Bilateral Carotid Artery Dissection after High Impact Road Traffic Accident

Ankur Srivastava[1*], Marcus Bradley[2], Michael Kelly[3]

1. Department of Medicine, Southend Hospital, Essex, UK

2. Department of Radiology, Frenchay Hospital, Bristol, UK

3. Department of Surgery, Frenchay Hospital, Bristol, UK

* **Correspondence:** *Dr. Ankur Srivastava,, Department of Medicine, Southend Hospital, Prittlewell Chase, Westcliff-on-Sea, Essex SS0 0RY, UK (Ankur.Srivastava@southend.nhs.uk)*

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ABSTRACT

A 58 year old man was involved in a high impact road traffic incident and was admitted for observation. Asymptomatic for the first 24 hours, he collapsed with symptoms and signs consistent with a cerebrovascular accident. Computed tomography angiogram (CTA) and Magnetic resonance angiogram (MRA) demonstrated bilateral internal carotid artery dissections and a left middle cerebral artery infarct. It was not considered appropriate to attempt stenting or other revascularistation. The patient was treated with heparin prior to starting warfarin. He made a partial recovery and was discharged to a rehabilitation facility. This case is a reminder of carotid dissection as an uncommon but serious complication of high speed motor vehicle accident, which may be silent initially. Literature Review suggests risk stratification before relevant radiological screening at risk patients. Significant advances in CTA have made it the diagnostic tool of choice, but ultrasound is an important screening tool.

CASE REPORT

Introduction:

The most common cause of blunt traumatic injury to the carotid vasculature is motor vehicle accidents (1). Blunt trauma can be classified as direct, for example strangulation, or indirect, where deceleration forces cause injury, as found in motor vehicle accidents and bungee-jumping (2). Dissection of the carotid arteries is rare with recent data suggesting rates in patients with blunt head trauma in the range of 0.86% (3). Whilst blunt trauma causes only a tenth of cervical vessel injuries, it accounts for 80% of cases with neurological defects (4), confirming the seriousness of this type of injury.

Diagnosis is frequently delayed as symptoms and signs are often absent initially, and the task is to identify susceptible patients and devise suitable radiological screening methods. Debate continues on the merits of screening people after involvement in high speed road traffic accidents, and which is the best screening modality

Reported herein is a case of bilateral carotid dissection due to a high speed motor vehicle accident. The patient was initially completely asymptomatic and the diagnosis was not considered until he collapsed 24 hours after the injury with hemiplegia.

Case Report:

A 58 year old man was admitted following a high speed (exceeding 70 mph) motor vehicle collision which resulted in a fatality in the other vehicle. He was a generally fit and well man with no pre-existing conditions On admission he was well and complained only of abdominal pain Examination showed normal vital signs, a Glasgow Coma Scale (GCS) of 15 and minor skin abrasions. He had mild generalized abdominal tenderness and neurological examination was entirely normal.

Initial investigations included a normal blood count and serum biochemistry. Chest, abdominal and cervical spine X-rays were all normal. Ultrasound of the abdomen the following day showed normal intra-abdominal organs with no evidence of any free fluid.

After twenty four hours of uneventful observation, as the patient was being prepared for discharge, he collapsed in the ward. Vital signs were normal and respiratory, cardiovascular and abdominal examinations were unremarkable. However, neurological examination showed a Glasgow Coma Scale score of 11, a total right hemiparesis (grade 0 power) and both receptive and expressive aphasia. There were no carotid bruits. An urgent computed tomography (CT) scan of the brain was performed, an hour after the collapse and was entirely normal.

A CT angiogram (CTA) was concurrently performed, which confirmed tapering occlusion of the left internal carotid artery consistent with arterial dissection (figure 1). A dissection was also demonstrated in the right internal carotid.

