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Carotid canal fracture with internal carotid artery transection: A deadly trauma

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Abstract

Carotid artery injuries are rare with an incidence of 1%–2.6% in trauma patients. They are associated with high morbi-mortality rates, with mortality ranging from 19% to 43%. The diagnosis relies mainly on computed tomography angiography in the emergency setting; however, it is fundamental to be able to suspect carotid artery injuries on non-contrast computed tomography, as the latter is the routine imaging tool for trauma patients. We report the case of a young male, victim of a blunt high velocity motor-vehicle trauma. He was unconscious, with abundant epistaxis and hypovolemic shock. A fracture of the left carotid canal on non-contrast computed tomography was seen, raising concern for an arterial injury. A computed tomography angiography was performed subsequently revealing a transection of the internal carotid artery. This type of injury is highly lethal, and its management relies on urgent surgical intervention, and endovascular treatment, with the purpose of controlling the hemorrhage.

Keywords

Internal carotid artery, carotid canal fracture, trauma, hemorrhage, computed tomography

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Introduction

Carotid artery (CA) injuries are rare with an incidence of 1% to 2.6% in trauma patients.

They are associated with high morbi-mortality rates, with mortality rates ranging from 19% to 43%.[1]

Our case draws attention to the clinical signs and non-contrast computed tomography (CT) signs that should raise concern for an arterial injury and highlights the importance of availability of endovascular treatment in the emergency setting.

Case report

A 36-year-old male motorcyclist, with unremarkable history, victim of a high-velocity motor-vehicle collision while not wearing a helmet, arrived at the emergency room. The physical examination found an unconscious patient (with a Glasgow Coma Scale score of 3), with multiple blunt cranio-facial injuries with abundant epistaxis, and hemodynamic instability with low blood pressure (100 mm Hg) and accelerated heart rate (105 bpm).

A total-body CT scan was performed, within 6 h post-trauma, showing in the non-contrast brain scan, multiple cranio-facial fractures, including a bilateral fracture of the carotid canals. The fracture in the left carotid canal was slightly displaced (Figure 1). Left fronto-parietal extra-dural hematoma, intra-parenchymal hematoma, subarachnoid hemorrhage and diffuse left hemispheric edema were associated (Figure 2).

The carotid canals fractures raised worries about a potential internal CA injury (especially in the left, due to the fracture being displaced and due to the diffuse left hemispheric edema); therefore, post-contrast brain CT acquisitions in the arterial, venous, and delayed phases were performed, showing in the arterial phase a loss of opacification in the petrous, cavernous, and intradural segments of the left internal CA (Figure 3), and showing in the delayed phase, a voluminous contrast extravasation into the extradural collection and into the homolateral sphenoidal sinus (Figure 4(a) and (b)),

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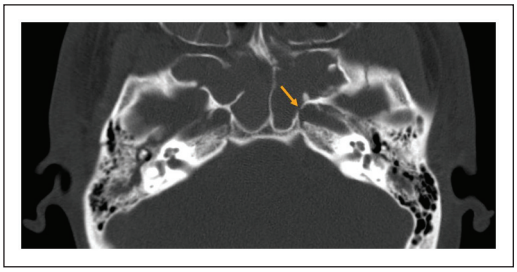

Figure 1. Axial CT scan image in the bone window showing bilateral carotid canal fractures, slightly displaced in the left (arrow).

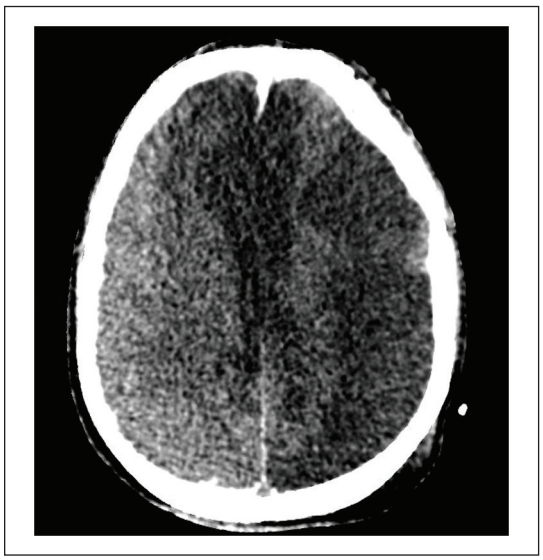

Figure 2. Axial CT scan image in the brain window, showing diffuse left hemispheric edema with mass effect on the falx cerebri, probably linked to infarction.

testifying of a left internal CA transection with a massive active bleeding.

The patient unfortunately died soon after CT was performed, due to hypovolemic shock and multiorgan failure despite maximal supportive measures. The location of the vascular lesion made it surgically not accessible, and the endovascular approach, unfortunately, was logistically not possible.

Discussion

Non-contrast CT scan is a very useful routine imaging tool in screening for intra-cranial lesions in trauma patients. CT after contrast injection (angiography), magnetic resonance angiography, and digital subtraction angiography (the gold standard) are more sensitive diagnostic tools for identifying CA injuries.[2] However, in practice, CT angiography is used more than the two other imaging tools, as they are often not accessible urgently.

