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Endovascular treatment of traumatic Carotid Cavernous fistula: A case report

Vojsava Leka[1], MD, Aida Agastra[1] MD, Oneda Cibuku[2] MD, Stela Dodaj[1] MD Mirel Grada[1], MD, Arben Rroji[1] MD, PhD, Eugen Enesi[1] MD, PhD Mentor Petrela[1], MD, PhD, IFAANS, WANS*

[1] Department of Neurosurgery and Interventional Neuroradiology, American Hospital 3, Tirana, Albania
[2] Department of Stroke Unit, UHC Mother Theresa, Tirana, Albania

***Corresponding author:**

MENTOR PETRELA

American Hospital 3, Tirana, Albania
Email: *mentor.petrela@gmail.com*

Abstract

Background: Direct carotid cavernous fistulas (dCCF) result from disruption of the cavernous internal carotid artery (ICA) causing direct shunting into the cavernous sinus[1,2]. Trauma is the most common cause of dCCF, as in our patient. The clinical presentation is a triade of chemosis, proptosis, and exophthalmos._Endovascular management is the current treatment for CCFs. Trans arterial or trans venous route are used for the endovascular treatment [1,3].

Case presentation: A 75-year-old male presented one month after a traumatic brain injury from a syncope due to high blood pressure with bilateral exophthalmos, proptosis and eyelid swelling more expressed on the right side. Neurologic examination revealed: bilateral ophthalmoplegias, amaurosis OD, right afferent pupillary reflex and right corneal reflex was absent, left hyporeactive corneal reflex, left pupilar reflex was present, in the auscultation bilateral temporal systolic noise and supraorbital pulsation were evident.

On ophthalmologic evaluation bilateral papilledema with high intraocular pressure was noticed. A cerebral Digital Substraction Angiography (DSA) was performed.

Direct right CCF, Type A according to Barrow was confirmed and endovascular treatment was suggested.

Intervention: Endovascular treatment with direct detachable balloon occlusion of the right CCF under general anesthesia through arterial femoral access.

Embolization of the cavernous sinus with coiling under the protection ICA with a balloon.

Outcome:. After a first clinic impromevent, the conditions worsened with progression of exophthalmos and proptosis bilaterally.

Another DSA was performed which showed re-opening of the right dCCF so embolization of the cavernous sinus with coiling was decided.

After the procedure, he was without systolic noise and supraorbital pulsation on auscultation bilaterally. Exophthalmos and proptosis were reduced and after 14 days visual acuity, ophthalmoplegia on the left eye improved.

Conclusion. dCCFs are a rare but treatable cause of cranial trauma. Endovascular embolization of CCFs with coiling or liquid agents is the only treatment.[2]

Key words: Carotido-Cavernous fistulas (CCF), trauma, endovascular, ophthalmoplegia

Introduction

Direct carotid cavernous fistulas (dCCF) results from disruption of the cavernous internal carotid artery (ICA) causing direct shunting into the cavernous sinus[1,2]. Trauma is the most common cause of dCCF, as in our patient, accounting for up to 75% of all CCFs. They have been reported to occur in 0.2% of patients with craniocerebral trauma and in up to 4% of patients who sustain a basilar skull fracture[7,8] . Less frequently, spontaneous dCCFs occur because of rupture of a cavernous carotid aneurysm or rupture of a weakened carotid wall in patients with connective tissue diseases. The clinical presentation is a triade of chemosis, proptosis, and exophthalmos. Other findings include headache, diplopia, ophthalmoplegia, and decreased visual acuity[3]. Once routinely treated with open surgical procedures, including carotid ligation or trapping and cavernous sinus exploration[6]. Endovascular management is the current treatment for CCFs, but recurrence may happen. Trans arterial or trans venous route are used for the endovascular treatment [1,3]. In Albania endovascular treatment started up in 1982, when a CCF was treated with Fogarty closure.

Case report

A 75-year-old male presented one month after a traumatic brain injury from a syncope due to high blood pressure, with right otorrhagia, right exophthalmos and proptosis, immediately after trauma. At the time, he was transferred to University Hospital of Trauma and was discharge after one week with diagnosis: SAH and Right frontal contusion.

He was transferred at our hospital, with bilateral exophthalmos, proptosis and eyelid swelling more expressed on the right side (fig 1) bilateral ophthalmoplegias, amaurosis OD, right afferent pupillary reflex and right corneal reflex was absent, left hyporeactive corneal reflex, left pupilar reflex was present, in the auscultation bilateral temporal systolic noise and supraorbital pulsation were evident.

On ophthalmologic evaluation bilateral papilledema with high intraocular pressure (16 mmHg on left eye,18 on right eye) was noticed. Amaurosis OD and significant reduction of visual acuity OS (can detect only light).

A cerebral Digital Substraction Angiography (DSA) was performed.

