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Case report

Traumatic Thrombosis of the Intra-Petrous and Cavernous Segment of the Internal Carotid Revealed by Otological Symptoms: A Case Report

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Abstract

Introduction: Thrombosis of the internal carotid following a non-penetrating blunt trauma is rare and unusual, implicating mostly intimal and medial vessel wall damage. With the lack of proper clinical presentation, it's often diagnosed by arteriography while searching for expansive cerebral hemorrhagic complications. ***Case report:*** Hence, the report of this rare case of a post blunt trauma thrombosis of the intra-petrous and cavernous segment of the internal carotid in a 26-years-old male patient was revealed 1 year later by otological symptoms. CT scan, MRI, and angiography confirmed the diagnosis. ***Conclusion:*** Management of this serious injury is controversial, to say the least among authors involving both medical and surgical treatment.

Keywords: ***Traumatic thrombosis, Tinnitus, Conductive hearing loss.***

Introduction

Occlusion of the internal carotid; following a non-penetrating trauma; is considered an uncommon entity [1].

Carotid thrombosis at the skull base, especially in its petrous portion; accounts for less than 10% of cases, thus the diagnosis delay and which in most cases diagnosed after postmortem autopsy [2.3.4].

We report a rare and unusual presentation of thrombosis of the intra-petrous and cavernous segment of the internal carotid following a blunt trauma of the head in a 26-years-old patient, revealed 1 year later by spontaneous acute right ear hemorrhage, hypoacusis, and tinnitus. Radiologic imaging including CT scan, MRI, and angiography confirmed the diagnosis and the localization of the thrombosis.

Case Report

A 26-year-old patient was admitted into the ENT emergency room, for a spontaneous acute right ear otorrhagia, hypoacusis, and tinnitus (hissing sound), without any other signs including no facial palsy or any neurological deficit.

The patient had a history of blunt non-penetrating craniofacial trauma 1 year before the symptomatology starts. The initial CT scan, performed in the ER right after the trauma, showed a Non-displaced comminuted fracture of the right temporal bone, a fracture of the zygomatic process, and a fracture of the frontal bone extending to the outer wall of the right orbit. The patient then refused any additional treatment and left the facility.

The examination showed a complete stenotic ear canal with scabs of blood. The rest of the clinical examination was normal.

Tonal audiometry Showed a 50-dB conductive hearing loss (**Figure 1**). A new CT scan was performed and showed a large bony defect of the temporal bone with a poorly limited filling of the external auditory canal which extends to the middle ear, an eroded aspect of the tegmen tympani and antri, and lysis of the facial canal, jugular foramen and the carotid canal (**Figure 2**).

An MRI was performed and showed a lack of opacification of the right intrapetrous and cavernous segments of the right internal carotid artery, evoking the diagnosis of thrombosis (**Figure 3**). This diagnosis was eventually confirmed by angiography performed via a right femoral puncture with selective catheterization, the right hemisphere was vascularized by the left internal carotid, vertebral, and basilar arteries (**Figure 4**).

The patient was put under medical treatment using an Anticoagulant with close monitoring.

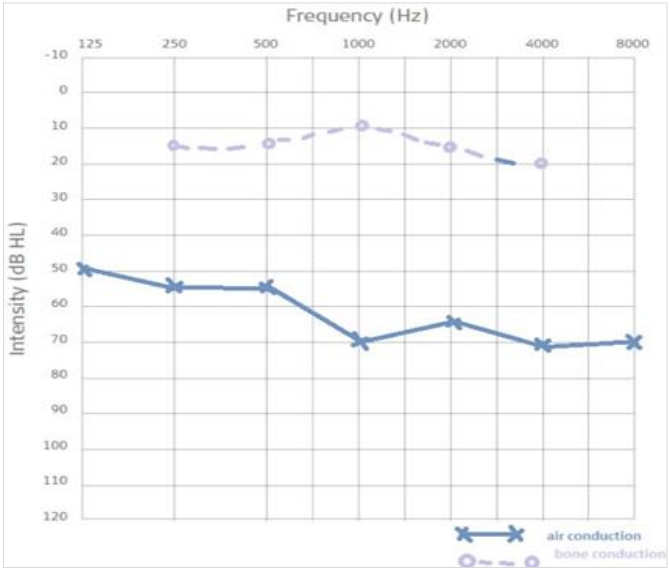

Figure 1: audiometric result revealing a 50-dB conductive hearing loss of the right ear.

