising medications) [2,8,10,23–26,56]. Viral causes include human immunodeficiency virus (HIV), herpes simplex, measles, and hepatitis [4,38,44]. Other causes include thrombophilia, maxillofacial/ocular surgery or trauma, dehydration, systemic disease including sepsis or autoimmune disease, intracranial tumors, and medications, though these are less common [4,5,37,44,45,55]. Thrombophilia includes those on oral contraceptives, malignancy, genetic clotting disorders (factor V Leiden mutation, prothrombin G20210A mutation, protein C or S deficiency, or increased factor VIII), pregnancy, or hormone replacement [4,5,44,45,57,58]. Idiosyncratic drug reactions associated with calcineurin inhibitors and tacrolimus used for solid organ transplantation are another etiology [4,5,44,59]. Of note, an underlying source is not found in up to 25–30% of cases [44,60].

3.2. What are reliable findings of CST based on the history and examination?

The majority of patients have a latent period of 1–21 days (mean 5–6 days) between the predisposing infection and the development of signs and symptoms of CST [4,5,27,28]. The most common presenting signs and symptoms of CST include headache, fever, and ocular manifestations, and patients often present with evidence of sepsis (tachycardia, hypotension). However, a variety of signs or symptoms including mental status changes and nausea and vomiting may occur, and patients may also present with a more indolent course [4,5,22]. Up to 90% of patients will experience headache, most commonly unilateral with a frontotemporal or retro-orbital distribution [1,4]. Fever affects up to 94% of patients with CST; however, it may be intermittent, interspersed with periods of normothermia [1,4,5]. Up to 40% of patients demonstrate nuchal rigidity [4,5,27,28].

Ocular manifestations are present in almost all patients due to the underlying pathophysiology, though findings are often subtle in the early course of the disease [1,4,8]. Unilateral symptoms including chemosis, periorbital edema, eyelid erythema, ptosis, proptosis, and ophthalmoplegia occur in 80–100% of cases, and symptoms may progress to involving both eyes within 48 h of the initial symptom onset [1,4,17,30,39]. Unilateral periorbital edema is typically the earliest finding on physical examination [1,4,17,30,39,61]. Ophthalmoplegia is present in 50–88% of patients, which may occur due to mechanical restriction associated with venous congestion, inflammation of the extraocular muscles, and involvement of the CNs [4,16,17,22]. Lateral rectus palsy with limited eye abduction is one of the most common findings due to the course of CN VI [4,61]. CN III and IV course through the lateral wall and are less susceptible to injury due to their protection with a thick layer of dura [30,61]. A sluggish or non-reactive pupil may be found due to parasympathetic nerve fiber damage of CN III, or due to dysfunction of the sympathetic nerve complex [29-32]. Those with parasympathetic nerve damage will present with mydriasis, while those with sympathetic nerve injury will present with miosis. Involvement of ophthalmic and maxillary branches of the trigeminal nerve may lead to pain or sensory changes in the upper and middle thirds of the face [4,22]. There may be loss of the corneal reflex due to damage of the ophthalmic branch of CN V, and Horner syndrome may occur in those with sympathetic fiber dysfunction [1,8]. Fundoscopic examination will reveal papilledema in up to 66% of patients [4,22]. Vision changes are common, with blindness ultimately occurring in 8–15% of cases [29-32]. These may be due to corneal abrasions and ulcerations as a result of loss of the corneal reflex, orbital congestion, proptosis, central retinal artery occlusion, ischemic optic neuropathy, or embolism [29-32].

Thrombophlebitis may spread to the internal jugular vein (Lemierre's syndrome), presenting with sore throat, neck tenderness/redness, cervical lymphadenopathy, ear pain, dyspnea, chest pain, or trismus [31,62,63]. Pneumonia and empyema may occur [1,4,6,64-66]. The disease may spread intracranially, leading to brain abscess, subdural empyema, meningitis, and encephalitis [1,6,64-68].

Ultimately, CST should be considered in patients with prior head/neck infection or other risk factors for CST (e.g., thrombophilia, head/neck operation or trauma, dehydration, etc.) who present with headache, fever, ocular findings (proptosis, chemosis, ophthalmoplegia, vision changes), CN abnormalities (CN III-VI), or meningeal findings.

