Clin Neuroradiol (2019) 29:371–374
https://doi.org/10.1007/s00062-018-0729-6

CORRESPONDENCE

Lateral rectus atrophy in cavernous sinus thrombosis

Bertie Geng[2] · Xiao Wu[2] · Ajay Malhotra[1]

Received: 6 May 2018 / Accepted: 15 September 2018 / Published online: 19 October 2018

Introduction

Septic cavernous sinus thrombosis (CST) is thrombophlebitis in the cavernous sinus, which may occur as a complication of midfacial infections, paranasal sinusitis, pharyngitis, and rarely otitis media or dental infections. Although mortality from CST has dropped from almost 100% to less than around 30% with the advent of antibiotics, CST remains a devastating disease with many long-term sequelae including cerebral infarcts, pituitary insufficiency, blindness and external ophthalmoplegia [1]. This article presents a unique case of CST with irreversible internal carotid artery (ICA) narrowing and isolated, acute lateral rectus muscle denervation with subsequent atrophy.

Case Presentation

A young woman with a history of recurrent acute pansinusitis presented with fever and altered mental status. On examination, the patient had meningismus, proptosis and an abduction defect in the left eye consistent with cranial nerve VI palsy. The patient had no previous visual or neurologic issues, or any other relevant past medical history. Computerized tomography (CT) scan (Fig. 1) revealed paranasal sinus opacification in the sphenoid sinus, left maxillary sinus and bilateral ethmoid air cells. Asymmetric bulging of the left cavernous sinus was noted on noncontrast images. Postcontrast images showed poor contrast enhancement of the left cavernous sinus, and a thickened, nonopacified left superior ophthalmic vein, likely due to thrombosis. Magnetic resonance imaging (MRI) showed approximately 70% stenosis of the cavernous segment of the left ICA (Fig. 2). Proptosis was noted in the left orbit with enlargement, T2 hyperintensity and asymmetric enhancement of the left lateral rectus muscle (Fig. 3). The patient was diagnosed with sinusitis that led to bacterial meningitis of unknown microbiological cause and cavernous sinus thrombosis. No definite leptomeningeal enhancement, however, was appreciated on MRI brain study. The patient underwent endoscopic sinus surgery and was treated with anticoagulation and prednisone for the CST, as well as with ceftriaxone, ertapenem, metronidazole, and fluticasone to treat the infection. The clinical condition improved during hospitalization, and warfarin and ertapenem were continued after discharge for 1 month.

After 2 months following discharge, the patient was alert and oriented with normally reactive pupils, intracranial pressure, no proptosis, and unremarkable physical examination except for a large left esotropia in the primary gaze. Left lateral rectus muscle atrophy and persistent narrowing in the left ICA was revealed by MRI (Fig. 4). The computed tomographic angiography (CTA) showed approximately 60% stenosis compared to the right ICA. The patient was referred to a strabismus specialist for surgery to correct the esotropia.

Discussion

A CST is a much rarer disease in the post-antibiotics era but it can have a severe and lasting impact. This report is unique in demonstrating a case of CST causing irreversible ICA stenosis as well as acute denervation followed by atrophy of the lateral rectus muscle. Cases of CST often present with a high fever. Common ophthalmic signs include swelling of the orbit that eventually expands to the swelling of the face on the affected side and can expand to involve cranial nerves III, IV, V1, V2 or VI due to the anatomy of the region [2–4]. Bilateral eye involvement can

Bertie Geng
bertie.geng@yale.edu

Xiao Wu
xiao.wu@yale.edu

✉ Ajay Malhotra
ajay.malhotra@yale.edu

[1] Department of Radiology and Biomedical Imaging, Yale School of Medicine, 333 Cedar St, New Haven, CT 06520-8042, USA

