The diagnosis and management of septic cavernous sinus thrombosis in a previously healthy paediatric patient: case report

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Research Article

Keywords: Ocular trauma, cavernous sinus thrombosis, ptosis, ophthalmoplegia, paediatric ophthalmology

Posted Date: August 1st, 2022

DOI: https://doi.org/10.21203/rs.3.rs-634667/v1

Abstract

Background

Septic cavernous sinus thrombosis (CST) is a rare complication of local or systemic infection. Despite aggressive antibiotic treatment, it can be associated with significant rates of mortality and morbidity.

Case Presentation

We report a left sided traumatic septic CST and internal carotid artery (ICA) occlusion complicated by third, fourth and sixth nerve palsies in a healthy boy. This was treated conservatively with intravenous antibiotics and anticoagulants with near complete resolution of ocular movement deficit

Conclusion

Septic cavernous sinus thrombosis is a rare complication of the spread of untreated infection, predominantly of facial or paranasal origin. A high clinical suspicion in at-risk patients of early symptoms and signs, such as persistent fever and meningism, should prompt diagnostic neuroimaging and early intervention prior to the onset of advanced features such as cranial nerve palsies. In our experience, a timely multi-disciplinary approach with intensive parenteral antibiotics and anti-coagulants is associated with good clinical outcomes.

Background

Septic cavernous sinus thrombosis is a rare complication of the spread of untreated infection, predominantly of facial or paranasal origin. A high clinical suspicion in at-risk patients of early symptoms and signs, such as persistent fever and meningism, should prompt diagnostic neuroimaging and early intervention prior to the onset of advanced features such as cranial nerve palsies.

Other previously reported cases in a paediatric setting have been reported in patients with underlying health conditions with significant functional impairment on treatment. Here, we describe the timely identification and conservative treatment of septic CST in a healthy paediatric patient with significant improvement in ophthalmoloplegia and ptosis.

Case Presentation

A previously fit and well 12-year-old boy was referred with a traumatic left upper lid laceration and an unreactive pupil following a fall whilst running where he landed on his head. He described instant severe left sided ocular and facial pain with loss of vision.

Clinical examination demonstrated reduced visual acuity in the left eye (6/60 best corrected compared with 6/5 in the right eye) with elevated intra-ocular pressure (30mmHg with Goldmann applanation tonometery). There was a 10mm laceration extending inferiorly from the brow to the left upper lid which

was grossly oedematous. The left globe was proptosed by 3mm with subconjunctival haemorrhage. The left pupil was fixed and dilated. Eye movements were painful. The remainder of the examination of the globe and adnexa was unremarkable.

A left sided retrobulbar haemorrhage with upper lid injury was diagnosed and an urgent lateral canthotomy and cantholysis performed. Intravenous metronidazole and cefuroxime were commenced in combination with topical chloramphenicol ointment. A computed tomography (CT) scan later demonstrated a left sided retro-bulbar haematoma centred supra-temporally with lateral rectus swelling (Figure 1). A sustained improvement was noted subjectively and objectively over the next few days (BCVA 6/36, IOP 14mmHg) whilst the patient remained on intravenous antibiotics. The brow laceration healed well by secondary intention.

Six days following the initial injury, the patient complained of a severe frontal headache and later developed diplopia. Ductions of the left eye were significantly reduced (-4) in all directions with a complete ptosis (Figure 2). The remaining cranial nerves functioned normally on clinical examination. Magnetic Resonance Imaging (MRI) demonstrated a collection measuring 13mm x 8mm within the left orbit (Figure 3A). This ran along the superolateral wall, extending posteriorly into the orbital apex and cavernous sinus (Figure 3B). In addition, the left internal carotid artery was occluded along its entire length (Figure 3C). MRI head and orbits were performed at the time of diagnosis to confirm a suspected orbital collection, however vascular imaging with Magnetic Resonance Angiography (MRA) or CT Angiography (CTA) were not done, as ICA thrombosis and CST were not suspected when imaging was initially requested. MRI clearly showed a left orbital collection extending into the cavernous sinus and a diagnosis of left ICA thrombosis, therefore, further vascular imaging was deemed unnecessary at this stage. The neurosurgical team reviewed the case and felt there was no scope for surgical intervention. The patient was transferred to an intensive care unit and commenced on a heparin infusion with input from the haematology team.