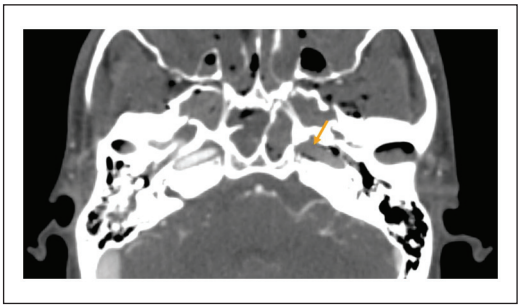

Figure 3. Axial post contrast CT image in the arterial phase, in the soft tissue window, showing a lack of opacification in the left internal carotid artery (Arrow).

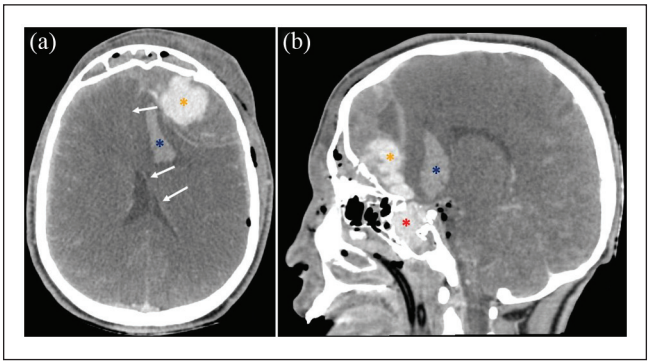

Figure 4. (a) Axial post contrast CT image in the delayed phase, showing extravasation of contrast into the left frontal extra-axial hematoma (orange asterisk). Note the left frontal intra-parenchymal hematoma (blue asterisk), and the mass effect on the homolateral lateral ventricle and the falx cerebri (arrows). (b) Sagittal reconstruction post-contrast CT image in the delayed phase, showing extravasation of contrast into the extra-axial hematoma (orange asterisk) and into the left sphenoidal sinus (red asterisk). Also note the intra-parenchymal hematoma (blue asterisk).

It is important to be able to predict CA injuries clinically and on non-contrast brain CT since post-contrast CT is not systematically performed in the trauma context.

Clinically, a Glasgow Coma Scale score below 8, neurologic symptoms unexplained by known injuries, or epistaxis from a suspected arterial source should raise concerns about a vascular lesion.[3]

Injuries on non-contrast CT that are predictive of CA injuries, and that impose to carry out post-contrast imaging are as follows: a fracture traversing the carotid canal, a penetrating injury, when there is concern for post-traumatic vasospasm, and atypical intracranial hemorrhage pattern for trauma (such as subarachnoid hemorrhage when it is isolated in the basilar cisterns, when it is isolated and of large volume in the sylvian fissure, and when it is isolated in the anterior inter-hemispheric fissure. These atypical patterns raise the suspicion for a ruptured aneurysm). Moreover, a large hematoma associated with calcifications or prominent veins should raise suspicion for vascular malformation.[4]

Consider that, generally, basilar skull fractures, cervical fractures, spine fractures, and thoracic injuries have a higher risk of CA injuries.[1]

Edema in a vascular territory linked to a cerebral infarction can also reveal an internal carotid injury in the homolateral side.[5]

Diffuse cerebral edema can result from head injury, possibly secondary to disruption of cerebral autoregulation and blood–brain barrier. The release of various mediators can increase blood–brain barrier permeability driving to vasogenic and cytotoxic edema.[4]

It should be noted that blunt carotid artery injuries can be seen even in the absence of a fracture, occurring due to a rapid deceleration with hyperextension and rotation of the neck, which stretches the internal CA over the transverse process of the superior cervical vertebrae, driving to an intimal laceration.[5]

Arterial injuries include arterial dissection, pseudoaneurysm, vascular occlusion, active extravasation, and traumatic fistula (carotid-cavernous or dural arteriovenous fistula).[4]

The injuries can be graded as follows:

Grade I: Intimal irregularities with less than 25% luminal narrowing.

Grade II: More than or equal to 25% luminal narrowing, intraluminal thrombus, or raised intimal flap.

Grade III: Pseudoaneurysm.

Grade IV: Occlusion.

Grade V: Vessel transection with active extravasation: has the highest mortality rate (50%).[1,3,6]

Management of Grade V lesions (transection with extravasation) relies on immediate surgical exploration.[3] Emergent endovascular treatment of arterial transection is mainly practiced when lesions are not accessible surgically.[6]

The endovascular techniques include embolization and balloon occlusion.[1] Stent grafting on the other hand demonstrates a 95% efficacy rate for endovascular covered stents in controlling acute hemorrhage with a reported rebleed rate of 30%.[6]

The best approach in our case would have been to stent graft the left internal carotid artery from its cervical segment to its supra-clinoid segment, or to sacrifice the artery if the circle of Willis is complete.

Conclusion

Blunt post-traumatic carotid artery transection with active bleeding is rare, but with poor survival rates.

CT angiography is the practical tool to diagnose and grade these types of injuries in the emergency setting.

On non-contrast CT, basilar skull fracture especially when traversing the carotid canal and hemispheric infarction are clues to the diagnosis.

Making the endovascular treatment accessible and logistically possible in the emergency setting will help reduce the mortality rates in this highly lethal type of injury.

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Author contributions

A.N., M.B., and I.D.D. contributed to the conception, acquisition, analysis, interpretation of data, and drafted the manuscript.
O.E.A., F.Z.L., and L.J. critically revised the manuscript and approved it.

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Ethical approval

No ethical approval is required for de-identified single case reports based on our institutional policies.

Informed consent

Written informed consent was obtained from a legally authorized representative(s) for anonymized patient information to be published in this article.

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