[2] Yale School of Medicine, New Haven, USA

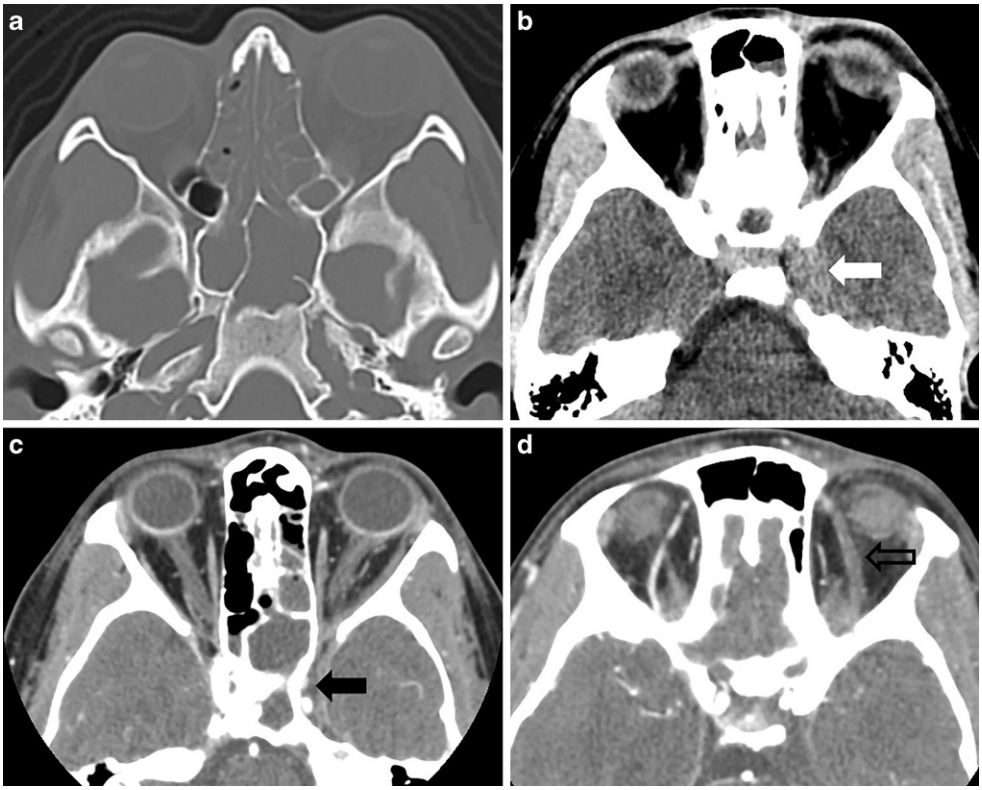

Fig. 1 Axial noncontrast CT images show extensive paranasal sinus mucosal disease and near complete opacification of ethmoid and sphenoid sinuses (**a**) with subtle, asymmetric bulge in the left cavernous sinus (*white arrow*; **b**). Postcontrast images show nonfilling of left cavernous sinus (*black arrow*; **c**) with enlarged, nonopacified left superior ophthalmic vein (*blank arrow*; **d**)

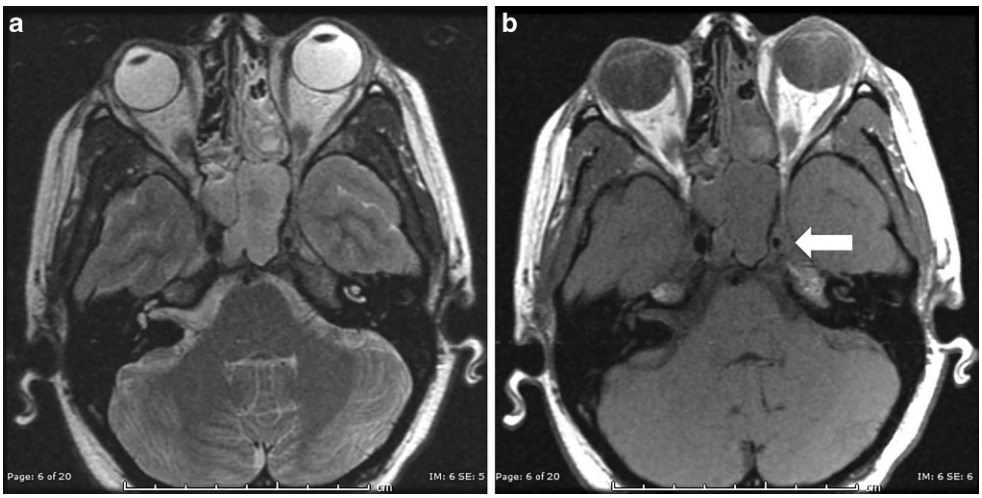

Fig. 2 Axial T2 (**a**) and T1 WI (**b**) show extensive paranasal sinus disease including the sphenoid sinus and ethmoids. Asymmetric soft tissue is seen in the left cavernous sinus, with narrowing of left cavernous internal carotid artery (ICA) (*arrow*; **b**)

occur in cases of septic CST because the cavernous sinuses are connected by intercavernous venous channels. The predominant pathogens in septic CST include Gram-positive cocci including *Staphylococcus aureus* and streptococcal species, similar to those found in primary sites of infection. Blood and surgical specimens can be used to identify the causative organism in 70% of cases of CST [5]. Although the microorganism causing the patient's infection was never identified, the patient was started on empirical high-dose IV antibiotics in order to reduce mortality and long-term sequelae related to the disease. The duration of appropriate antibiotic treatment is not well established, but commonly used regimens range in duration from 3 to 4 weeks [6]. The efficacy of anticoagulants in the treatment of CST has yet to be proved in the literature but anticoagulants are commonly prescribed as they theoretically should prevent further thrombus formation [7].

