Case Report

Bony Protuberances on the Anterior and Posterior Clinoid Processes Lead to Traumatic Internal Carotid Artery Aneurysm Following Craniofacial Injury

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Traumatic intracranial aneurysms are rare, comprising 1% or less of all cerebral aneurysms. The majority of these aneurysms arise at the skull base or in the distal anterior and middle cerebral arteries or their branches following direct mural injury or acceleration-induced shearing force. We present a 50-year-old patient in whom subarachnoid hemorrhage (SAH) was developed as a result of traumatic aneurysm rupture after a closed craniofacial injury. Through careful evaluation of the three-dimensional computed tomography and conventional angiographies, the possible mechanism of the traumatic internal carotid artery trunk aneurysm is correlated with a hit injury by the bony protuberances on the anterior and posterior clinoid processes. This traumatic aneurysm was successfully obliterated with clipping and wrapping technique. The possibility of a traumatic intracranial aneurysm should be considered when patient with SAH demonstrates bony protuberances on the clinoid process as a traumatic aneurysm may result from mechanical injury by the sharp bony edges.

Key Words: Anterior clinoid process · Posterior clinoid process · Traumatic aneurysm · Craniofacial injury.

INTRODUCTION

Traumatic intracranial aneurysms occur rarely, constituting less than 1% of all aneurysms. The majority of these aneurysms arise at the skull base or in the distal anterior and middle cerebral arteries or their branches following direct mural injury or acceleration-induced shearing force. The mortality rate for patients with traumatic intracranial aneurysms can be as high as 50%.

We present a case of aneurysmal subarachnoid hemorrhage (SAH) immediately following closed craniofacial injury in which the development of traumatic intracranial aneurysm was likely due to mechanical pressure adjacent to the sharp tips of the anterior and posterior clinoid processes with subsequent brain shifting.

CASE REPORT

A previously healthy 50-year-old woman fell down while participating in recreational climbing and suffered closed craniofacial injury. Upon arrival to the emergency room, she was found to be drowsy and complained of a severe headache and dizziness. Multiple abrasions and swelling over the face and a fracture of the left maxilla were noted. Non-enhanced computed tomography (CT) scans of the brain revealed SAH predominantly locat-
ed in the right sylvian and basal cisterns and soft tissue swelling on the forehead and left periorbital area (Fig. 1). Given a high degree of suspicion for cerebral vascular injury, three-dimensional CT angiography was performed immediately demonstrating an aneurysm of the right internal carotid artery (ICA) trunk (Fig. 2A) in close proximity to the bony protuberances on the anterior and posterior clinoid processes and a vertical fracture line on the presphenoid portion (Fig. 2B). Based on the history of head trauma, clinical presentation, and radiological findings, a traumatic intracranial aneurysm was suspected. For further evaluation of possible traumatic ICA trunk aneurysm, four-vessel digital subtraction angiography (DSA) was performed three days after admission. It demonstrated an aneurysm size increase with diffuse M1 vasospasm (Fig. 3). The patient was taken to surgery, during which the sharp tips of the anterior and posterior clinoid processes were observed to be close to the non-branching site of the right supraclinoid ICA trunk. A complete vascular wall tearing occurred on the mid-portion of the right distal ICA trunk with pseudosac formation (Fig. 4A). In an effort to salvage the parent vessel lumen, the first curved aneurysm clip was applied along the long axis of the tear under the control of temporar-

Fig. 2. Preoperative three-dimensional computed tomographic angiogram (3-D CT). A: Anteroposterior view of the preoperative 3-D CT of the right internal carotid artery (ICA) obtained immediately following cranial fracture shows an aneurysm on the lateral side of the supraclinoid ICA. B: Superior view of the 3-D CT skull base reconstruction image shows sharp bony protuberances on the tops of the anterior and posterior clinoid processes (large and small arrowheads) and a vertical linear fracture line on the presphenoid bone (double arrow).

Fig. 3. Conventional angiogram obtained three days following the trauma shows a growing aneurysm, approximately 5 mm in diameter, arising from the non-branching site of the right internal carotid artery (white arrow) and a diffuse vasospasm on the right M1 portion.

ry clipping (Fig. 4B). The wrapping technique with a cottonoid and right-angled clip was added to secure the torn portion (Fig. 4C).