3.3. What testing modalities are most reliable for diagnosis?

Laboratory testing can demonstrate clues to the diagnosis of CST but may be unremarkable early in the course of the disease. Complete blood count may reveal polymorphonuclear leukocytosis in up to 90% of patients [4,22,28,36]. Elevated inflammatory markers (e.g., CRP, ESR) and D-dimer are common, but negative values should not be used to exclude the diagnosis [1,2]. Blood cultures should be obtained and will yield an organism in up to 70% of cases [69]. Lumbar puncture is recommended if concern for meningitis or encephalitis is present (i.e., nuchal rigidity, altered mental status) [67]. CSF analysis typically demonstrates pleocytosis, elevated protein, and low glucose, with CSF pleocytosis present in 80–100% of cases [4,22,38,70]. However, CSF culture is positive in only 20% of patients [4,5].

Imaging is an important component of the evaluation [4,5,18,33-35,60]. CT of the orbits and head with IV contrast delayed phase should be obtained in the ED if the clinician suspects CST [33-35,60]. Non-contrast head CT is most commonly unremarkable, though a high-density thrombus may be present in the cavernous sinus in up to 25% of cases (Fig. 2) [4,60]. Direct findings of CST on CT of the orbits with IV contrast include expansion of or filling defects within the cavernous

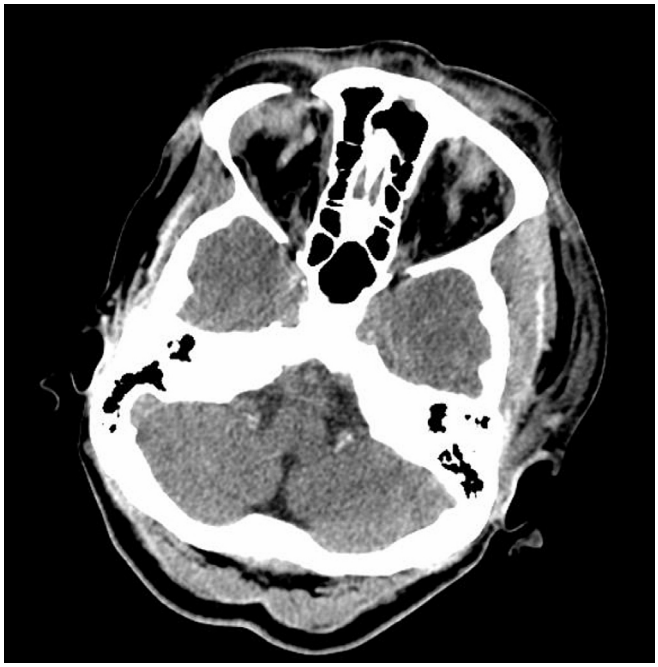

Fig. 2. Non-contrast CT demonstrates dense, distended bilateral superior ophthalmic veins, consistent with CST. Case courtesy of Andrew Dixon, Radiopaedia.org, rID: 10619.

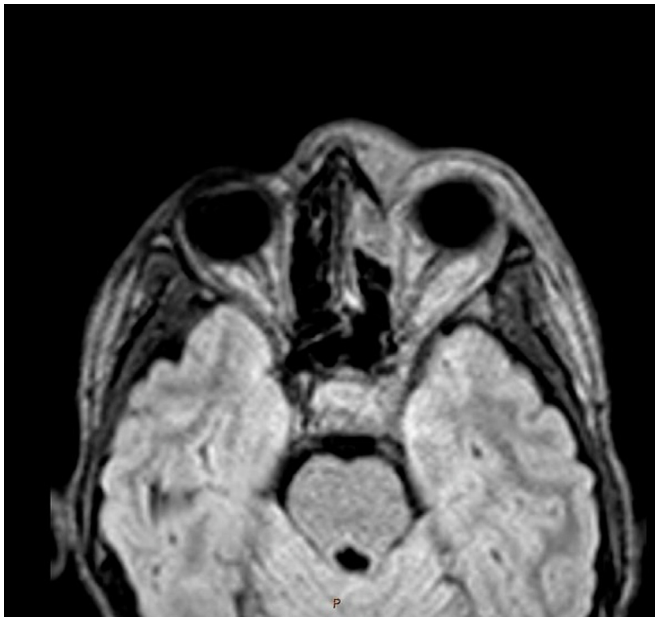

Fig. 4. MRI demonstrating left superior ophthalmic vein swelling and abnormal FLAIR signal in left cavernous sinus, diagnostic of CST. Case courtesy of Derek Smith, Radiopaedia.org, rID: 87888.

