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Can J Ophthalmol 2016;51:e72–e75
0008-4182/16/$-see front matter
http://dx.doi.org/10.1016/j.jcjo.2015.12.008

Bilateral septic cavernous sinus thrombosis, congestive orbitopathy, and ischemic optic neuropathy

A 73-year-old woman who experienced worsening headache and fever for 2 weeks presented with left congestive orbitopathy with globally limited motility and optic neuropathy but intact trigeminal function. Her visual acuity (VA) was 20/30 OD and 20/70 OS with a 0.9 log unit relative afferent pupillary defect (RAPD) OS. Intraocular pressures (IOPs) were 16 mm Hg OD and 22 mm Hg OS. Both fundi appeared normal.

Opacification of the sphenoid and right posterior ethmoid sinuses and fat stranding within the right posterior orbit were noted on computed tomography (Fig. 1A). Magnetic resonance imaging (MRI) demonstrated bilateral cavernous sinus thrombosis (CST) (Fig. 1B), bilateral superior (Fig. 1C) and left inferior ophthalmic vein thrombosis, and left transverse sinus thrombosis.

Because of a penicillin and cephalosporin allergy, she was treated with intravenous vancomycin 1500 mg/12 hours for gram-positive coverage, aztreonam 2 g/8 hours for gram-negative, and clindamycin 900 mg/8 hours for anaerobic coverage. She underwent emergent sphenoidotomy and right posterior ethmoidectomy under general anaesthesia. The mean arterial blood pressure fell to 60-70 mmHg twice. The nasal mucosa was injected with 2% lidocaine with 1:80 000 epinephrine and sprayed with oxymetazoline. The IOP OS increased to 31 mm Hg. Fundoscopy revealed mild venous tortuosity and a perfused central retinal artery (CRA), but no optic disc edema OU. A left canthotomy and inferior cantholysis was performed intraoperatively.

Eight hours postoperatively, the VA was no light perception (NLP) OS and 20/160 OD with moderate congestive orbitopathy and a >1.2 log unit RAPD OS. IOP was normal bilaterally. There was no optic disc edema or retinal artery occlusion. Repeat MRI demonstrated restricted diffusion within the left optic nerve (Fig. 1D, E). Sinus cultures were positive for *Streptococcus anginosus*. Aztreonam and clindamycin were discontinued and metranidazole 500 mg/ 8 hours was added. Methylprednisolone was administered intravenously once daily for 4 days (250, 250, 100, and 60 mg). Within hours of the first dose, the

