Case Report

Traumatic Carotid Artery Pseudoaneurysm Mimicking A Peritonsillar Abscess

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Abstract

Carotid artery pseudoaneurysms can occur in many different etiologies including blunt or penetrating trauma and iatrogenic causes which happen during vascular procedures. Although there are no external signs of neck trauma in 50% of cases, neck hematoma, bruits, pulsatile neck mass, or a palpable thrill may be found on physical examination. Patients with pseudoaneurysm of the internal carotid artery are usually present with neurologic complaints which could be occurred for hours to weeks following the initial injury. Here, we report a case with a traumatic pseudoaneurysm of the internal carotid artery after blunt trauma which was initially misdiagnosed as a peritonsillar abscess.

Keywords: Pseudoaneurysm, blunt trauma, internal carotid artery, stroke

INTRODUCTION

Traumatic injury to the carotid artery is a rare in the recent literature. Following blunt trauma, serious complications including dissection, complete disruption, thrombosis and pseudoaneurysm formation can develop. Pseudoaneurysm formation which occurs in nearly one-third of blunt carotid injuries (BCI) should be included in differential diagnosis of masses located within the carotid space.

Here, we present a case with cerebral infarct due to the left carotid pseudoaneurysm after blunt trauma to the carotid artery was initially misdiagnosed as a peritonsillar abscess.

CASE PRESENTATION

A 63-year-old woman with a history of arterial hypertension and hyperlipidemia presented to the our emergency department with a sudden onset of speech disturbance and weakness of her right arm. On admission she had no other complaints.
When she came the emergency department, she only had been taking antibiotics since she had been diagnosed as peritonsillar abscess (PTA) in a different medical center.

On physical examination, the patient was noted to have temperature of 36.8 ºC, pulse of 72 beats/min and blood pressure of 140/80 mmHg. Examination of the neck revealed pulsatile mass in the left submandibular area but no obvious lymphadenopathy of the posterior or anterior cervical chain. The patient's heart, lung and abdominal examinations were all normal. On her neurologic examinations, sensorimotor dysphasia, right central facial paralysis, decreased deep tendon reflexes and Babinski sign on right side and right hemiparesis were found. Cerebellar tests were normal on left side but couldn't evaluated on the opposite side because of paresis. Sensorial examinations could not be performed due to sensorimotor dysphasia.

Serum electrolytes, glucose, blood urea nitrogen and creatinine were all within normal range. There was no abnormality in her electrocardiography and brain CT examination. She was referred to our neurology department for treatment, rehabilitation and investigation of stroke ethiology.

In her brain magnetic resonance imaging (MRI), revealed acute infarction at left frontoparietal area (Figure 1). Carotid Doppler ultrasonography, shows occlusion and dilatation suggestive of thrombosed aneurysm in left internal carotid artery. Left internal carotid artery occlusion and a lesion which is compatible with pseudoaneurysm in left carotid bulb were reported in neck MR angiography (Figure 2). Anticoagulation therapy was started. After questioned by doctors again, she said that approximately 3-4 weeks ago, she had fallen down the stairs. After two weeks she had admitted to otorhinolaryngologist because of puffiness in her neck. She had treated with antibiotics because of a presumptive diagnosis of peritonsillar abscess by the otorhinolaryngologist. Although antibiotic treatment was going on, there was no change at puffiness in her neck. While these examinations were done, she started to the rehabilitation programme and an improvement on her paresis was seen. On her digital subtraction angiography, left internal carotid artery was found to be occluded 1 cm after the carotid bulb, and anterior to this level, a slow filling pseudoaneurysm, which was 1.5 x 1.5 cm in diameter was present (Figure 3). Intracranial carotid system was supplied by the right internal carotid artery, via the communicating arteries.

Figure 1: Diffusion weighted MR images show acute infarction on left frontal and parietal lobes.
DISCUSSION
Most common causes of extracranial carotid artery aneurysms are trauma and spontaneous dissection. Arteriosclerosis, fibromuscular dysplasia, Ehler-Danlos syndrome, deep neck space infections, surgical injury and radiotherapy are other less frequent causes.