The patient gradually improved over the next few weeks with conservative treatment. Repeat MRI scan confirmed improvement compared to the previous, there was flow at the centre of the left ICA and reduction in the size of the collection, which no longer involved the cavernous sinus. Two months following the initial injury, the patient’s symptoms had markedly improved. Best corrected visual acuity was 6/12 but the left pupil remained fixed and dilated. The ptosis reduced (4mm palpebral aperture) and there was a subtle but persistent restriction of left elevation (-1) and adduction (-1) with full abduction and depression. Formal vascular imaging was also performed at this time with MRA, which demonstrated recanalisation of the left ICA (Figure 4).

Discussion And Conclusion

The cavernous sinus is a paired dural sinus in the middle cranial fossa related inferiorly to the optic chiasm and superolaterally to the sphenoid air sinuses. The cavernous sinus drains the orbits through the superior and inferior ophthalmic veins and the brain through the sphenoparietal sinus and the middle and

inferior cerebral veins. They are drained by the superior and inferior petrosal sinuses into systemic venous circulation via the sigmoid sinus and internal jugular vein respectively. Of key clinical importance, its walls transmit the oculomotor, trochlear and the ophthalmic (V1) and maxillary (V2) branches of the trigeminal nerve. The abducens nerve travels within the cavernous sinus lateral to the internal carotid artery.

Septic CST is a rare thromboembolic complication of infectious aetiology. Bacteria are known to produce several pro-thrombotic mediators and are well incubated within the thrombus [1, 3]. Involvement of the cavernous sinus may arise through antegrade or retrograde venous circulation; via the superior and inferior ophthalmic veins or the pterygoid venous plexus respectively, or through direct invasion from adjacent structures [1]. Infection most commonly originates from nearby facial tissue, such as the paranasal air sinuses although septic embolisation from distant tissue has been reported[4]. In a review of 88 cases, Weerasinghe et al reported 57% of cases were related to a paranasal sinusitis (most commonly sphenoidal). The next frequent sources of infection were mid-facial (13%) and dental infections (12%). Causative organisms are diverse; methicillin-resistant staph.aureus is a common bacterial culprit with Aspergillus fumigatus was the most common fungal growth, predominantly in immunocompromised patients [5]. In our case, blood cultures did not demonstrate a causative pathogen, most probably because empirical antibiotics had been started immediately following the injury on presenting to hospital and prior to the onset of systemic symptoms suggestive of sepsis and subsequent collection of blood cultures.

Early systemic features of septic CST include persistent fever, meningism and headache. Ophthalmic signs develop secondary to venous congestion and raised intracranial pressure and include; proptosis, peri-orbital oedema, hyperaemia, chemosis and disc swelling [1]. Such features are typically initially unilateral. Due to the intimate anatomical relationship, third, fourth, sixth and fifth (V1 and V2) cranial nerve palsies are typical later features of septic CST. The sixth nerve is particularly vulnerable given its intraluminal position within the cavernous sinus [2].

Anti-microbial therapy has resulted in a significant improvement in the outcomes of patients with septic CST [6]. Despite this, morbidity and mortality rates remain high, particularly in vulnerable groups such as paediatric and immunocompromised cohorts [5, 6]. One study reported all paediatric patients with septic CST and radiographic-proven orbital involvement had significant visual impairment despite treatment (hand movements or worse) [7].

