TRAUMATIC CAROTID-CAVERNOUS FISTULA: CLINICAL PRESENTATION AND OUTCOME

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ABSTRACT

Objective: To evaluate the presentation, clinical course and outcome of traumatic carotid-cavernous fistula (CCF) with endovascular treatment at our institution during the last five years. **Design:** Retrospective descriptive study. **Setting:** The study included patients seen at Aga Khan University Hospital from January 2000 to December 2005. **Methods:** Retrospective analysis based on data retrieval from medical records using ICD coding system. Only those cases who had traumatic CCF and underwent endovascular treatment were included in the study. **Results:** A total of 8 patients were diagnosed with post-traumatic CCF and 11 procedures were done. Mean age at presentation was 35.6 years; mean duration of symptoms was 23 weeks after trauma; 6 patients were male and 2 female. Patients presented from 1 week to 2 years after the trauma; 7 had high-flow fistulas and 1 had low-flow fistula. Proptosis was the most common symptom (6 patients); decreased vision was present in 3 patients; 4 patients had an accompanying skull base fracture; and 1 patient had bilateral CCF. All patients were treated by endovascular procedures; 2 patients had recurrences, seen within 1 month of initial treatment and subsequently successfully treated. In 1 patient, the procedure failed due to the small size of the fistula. **Conclusion:** High success rate with minimal complications as seen in our series supports endovascular treatment as the leading option for CCF management.

Carotid-cavernous fistula is an abnormal communication between the carotid artery and the cavernous sinus. Classification of this condition is problematic. The angiographic classification depends on the velocity of blood flow across the shunt and the anatomical origin of the arteries supplying the cavernous sinus fistula. Barrow described the classification of CCF according to which Type A fistulas are high flow, usually traumatic in origin.[1] Post-traumatic fistulas are a rare complication of moderate to severe head injury. The incidence and pathophysiology of these fistulas has not been clearly ascertained. Direct injury in the base of the skull, torsion or stretching of the carotid siphon during impact, and impingement of vessels upon nearby bony prominences are a few possible explanations for the abnormal communication.

Modern neuroradiological techniques have made the therapeutic goals of obliteration of the fistula with preservation of internal carotid artery (ICA) patency possible in most cases. With improvement in radiological techniques and sophisticated angiographic equipment, especially Guglielmi detachable coils (GDC) and intra-arterial detachable balloons by Serbinenko and Deburn, endovascular embolization has replaced surgery as the first line of treatment.[2-5]

Surgical treatment, although feasible and successful, is often not possible because of the involved region's anatomical complexity. Moreover, it is extensive, invasive and carries considerable risks even in the best hands.[6] Considering the rarity of this entity, the difficulties in conventional surgery and the improvement in treatment techniques, we thought it is useful to present our experience regarding traumatic CCF.[7-9]

METHODS

All patients included in our series were admitted with a history of trauma and presented with suspected features of raised ocular pressure, possible cranial nerve dysfunction or unexplained stroke-like features irrespective of mechanism and duration of symptoms. History, neurological examination and essential hematology workup were

recorded. Radiological investigations (CT, MRI and digital subtraction angiography) were reviewed. Patients were diagnosed on the basis of clinical suspicion, radiological evidence (CT/MRI or both) and positive angiographic findings. All proven cases were reviewed with an interventional neuroradiologist.

Endovascular procedures were done under standard general anesthesia with monitoring. Pre- and post-procedure neurological examinations were documented. Vascular access was gained from the femoral artery. Standard diagnostic catheter (5-10 Fr) was used for initial angiographic evaluation, and the catheter advanced and positioned in the ICA. A vascular sheath was inserted and a small micro-catheter passed. Microcoils, liquid particulate material or balloons were used to obliterate the fistula according to individualized case circumstances. Additional venous access through internal jugular vein was achieved in cases where it was necessary. Throughout the procedure heparin infusion was maintained to keep the aPTT prolonged by 1.5 to 2-fold. Pressure was applied on the femoral puncture site for 4 hours after finishing the procedure for the prevention of hematoma. Serial neurological examinations were done in follow-up.

RESULTS

There were a total of 8 patients, with 6 males and 2 females. Mean age was 35.6 years and the mean duration of onset of symptoms was 23 weeks after trauma. Road traffic accident was the most common mechanism of trauma, present in 5 patients; 2 patients suffered form gunshot wounds while 1 patient fell from a height of 10-15 feet.

Proptosis was the leading clinical manifestation (seen in 6), followed by decreased vision in 4 patients and headache in 2 patients. Bruit was audible in all patients. Seven patients had unilateral symptoms while 1 had bilateral symptoms. In 4 patients there was an accompanying base of skull fracture.

