



A Case of Eye Pain and Confusion

Daniel G. Murphy, MD

A 24 year-old female presented to the ED with a headache for 2 weeks that had increased in severity for 2 days. At triage, her vital signs were BP 104/76, HR 81, RR 18, T 98.1F (orally). The headache was localized to the right frontal forehead, was non-radiating, sharp in quality, constant but waxing and waning in intensity. It was associated with nausea but no vomiting. There was no photophobia, neck pain, fever, or visual complaints. There was no history of recent trauma. The patient had tried ibuprofen, aspirin, and Tylenol PM at different times over the last few days to relieve the pain, but with no success. The headache worsened when the patient moved.

The patient smoked 1 pack per day of tobacco and occasionally drank alcohol (2-3 beers/week). She did not use illicit drugs. She lived at home with her parents and was a part-time sales clerk. Her past medical history was unremarkable and did not include a history of headaches. Her past surgical history included spinal surgery for scoliosis as a child. Her family history was significant for non-insulin dependent diabetes mellitus and coronary artery disease (father). Her last menstrual period was approximately 2 weeks prior to presentation and a UCG performed during triage was negative. She had no known allergies and had no known exposures to others who were ill. She took no medications other than those used to relieve this headache.

On exam, the patient was in moderate discomfort due to her headache. She was alert and oriented. She appeared well hydrated and nourished. Her face appeared normal; her pupils were equal, round and reactive to light. All extra-ocular movements were intact bilaterally. Fundoscopic exam revealed normal vessels and no papilledema. Her head and face exam were normal with normal TMs and mastoids. The patient's neck was nontender and freely moveable. Her neurologic exam was normal, including gait.

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The patient was treated with intravenous prochlorperazine, 10 mg, intravenously, and 2 tablets of acetaminophen/oxycodone (325/5) orally. A chem7 profile and complete blood count were sent to the lab and a CT scan of the brain without contrast was ordered. The patient's pain responded

to the medication and the CT was reviewed by the ED attending and radiology resident-on-call, who both noted no abnormal intracranial processes. Her CBC revealed a slightly elevated WBC count of 12.4K and an automated differential of 76% neutrophils, no other labs were or indices were outside the normal range. The patient was discharged with a diagnosis of headache and was given prepared discharge instructions for migraine headache and a prescription for Fioricet (acetaminophen/butalbital/caffeine).

Two days later, on a Monday morning, an attending radiologist reviewed the case and concurred that there was no acute intracranial disease, but noted opacification of the right ethmoid and right sphenoid sinuses with expansion of the sphenoid septations toward the left. After the emergency department was alerted to the over-read and discrepancy by radiology, the patient was contacted. The patient went to a private physician in the community who prescribed levofloxacin by mouth and sent her home.

That evening, the patient developed swelling around her left eye. Overnight, she slept poorly and her family thought that she was confused, possibly hallucinating. The next morning, her family returned her to the emergency department. Her vital signs were BP 100/80, HR 96, RR 18, T 101.9F (orally). She had prominent left peri-orbital edema and erythema, left proptosis, and excruciating pain with left eye movement. She was hemodynamically stable, breathing comfortably and able to communicate that her headache was worse on the right side with left eye pain. She followed simple commands well. However, she was mildly confused and answered simple questions slowly or incorrectly, with an apparent inability to focus on the interview. She seemed fatigued, lethargic, and mildly obtunded.

She preferred to keep both eyes closed. Visual acuity was not tested. There was no rhinorrhea. Her pupils were equal and reactive and her TMs were negative. Her left eye had chemosis. She cried out in pain when her neck was flexed. A cursory neurologic exam was negative. She had walked into the ED without a limp or abnormal upper extremity posture.