Direct right CCF, Type A according to Barrow was confirmed (fig 2). Immediate endovascular treatment was performed with direct detachable balloon occlusion of the right CCF under general anesthesia through arterial femoral access. Through balloon protection of cranial ICA, another proper size balloon was blown inside the fistula. At the end of the procedure control confirmed the complete closure of the fistula (fig 2).

His clinical condition immediately improved with significant reduction of exophthalmos and proptosis, and the bitemporal systolic noise with conic and plane stethoscope auscultation disappeared. Three days after discharge, the condition worsened with progression of exophthalmos and proptosis bilaterally, the systolic noise and supraorbital pulsation on the both sides reappeared in auscultation.

Another DSA was performed which showed re-opening of the right dCCF, with direct communication between the cavernous portion of the ICA and the cavernous sinus. (fig.3). Balloon in the cavernous sinus placed in the previous procedure was noted, but it seemed to allow the passage of the contrast from the ICA to the sinus. In these conditions, embolization of the cavernous sinus with coiling under the protection ICA with a balloon was decided. After placing the coils in the cavernous sinus, closure of the fistula was confirmed (fig.3). After the procedure, he was without systolic noise and supraorbital pulsation on auscultation bilaterally. After 24 hours, exophthalmos and proptosis were reduced and after 14 days visual acuity, ophthalmoplegia on the left eye improved. (fig 1).

Figure 1: Exophthalmos and proptosis on admission; Exophthalmos and proptosis 14 days after the second endovascular treatment

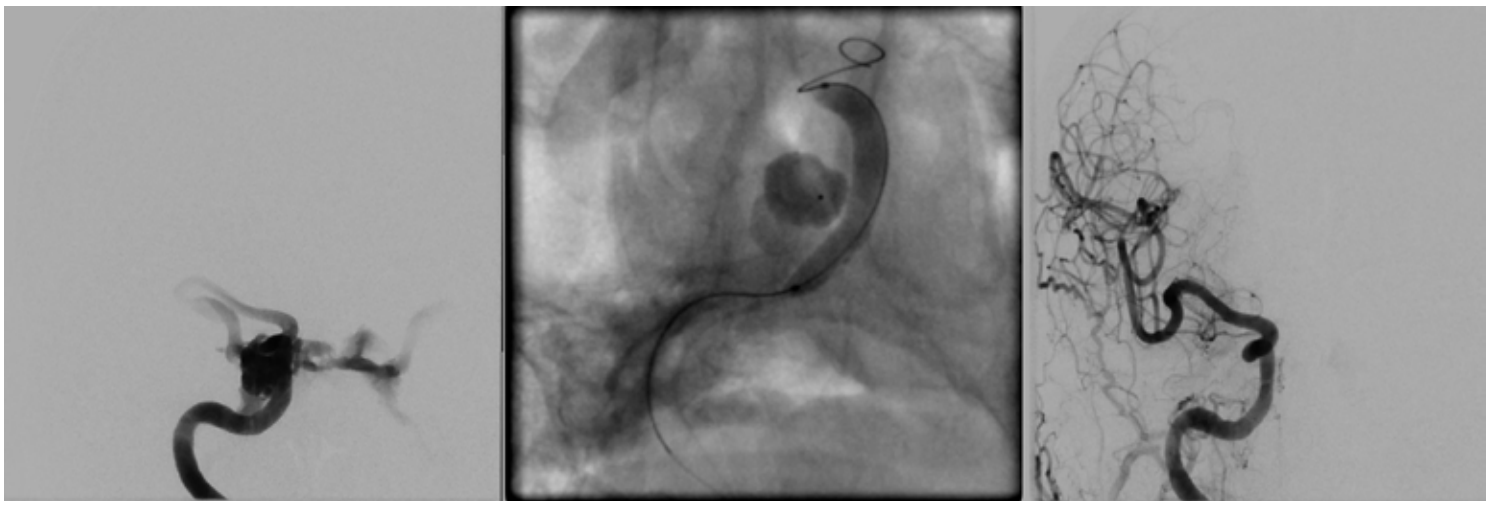

Figure 2: Direct right Carotido-Cavernous fistule Type A according to Barrow; Procedure: ballon in the right cavernous sinus; First DSA, Closure of the direct communication between right cavernoues sinus and ICA