Complications included post-angiographic anaphylactic shock (1 patient) which was successfully treated. In 1 patient the procedure failed due to the small size of the fistula. Recurrence of the fistula was seen in 2 patients after the first treatment.

DISCUSSION

Clinical presentation of CCF is dependent on size, duration, location, venous drainage patterns, and collateral vascular anatomy. Neoplastic, infective and other etiological conditions may lead to a similar clinical presentation. A vigilant approach is mandatory to exclude other causes of CCF while dealing with the cavernous sinus syndrome, orbital apex syndrome, superior orbital fissure syndrome or orbital symptoms. Radiological investigations such as MRI and CT (head and orbit) help exclude these possibilities.

Hallbach et al have identified high-risk features in 155 cases of CCF including 127 direct fistulas; they list important risk factors responsible for acute deterioration including increased intraocular pressure (8.7%), decreased visual acuity (32.3%), rapidly progressive proptosis (1.6%) and cerebral ischemia and hemorrhages (11%).10 Blindness was seen in 3.1% of their cases and 3.9% of their patients had a fatal outcome. Based on this study they concluded that the risks are more in direct fistula cases (which are mainly traumatic in origin) and recommended urgent therapy for the cases manifesting these high-risk features.[10]

CT and MRI of the brain have a role in the identification of associated traumatic injuries such as skull fractures, intracranial hemorrhage, contusions and pneumocranium. Base of skull fractures have been linked strongly with the development of CCF in trauma patients. In our series 50% of patients had evidence of a skull base fracture suggesting it as an important associated feature, supporting observations made by others.

Trauma in the form of gunshots, traffic injury, blasts and disaster is becoming an important burden of emergency medical units around the globe, with head injury as the leading cause of morbidity and mortality affecting the segment of population in their most productive years of life.[11,12] The calculated annual rate of head injury patients in Pakistan is 81 per 100,000 with a mortality rate of 15 percent.[11,12] About 0.2 % of all head injury patients may end up with CCF.[14] Our cases also comprised predominantly young males, with road accidents and gun shots being the leading cause of injury.

Cerebral angiography is the 'gold standard' for the diagnosis of CCF. Chen and his colleagues from Taiwan retrospectively reviewed 53 cases in which CCF proven on digital subtraction angiography (DSA) was compared with CT angiography (CTA) and MR angiography (MRA). They found a valuable diagnostic role for CTA and MRA, with CTA comparable to DSA in selective cases as a diagnostic tool for CCF.[13] Another study from China has mentioned the useful role of colored Doppler ultrasonography in 33 cases studied.[14]

As in most intracranial pathologies, thorough clinical examination cannot be replaced for the diagnosis and follow-up of these cases. Detection of bruit and elevated

ocular pulse amplitude, especially asymmetrical elevation in the affected side, are the features highly in favor of CCF. In our series, all the patients were suspected to have CCF on the basis of history and neurological examination; 3 cases were referred with an established diagnosis. All patients underwent CT brain and diagnostic DSA.

Historically, angiography is in use for the treatment of intracranial vascular and other multiple pathologies since at least the 1960s.[15,16] High-resolution DSA coupled with advancement in catheter technology and 'road-mapping' capability has made endovascular treatment of CCF the method of choice. Endovascular CCF treatment history has evolved tremendously since 1950s. Over time, there has been continuous innovation in angiographic equipments and catheter technologies. Introduction of silicon and latex as ballooning tools and availability of GDC (Guglielmi Detachable Coils) have expedited the ongoing use and interest of endovascular technology in neurovascular pathologies.[4] The field is ever growing. What we learn and apply today may become obsolete tomorrow due to rapid advancements in this area.

Six (75%) patients in our series were male and the mean age was 35.6 years, while Moron at el showed the same male predominance in their series with 60% males and 42 years as the mean age.[17] Most of our patients were in the second and third decades of life, which corresponds with the literature.[18] An interesting and perhaps important finding we noted is the late presentation in our cases which ranged from 7 days to as long as 2 years with the mean duration of 20 weeks. In 1988, Batjer reported that all cases in his series developed signs and symptoms of CCF within 8 weeks.[19] However, this discrepancy can be due to lack of ideal neurosurgical care and non-referral in our health care setup.

CONCLUSION

CCF is a rare but important complication associated with trauma. It may lead to acute visual decline, neurological deficits and hemorrhage, which can be fatal. Early recognition and appropriate treatment is essential to prevent morbidity and mortality. Treatment options have evolved remarkably in the last few decades and include endovascular and surgical approaches. Endovascular treatment with detachable silicone balloons or transvenous coil embolization are safe and effective therapy. Surgery carries a definite high risk even in the best hands. Optimal management of CCF requires multi-disciplinary collaboration between neurologists, neurosurgeons, neuro-ophthalmologists and interventional neuroradiologists in order to ensure excellent outcomes.

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