Two grams of ceftriaxone were immediately given by vein and a head CT was repeated, which revealed right pan-sinusitis (frontal, ethmoid and maxillary). An LP was performed and yielded cloudy or sedimented CSF with 1800 wbcs (100% neutrophils), 1200 rbcs, and no organisms. The CSF glucose was 61 and protein 93. No CSF bacterial antigens were detected by CIE. Her blood WBC count was 20K with 88% neutrophils.

After the LP, a neurologic motor exam revealed mild left upper and lower extremity weakness. Her mental status and neurological deficit worsened over the next few hours as MICU, ID, ENT and neurosurgical consults were performed in the ED. Vancomycin and metronidazole were added to the antibiotic regimen. The patient was admitted to the MICU.

Dural and Cavernous Sinus Thromboses

Background, Risk Factors and Epidemiology:

Suppurative intracranial thrombophlebitis or septic dural sinus thrombosis is an infected venous thrombosis of cortical veins and sinuses. This disease can occur as a dramatic complication of bacterial meningitis, subdural empyema, epidural abscess, or an infection of facial skin, paranasal sinus, middle ear, mastoid, maxillary teeth or neck. Iatrogenic cases of dural sinus thrombosis have been associated with rhinoplasty, hip surgery and oral/dental surgery.

Septic dural sinus thrombosis is a rare disease. One review noted that from 1940 to 1984, 19 cases of septic dural sinus thrombosis were diagnosed at the Massachusetts General Hospital, and some 136 cases were reported from other institutions (total = 155 over 44 years). Of these, septic thrombosis most frequently involved the cavernous sinuses (96 cases, 62%)(1).

Septic dural sinus thrombosis is generally a fulminant process with high rates of morbidity and mortality. Fortunately, the incidence of this disease has decreased significantly in the antibiotic era. Prior to effective antimicrobial agents, the mortality rate from cavernous sinus thrombosis (CST) was effectively 100%. With aggressive management, the mortality rate is now less than 30%. Morbidity, however, remains high, indeed complete recovery is rare. Roughly one sixth of patients are left with some degree of visual impairment, and one-half have residual cranial nerve deficits. The mortality rate in patients with septic thrombosis of the superior sagittal sinuses remains a bleak 78% (1,2).

There is no predilection for particular racial or ethnic groups. Both sexes are affected equally. All age groups are affected, with the mean age being 22 years. But again, the documented number of cases has been very small.

Most cases of septic CST occur as a consequence of an acute infection or injury in an otherwise healthy individual. However, patients with chronic sinusitis or diabetes mellitus may be at a slightly higher risk (3-5).

Anatomy and Pathophysiology

Intracranial veins and venous sinuses have no valves. Blood within them can flow in either direction. The superior sagittal sinus, the largest of the venous sinuses, receives blood from the frontal, parietal, and occipital superior cerebral veins and the diploic veins, which communicate with the meningeal veins. Bacterial meningitis is a common predisposing condition for septic thrombosis of the superior sagittal sinus. The diploic veins provide a route for the spread of infection from the meninges to the superior sagittal sinus. Infection can also spread to the superior sagittal sinus from nearby subdural empyema or epidural abscess.

Non-infectious causes of venous sinus thrombosis include dehydration from vomiting, hypercoagulable states, and immunologic abnormalities, including the presence of circulating antiphospholipid antibodies. Thrombosis may extend from one sinus to another. Thrombosis of the superior sagittal sinus is often associated with thrombosis of superior cortical veins and small parenchymal hemorrhages.

Regardless of location and cause, if recanalization of a thrombosed dural sinus does not occur, venous hypertension will lead to cerebral edema, infarction, and hemorrhage.