sinus (Fig. 3). Bulging of the sinus lateral wall is one of the most frequent and sensitive findings [8,18,27,33-35]. Indirect signs include narrowing or occlusion of the intracavernous segment of the internal carotid artery, venous flow reversal, increased dural enhancement within the cavernous sinus, fat pad edema, exophthalmos, and venous congestion (Fig. 3) [8,18,27,33-35,71-74]. Secondary thrombosis of the superior ophthalmic vein, petrosal, sphenoparietal, or sigmoid sinus may also be found [8,18,27,33,71]. The most sensitive modality is MRV (95%), as this can diagnose CST in all stages (Fig. 4). However, MRV is unavailable in many EDs (Fig. 2) [4,14,27]. If the disease is suspected but CT with IV contrast is not diagnostic, further imaging with MRV is recommended.

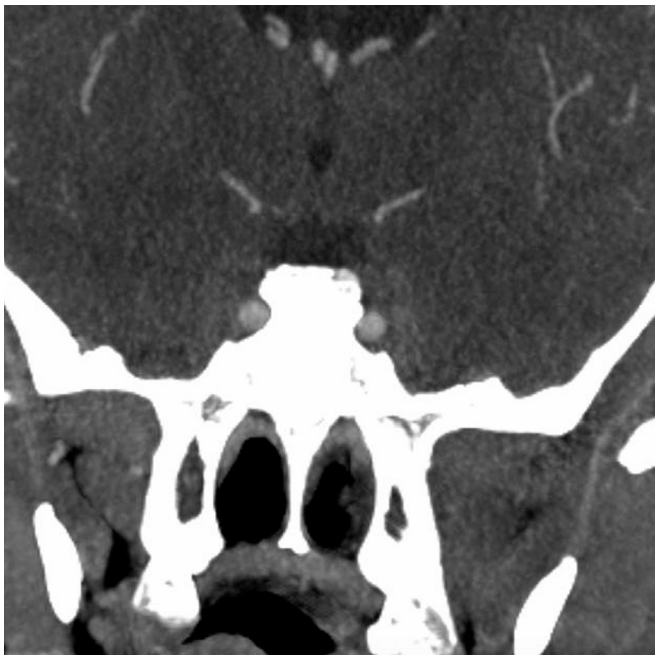

Fig. 3. CT venogram with dense, distended bilateral superior ophthalmic veins, consistent with CST. Case courtesy of Andrew Dixon, Radiopaedia.org, rID: 10619.

3.4. What are the key components of management?

There are limited high-quality data concerning management of CST, with most recommendations based on retrospective studies and expert specialist opinion. However, the primary tenets of management include resuscitation in those with evidence of sepsis, along with broad-spectrum antibiotics, anticoagulation, and specialist consultation (e.g., otolaryngology, neurosurgery for intracranial complications, ophthalmology for ocular complications). Broad-spectrum antibiotics should be administered once the diagnosis is suspected. These include ceftriaxone 2 g IV or cefepime 2 g IV plus vancomycin 20 mg/kg IV or linezolid 600 mg IV. If a dental or sinus infection is suspected on evaluation, metronidazole 500 mg IV should be added [4,5,18,37-39,75,76]. Antifungal agents should be administered in those with immunocompromise or poorly controlled diabetes (voriconazole, itraconazole, or amphotericin b) [4,18,37-39,76,77].

