



Mycotic Aneurysm of External Carotid Artery following Traumatic Brain Injury: Case Report and Review of Literature

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ABSTRACT

Mycotic aneurysm of external carotid artery is extremely rare. We herein report a case of external carotid artery (ECA) aneurysm following severe traumatic brain injury. A 24-year-old man with severe traumatic brain injury (TBI) following a car accident was referred to Rajaei Trauma Center Emergency Room affiliated to Shiraz University of Medical Sciences in Shiraz, Iran. He underwent ventriculostomy on arrival for intracerebral pressure (ICP) monitoring and for a second time due to hydrocephalus following decompressive craniectomy. He developed fulminant meningitis and ventriculitis during his hospital course. A bulged pulsatile lesion under the frontotemporal scalp resulted into the suspicion to underlying vascular pathology. Six-vessel angiography of brain was done which revealed mycotic aneurysm of external carotid artery. The patient underwent a two-week course of a combination of intravenous antibiotics. Follow-up angiography was performed which confirmed successful treatment of mycotic aneurysm of ECA. Mycotic aneurysm of ECA is extremely rare. To our knowledge, this is the first report of mycotic aneurysm of ECA following severe TBI which was successfully treated with antimicrobial therapy.

Keywords: Mycotic aneurysm; Traumatic brain injury; External carotid artery.

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Introduction

Mycotic aneurysm of external carotid artery is extremely rare [1,2]. Most of previously reported cases had presented following acute bacterial endocarditis and they tended to happen in immunocompromised individuals [2,3]. We herein report a case of external carotid artery aneurysm following severe traumatic brain injury, which was successfully, treated with non-invasive measures i.e. intravenous antibiotics alone.

Case Report

A 24-year-old, previously healthy man was brought to Rajaei Trauma Center Emergency Room affiliated to Shiraz University of Medical Sciences in Shiraz, Iran by EMS an hour after a car-car accident. On arrival, his Glasgow coma scale (GCS) was 4 and his pupils were midsized but fixed and non-reactive to light. His vital signs were as follows: PR: 134/min, BP: 88/46 mmHg. FAST ultrasonography revealed moderate

to severe free fluid with suspicion to splenic rupture in the abdominal cavity. So he was emergently transferred to operation room for laparotomy. As the patient's general condition did not allow us to perform a brain computerized tomography (CT) scan prior to operation, we decided to investigate his possible brain injury (during laparotomy by general surgeons) through pneumocephalography. So a ventriculostomy tube was inserted in right Kocher point and after draining cerebrospinal fluid (CSF), air was injected and the x-ray was taken which revealed no significant shift of midline. After a few hours of post-op intensive care unit (ICU) and fluid resuscitation the patient was stabilized and he underwent a brain CT scan which revealed multiple brain contusions and a contusional intracerebral hemorrhage (ICH) in right (Rt.) parietooccipital area. However ambient cistern was open and there was no shift of midline at the level of foramen of Monro. Pneumoventricle within left (Lt.) lateral ventricle was also noted.

On the fourth day of hospital admission, his intracerebral pressure (ICP) monitoring revealed a high ICP which was unresponsive to sedation and drainage. So an emergency brain CT scan was done which showed a tight brain and slit lateral ventricles. The patient was emergently transferred to operation room (OR) and right frontotemporoparietal decompressive craniotomy was done for him. Post-op images revealed hydrocephalus, so left anterior ventriculostomy tube was placed in order to control hydrocephalus and monitor ICP.

During 3 days of ICP monitoring, although high ICP was recorded but it responded to CSF drainage. Unfortunately on the 4th day of ICP monitoring, the patient became febrile and CSF analysis became active and we noted periventricular edema in his brain CT scan. So triple antibiotic therapy with meropenem, vancomycin and amikacin was started for him but the patient did not show any clinical response and the high grade fever did not disappear. So ventriculostomy tube was disconnected and we decided to perform daily lumbar puncture with tapping the CSF to dry followed by injection of intrathecal antibiotics

