Scholars Journal of Medical Case Reports
Abbreviated Key Title: Sch J Med Case Rep
ISSN 2347-9507 (Print) | ISSN 2347-6559 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

Radiology

Ischemic Stroke Secondary to A Traumatic Carotid Artery Dissection

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DOI: https://doi.org/10.36347/sjmcr.2025.v13i11.041 | **Received:** 15.09.2025 | **Accepted:** 03.11.2025 | **Published:** 20.11.2025

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Abstract | **Case Report**

Background: Internal carotid artery dissection is an uncommon yet important cause of ischemic stroke in young individuals without any known cerebrovascular risk factors, often resulting in high morbidity and mortality. When patients exhibit significant neurological deficits following blunt trauma to the head or neck, carotid artery injury should be suspected until ruled out. Early detection and intervention guided by a structured algorithm, can help prevent severe brain damage.

Keywords: Internal carotid artery dissection, Ischemic stroke, Traumatic brain injury (TBI), Epidural hematoma, Cerebral angiographic study, Glasgow Coma Scale (GCS).

INTRODUCTION

Traumatic brain injury (TBI) is a significant public health concern and a major cause of death and disability, it increases the lifetime risk of acute ischemic stroke through several mechanisms, often involving the disruption of cerebral blood flow. The primary pathophysiological processes include traumatic vascular injury, cerebral contusion or hematoma, and embolism [1]. Carotid artery dissection is a rare but life-threatening condition that can occur following a head and neck trauma. There should be a high index of suspicion in patients with a mechanism of injury that places the internal carotid artery at risk because blunt vascular injury may show delayed onset with no initial symptoms of vascular damage. [2]

Dissection of the internal carotid artery in both its extracranial and intracranial segments represents approximately 2% of ischemic strokes, especially in younger individuals with no known cerebrovascular risk factors. Research indicates that this condition is more common in people aged 30 to 50, with no significant gender differences. The extracranial segments are more commonly involved, which can be attributed to their increased mobility.[3].

CASE REPORT

We report the case of a 30-year-old male patient, a victim of a road traffic accident, admitted to the emergency department for a severe traumatic brain injury. A body scan was performed after stabilizing the patient. At the cerebral level, the CT scan revealed an acute right fronto-parietal epidural hematoma with a maximum thickness of 10 mm, without any mass effect on the median structures, associated to a meningeal hemorrhage. On the bone window, a left frontal fracture line extending to the right fronto-parieto-temporal region was detected, along with a left temporomandibular joint subluxation. Due to the deterioration of the patient's neurological status, characterized by a decline in the Glasgow Coma Scale (GCS) score and the appearance of miosis, a cerebral angiographic study was performed. Imaging revealed an acute ischemic stroke involving the right cerebral hemisphere, with evidence of mass effect resulting in right-to-left falcine herniation. The infarction predominantly affected the right temporal and central regions and was associated with diffuse cerebral edema. Additionally, there was a suspected endoluminal hypodensity involving the cervical segment of the right internal carotid artery, extending into its petrous portion, suggestive of a possible intraluminal thrombus or dissection. **[Figure 1]**.

The patient was transferred to the neurosurgical operating room for surgical evacuation of the hematoma, followed by admission to the surgical intensive care unit for postoperative monitoring and management. Subsequent cerebral magnetic resonance imaging (MRI) confirmed the presence of a right internal carotid artery dissection, which was responsible for a compressive ischemic stroke involving the entire vascular territory of

the affected artery. The MRI also demonstrated multiple additional ischemic lesions within the right frontotemporal and mesencephalic regions, as well as in the left cerebellar hemisphere **[Figures 2 and 3].**

