Cavernous Sinus Thrombosis As A Complication Of Sphenoid Sinusitis: A Case Report And Review Of Literature

R Ahmad, R Salman, S Islam, A Rehman

Citation

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Abstract

Reported is a rare case of cavernous sinus thrombosis complicated by sphenoid sinusitis leading to right sided ophthalmoplegia and ipsilateral visual field involvement. An attempt is made to correlate the aetiopathology with the clinical features.

INTRODUCTION

Cavernous sinus thrombosis (CST) was initially described by Bright in 1831 as a complication of epidural and subdural infections. CST is usually a late complication of an infection of the central face or paranasal sinuses. Other causes include bacteremia, trauma, and infections of the ear or maxillary teeth. CST is generally a fulminant process with high rates of morbidity and mortality. Septic cavernous sinus thrombosis is now a rare dis¬ease as the incidence of CST has been decreased greatly with the advent of effective antimicrobial agents[1]. Blindness as a result of cavernous sinus throm¬bosis is uncommon, occuring in less than 10 per cent of cases[1] . Several mechanisms may be implicated. Cor¬neal ulceration may occur from failure of lid closure[2]. Occlusion of the central retinal artery may develop from pressure at the orbital apex[3] or as the result of embolium[4]. Focal arteritis leading to occlusion of the inter¬nal carotid artery may cause ophthalmic artery ischaemia[5]. Toxic neuritis of the optic nerve has been suggested as a possible cause of blindness, secondary to 'adjacent purulent inflammation'. Ischaemic optic neuropathy has been thought to be the cause of blind¬ness in a case reported by Friberg and Sogg[6]. Thrombosis of the cavernous sinus may be due to extension of retrograde thrombosis from various sour¬ces. Because of the communications of the cavernous sinus with other venous channels, infection may occur via the orbital veins (as in septic lesions of the face, mouth, pharynx, ear, nose and paranasal sinuses), or as a metastasis in infectious diseases or septic condi¬tions[7].

CASE REPORT

A 22 year old female patient was admitted in the ENT ward with the chief complaints of right sided proptosis and pain, fever and headache for 5 days. The patient subsequently developed periorbital puffiness and lid edema in the left eye.

The patient had a history of headache . A day after, she started having high-grade fever associated with chills or rigors. This was followed by progressive swelling and redness of both eyelids in the right eye with proptosis and opthalmoplegia developing subsequently. The patient after admission developed periorbital puffiness and lid edema of the left eye also. There was no history of previous ocular disease, injury, hypertension or diabetes.

General Physical Examination: On the day of admission the patient was semiconscious, not anaemic, jaundiced or cyanosed. There was neither lymphadenopathy nor oedema. The pulse was 100/ minute and regular. The blood pressure was 120/70 mm. Hg. and the temperature was 102° F. Systemic Examination : Revealed nothing abnormal . Ocular Examination : Visual acuity was 6/6 J1 in both eyes. Left eye: Showed normal occular movements, slight prop¬tosis, but absent consensual pupillary reaction. Right eye showed proptosis. There was complete ptosis of the upper lid and total ab¬sence of ocular movements in all directions. The pupil was semi - dilated and non-reacting. The optic disc was pale with clear-cut margins and normal optic cup. Visual fields of the right eye showed concentric constric¬tion, with lower nasal quadrantic defect.

The patient (Image 1) after admission was put on high dose

antibiotics, but did not show any response to the treatment. A CT scan of the nose and paranasal sinuses was adviced which revealed radio-opacity involving the sphenoid sinus and an orbital abscess on the right side (Image 3). A subsequent MRI of the patient confirmed the findings of sphenoid sinusitis and phlegmon in the right orbit and also revealed engorgement of the cavernous sinus, ophthalmic veins and extraoccular muscles (Images 4 & 5). The signal from the abnormal cavernous sinus was heterogenous with an obvious hyperintense signal of the thrombosed vascular sinus on the right side. A diagnosis of cavernous sinus thrombosis of the right side with ipsilateral ophthalmoplegia and visual field involvement secondary to sphenoid sinusitis was made.