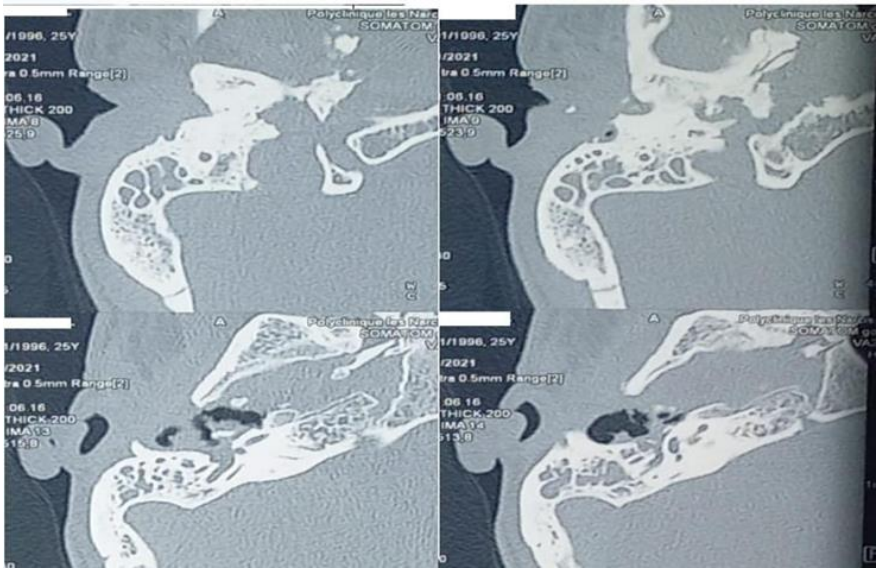

Figure 2: Axial Ct scan images showing a large bony defect of the temporal bone with lysis of the facial canal, jugular foramen, and the carotid canal.

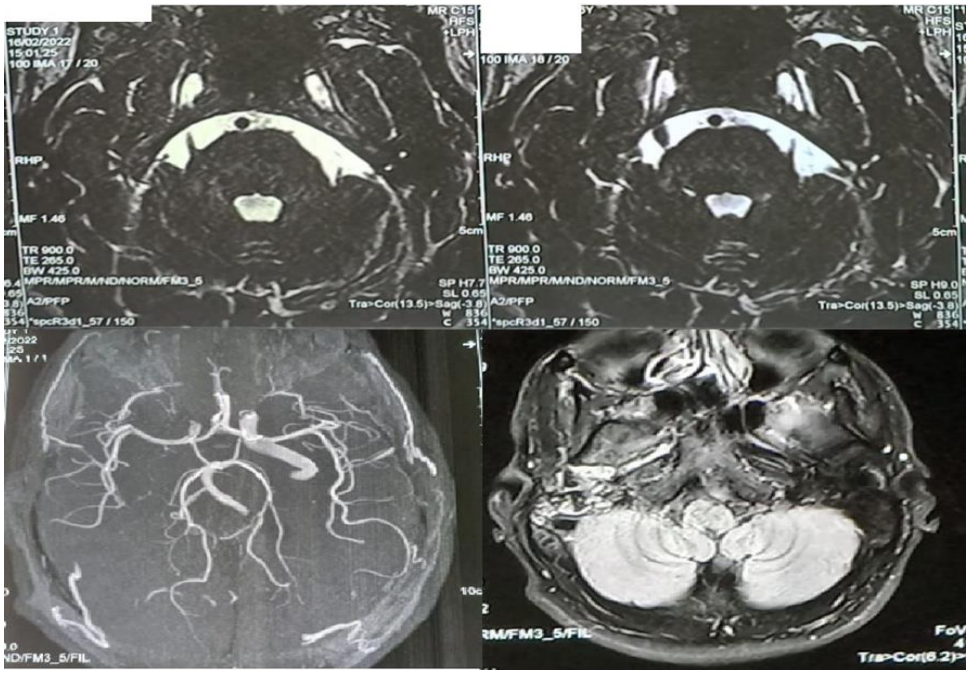

Figure 3: different MRI sequences revealing showing a lack of opacification of the right intrapetrous and cavernous segments of the right internal carotid artery

