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Vol: 2 & Iss: 2

Case Report

Traumatic Internal Carotid Artery Dissection: Unveiling the Silent Catastrophe: A Case Report

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Citation: Pendse T, Joshi P, D'Souza J, Murumkar V. Traumatic Internal Carotid Artery Dissection: Unveiling the Silent Catastrophe: A Case Report. *Curr Res Altern Complement Integra Med 2025;* 2(2): 131-133.

Received: 29 September, 2025; **Accepted:** 13 October, 2025; **Published:** 15 October, 2025

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ABSTRACT

Traumatic internal carotid artery dissection, as a cause of stroke, is infrequent yet significant, being responsible for 20% of ischemic strokes in patients under 50 years of age. It is a potentially devastating condition that occurs as a consequence of blunt or penetrating trauma to the neck region. It can present with a wide spectrum of symptoms, ranging from mild neck pain and headache to more severe manifestations. The non-specific nature of these symptoms often poses a diagnostic challenge.

Objectives: To help identify early imaging features of traumatic internal carotid artery dissection and to facilitate early intervention.

Imaging findings: Case: A 17-year-old male, met with a road traffic accident, came with drowsiness and multiple craniofacial fractures. One of the fracture lines was traversing through the right carotid canal. CT brain revealed hyperdense right MCA and ICA with evolving right MCA territory infarct. CT angiography revealed smooth tapering of right ICA at cervico-petrous junction, likely due to dissection and complete occlusion of communicating segment of ICA.

Conclusion: High index of suspicion is required to diagnose traumatic dissection of the ICA and prevent subsequent catastrophic outcome.

Heightened awareness among healthcare professionals is crucial for early diagnosis and timely intervention.

Keywords: Internal carotid artery dissection, Stroke, Traumatic dissection

1. Introduction

Traumatic internal carotid artery dissection (TIAD) is a rare and potentially life-threatening condition that occurs as a result of trauma to the neck region.

It involves a tear or injury to the inner lining of the internal carotid artery, one of the major blood vessels supplying the brain. TIAD may result from a direct blow to anterolateral aspect of the neck or an extreme extension and rotation of the neck. Trauma mechanisms involved are variable, ranging from high-speed motor vehicle accidents to trivial traumas in certain groups of patients (for example patients with hypertension or connective tissue diseases)[1].

TIAD evolves into stroke in 80% of cases within the first week of the trauma. The common cause of stroke is arterial thrombosis resulting in permanent neurological deficits, with a mortality rate approaching 40%.

Clinically, TIAD can present with a variety of symptoms that can range from mild neck pain and headache to more severe

manifestations such as focal neurological deficits or signs of cerebral ischemia.

However, the clinical presentation can be nonspecific and easily overlooked, making the timely and accurate diagnosis of TIAD challenging.

Diagnostic approaches, including imaging techniques such as computed tomography angiography (CTA), magnetic resonance angiography (MRA) and Doppler ultrasound, play a critical role in identifying and confirming TIAD.

Hence the objective of this study is to help identify early imaging features of traumatic internal carotid artery dissection.

2. Case Presentation

A 17-year-old male was brought to the emergency department following a high-velocity road traffic accident. On arrival, he was drowsy with a low Glasgow Coma Scale (GCS) score and had multiple craniofacial fractures.

2.1. Initial imaging

Non-contrast CT brain and cervical spine revealed a fracture line traversing the right carotid canal, with hyperdense right internal carotid artery (ICA) and middle cerebral artery (MCA), suggestive of acute thrombosis. Evolving hypodensity in the right MCA territory indicated an acute infarct.

2.2. Clinical course

The patient's neurological status deteriorated, necessitating intubation. Repeat CT angiography demonstrated complete occlusion of the right ICA at the communicating segment, with smooth, flame-shaped tapering of the distal cervical ICA extending to the cervico-petrous junction. Features were consistent with traumatic dissection and intramural hematoma. A large right MCA territory infarct with midline shift and transtentorial herniation was noted.