(cholistine, vancomycin and amikacin). Although the patient was treated with high dose of intravenous and intrathecal antibiotics, and the daily CSF analysis was going to be cleared, he did not become afebrile. Therefore, we started a full investigation to identify the source of his fever. As his blood culture with BACTEC™ tube became positive for *Staphylococcus aureus*, we requested an echocardiography to rule out bacterial endocarditis. However, no vegetation or any other sign of bacterial endocarditis was noted within two separate echocardiographic evaluations by two different cardiologists. During his 31st day of admission, we noted a 2x2 pulsatile mass under his left frontotemporal scalp. So we requested a brain CT-angiography with suspicion to a vascular pathology. A vascular lesion was noted in the above mentioned area which led us to perform a 6-vessel digital subtraction angiography of brain which highlighted the vascular lesion to be a mycotic aneurysm of external carotid artery (ECA) (Figure 1). So a new course of intravenous (IV) antibiotic therapy with cholistine, vancomycin, meropenem, amikacin and amphotericin was started for him. After two weeks of therapy, the pulsatile mass disappeared and the patient became totally afebrile. Another 6-vessel DSA of brain was done which confirmed successful treatment of ECA mycotic aneurysm with this antibiotic regimen (Figure 2).

Discussion

The mycotic aneurysms of the carotid arteries are rare complications of traumatic injuries or systemic infections. These are false aneurysms caused by different species of fungi, whether intravascular or systemic [1]. ECAs are rare especially with introduction of broad spectrum antibiotics and anti-fungal agents. The incidence has been steady, approximately 20 cases reported per decade over the past 30 years. Previously, Knouse and co-workers [2] analyzed 99 cases of ECA mycotic aneurysm which was reported until 2002 in the literature [3].

There are three pathophysiologies for mycotic aneurysms of carotid arteries. The most important one

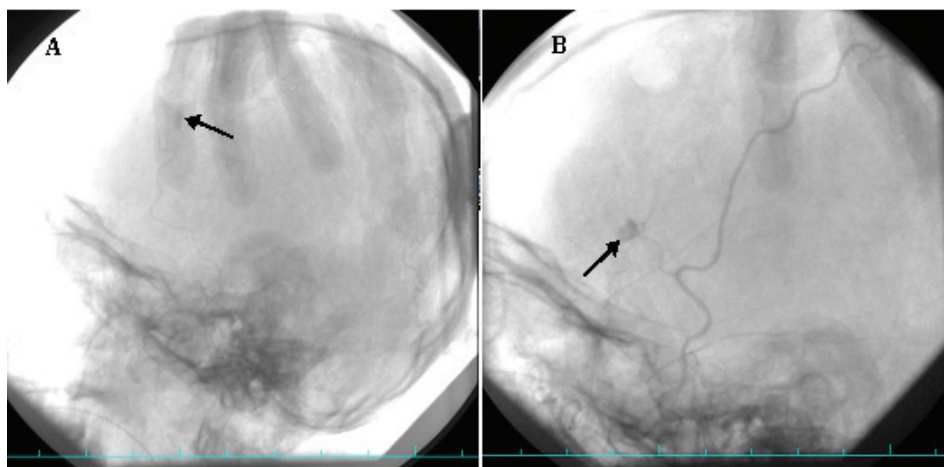


Fig. 1. Sagittal (A) and right oblique (B) view in conventional angiography of the patient with post-traumatic mycotic aneurysm of right external carotid artery (flash).

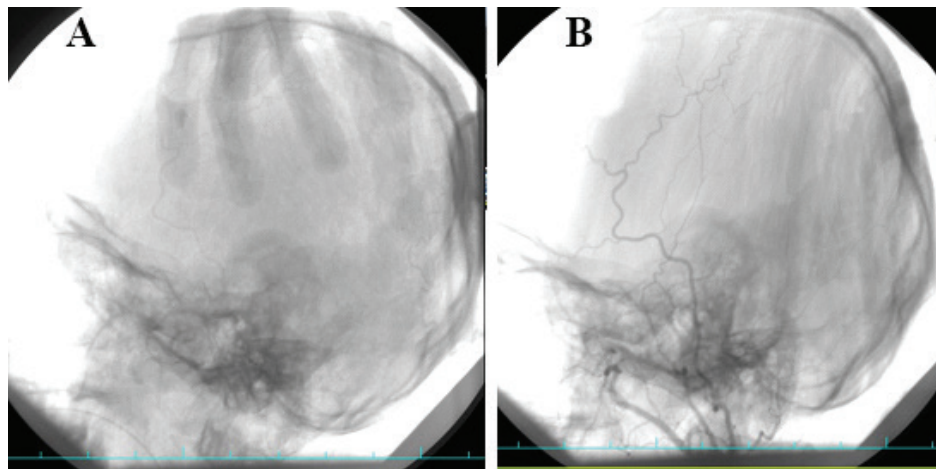


Fig. 2. Sagittal (A) and right oblique (B) view in conventional angiography of the patient after conservative management. The mycotic aneurysm has resolve completely.