Delayed diagnosis of septic CST may be a factor in the poor visual outcomes and other complications such as meningitis, brain abscess, cerebral infarction or, such as in this case, occlusion of the internal carotid artery secondary to arteriospasm, thrombosis or arteritis [6]. Neuroimaging, ideally with MRI scanning, is essential to exclude differential diagnoses (e.g. orbital cellulitis) and to correctly diagnose and identify sequalae of septic CST. Blood and tissue cultures should ideally be performed prior to commencing anti-microbial therapy but, like neuroimaging, should not delay their commencement. In the absence of targeted anti-microbial therapy, high dose, extended broad-spectrum intravenous antibiotics

with close monitoring is essential. Anti-fungal agents are added depending on clinical circumstances. For example, failure to improve with antibiotics or in cases of immunosuppression [2]. Anti-coagulants, usually low molecular weight heparins, are used in a significant proportion of patients. In one review, the use of anti-coagulants was associated with a lower mortality rate (12% versus 28%) [5]. Such findings concur with earlier historical studies [6]. The role of corticosteroids is more unclear but may reduce oedema and inflammation although there are risks to consider such as immunosuppression and hypercoagulability [1, 2]. Evidence suggests that mortality and morbidity rates are broadly similar irrespective of corticosteroid therapy use [5]. Surgical intervention is generally reserved for evacuation or removal of infected tissue where possible [1].

In this instance, septic cavernous sinus thrombosis originated from extension of an orbital abscess and responded to parenteral anti-coagulants and intensive intravenous antibiotics without the need for surgical intervention. The patient regained significant vision (best corrected visual acuity 6/12) and ocular motility (-1 left elevation and adduction). The patient and his guardian were pleased with the functional and cosmetic outcome. In contrast to other reports, there was no pre-existing medical condition identified despite extensive investigation.

Abbreviations

CST – cavernous sinus thrombosis

ICA – internal carotid artery

MRA - Magnetic Resonance Angiography

CTA – CT angiography

Declarations

Funding: No funding was obtained for this report.

Competing Interests: The authors declare that there is no conflict of interest regarding the publication of this article.

Ethical approval: No ethical approval was required.

Consent for publication: The patient and his guardian have provided written conset for publication.

Consent for participation: Not applicable

Availability of data and materials: Not applicable

Code availability: not applicable

Author Contributions:

MGM – Writing of paper, KA: review of paper, obtaining images DR: surgeon involved in care, review of paper, TK: review and writing of paper, AK: senior consultant, supervising of project. All authors have reviewed and approved the manuscript.

Acknowledgements: We thank Dr Stuart Viner for the selection of images for inclusion in this report.

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Figures

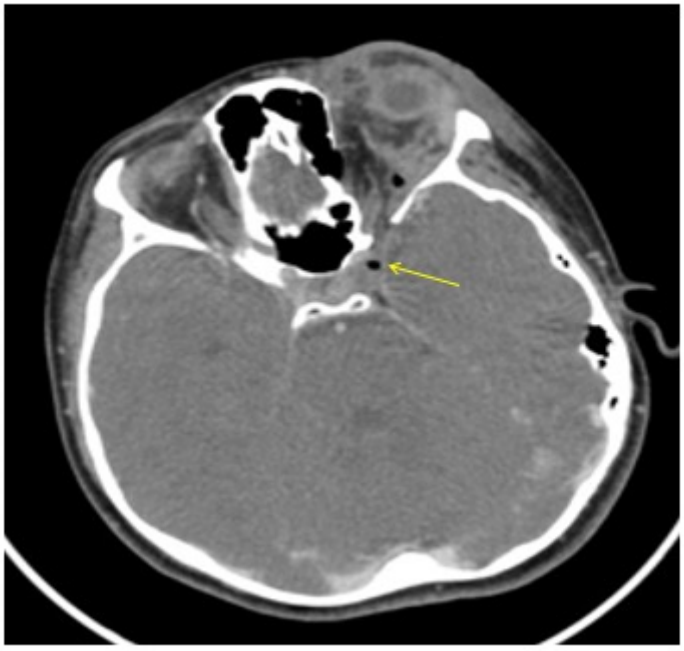

Figure 1

Axial post contrast CT orbits. Marked left sided proptosis with inflammatory changes in orbit. Multiple low density foci in orbit, extending into cavernous sinus (yellow arrow), felt likely to be air related to trauma or recent canthotomy. Wooden foreign bodies may have similar appearances on CT.