After the initial diagnosis had been confirmed, a magnetic resonance angiogram (MRA) was arranged. The MRA was performed on a 1.5 Tesla scanner (Intera 11.1.4, Phillips) using a Standard Head Coil. Standard image sequences were obtained with the most useful being the fast spin echo T2-weighted images using the following parameters TR 4959, TE 100; matrix 512 x 512. T1 weighted images were obtained using the TR 703, TE 150 and 512 x 512 matrix settings. The bilateral dissections were visualized (figure 2), the left dissection extending as far as the cavernous sinus and the right up to C1/ C2 level. There was a cerebral infarct in the left MCA territory. The dissections were easily traced on the MRA brain images (figure 3), which demonstrated that the dissection persisted on the left but was not present distally on the on the right (figure 4).

Angiography was undertaken to assess the stenosis in the right carotid and to deduce if revascularisation was appropriate. Initial angiography confirmed a completely occluded left internal carotid with a tapering narrowing from the bifurcation. The left external carotid demonstrated some collateral flow into the distal left internal carotid via the left ophthalmic artery (figure 5) and the right internal carotid displayed a narrow non-occlusive dissection (figure 6).

After careful thought and discussion, in view of his satisfactory clinical status with no symptoms referable to his right hemisphere, and the risks associated with stenting, it was decided to manage him conservatively.

A diffusion weighted MRI scan was performed to evaluate the perfusion status of the hemispheres. Figure 7 demonstrates on this scan a well defined focus of restricted diffusion in the left parietal region and left frontal area, indicating of ischaemia. However, elsewhere, the cerebral blood flow through the cortical mantle appeared satisfactory. To confirm that the diffusion abnormalities are due to restricted diffusion and not artifact, known as T2 shine through, an apparent diffusion coefficient scan was performed which confirmed lesions in similar places, indicating acute infarction.

He was started on an intravenous heparin infusion and warfarin was started and continued subsequently for three months. The patient made significant progress during his 6 week inpatient stay. By the time he was transferred to a local rehabilitation centre closer to his home, the right upper and lower limb function had fully recovered with no discernable difference between right and left. He made considerable progress from his initial dense expressive dysphasia, his vocabulary grew daily and was able to express himself with respect to his care needs.

DISCUSSION

This case report is a reminder of the sub-acute presentation of carotid dissection injuries after high impact traumas. The patient in this report presented with no specific complaints despite significant injury. There was no screening protocol in place for detection of carotid injury in the absence of symptoms after high speed motor vehicle accidents. Suspicion was only raised after deterioration in GCS. The use of GCS is very important when monitoring any patient involved in trauma. The scale provides an objective, reliable way in quantifying consciousness and is particularly valuable when used as a continuous evaluation. Despite a normal CT brain, CT angiogram demonstrated bilateral internal carotid dissections, with total occlusion on the left. Further imaging was obtained with MRA and formal angiography, before a clinical decision was made to manage conservatively.

The mechanism of dissection in road traffic accidents (RTA) is thought to be based on tears in the intima with blood flow into the arterial wall, creating a haematoma that dissects the vessel wall (5). The tear is instigated by the decelerating forces involved in high impact incidents. The dissection may be initially silent but cerebral hypoperfusion and embolism can lead to stroke (6, 7).

The identification of the presence of carotid artery injury before the development of neurological symptoms is a significant challenge (8). Screening protocols have been devised with patients at high risk thought to include those with severe hyperextension or flexion injuries, cervical spine or basilar skull fractures with carotid canal involvement and those with diffuse axonal injury with GCS < 6. Other important factors include patients with cervical bruits, expanding cervical haematomas and focal neurological deficits (9). Screening yields have been quoted at 30% using angiography and protocols based on the principles outlined above (10).

Studies have suggested imaging the vasculature can allow carotid injury to be classified into grades which could help guide management and prognosis. For example, Biffl and colleagues (11) devised a system where a grade I injury suggested intimal injury where luminal narrowing did not exceed 25%. Two thirds of such injuries will heal regardless of treatment option. A visualized dissection flap, or luminal narrowing exceeding 25% would represent a grade II injury, and anticoagulation only would be sufficient in a third of cases. Grade III injuries indicated the presence of pseudoaneurysms, and the authors suggested stent placement

to be beneficial. Grade IV injuries and grade V injuries, signified by vessel occlusion and vessel transsection respectively, were both associated with significant morbidity and mortality.