is the septic emboli from the heart and the endocardium which tends to lodge at the distal branches of the carotid arteries. This infects the endothelial and weakens the arterial wall leading to false aneurysm formation [4]. Another pathomechanism of mycotic aneurysm formation in carotid arteries is chronic inflammation and infection of the cranial structures leading to infection and inflammation of the surrounding tissues such as periarterial lymphatics and the vasa vasorum. Cervical lymphadenitis is the most common cause of carotid artery aneurysms in children [5]. The last pathophysiology could be the iatrogenic vascular manipulations or vascular traumas leading to endothelial infection and inflammation. Previous reports have indicated that endovascular procedures could be associated with external carotid mycotic aneurysms [6] which may also occur in immunocompromised patients [7]. In the present study, we reported a patient who developed ECA mycotic aneurysm secondary to septic meningitis following traumatic brain injury. The possible mechanism in our reported case could be the infection of the surrounding structures (septic meningitis) along with the direct vascular trauma to external carotid artery which made it susceptible for metastatic infection. The spread of the infection from the septic meningitis as well as vascular metastasis through venous return lead to infection of traumatized external carotid artery resulting in mycotic aneurysm.

Syphilis, tuberculosis, and untreated endocarditis were among the most common causes of carotid artery mycotic aneurysms before introduction of the antibiotics [8]. But currently, the most common cause of these aneurysms (based on 76 cases reported in the English literature) are *Staphylococcus aureus* (28 cases), *Salmonella* (13 cases), *Streptococci* (13 cases), *Escherichia coli* (6 cases), *Klebsiella* (5 cases), *Aspergillus* (3 cases), *Mycobacterium tuberculosis* (2 cases), and *Proteus*, *Yersinia*, *Pseudomonas*, *Enterococcus*, *Bacteroides*, and *Mycobacterium bovis* (1 case each). In 23 cases from the literature, the bacterial pathogen was not specified. In our case,

Staphylococcus aureus was detected which is in consistent with previous reports. In addition, it can be concluded from the literature that those developing carotid artery mycotic aneurysms following trauma and vascular manipulation were more susceptible to be infected with *Staphylococcus aureus*.

The untreated mycotic aneurysm of carotid arteries is associated with high mortality and morbidity. There are several severe complications associated with this aneurysm such as rapid enlargement resulting in rupture, hemorrhage, and septic embolization leading to neurologic sequelae and arterial occlusion [9]. Previously, non-operative treatments were associated with a mortality rate of 70-90% [2]. But currently with implication of surgical techniques, the mortality rate has been decreased to 10% [4]. The available surgical techniques include ligation, open arterial reconstruction, and endovascular techniques [1]. Currently, surgical treatment of a mycotic aneurysm is always associated with long-term antibiotic therapy, which begins in preoperative period and is based on the results of sensitivity studies. Postoperatively, antibiotic therapy is generally recommended for at least 6 weeks [4] with some authors recommending it for 6 months [10]. However, we did not apply the surgical procedure and the patient was treated successfully with intravenous antibiotics. As the pathogen was found to be *Staphylococcus aureus*, a broad spectrum regimen was selected including cholistine, vancomycin, meropenem, amikacin and amphotericin. The patient responded well after 2 weeks of therapy and did not experience any neurologic sequelae.

In conclusion, mycotic aneurysm of ECA is a rare complication of traumatic brain injury and septic meningitis which could be successfully treated with broad spectrum intravenous antibiotic. To the best of our knowledge, this is the first report of mycotic aneurysm of ECA following severe TBI which was successfully treated with antimicrobial therapy.

Conflict of Interest: None declared.

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