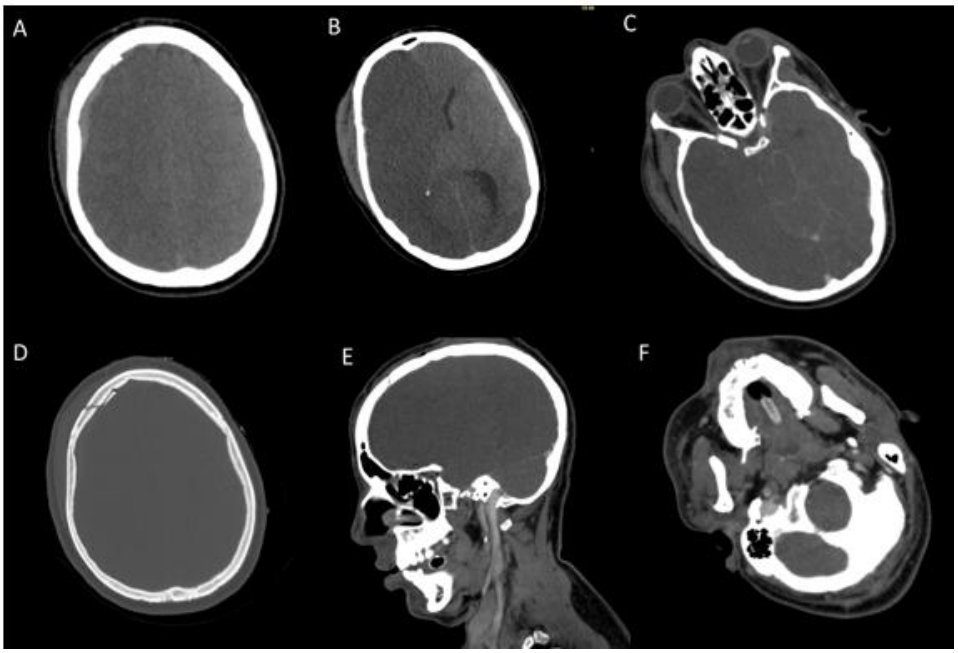

Figure 1: Brain CT scan before contrast injection (A, B, C): parenchymal window: Axial section (A, B, C) showing a right frontal extradural hematoma associated to a right hemispheric hypodensity exerting a mass effect on the median structures with contralateral diffuse edema and an anteroposterior elongation of the mesencephalon related to a central herniation. Bone window axial section (D) showing a right frontal fracture line. Parenchymal window, after contrast injection sagittal (E) and axial section (F): showing a linear endoluminal hypodensity of the right internal carotid artery likely related to a carotid dissection.

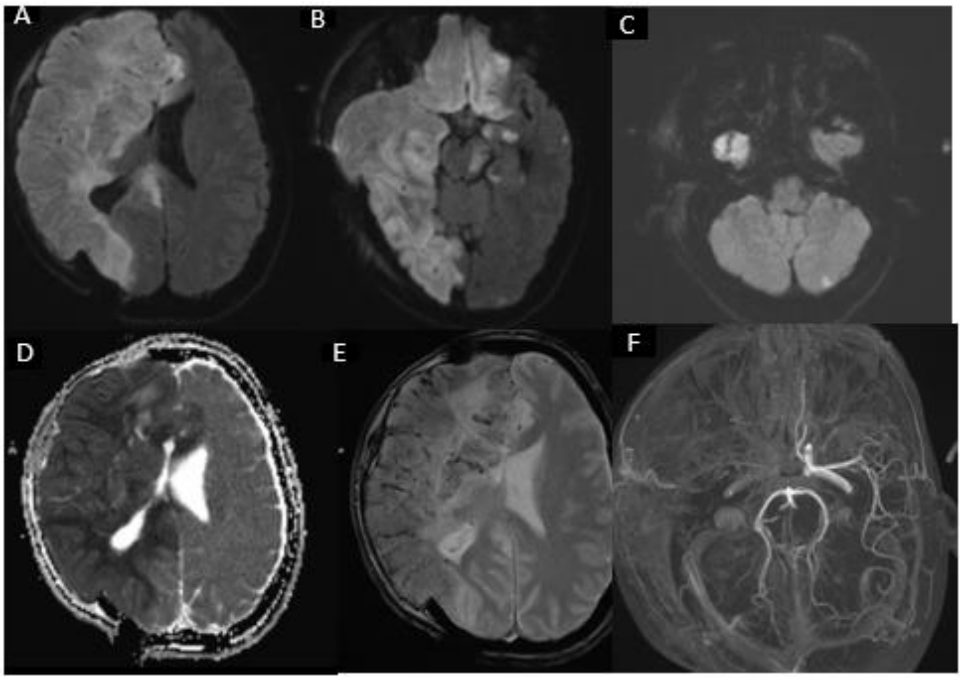

Figure 2: Brain MRI images (A–C) in axial diffusion-weighted sequences demonstrate a right hemispheric hyperintense signal consistent with an acute ischemic process, exerting a compressive effect and extending to involve the left fronto-temporo-occipital region, as well as the left mesencephalic and cerebellar areas, with corresponding ADC restriction (D). Image (E), an axial T2-weighted echo sequence, reveals areas of hemorrhagic transformation within the infarcted regions. Image (F), a time-of-flight magnetic resonance angiography (TOF-MRA), shows an abrupt cutoff of the right internal carotid artery (ICA) with non-visualization of its distal branches, consistent with arterial occlusion secondary to dissection.