Anticoagulation is controversial but typically utilized, though there are no high-quality randomized data. Proposed benefits include preventing clot propagation, reduction in inflammation, platelet inhibition, and assisting with antibiotic penetration into the thrombus [1,4,22,37,39,40]. Risks include intracranial or systemic hemorrhage; however, this risk is likely low [1,4,15,22,39]. Most of the data supporting anticoagulation are based on retrospective studies. One study found reduced mortality in patients receiving unfractionated heparin (UFH) compared to no anticoagulation (34% vs. 50%) [22]. Another study found that patients who survived and received anticoagulation within 7 days of diagnosis and admission had improved neurological outcomes compared to anticoagulation after 7 days [40]. A study of 88 cases evaluating anticoagulation (UFH, low molecular weight heparin [LMWH], or dalteparin) found greater likelihood of full recovery (53.6% vs. 32%) and reduced mortality (12% vs. 38%) with anticoagulation compared to no anticoagulation [10]. A Cochrane review including 79 patients found anticoagulation (UFH or LMWH) was not associated with reduced death (relative risk [RR] 0.33; 95% confidence interval [CI] 0.08–1.21) or death or dependency (RR 0.46; 95% CI 0.16–1.31)

[78]. No patient experienced an intracerebral hemorrhage, though one patient experienced a gastrointestinal hemorrhage. Authors of this review state anticoagulation is safe, with potentially a reduction in risk of death or dependency [78]. While the data are limited, anticoagulation is likely safe and may reduce morbidity and mortality. There are several options for anticoagulation. UFH can be reversed quickly, while LMWH may cause less bleeding [1,4,78-80]. Ultimately, the decision to anticoagulate should be made with conjunction with the admitting physician and specialist. Anticoagulation is typically continued for at least 4–6 weeks, and in many cases 3 months [1,4,5,18,39].

Corticosteroids may have a role, though they remain controversial. Steroids may decrease orbital inflammation and CN and vasogenic edema [4,5,37]. One study of 88 patients with CST found no improvement in survival, recovery, or survival with disability with steroids [10]. However, steroids are beneficial in patients with adrenal or pituitary insufficiency [4,5,42]. At this time, steroids should only be administered if the patient has adrenal or pituitary insufficiency, or at the request of the specialist [4,5,42].

Surgical intervention is rarely performed and technically difficult due to the anatomical location of the infection. It is typically reserved for treating the primary site of infection and is often performed endoscopically. Surgical intervention may include sphenoidectomy, maxillary antrostomy, mastoidectomy, ethmoidectomy, orbital decompression, or craniotomy and ventricular shunt placement (for subdural empyema or abscess) [1,4,5,37,39,48,66,77,81].

3.5. What conditions mimic CST?

Due to the nonspecific signs and symptoms early in the course of the disease, there are a variety of mimics. These conditions may also occur concomitantly with CST. Mimics or other concomitant conditions include orbital cellulitis, meningitis, and mass-like lesions near or within the cavernous sinus. Table 1 summarizes the primary mimics, while Table 2 provides pearls concerning the evaluation and management of CST.

4. Conclusion

CST is a thrombophlebitic disease involving the cavernous sinuses associated with significant morbidity and mortality. Underlying etiologies include sinusitis or other facial infection, maxillofacial trauma or surgery, thrombophilia, dehydration, or medications. Fever, headache, and ocular manifestations are the most common presenting signs and symptoms. Laboratory testing may demonstrate elevated inflammatory markers. While the disease can be diagnosed clinically, imaging including CT with IV contrast of the head and orbits or MRV is typically

Table 1
Mimics of CST.