Duplex ultrasound has been postulated as a possible screening tool (1), because it is non invasive, inexpensive procedure and no contrast agents are used. Ultrasound has been shown to have 86% recognition rates for detection of carotid injuries (12). However, this was a small study which has not been prospectively investigated. Ultrasound is operator dependant and the presence of subcutaneous emphysema or cervical collars can reduce its ability in detecting cervical vessel damage. The diagnostic quality of CT and MRI has been demonstrated in this case report, with several excellent images obtained. Rogers and colleagues (13) have demonstrated that when CTA is used as a screening tool, this increases rates of detection. In addition, CTA allows for visualization of non-vascular injuries. Comparing CTA with the gold standard of catheter angiography, one study demonstrated CTA had sensitivity of 70%, specificity of 67%, positive predictive value of 65% and negative predictive value of 65% (14). Recent advances in this modality have seen results improve (8). MRA has equally, if not better, sensitivity and specificity (15). Studies have demonstrated that MRA images are able to detect ischemic brain changes earlier than CTA, which might alter therapy (8). In summary, CTA is beneficial in acute diagnoses, displaying good anatomical accuracy and allowing diagnosis to be made. However, MRA has the potential to provide improved information regarding the state of the brain and age of the injury, and both modalities can be used in tandem to aid management.

Anticoagulation remains the mainstay of medical treatment of carotid dissection Studies have shown that ischemic events can be avoided in most cases if early diagnosis and treatment with anticoagulants is implemented (9). A recent meta-analysis highlighted the lack of randomized trials in this area. Based on evidence from non-randomized trials, anti-thrombotic and anticoagulants have an important role in artery dissection with the aim to prevent mortality and morbidity (16). The risk-benefit ratio of anticoagulation in established stroke is another controversial area. Knowledge of thromboembolism pathophysiology and clinical experience leads to the theory that heparins will prevent red thrombus formation, propagation and embolism. Unfortunately, effects of reduction of recurrent stroke risk may be counterbalanced by a substantial increased risk of intracerebral haemorrhage with intravenous anticoagulation (17). Overall, the present rationale in the stable patient is to commence anticoagulation, with subsequent angiography, repeat CTA scans with contrast or repeat MRI scans, as well as clinical progress determining further management. The surgical and interventional radiological techniques are beyond the scope of this report.

TEACHING POINT

This report is a reminder that carotid dissection is a rare but serious complication of high speed motor vehicle accidents.

A high index of suspicion of carotid injury should be maintained in any traumatic injury as patients may be asymptomatic on admission. Morbidity from this injury can be significant.

Ultrasound is a useful screening tool. However, CT angiogram or MRI angiogram are the gold standards. Sophisticated imaging may be required to plan optimal treatment which will need to be highly individualized.

ABBREVIATIONS

CTA = Computed tomography angiogram
MRA = Magnetic resonance angiogram
MPH = Miles per hour
GCS = Glasgow coma scale
TE = Time of echo
TR = Time of repetition
ADC = Apparent diffusion coefficient
MCA = Middle cerebral artery
RTA = Road traffic accident