The mechanism of BCI is usually direct trauma resulting from motor vehicle accidents, assault and falls. Cervical hyperextension/rotation, intraoral trauma and basilar skull fracture are other recognized mechanisms for BCI. The diagnosis of BCI was more common in patients with an essentially normal neurologic examination (49%) than in individuals with a Glasgow Coma Scale of <8 (37%). Neurological morbidity and mortality rates of 40-80% and 5-40% respectively in all patients presenting with blunt carotid artery injury.
Types of vascular injury found associated with BCI include intimal flap/dissection, carotid-cavernous fistula formation, complete transection, occlusion/thrombosis, pseudoaneurysm or a combination of these lesions\(^{(6)}\). Carotid artery injuries can lead the complications from hemorrhage, thrombosis and/or embolization. Cerebral ischaemia following BCI may result from one of the two mechanisms. First, damage to the arterial wall can cause internal carotid artery dissection resulting in haemodynamic disturbance. Second explanation could be that damage to the arterial intima exposes subendothelial collagen, a thrombogenic surface and potent platelet aggregator\(^{(1)}\). This can cause transient cerebral or retinal ischaemic attacks. Most cerebral infarctions are caused by this phenomenon, rather than by haemodynamic disturbance\(^{(5)}\).

Many individuals with blunt carotid artery injury are asymptomatic at presentation and the diagnosis is usually made after onset of neurological symptoms and signs. Clinical symptoms and signs following BCI are highly variable ranging from simple unilateral headache, cerebral ischaemia, Horner's syndrome, cranial nerve palsy, neck pain, syncope, scalp or ICA tenderness and swelling in the neck\(^{(16)}\). These symptoms may develop hours or weeks after the injury\(^{(11)}\). On physical examination, neck hematoma, bruits, pulsatile neck mass or a palpable thrill may be found. Less frequent presentations are including a mass mimicking a peritonsillar abscess or nasopharyngeal/pharyngeal mass\(^{(4,15)}\). However most of cases have no external signs of neck trauma. On physical examination our patient had only a pulsatile mass in her neck. She had forgotten that she had fallen down from the stairs because she didn't feel anything except for neck pain which was going on for a short time. Approximately two weeks later, she had admitted to the otorhinolaryngologist due to puffiness in her neck. Although she had no fever and leukocytosis, she had been diagnosed a peritonsillar abscess due to swelling of the peritonsillar area and sore throat. The otorhinolaryngologist had begun the antibiotherapy to her for the treatment of PTA. When she came to our emergency service with right hemiparesis, she had been taken antibiotherapy.

In a prospective study, the incidence of 1.03 % of BCI is identified by aggressive screening using four-vessel cerebral angiography and helical CT angiography in patients with blunt head and neck trauma\(^{(9)}\). Conventional carotid angiography demonstrates not only the extent of proximal and distal injury, associated vertebral injuries and intracranial thromboembolic material but also the anatomy of the intracranial circulation. Typical findings are irregular and often tapered stenoses which usually begins 2-3 cm distal to the bifurcation with a characteristic ‘string sign’. The pathognomic sign of a carotid double lumen is rare\(^{(11)}\). Other typical findings are aneurysmal dilatations or distal branch embolisations\(^{(16)}\). Occasionally, irregularity of the vessel wall may be the only findings. It has been shown that transcranial and extracranial Doppler ultrasonography have 86% sensitivity for the identification of carotid vessel injury\(^{(12)}\). Our patient's pseudoaneurysm was first identified using Doppler ultrasonography. We did conventional angiography to confirm the presence of pseudoaneurysm which is typically considered to be the gold standart in diagnosis\(^{(11)}\).

Available treatment modalities of posttraumatic carotid artery pseudoaneurysm are supportive management, anticoagulation and surgery include ligation, vascular bypass and embolization. Treatment must be considered on an individual patient basis. Anticoagulation seems to be the treatment of choice in most cases and surgical intervention is rarely indicated\(^{(11)}\). During
anticoagulation, our patient's paresis improved completely but she had only mild sensorimotor dysphasia.

Early diagnosis and treatment is important for prognosis. Complete or almost complete recovery occurs in 23-85%, but severe persistent deficits occur in 16-37% of patients. The prognosis is variable in different studies due to selection of patient and various methods of treatment.

Patients who experience BCI, usually develop focal neurologic deficits prior to diagnosis. Clinical suspicion is essential in any patient presenting with neck or throat complaints following trauma.

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