The superior sagittal sinus drains into the transverse (or “lateral”) sinuses. The transverse sinuses also receive venous drainage from small veins from both the middle ear and mastoid cells. The transverse sinus becomes the sigmoid sinus before draining into the internal jugular vein. Septic transverse/sigmoid sinus thrombosis can be a complication of acute and chronic otitis media or mastoiditis (6). Infection spreads from the mastoid air cells to the transverse sinus via the emissary veins or by direct invasion. The cavernous sinuses are inferior to the superior sagittal sinus at the base of the skull. The cavernous sinuses receive blood from the facial veins via the superior and inferior ophthalmic veins. Bacteria in the facial veins enter the cavernous sinus via these veins. Bacteria in the sphenoid and ethmoid sinuses can spread to the cavernous sinuses via the small emissary veins. The sphenoid and ethmoid sinuses are the most common sites of primary infection resulting in septic cavernous sinus thrombosis.

The cavernous sinuses are reticulated structures, traversed by numerous interlacing filaments. They are of irregular shape, one on either side of the body of the sphenoid bone, extending from the superior orbital fissure to the apex of the petrous portion of the temporal bone. They lie on either side of the sella turcica, are just lateral and superior to the sphenoid sinus, and are immediately posterior to the optic chiasm. On the medial wall of each sinus is the internal carotid artery, accompanied by filaments of the carotid plexus; near the artery is the abducens nerve; on the lateral wall are the oculomotor and trochlear nerves, and the ophthalmic and maxillary divisions of the trigeminal nerve. These structures are separated from the venous blood flowing along the sinus by the lining membrane of the sinus. The two sinuses communicate with each other by means of the anterior and posterior intercavernous sinuses.

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 sinuses. Again, this complex web of veins contains no valves; blood can flow in any direction depending on the prevailing pressure gradients. This intimate juxtaposition of veins, arteries, nerves, meninges, and paranasal sinuses accounts for the characteristic etiology and presentation of CST.

The major pathogens associated with cavernous-sinus infection include *Staphylococcus aureus*, other gram-positive organisms, and anaerobes. Septic lateral-sinus thrombosis (64 cases) is almost exclusively a complication of otitis media and/or mastoid infection. Organisms causing this infection include *Proteus* species, *Escherichia coli*, *S. aureus*, and anaerobes. Septic thrombosis of the superior sagittal sinus (23 cases) most frequently accompanies bacterial meningitis or air sinus infection. Causative organisms include *Streptococcus pneumoniae*, *S. aureus*, other streptococci, and *Klebsiella* species (1).

ED Presentation

Septic thrombosis of the superior sagittal sinus presents as headache, nausea and vomiting, confusion, and focal or generalized seizures. There may be a rapid development of stupor and coma. Weakness of the lower extremities with bilateral Babinski signs or hemiparesis is often present. When superior sagittal sinus thrombosis occurs as a complication of bacterial meningitis, nuchal rigidity and Kernig's and Brudzinski's signs may be present.

Patients with cavernous sinus thrombosis (CST) generally have sinusitis or a midface infection (most commonly a furuncle) for 5-10 days. In as many as 25% of cases in which a furuncle is the precipitant, it will have been manipulated in some fashion (e.g., squeezing, surgical incision).

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The symptoms of septic cavernous sinus thrombosis are fever, headache, malaise, retro-orbital pain and diplopia, which generally precede the development of ocular findings. The classic signs are ptosis, proptosis, chemosis, eyelid edema, peri-orbital edema and extra-ocular dysmotility due to deficits of cranial nerves III, IV, and VI. Hypo- or hyperesthesia of the ophthalmic and maxillary divisions of the fifth cranial nerve and a decreased corneal reflex may be detected. There may be evidence of dilated, tortuous retinal veins and papilledema. Meningeal signs may be noted, including nuchal rigidity and Kernig and Brudzinski signs. In some patients, periorbital findings do not develop early on, and the clinical picture is subtle.

Without effective therapy, signs appear in the contra-lateral eye by spreading through the communicating veins to the contralateral cavernous sinus. This is pathognomonic for CST. The patient rapidly develops mental status changes from CNS involvement and/or sepsis. Death follows shortly thereafter (5).