Endoscopic examination of the patient was undertaken and pus was drained from the right sphenoid sinus.The patient showed dramatic response after drainage with recovery of conciousness and complete resolution of orbital symptoms (Image 2).

Investigation: The blood examination showed leucocytosis with 83 per cent polymorphs. Blood culture was negative. Urine examination showed no abnormality.

Lumbar puncture of the patient showed clear fluid under pressure. X-ray of the skull, lateral view did not show any abnor¬mality.

The patient was managed on intravenous antibiotics, including Pippercillin + Tazobactum and Vancomycin, and local decongestants.

Figure 1

Image 1: Proptosis of right eye with bilateral lid edema., Image 1: Pre-operative photograph of patient.

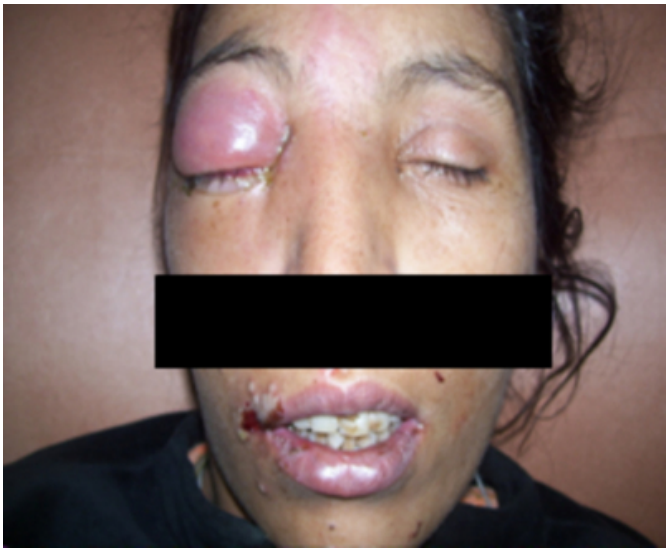


Figure 2

Image 2: Same patient after recovery., Image 2: Post-op image.

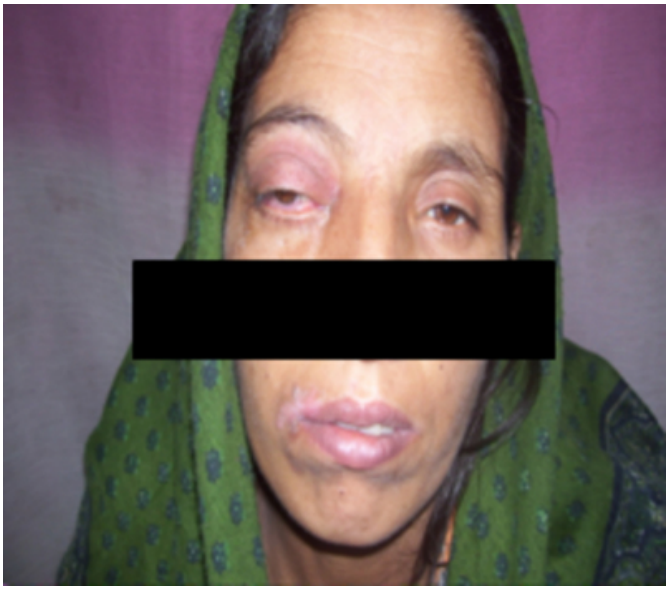


Figure 3

Image 3: CT scan revealing radio-opacity involving the sphenoid sinus and an orbital abscess on the right side.