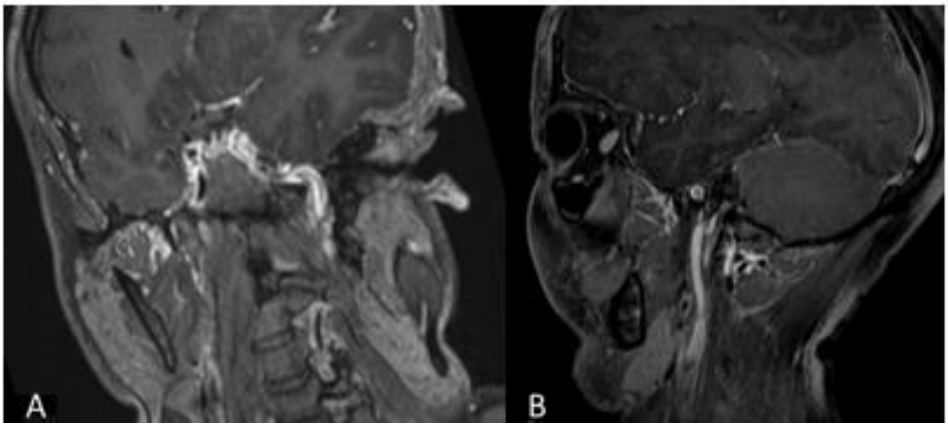

Figure 3: Brain MRI coronal (A) and sagittal (B): T1 FAT SAT sequences after gadolinium injection showing an intimal flap of the right internal carotid artery (ICA)

DISCUSSION

Ischemic stroke is observed acutely in 2.5% of moderate to severe TBI survivors and predicts worse functional and cognitive outcome. Half of TBI patients with AIS were aged ≤40 years, and AIS patients more often had cervical dissection.[4]

Internal carotid artery dissection's pathogenesis remains unclear. It generally occurs when an intimal tear permits blood to enter the potential space between the vessel intima and media. However, arterial dissection, cerebral vasospasm, and thrombosis have been some of the theories discussed in the pathogenesis of cerebral infarct secondary to carotid artery dissection after trauma injuries [5-6]. In general, clinical findings seem to be different when comparing traumatically induced ICAD with spontaneous ICAD. In the traumatic group, focal cerebral ischemic symptoms were the most common manifestations. In the spontaneous group, unilateral headaches were the most commonly experienced symptom, often in association with Horner syndrome. [6-7] Like spontaneous ICAD, the onset of ischemic signs and symptoms may be delayed in traumatic cases.

Neuroimaging plays an essential role in establishing the diagnosis of post-traumatic carotid artery dissection (PTCAD) and its complications. Non-contrast enhanced CT scan was shown to be insensitive for PTCAD, however this imaging modality is able to show skull base fracture, which is commonly associated with PTCAD in children and adults.[8] In adults, air extending along the carotid canal was shown to predict injury of the ICA with a sensitivity of 60% and specificity of 67%. Although sensitivity and specificity are rather low, air inclusions extending along the carotid canal in combination with skull base fractures alert the radiologist to examine the ICA region carefully to rule out PTCAD, [9] While experience remains limited, studies have reported that CTA can be sensitive as MRA in detecting carotid artery dissection (CAD). CAD findings in CTA typically include eccentric mural thickening in stenotic regions and a narrowed central or eccentric enhancement from a residual lumen surrounded by hypodensity secondary to a mural hematoma. [10] MRI and MRA have been shown to be as sensitive and specific as conventional angiography. MRI findings of PTCAD include absence of the normal flow void or altered luminal signal intensity in and narrowing of the arterial lumen with hematoma within the arterial wall [8]. The imaging characteristics of the thrombus within the carotid sheath are the diagnosis key. The thrombus will appear as a bright crescent (isointense with fat) encircling the ICA lumen on both T1 and T2 fast spin-echo axial MRI this crescent luminal sign (corresponding to the true lumen with the void signal), surrounded by the false lumen or intramural hematoma. No appreciable enhancement will occur after the administration of intravenous gadolinium. The pattern of ischemic lesions most commonly associated with extracranial internal carotid dissection, according to the literature, includes cortical lesions in 83% of cases and subcortical lesions in 60%. [3-6-11]

The Management is influenced by several factors such as symptoms, injury location, severity grade, and any associated injuries, different treatment options aim to reduce neurological deficits and restore cerebral circulation, including thrombolysis, antithrombotic therapy, endovascular procedures, and open carotid artery surgery. If there are no contraindications, such as active hemorrhage, treatment recommendations for antithrombotic therapy are tailored based on the injury grade [[12-13]. Follow-up imaging, typically with CT angiography, is recommended 7 to 10 days after the initial identification of the cerebrovascular injury, with additional imaging after 3 months to assess the need for long-term antithrombotic therapy.

CONCLUSION

Ischemic stroke following traumatic brain injury (TBI) can be detrimental, whether it occurs immediately or later. However, ischemic stroke that develops shortly after TBI requires special attention due to its strong link to poorer patient outcomes, which may be preventable with prompt intervention. Early clinical suspicion, supported by radiological findings, enables

timely neurovascular treatment aimed at preserving viable brain tissue during the critical hours following the injury.

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