Headache and earache are the most frequent symptoms of transverse or lateral sinus thrombosis. A transverse sinus thrombosis may also present with Gradenigo's syndrome characterized by otitis media, sixth nerve palsy, and retroorbital or facial pain. Sigmoid sinus and internal jugular vein thrombosis may present with neck pain (4).

Lab Studies

CST is a clinical diagnosis and lab studies are seldom specific. Most patients exhibit a polymorphonuclear leukocytosis, often marked with a shift toward immature forms. Examination of the cerebrospinal fluid is consistent with either a parameningeal inflammation or frank meningitis. Blood culture results generally are positive for the offending organism.

Imaging Studies

Historically, a number of techniques have been used to image CST, including plain sinus radiography, carotid angiography, and orbital venography. In current practice, computed tomography (CT) scan – preferably with contrast, MRI or MR venography are the modalities of choice to confirm the diagnosis of dural sinus thrombosis. The diagnosis of septic venous sinus thrombosis is suggested by an absent flow void within the affected venous sinus on MRI and confirmed by magnetic resonance venography or the venous phase of cerebral angiography (7-9,11).

On CT, there may be enlargement of the cavernous sinus with internal filling defects and incomplete enhancement of the sinus. There may be periorbital edema, prominence of the superior ophthalmic vein, and exophthalmos (14). Sphenoid sinusitis may be present with air-fluid levels.

Thrombosis of the cavernous sinus can be appreciated as increased density. The introduction of intravenous contrast can reveal filling defects within the cavernous sinus. Nevertheless, CT scan findings may be subtle, and a negative CT scan cannot rule out CST reliably when the clinical suspicion is high.

MR images may show high signal thrombus in the cavernous sinus. Because thrombus is often high in signal intensity, contrast medium administration is of little help in the evaluation.

Procedures

Lumbar puncture may be helpful in distinguishing CST from more localized processes (e.g., sinusitis, orbital cellulitis).

Emergency Department Care

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.

Anticoagulation with heparin may be considered and generally is supported by most recent literature (12, 13). Retrospective analysis suggests that treatment with heparin may reduce mortality in carefully selected cases of septic cavernous-sinus thrombosis. Anticoagulation is not recommended in other forms of septic dural-sinus thrombosis. Anticoagulation with dose-adjusted heparin has been reported to be beneficial in patients with aseptic venous sinus thrombosis; it is also used (as a last ditch effort?) in the treatment of septic venous sinus thrombosis complicating bacterial meningitis in patients who are worsening despite antimicrobial therapy and intravenous fluids. The presence of a small intracerebral hemorrhage from septic thrombophlebitis is not an absolute contraindication to heparin therapy. Successful management of aseptic venous sinus thrombosis has been reported with urokinase therapy and with a combination of intrathrombus recombinant tissue plasminogen activator (rtPA) (10) and intravenous heparin, but there is yet no reported experience with these therapies in septic venous sinus thrombosis.

Corticosteroids may help to reduce inflammation as an adjunctive therapy. When the course of CST leads to pituitary insufficiency, however, corticosteroids definitely are indicated to prevent adrenal crisis.

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 (e.g., sphenoid sinusitis, facial abscess).

Consultations

If drainage is indicated, make arrangements for intensive care and request the appropriate surgical consultation.

Summary

Septic dural sinus thrombosis, including cavernous sinus thrombosis, is a rare and deadly emergency. Septic cavernous sinus thrombosis can affect people suffering from sphenoid or ethmoid sinusitis and can mimic orbital cellulitis by blocking venous return from the orbit. Contralateral proptosis to an affected sphenoid or ethmoid sinusitis is pathognomonic for CST.

Early broad-spectrum antibiotics adequately covering *S. aureus* and consideration of surgical drainage of the infected sinus should be done. Anticoagulation, steroids and even thrombolytics may prove beneficial, but prospective, randomized trials are not feasible.