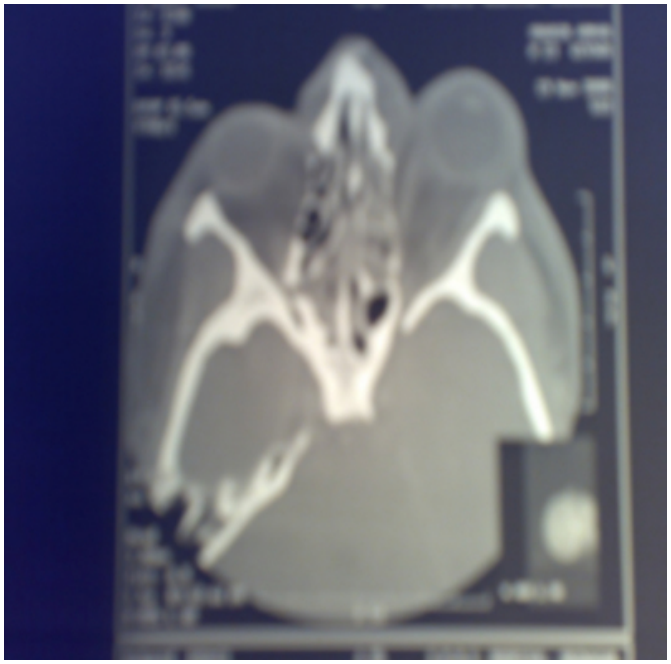


Figure 4

Image 4: A Magnetic resonance image of the cavernous sinuses ,with an axial, T2 – weighted, postgadolinium image, revealing proptosis of right eye with thrombosis in right superior ophthalmic vein and right cavernous sinus with sphenoid sinusit

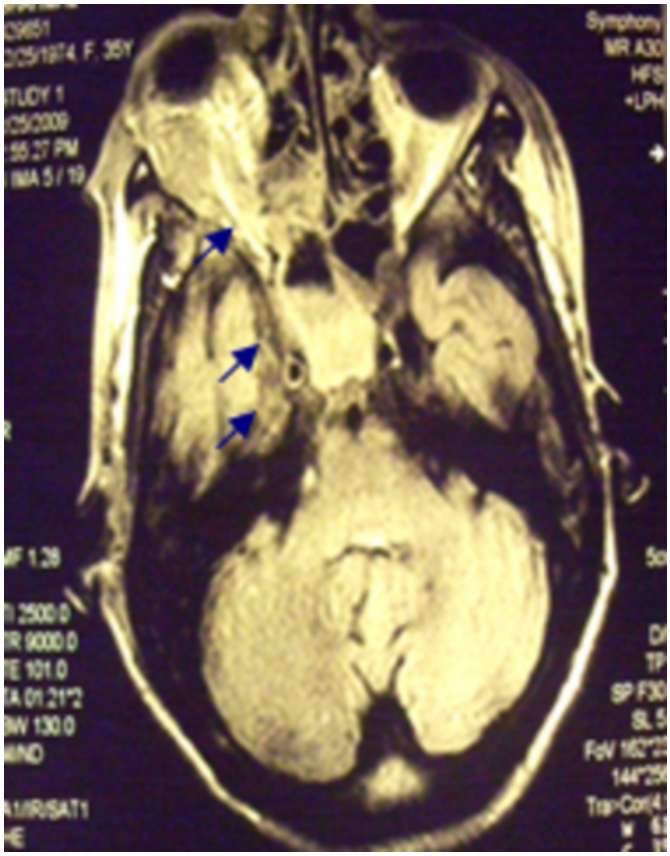
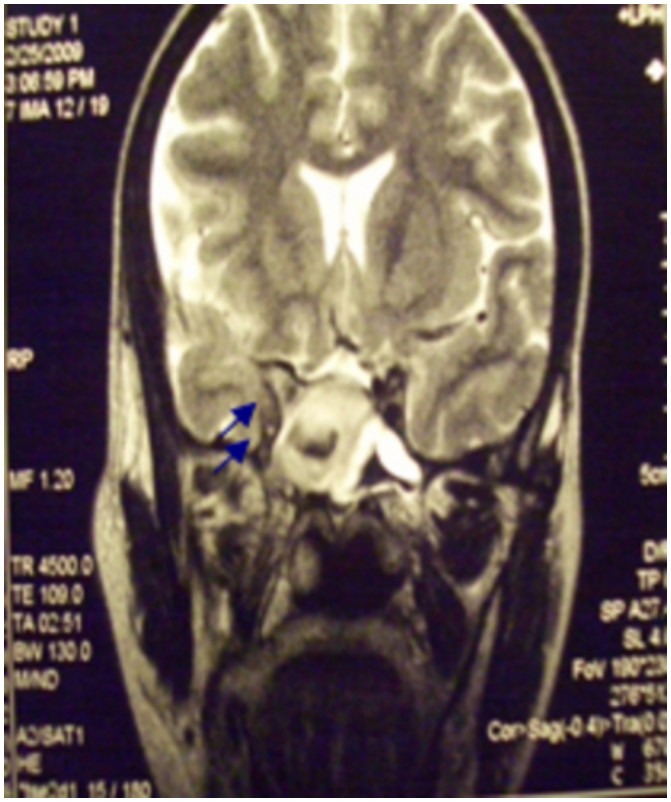


