

Cavernous sinus thrombosis after follow-up cerebral angiography

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Keywords: Carotid cavernous sinus fistula, cavernous sinus thrombosis, cerebral angiography

Introduction

Cerebral vein and dural sinus thrombosis is less common than most other types of stroke, being responsible for 1% to 2% of all strokes in adults.¹ It has diverse clinical and imaging features, risk factors, and variable outcomes.² Furthermore, data on natural history, management and prognosis is limited when compared with arterial stroke.³ Iatrogenic cavernous sinus thrombosis (CST) is extremely rare with very few cases reported in the literature.^{4,5}

Case report

A 66-year-old male was submitted to embolization of a direct left carotid-cavernous fistula (CCF) one month after a suicide attempt by gunshot to the head. No complications were noted during the procedure, and the patient was discharged with a mild left proptosis.

He was admitted for follow-up cerebral angiography nine months later. The angiography showed incomplete closure of the CCF and normal patency of the ipsilateral cavernous sinus (Fig. 1A). Apart from two episodes of vomiting of unknown etiology a few hours before and after the angiography, no other complications occurred. The vomiting ceased with antiemetic medication (ondansetron 4 mg) and caused no electrolyte or other analytic disturbances, even though it led to the need of fluid therapy for 24 hours while restarting oral feeding. On the following day, worsening of the proptosis was noted and was accompanied by pain, hyperaemia and raised intraocular

pressure (Fig. 1B). There was no bruit over the left orbit. Ophthalmological evaluation demonstrated marked visual acuity loss associated with left mono-ocular hypertension of up to 54 mmHg. Although no carotid bruit was present and no further intervention was made to the residual CCF, suspicion of reopening of the direct CCF was raised. Alternative diagnosis such as an increased flow rate of the fistula or mispositioning of the coils were also considered. Cerebral angiography performed afterwards (Fig. 1C) excluded these differentials and showed the presence of a complete ipsilateral CST.

A decision to start anticoagulation was made and enoxaparin was started on the same day (1 mg/kg twice a day). Clinical improvement was evident within the first 48 hours. The patient's symptoms kept improving, allowing for hospital discharge eleven days after admission, with normal intraocular pressure and only mild proptosis and chemosis (Fig. 1D).

On the follow up, almost 2 years after the CST, the patient presented with progressive left sided pleuritic chest pain which led to the diagnosis of a malignant pleural mesothelioma.

Discussion

CST represents an unusual but damaging disease, with a myriad of clinical presentations, and can be associated with significant long-term patient morbidity and/or mortality. The prompt recognition and management of this problem is critical. Nevertheless it might be a challenging diagnostic process.

CST after cerebral angiography is a particularly uncommon phenomenon. Although the underlying pathophysiology is not fully understood, there is some evidence that angiographic contrast media may exacerbate the process of leukocyte adhesion in the presence of venous stasis and tissue damage.⁴ In our patient, in addition to the contrast-induced hyperviscosity, the presence of vomit and subsequent fastening induced dehydration^{4,5} and the presence of a thrombogenic coil mass packing the left cavernous sinus may have further aggravated the prothrombotic status leading to in-situ thrombosis⁶ in an anatomically disrupted cavernous sinus. Although a malignant pleural mesothelioma was diagnosed almost 2 years afterwards, probably related to professional asbestos exposure (contractor), it is not likely that this may have contributed to the prothrombotic status since there was no clinical nor imaging evidence of this malignancy at the time of the CST. Besides, the association of mesothelioma and venous thrombosis is not commonly described in literature and the cases described are usually associated with direct compression of the veins by the tumor tissue⁷ which was not the case. In conclusion, CST may be a very rare and potentially serious complication of catheter angiography warranting prompt diagnosis and treatment.

Sponsorships or competing interests that may be relevant to content are disclosed at the end of this article.

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Porto Biomed. J. (2020) 5:6(e097)