2.3. Outcome

Despite supportive measures, the patient developed worsening neurological deficits and ultimately succumbed to the complications of massive infarction secondary to ICA dissection **(Figures 1-3)**. **(Figure 1)** showing findings of CT brain plain. Clinically, the patient deteriorated and his GCS dropped further, warranting a CT brain angiography.

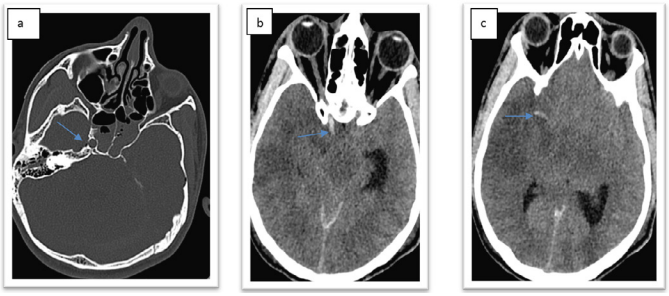

Figure 1: CT brain plain shows:

a-Bone window showing fracture of the right carotid canal.

b-Hyperdense right ICA suggestive of thrombosis.

c- Hyperdense right MCA suggestive of thrombosis with surrounding loss of grey white matter differentiation and hypodensity in right MCA territory, likely suggestive of infarct.

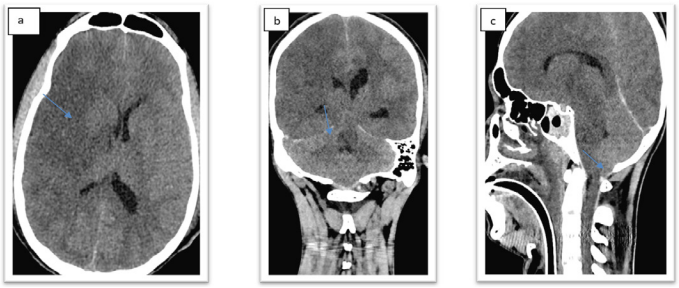

Figure 2: shows CT brain angiography findings, which was done after clinical deterioration.

a- Large right MCA territory infarct causing midline shift of 8.3 mm to the left.

b,c- Central descending transtentorial herniation noted (7.3 mm below the foramen magnum).

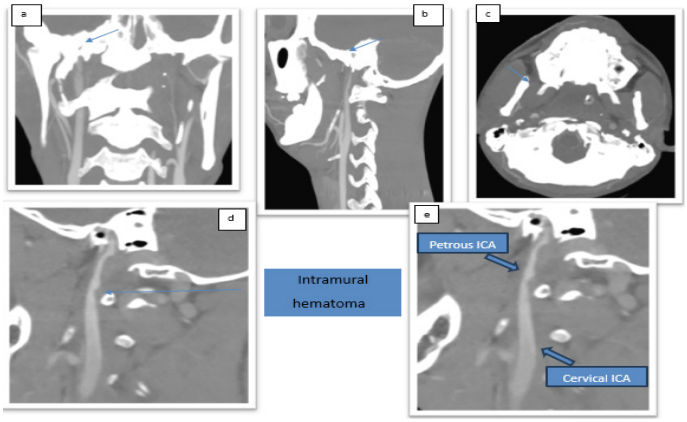

Figure 3 demonstrates:

a,b-Smooth flame shaped tapering of right distal cervical ICA extending till the cervico-petrous junction and sparing of carotid bulb, likely due to dissection.

c,d,e- extrinsic compression from posterior wall on the residual lumen of ICA likely due to intramural hematoma.

3. Discussion/Summary

Traumatic internal carotid artery dissection is characterized by the formation of an intramural hematoma, which can lead to partial or complete obstruction of blood flow and subsequent ischemia or even arterial rupture.

Extracranial internal carotid artery dissection is more common than intracranial internal carotid artery dissection. It typically occurs 2-3 cm superior to the common carotid bifurcation.

Traumatic ICAD is suspected and diagnosed when neurological symptoms occur unexpectedly after a trauma.