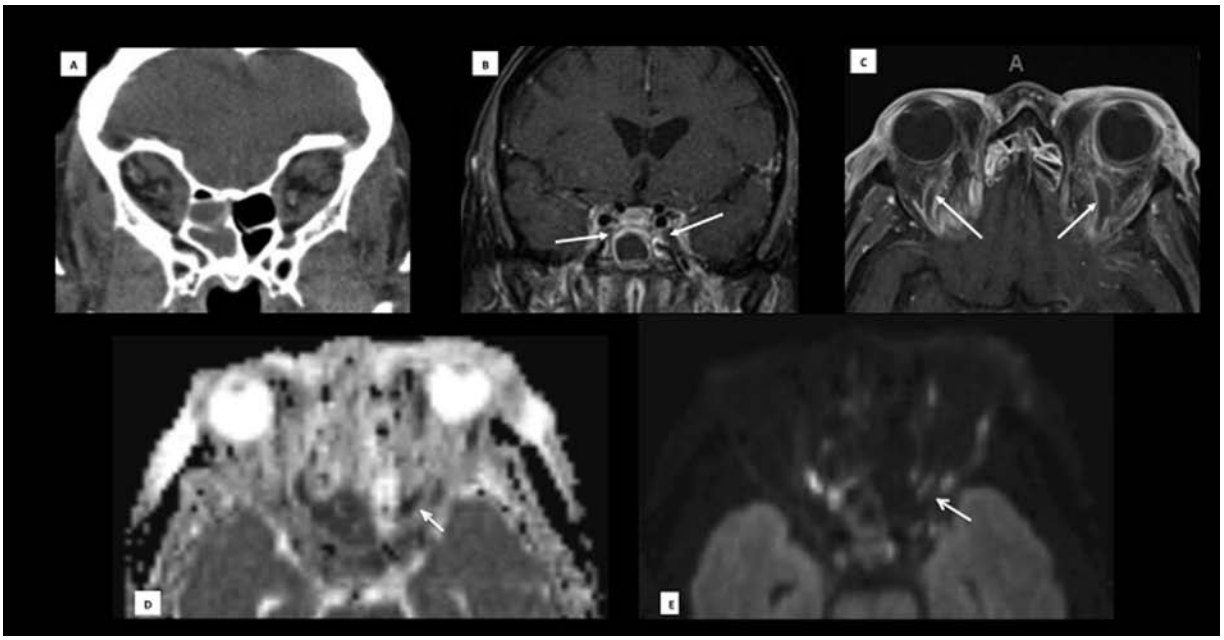

Fig. 1—(A) Coronal contrast-enhanced maxillofacial computed tomography scan showing right posterior ethmoid sinus opacification and fat stranding within the right posterior orbit. (B) Coronal T1-weighted contrast-enhanced brain magnetic resonance imaging (MRI) demonstrating enlargement of both cavernous sinuses with convexity of the lateral walls and filling defects (arrows) within the sinuses corresponding to areas of thrombosis. (C) Axial T1-weighted contrast-enhanced orbital MRI showing filling defects within both superior ophthalmic veins (arrows). (D) The axial diffusion-weighted MRI and (E) the corresponding diffusion coefficient map demonstrating restricted diffusion in the left optic nerve (arrows).

orbital congestion was reduced bilaterally, and VA improved to 20/30 OD but remained NLP OS. Low-molecular-weight heparin (LMWH) was started 36 hours postoperatively.

On day 5, VA OD recovered to 20/25, with an inferonasal visual field defect, and remained NLP OS. The patient was discharged on an oral prednisone taper, 3 months of warfarin, and 10 weeks of oral moxifloxacin. Three weeks postoperatively, all orbital congestion had resolved and motility was full, but VA remained unchanged. Optic disc pallor was present bilaterally. Eighteen months after discharge, the patient remained stable. This report has adhered to the principles outlined in the Declaration of Helsinki.

Before the antibiotic era, septic CST, a thrombophlebitic process caused by infection of the paranasal sinuses, was fatal. Currently, it is associated with 15% to 30% mortality.[1] In atypical cases of unilateral orbital pathology, expedited neuroimaging is vital for establishing the diagnosis of CST; the poor prognosis for septic CST may be due to delays in diagnosis.[2] In those who survive, morbidity from cranial neuropathies occurs in up to 50% and optic neuropathy in approximately 17%.[3,4]

Visual loss in CST may result from thromboembolism of the CRA[5] or orbital compartment syndrome (OCS) causing CRA occlusion or ischemic optic neuropathy.[6–10] A canthotomy and cantholysis OS was performed prophylactically in view of the impending OCS. The visual loss was likely caused by posterior ischemic optic neuropathy (PION), supported by an initial normal-appearing optic disc and restricted diffusion within the left optic nerve. Restricted diffusion in both optic nerves and retinas has been reported in a patient with bilateral blindness after CST.[10] PION results from reduced blood flow to the posterior intraorbital optic nerve, which is supplied by multiple small collateral arteries usually arising directly from the ophthalmic artery and less often from other arteries. The rise in orbital congestion and venous pressure caused by CST and ophthalmic vein thrombosis produced reduced perfusion pressure in our patient and, hence, decreased flow within these vessels. This decreased flow was exacerbated by administration of labetolol when the mean arterial pressure rose to 150 mm Hg and by episodes of intraoperative arterial hypotension. Vasoconstriction of terminal arterioles was likely produced by intraoperative vasopressor agents,[11] and topical epinephrine and oxymetazoline may have diffused from the sinonasal mucosa across the lamina papyracea, causing vasoconstriction of orbital vessels. These multiple risk factors in combination are likely to have created the ideal conditions for PION. Surgical PION tends to cause severe bilateral visual loss that is usually permanent.[11]

Treatment with high-dose steroid during the acute stages of nonarteritic PION may produce improvement of VA and visual fields.[11] Steroid treatment may have contributed to visual recovery OD, whereas the greater orbital venous congestion OS resulted in irreversible ischemia. The role of steroids in septic CST, other than prevention of an Addisonian crisis, is unclear, however.[12] The immunosuppressive effects of steroids are potentially harmful, but, in cases such as ours in which the infectious organism has been identified and treated with appropriate antibiotics, their anti-inflammatory properties may help reduce congestive orbitopathy and cranial neuropathy.[4,9,12]

In septic CST, antibiotics should be given for an extended period to treat sequestered bacteria within the thrombus. The *S. anginosus* groups, also known as *Streptococcus milleri*, were isolated in our patient and are a subgroup of *Streptococcus viridans*. Their ability to cause abscesses is unique from other pathogenic streptococci.[13]

We started LMWH to prevent further thrombus propagation and because heparin has antiplatelet and anti-inflammatory properties.[14] If started early, anticoagulation can improve morbidity in septic CST, rarely with hemorrhagic complications[15] if overanticoagulation is avoided.[2] Anticoagulation should be continued until there is radiological confirmation of thrombus resolution.

Disclosures: The authors have no proprietary or commercial interest in any materials discussed in this article.

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***Can J Ophthalmol* 2016;51:e75–e77**
0008-4182/16/$-see front matter
http://dx.doi.org/10.1016/j.jcjo.2015.12.007