Received: 5 January 2020 / Accepted: 5 October 2020

<http://dx.doi.org/10.1097/j.pbj.000000000000097>

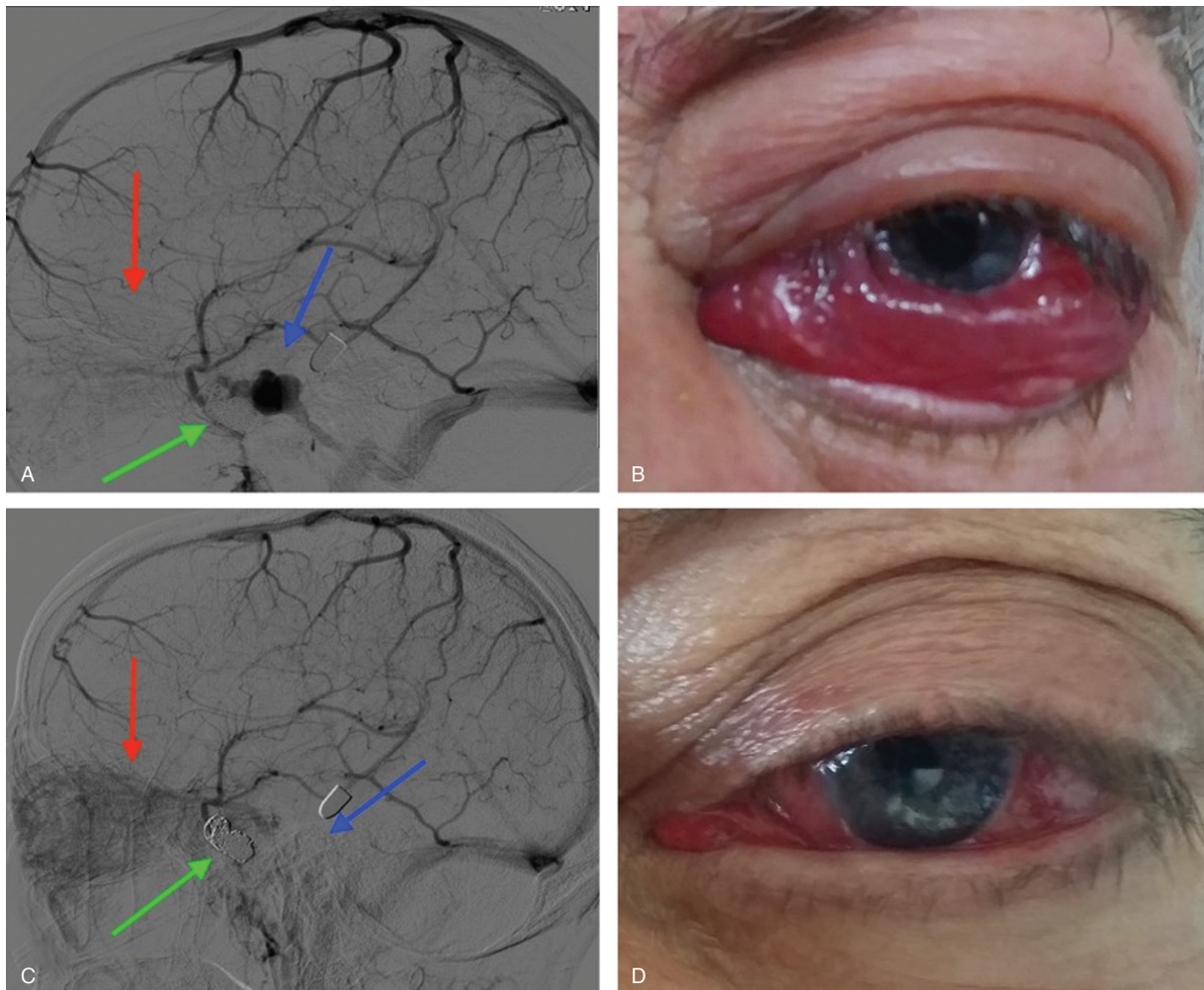


Figure 1. (A) Cerebral digital subtraction angiography, late venous phase, showing patency of the left cavernous sinus 9 mo after partial embolization of a left carotid cavernous fistula (blue arrow) with retrograde flow to the inferior petrosal sinus and residually to the ophthalmic veins (red arrow). (B) 24 h after the procedure, the patient presented with progressive proptosis, hyperemia and raised intraocular pressure. (C) Cerebral digital subtraction angiography, late venous phase, 24 h after the beginning of symptoms, indicating pericircular venous stasis (red arrow) and thrombosis of the left cavernous sinus (blue arrow), and confirming the correct positioning of the coil mass (green arrow), as shown in the first figure. (D) Clinical improvement 8 d after anticoagulation.

Disclosure

The manuscript, as submitted or its essence in another version, is not under consideration for publication elsewhere, and will not be published elsewhere while under consideration by this journal. However, the work was partially presented in the form of oral communication in a local stroke course in Portugal (June 2017), previously to the diagnosis of pleural mesothelioma. The authors have no commercial associations or sources of support that might pose a conflict of interest. All authors have made substantive contributions to the study, and all authors endorse the data and conclusions. A written informed consent was obtained from the patient before submitting the manuscript.

Conflicts of interest

The authors declare no conflicts of interest.

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