The CST syndrome is a rare life-threatening emergency that should be considered when evaluating a patient with a meningitis syndrome, headache, fever, ethmoid or sphenoid sinusitis, altered

mental status or ipsi-lateral or contra-lateral eye findings similar to orbital cellulitis. A diagnosis of ethmoid or sphenoid sinusitis on CT or MR should prompt concern about CST as a co-morbidity or sequela, perhaps especially if the sinus is appears completely opacified with a mass effect.

Dural and Cavernous Sinus Thromboses

Reference List

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14. Larson TL: Petrous apex and cavernous sinus: anatomy and pathology. *Semin Ultrasound CT MR* 1993 (Jun); 14(3): 232-46
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Dural and Cavernous Sinus Thromboses

Outcome of Case

On the evening of admission, her neurologic and mental status worsened. She developed a left sided facial droop and complete left hemiparesis. She suffered a generalized seizure. She was intubated orotracheally and phenytoin was given. A repeat CT was done with contrast revealing possible cavernous sinus thrombosis. Heparin was initiated.

On hospital day 2, ceftriaxone was discontinued and ceftazidime was initiated. Initial blood and CSF culture results were negative. The patient was lethargic but opened her eyes and had motor function improvement on her left side.

The patient's mental status and left sided motor function relapsed by the end of day 2. A MR of the brain and MR cerebral venogram were performed, revealing pansinusitis, a right sided thalamic infarct, and thrombi in the cavernous sinus, the superior sagittal sinus and the left transverse sinus.

Drainage of the ethmoid and sphenoid sinuses was performed on hospital day 5. Electrolyte disturbances, an episode of bradycardia and unresponsiveness to painful stimuli were noted on day 6. Cerebral edema and transtentorial herniation were noted on repeat CT. The patient died after a steady deterioration of multiple organ systems and brain death on hospital day 19.

Diagnoses: Pan-sinusitis; Thromboses of the superior sagittal sinus, transverse sinus and cavernous sinus; Brain death.

Dural and Cavernous Sinus Thromboses

Annotated Bibliography

1. **Southwick FS, Richardson EP Jr, Swartz MN: Septic thrombosis of the dural venous sinuses. *Medicine* 1986 Mar; 65(2): 82-106**

This is a retrospective review of 19 cases of septic dural-sinus thrombosis that were diagnosed at the Massachusetts General Hospital between 1940 and 1984. The author also discovered that only 136 cases had been reported from other institutions in the same time period. Clinical findings, culture results and risk factors are evaluated. The analysis also suggests that treatment with heparin may reduce mortality in carefully selected cases of septic cavernous-sinus thrombosis, but not in other forms of septic dural-sinus thrombosis.

2. **Ben-Uri R, Palma L, Kaveh Z.: Septic thrombosis of the cavernous sinus: diagnosis with the aid of computed tomography. *Clin Radiol* 1989 Sep; 40(5): 520-522**

A case of septic cavernous sinus thrombosis is described which was observed with high resolution computed tomography (CT). The significant CT findings in cavernous sinus thrombosis are discussed, including unilateral or bilateral swelling of the orbital soft tissues and sinusitis.

3. **Guermazi A, Miaux Y, Williams M, et al: Dural sinus thrombosis: CT and MR imaging of different stages. *J Belge Radiol* 1997 Aug; 80(4):167-169**

Reports a case with different stages of thrombosed superior sagittal and right transverse dural sinuses demonstrated by CT and MR imaging.

4. **Hsu FP, Kuether T, Nesbit G, Barnwell SL: Dural sinus thrombosis endovascular therapy. *Crit Care Clin* 1999 Oct;15(4):743-753**

Discusses endovascular approaches using direct infusion of thrombolytic drugs into the occluded sinuses, hypothesizing recanalization and improved patient outcomes.