3.1. Pathophysiology

There are two types of arterial wall tears that can occur: subintimal and subadventitial dissections. In subintimal dissections, the tear happens between the innermost layer of the artery called the intima and the middle layer called the media. When blood enters between these layers, it can cause narrowing or stenosis of the artery[2].

On the other hand, in subadventitial dissections, the tear occurs between the middle layer (media) and the outer layer (adventitia) of the arterial wall. This type of tear may result in the dilation or enlargement of the artery, forming an aneurysm within its wall.

In some cases, an intramural hematoma can develop without an actual tear in the artery wall. This can occur particularly in arteries that are affected by arteriopathy, which refers to a disease or abnormality of the blood vessels.

In such cases, bleeding can originate from small blood vessels called vasa vasorum within the arterial wall, leading to the formation of an intramural hematoma **(Figure 4)**.

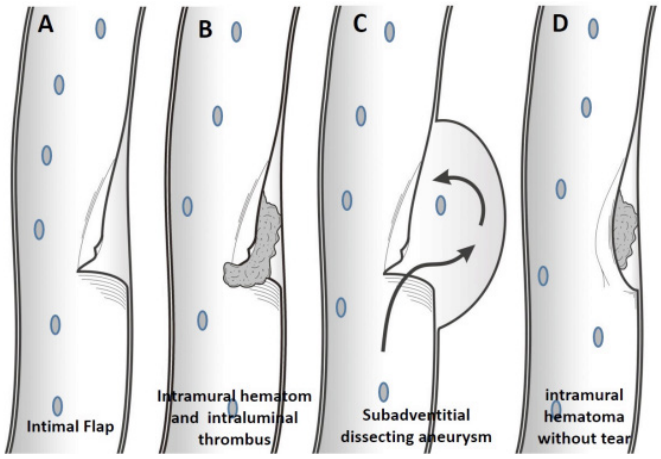

Figure 4: demonstrated various mechanisms of dissection[2].

A-shows an intimal flap.

B-Tear between the intima and media with collection of blood in between causing intramural hematoma.

C-Subadventitial dissecting aneurysm- tear between media and adventitia.

D- Intramural hematoma without tear.

3.1.1. Clinical presentation: TIAD is often underrecognized due to its nonspecific symptoms, which may include headache, neck pain, Horner's syndrome or transient ischemic events. Neurological deterioration, as in this case, is usually rapid once cerebral ischemia develops.

3.1.2. Imaging: Early and accurate diagnosis hinges on high suspicion and appropriate imaging. CT angiography is the first-line modality in trauma settings due to its wide availability, rapid acquisition and high sensitivity. Key features include flame-shaped tapering of the lumen, intramural hematoma and complete or partial occlusion. MR angiography, vessel wall imaging and duplex ultrasound are useful adjuncts, especially for follow-up. Differentiating TIAD from mimics such as atherosclerosis, vasculitis or compressive lesions is critical.

3.1.3. Management: Treatment strategies range from anticoagulation or antiplatelet therapy to endovascular stenting or surgical repair, depending on the severity, location and hemodynamic significance of the lesion. In unstable patients or those with major infarction, as in our case, options are limited and prognosis is poor. Early recognition prior to catastrophic infarction is the cornerstone of improving outcomes.

3.1.4. Comparison with literature: Previous reports highlight the diagnostic challenge posed by TIAD, particularly in young trauma patients with overlapping craniofacial injuries. Mehdi et al. described radiologic pitfalls and mimics of craniocervical dissections, emphasizing the need for careful review of the carotid canal in patients with skull base fractures. Similarly, Goodfriend et al. note that subtle imaging findings, if overlooked, may delay intervention and worsen outcomes.

4. Conclusion

This case underscores the devastating consequences of delayed recognition of TIAD. Radiologists play a pivotal role in early detection by carefully assessing the carotid canal and ICA in trauma imaging. High suspicion, timely use of CTA and multidisciplinary management are vital in mitigating morbidity and mortality.

5. References

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