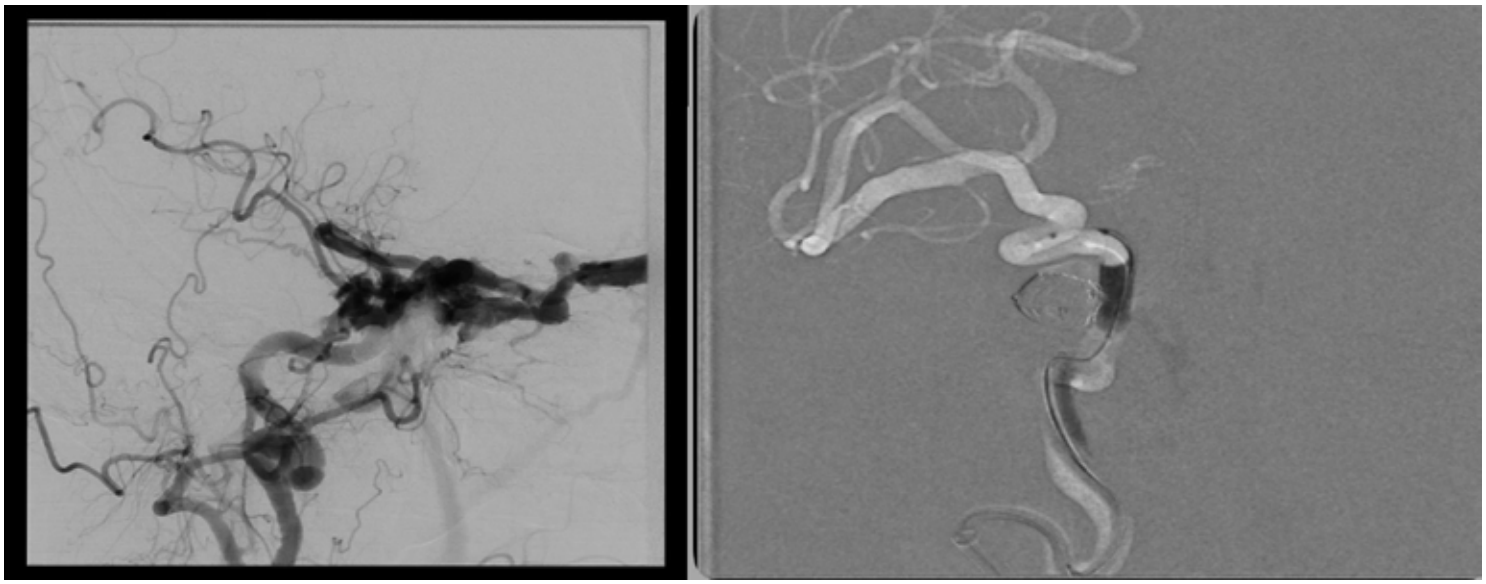

Figure 3: Second DSA the re-opening of the right dCCF, with direct communication between the cavernous portion of the ICA and the cavernous sinus; Second DSA and treatment, Embolization of the cavernous sinus with coiling blue arrow, under the protected ICA

Discussion

Traumatic dCCF is a direct connection between the intracavernous carotid artery and the surrounding cavernous sinus and it's a possibility consequence of any cranial trauma. Rupture of ICA aneurysms, arterial dissection, Ehlers-Danlos syndrome, fibromuscular dysplasia, pseudoxanthoma elasticum, or iatrogenic trauma from surgery[4] are second causes. dCCF are usually unilateral but can occur bilaterally in 2% of patients, as in our patient. The majority of dCCFs are high-flow lesions, with acute onset of venous congestive symptoms, blindness, conjunctival hyperemia and edema, pulsatile exophthalmos, and progressive cranial nerve symptoms and without any likelihood of resolving spontaneously[3,4]. DSA is the gold standard in the diagnosis of CCF and must be performed before any potential intervention[3]. Cure rates after endovascular repair of CCFs is approximate 80%[1], recurrences are reported. Transvenous embolization (TVE) and/or transarterial embolization using detachable coils to preserve the ICA has become the mainstay of treatment for dCCF; and a combination of balloon, stent, and liquid embolization materials has also been reported [4,5]. Symptoms of increased ocular pressure typically resolve within hours to days. Cranial nerve palsies may recover over several weeks and is dependent on the severity and duration of hemodynamic disruption[9].

Conclusion: dCCFs are a rare but treatable cause of cranial trauma. Endovascular embolization of CCFs with coiling or liquid agents is the only treatment.[2]

Authors contribution:

Conception and design: Mentor Petrela. Writing original manuscript: Vojsava Leka, Aida Agastra, Mentor Petrela. Formal analysis: Vojsava Leka, Oneda Cibuku, Mentor Petrela. Methodology: Eugen Enesi, Arben Rroji, Mentor Petrela. Review and editing: Mentor Petrela, Vojsava Leka, Mirel Grada, Aida Agastra, Stela Dodaj. Data curation: Vojsava Leka, Oneda Cibuku, Mirel Grada. Supervision: Mentor Petrela

Data Availability Statement

Data of this case report will be made available from the corresponding author upon reasonable request. Video of the endovascular treatment, Dr Eugen Enesi (eugenenesi@gmail.com)

Disclosures

The authors declare no conflict of interest.

Informed consent

Informed consent was obtained from the patient for this publication.

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Abbreviations used in this paper:

CA = carotid artery ; CCF = caroticocavernous fistula ; ICA = internal CA .

DSA = Digital Substraction Angiography; . TVE = Transvenous embolization; OD= Oculi dexter

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