Figure 5

Image 5: A Magnetic resonance image of the cavernous sinuses, with a coronal (frontal),T2-weighted, postgadolinium image, revealing a bulging lateral wall of the cavernous sinus and a large filling defect secondary to thrombus on the right side with sphenoid sinusitis



DISCUSSION

The cavernous sinuses are irregularly shaped, trabeculated cavities located at the base of the skull. The cavernous sinuses are the most centrally located of the dural sinuses and lie on either side of the sella turcica. These sinuses are just lateral and superior to the sphenoid sinus and are immediately posterior to the optic chiasm . Each cavernous sinus is formed between layers of the dura mater, and multiple connections exist between the 2 sinuses [1,8].

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. This complex web of veins contains no valves; blood can flow in any direction depending on the prevailing pressure gradients. Since the cavernous sinuses receive blood via this distribution, infections of the face including the nose, tonsils, and orbits can spread easily by this route.

The internal carotid artery with its surrounding sympathetic plexus passes through the cavernous sinus. The third, fourth, and sixth cranial nerves are attached to the lateral wall of the sinus. The ophthalmic and maxillary divisions of the fifth cranial nerve are embedded in the wall [1,2].

This intimate juxtaposition of veins, arteries, nerves, meninges, and paranasal sinuses accounts for the characteristic etiology and presentation of CST.

Ophthalmic signs of fully established cavernous sinus thrombosis include proptosis, chemosis, lid oedema, ophthalmoplegia, retinal venous engorgement (oc¬casionally associated with hemorrhages'), low- grade optic disc oedema, and at times, generalised retinal oedema, amd even corneal anesthesia with sub¬sequent ulceration. Visual acuity may be reduced during the initial stages as the result of pressure on the retinal artery at the orbital apex. The signs are unilateral to start with, but the other eye eventually becomes involved [9]. Cavernous sinus thrombosis produces ophthalmoplegia and papilloedema. Primary optic atrophy occurring in cavernous sinus thrombosis is not known.

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 and may include Aspergillus and Rhizopus species [1-4].

Occurrence of CST has always been low, with only a few hundred case reports in the medical literature. The majority of these date from before the modern antibiotic era. One review of the English-language literature found only 88 cases from 1940-1988.[10]

Prior to the advent of effective antimicrobial agents, the mortality rate from CST was effectively 100%. 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. There is no sex predilection and all ages are affected, with a mean of 22 years[1].

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Author Information

Rouf Ahmad, M.S.
Associate Professor, Department of ENT & HNS, Government Medical College

Raja Salman, MBBS
Post graduate scholar, Department of ENT & HNS, Government Medical College

Shafqat Islam, M.S.
Registrar, Department of ENT & HNS, Government Medical College

Ayaz Rehman, MBBS
Post graduate scholar, Department of ENT & HNS, Government Medical College