Figure 2

Presentation with complete left ptosis 6 days post initial injury

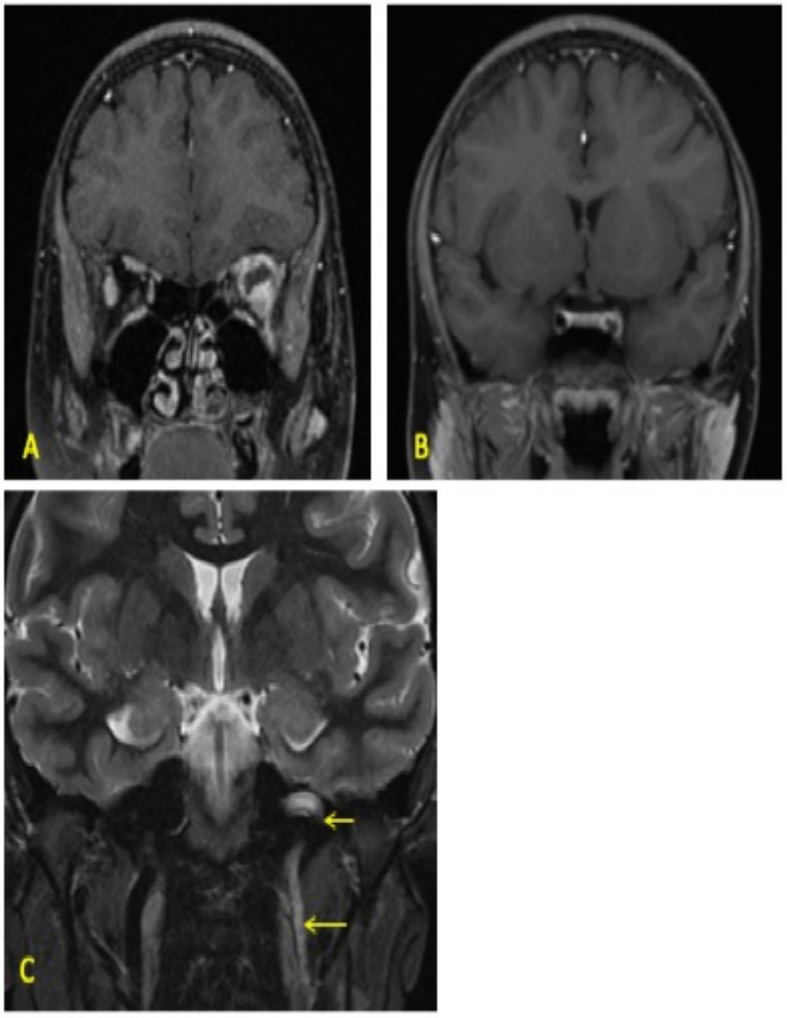

Figure 3

MRI: A and B. Coronal post contrast T1 with fat saturation. Peripherally enhancing collection in the superolateral orbit. Collection extends into the cavernous sinus.

C. Coronal T2 STIR. Absent flow void (yellow arrows) from left ICA indicating thrombosis.

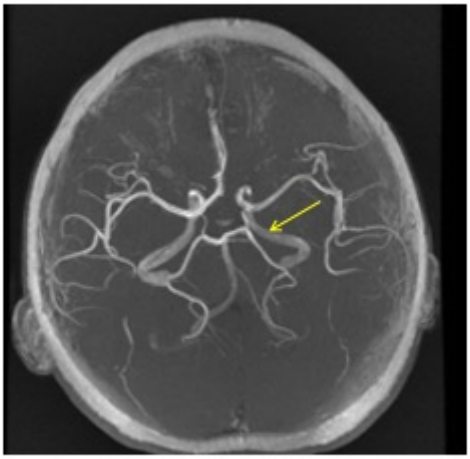

Figure 4

Time of flight MR Angiogram. Normal flow in the left ICA (yellow arrow).