Condition	Considerations
Acute angle-closure glaucoma	• Increased posterior chamber pressure due to obstructed aqueous outflow; ocular emergency • Abrupt onset of severe eye pain, blurry vision, halos around lights, frontal/orbital headache, nausea/vomiting, fixed/midposition pupil, hazy cornea, hard globe, conjunctival injection • Intraocular pressure > 20 mmHg; slit lamp examination with cell and flare
Aneurysmal dilation or rupture of internal carotid artery in cavernous sinus	• Aneurysm of the internal carotid arteries involving a portion of the vessel in the cavernous sinus; may rupture • More common in older patients, women > men • Diplopia, eye pain and/or headache, optic neuropathy and decreased vision • Diagnose with computed tomography angiography or magnetic resonance angiography
Carotid-cavernous fistula	• Abnormal vascular connection between the carotid artery or its branches and the cavernous sinus • Classified as low flow or high flow • May result from trauma, infection, collagen vascular disease, hypertension, atherosclerosis • Classic triad is chemosis, pulsatile exophthalmos, and ocular bruit; may also present with proptosis, diplopia, vision loss • Intraocular pressure may be elevated • Diagnose with computed tomography angiography or magnetic resonance angiography
Erysipelas	• Specific cellulitis involving dermis and subcutaneous tissues • Most cases caused by streptococcal species • Affects the face or lower extremities • Clinical diagnosis: erythema, warmth, swelling; well demarcated, raised borders • May present with systemic findings: fever, chills, malaise, headache, nausea/vomiting
Facial cellulitis	• Superficial soft tissue infection of the face; most commonly caused by *Streptococcal pyogenes* and *Staphylococcus aureus* • May have underlying etiology such as trauma, insect bite, radiation exposure/treatment, dental caries, foreign body • Risks include diabetes, immunosuppression, vascular injury • Clinical diagnosis: erythema, warmth, pain, edema
Meningitis or encephalitis	• Infection of the meninges (meningitis) or brain (encephalitis) • Bacterial, viral, or fungal etiologies • Headache, fever, meningismus, altered mental status • Patients may have seizure, rash, vomiting, photophobia, neurologic deficits • Diagnose via cerebrospinal fluid analysis obtained by lumbar puncture
Orbital cellulitis	• Postseptal infection of the orbit; ocular emergency • Fever may be present • Swelling and erythema of the eyelid and surrounding tissues • Patients have proptosis, limited or painful eye movements, double vision, vision changes, chemosis, and/or globe displacement • Intraocular pressure may be normal or elevated • Diagnose with computed tomography of the orbits, which will reveal postseptal infection
Periorbital cellulitis	• Soft tissue infection of the eyelid and other superficial tissues around the orbit • Fever may be present • Absence of proptosis, limited or painful eye movements, double vision, vision changes, chemosis, globe displacement • Intraocular pressure is normal • Computed tomography of the orbits will reveal preseptal infection and no orbital cellulitis
Sinusitis	• Infection of the sinuses; viral etiology most common • Clinical features include nasal congestion, facial pain/pressure, decreased smell, tooth pain, fever • Features suggestive of bacterial infection include symptoms >10 days, worsening or bimodal course, fever >39 degrees Celsius, purulent nasal discharge • Clinical diagnosis; computed tomography recommended only for critically ill patients

Table 2
CST pearls.

- CST is a rare but deadly thrombophlebitic disease affecting the cavernous sinus.
- The most common inciting event is a local infection (paranasal sinus, facial, odontogenic, otogenic, or pharyngeal infection); others include thrombophilia, maxillofacial surgery or trauma, dehydration, and idiosyncratic medication reactions.
- The most common signs and symptoms include fever, headache (frontotemporal or retro-orbital), and ocular manifestations (chemosis, periorbital edema, eyelid erythema, ptosis, proptosis, ophthalmoplegia, and vision changes). Cranial nerve palsies may occur, with CN VI most commonly affected (resulting in lateral gaze palsy).
- Leukocytosis occurs in up to 90% of cases, and elevated CRP and ESR are also common. However, normal laboratory testing should not be used to exclude the diagnosis.
- Blood cultures should be obtained and are positive in up to 70% of cases. CSF testing may reveal pleocytosis, elevated protein, and low glucose.
- CT of the heads and orbits with IV contrast delayed phase imaging may assist with diagnosis. MRV demonstrates the highest sensitivity for diagnosis.
- Treatment includes resuscitation, broad-spectrum antibiotics, anticoagulation, and specialist consultation.
- Antibiotics should cover staphylococcal and streptococcal species, gram-negative bacilli, and oral anaerobes. Fungal coverage is recommended in those with severe immunocompromise and poorly controlled diabetes.
- While there are no high-quality randomized data concerning anticoagulation in CST, it is likely safe and may reduce morbidity and mortality.

utilized. Treatment includes resuscitation, antibiotics, and anticoagulation with specialist consultation.

CRediT authorship contribution statement

Brit Long: Writing – review & editing, Writing – original draft, Visualization, Validation, Resources, Conceptualization. **Steven M. Field:** Writing – review & editing, Writing – original draft, Resources, Conceptualization. **Manpreet Singh:** Writing – review & editing, Visualization, Validation, Supervision, Resources, Conceptualization. **Alex Koyfman:** Writing – review & editing, Visualization, Validation, Supervision, Resources, Conceptualization.

Declaration of competing interest

None of the authors have submitted a review on this topic or published previously on this topic. No AI program was utilized to construct this review.

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