Involvement of the ICA was previously believed to be uncommon with CST [8]; however, recent literature documents frequent involvement. Press et al. found carotid artery narrowing in all 10 of the pediatric patients with CST, and 6/10 developed infarcts. The arterial narrowing re-

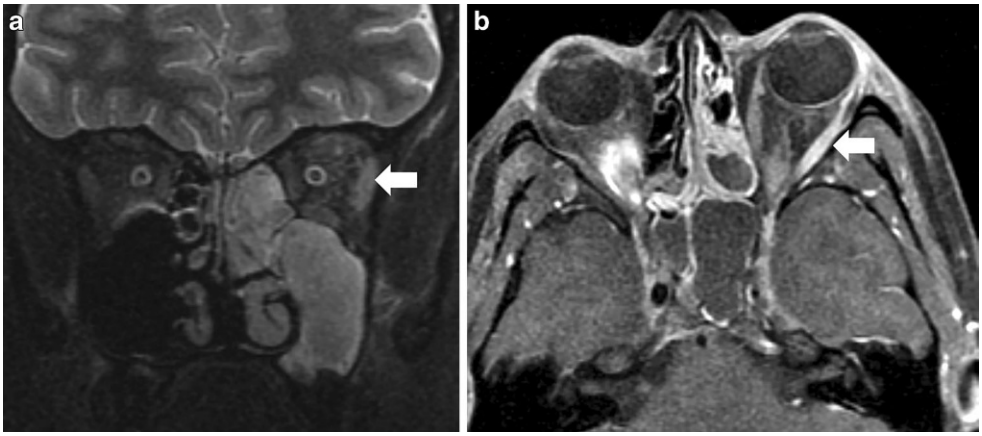

Fig. 3 Coronal T2 (**a**) and axial postcontrast (**b**) images show asymmetric left lateral rectus swelling, T2 hyperintensity and enhancement relative to other extraocular muscles (*arrows*)

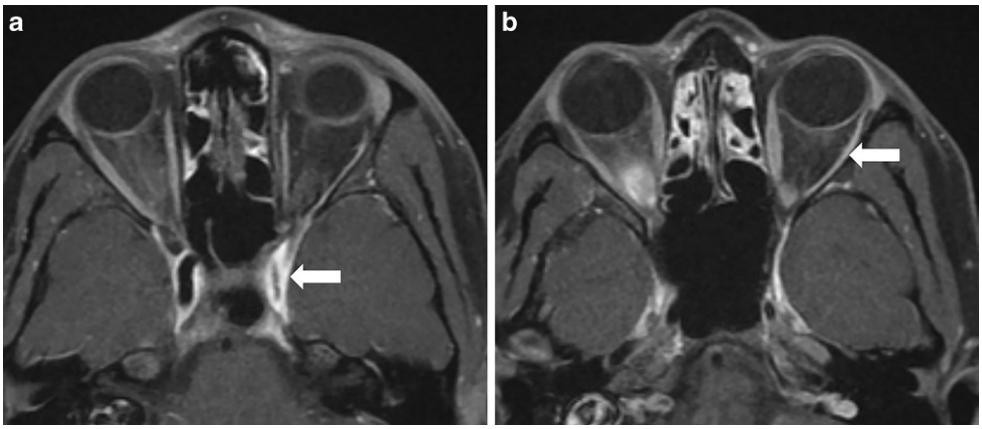

Fig. 4 Follow up imaging 2 months later—axial post-contrast images show persistent narrowing of the cavernous ICA (*arrow*; **a**) and interval atrophy of the left lateral rectus muscle (*arrow*; **b**)

solved in 60% of cases [9]. Orbital involvement was found in 8 patients with orbital cellulitis in 5 and optic neuritis in 3 patients, all of which were clinically significant. Smith et al. also found ICA abnormalities to be common (83%) on magnetic resonance angiograms (MRA), which varied from mild narrowing to complete occlusion, but no large arterial strokes; however, all cases of ICA narrowing improved on follow-up imaging with 70% resolution within 6 months [10]. Our patient had persistent, greater than 50% stenosis of ICA despite treatment, without evidence of cerebral ischemia.

Although cranial nerve involvement with CST has been reported, no case was found in the literature on imaging findings of acute denervation of extraocular muscles with CST. Frank et al. found motility deficits in 8/9 children with CST [11]. The majority of these patients had orbital cellulitis, but 2 patients had permanent ophthalmoplegia without orbital cellulitis. The mechanism of restriction of ocular movements in CST has been debated, with muscle swelling as a result of orbital edema and neural damage in the cavernous sinus being postulated causes. Most reported cases of ophthalmoplegia are reversible, and Walsh et al. showed histological evidence of infection in all six cases that came to necropsy [12]. Normal initial visual acuity may be a positive predictor of recovery of ophthalmoplegia [11]; however, as shown by this case, ophthalmoplegia can occur in the absence of orbital cellulitis due to cranial nerve dysfunction in CST. Radiologists should be aware of the possibility of acute denervation changes in extraocular muscles with CST.

Conflict of interest A. Malhotra, B. Geng and X. Wu declare that they have no competing interests.

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