5. **Levine SR, Twyman RE, Gilman S: The role of anticoagulation in cavernous sinus thrombosis. *Neurology* 1988 Apr; 38(4):517-522**

Reviews seven cases and the literature to determine the effectiveness of anticoagulation, complications, and morbidity among survivors. They found no conclusive evidence for reduction of mortality when anticoagulation is used in combination with antibiotics, but noted that early anticoagulation may reduce morbidity - blindness, stroke, ophthalmoplegia, hypopituitarism, focal seizures, and vascular steal syndrome.

- 6. Cipri S, Gangemi A, Campolo C, et al: High-dose heparin plus warfarin administration in non-traumatic dural sinuses thrombosis. A clinical and neuroradiological study. *J Neurosurg Sci* 1998 Mar; 42(1): 23-32**

Reports 7 cases of non-traumatic thrombosis of the dural sinuses, describing important radiographic findings, the indication & effectiveness of antithrombotic therapy, and outcome. Six patients had have a good quality recovery, and one patient a moderate disability, although follow up contrast-CT scans and Angio-MRI showed no re-canalization of the sinuses, in all patients.

- 7. Larson TL: Petrous apex and cavernous sinus: anatomy and pathology. *Semin Ultrasound CT MR* 1993 (Jun); 14(3): 232-46**

A detailed review of this anatomy, followed by a discussion of pathology.

Dural and Cavernous Sinus Thromboses

Questions

- 1. Which of the following is pathognomonic for cavernous sinus thrombosis?**
 - a. Fever with disorientation
 - b. Unilateral CN6 palsy with proptosis, edema and sinusitis
 - c. Expressive aphasia and sinusitis
 - d. Contra-lateral proptosis with sphenoid sinusitis
 - e. Vertigo, chemosis and proptosis

- 2. Which of the following is not consistent with a transverse dural sinus thrombosis?**
 - a. Fever, headache, malaise, retro-orbital pain and diplopia.
 - b. Headache, nausea and vomiting, confusion, and focal or generalized seizures.
 - c. Bilateral vision loss.
 - d. Headache and earache.

- 3. Which of the following therapies do not have proven benefit in cavernous sinus thrombosis?**
 - a. Early and aggressive antibiotics.
 - b. Surgical drainage of infected sphenoid sinus.
 - c. Heparinization.
 - d. Locally injected thrombolytics.

- 4. Which disease has the highest mortality rate?**
 - a. Cavernous sinus thrombosis.
 - b. Subarachnoid hemorrhage.
 - c. Superior sagittal sinus thrombosis.
 - d. Transverse sinus thrombosis.

- 5. Which of the following is false about cavernous sinus thrombosis?**
 - a. It is reliably visualized on CT.
 - b. It can occur in association with lateral and superior sagittal thromboses.
 - c. Can cause cerebral ischemia and stroke.
 - d. Is suggested by an absent flow void within the affected venous sinus on MRI.

6. What findings will likely be absent in a patient with a “swollen eye” due to CST and will probably be present in a patient with isolated orbital cellulitis?

- a. Peri-orbital and lid edema.
- b. Extra-ocular movement impairment.
- c. Proptosis.
- d. Palpable warmth.
- e. Fever.

Dural and Cavernous Sinus Thromboses

Answers

1. Answer d.

Contra-lateral proptosis with sphenoid sinusitis – pathognomonic.

2. Answer a, b, c.

Headache and earache or mastoiditis are associated with transverse sinus thrombosis.

3. Answer a, b, c.

Only antibiotics are known to effective based on morbidity and mortality data before and after the advent of antibiotics.

4. Answer c.

Superior sagittal sinus thrombosis. I suspect due to an association with various grim comorbidities.

5. Answer a.

CST is not reliably visualized on CT. CT scan findings may be subtle, and a negative CT scan cannot rule out CST reliably when the clinical suspicion is high.

6. Answer d.

In theory, there would only be “inflammatory” warmth, if any, on palpation. Orbital cellulitis feels hotter.