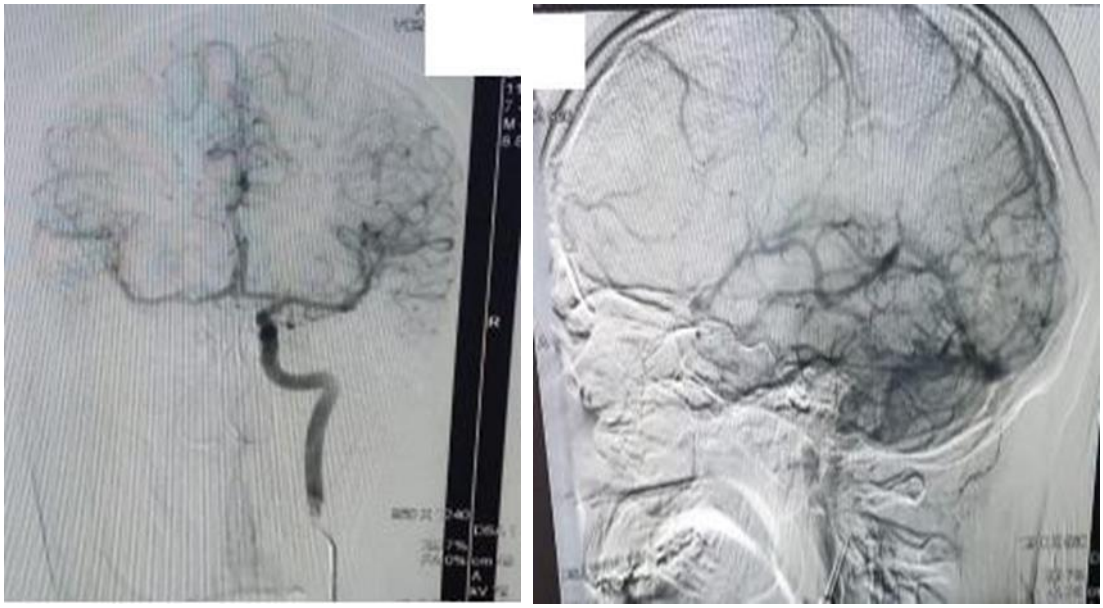

Figure 4: angiography images confirming the diagnosis of thrombosis of the internal carotid in its intrapetrous and cavernous segments.

Discussion

Thrombosis of the internal carotid following a penetrating wound is well documented, and it's an easily understood complication, unlike thrombosis resulting from non-penetrating blunt injury [1]. Closed traumatic carotid occlusion is quite rare and has different pathogenesis and mechanism. It mostly implicates an intimal and medial vessel's wall damage [1,2,5]:

- Directly by the presence of irregularities on the intima, following the trauma, responsible for clot formation
- Indirectly as a result of elongation and overstretching of the carotid or its crushing against the transverse processes of the vertebrae cervical.

Other authors reported the association between the intimal and medial wall damages and dissecting aneurysm, responsible for partial or total occlusion [6-8].

It is known that the obstruction of an internal carotid artery is generally well tolerated, and causes a-little-to-non cerebral ischemic damage [1]. Other causes of cerebral circulatory insufficiency, by the extension of the obstructive process to the cerebral vessels, or by insufficiency of anastomosis pathways of the polygon of Willis, or embolisms from the thrombosed area and spasm which expands to the cerebral arteries as a result of the irritation of the adventitia of the vessel thrombosis [1.2.5.9].

Traumatic carotid thrombosis does not have a proper clinical presentation [1-4], it is either demonstrated as extra-cerebral hemorrhage or typically detected while searching for expansive cerebral hemorrhagic complication by arteriography; as it is the only arsenal that provides the proper diagnosis and localization of the thrombosis and subsequently increases the number of post blunt traumatic thrombosis of the carotid artery reported in the literature [1,2].

There is a lucid interval of a few days [10,11] to a few weeks [12,13] between the trauma and the onset of clinical signs which equates to the necessary time for thrombus formation and eventually occlusion of the vessel [1,2]. In our cases, there was 1 year period of time between the trauma and the development of the symptoms which is extremely rare.

The treatment of traumatic thrombosis of the internal carotid is not uniform and is very much controversial some advocate the medical route; injecting procaine or nicotinic acid, anticoagulant medication. The surgical route is also variable; aiming at the restoration of the carotid circulation; which is exceptionally applicable due to the delay in both diagnosis and surgery, which is responsible for the extension of the thrombosis to cerebral vessels, or the carotid siphon [1.2.5.9.12.13].

The prognosis of traumatic thrombosis of the internal carotid artery is very poor; provoking either death or serious neurological defects [1.2.3.5.7.10.14].

Conclusion

Internal carotid thrombosis by blunt trauma to the skull, face, and neck is rare. The increasing application of carotid angiography in the evaluation of head and neck trauma made the diagnosis possible and well documented. Although, most often it affects young adults.

The prognosis is directly related to the collateral circulation that assures the blood supply. In the case of thrombosis, the insufficiency of blood circulation could lead to major neurological complications and even be fatal to the patient.

Conflict of interest

The authors declare no conflict of interest

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None

Consent

Verbal Informed consent was obtained from the patient

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