The pseudosac was found to be composed of the organized hematoma with fibrin admixed with white cell elements by histology (Fig. 5). No normal vessel wall elements were identified and resulting in a final diagnosis of a false aneurysm.

The patient did not demonstrate any postoperative neurological deficits and recovered uneventfully. A postoperative DSA showed complete obliteration of the traumatic aneurysm without compromise of the parent circulation (Fig. 6).

DISCUSSION

Traumatic intracranial aneurysms are rare complications of craniofacial trauma, accounting for less than 1% of all cerebral aneurysms[9]. Traumatic intracranial aneurysms are predominantly located in the anterior circulation and commonly occur in the distal cerebral vascular tree. The most frequent sites are the peripheral branches of the middle cerebral artery, followed by branches of the pericallosal artery[9].

Several mechanisms have been proposed to explain the formation of traumatic intracranial aneurysms, all of which involve direct vessel injury. However, indirect injury can produce a traumatic aneurysm as a result of the brain shifting or forceful stretching of the vessel by an adjacent structure, such as a falx, tentorium, or sphenoid ridge[9]. The brain and its vessels may also strike bony or connective tissue protuberances. The mechanism involved in our case was suspected to be direct injury by basal skull fracture, overstretching or torsion of the ICA wall due to
the different velocities of the brain and skull and tearing of the ICA by nearby sharp tips of bony structures. Remarkably, bony protuberances on the anterior and posterior clinoid processes were observed in our patient with tips in close proximity to the supraclinoid ICA. We assert that these bony protuberances of the clinoid processes pierced and may have torn the ICA secondary to brain shifting and the basal skull fracture sustained in the accident. Such a scenario, along with the supraclinoid ICA in close proximity to the clinoid processes, may explain why a closed craniofacial trauma led to the formation of traumatic aneurysm, particularly in atrophic brain. While traumatic intracranial aneurysms are more common in children than adults, Buckingham et al. report that 30% of all traumatic aneurysms occurred in patients younger than 20 years of age. In elderly, we suggest that the shifting of intracranial structures following craniofacial injury may be an important cause of traumatic aneurysm in elderly patients with atrophic brain. Traumatic intracranial aneurysms have been classified as true, false, and mixed aneurysms. It has previously been reported that most traumatic aneurysms are of the false variety. Asari et al. reviewed 23 cases of traumatic aneurysms, of which only four could be considered true aneurysms with the remaining 19 cases identified as false aneurysms. Angiographic characteristics of true aneurysm are a clear neck and regular contoured sac. However, angiographic findings of traumatic aneurysms reveal the absence of a clear neck, unusual locations for aneurysm (such as a non-bifurcation site or in intracranial peripheral vessels), and irregular aneurysm sac, associated with delayed filling and emptying. In our case, the pseudosac was found to contain an organized hematoma with fibrin and admixed white cell elements without the presence of normal vessel wall elements. Such appearances were consistent with a diagnosis of a false aneurysm.

The primary goals of managing traumatic intracranial aneurysms are early diagnosis and intervention to prevent delayed-onset rebleeding or thromboembolic complications. Traumatic intracranial aneurysms are often fragile and prone to rupture, and therefore present a challenging subset of vascular lesion for either surgical or endovascular intervention. The mortality rates for traumatic intracranial aneurysms not treated aggressively
varies from 32 to 50%\textsuperscript{334}. However, in surgically treated patients, the mortality rates range from 18% to 20%\textsuperscript{10}. Patients consistently experience superior outcomes after surgical management of traumatic aneurysms. Endovascular intervention, which includes placement of detachable balloons or coils, has also been successfully employed for the treatment of traumatic aneurysms\textsuperscript{11,12}. We believe that aggressive surgical or endovascular intervention is essential once the diagnosis has been established.

**CONCLUSION**

Traumatic intracranial aneurysm is a rare complication of closed craniofacial trauma. The possibility of a traumatic aneurysm should be considered when a patient with SAH demonstrates bony protuberances on the clinoid process as a traumatic aneurysm may result from mechanical injury by the sharp bony edges. A high index of suspicion in this setting allows surgeons to reduce the possibility of intraoperative catastrophic rupture.

**References**