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FIGURES

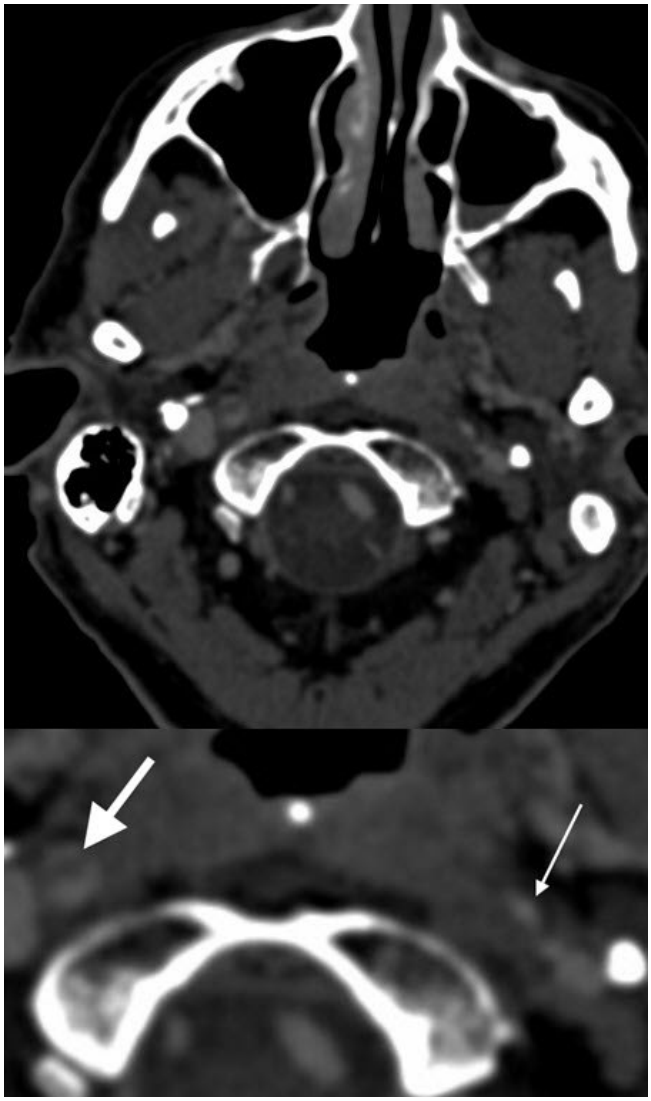

Figure 1: Computed Tomography Angiography, at the level of C1 showing a dissection flap (THICK arrow) across the right internal carotid artery just below the skull base. The left internal carotid artery (THIN arrow) shows a dissected vessel with intramural haematoma and a narrowed eccentric lumen.

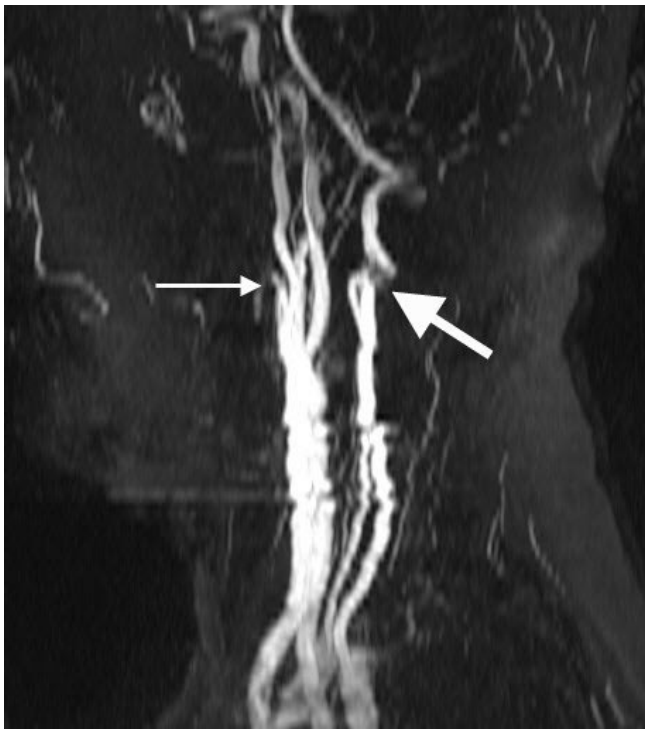

Figure 2: Magnetic Resonance Angiogram, taken as a T1 gradient echo time of flight caption, displaying bilateral carotid dissections. The left internal carotid shows stenosis with surrounding haematoma (THICK arrow) while the right internal carotid displays a false aneurysm formed by a dissection flap (THIN arrow) but remains patent throughout.

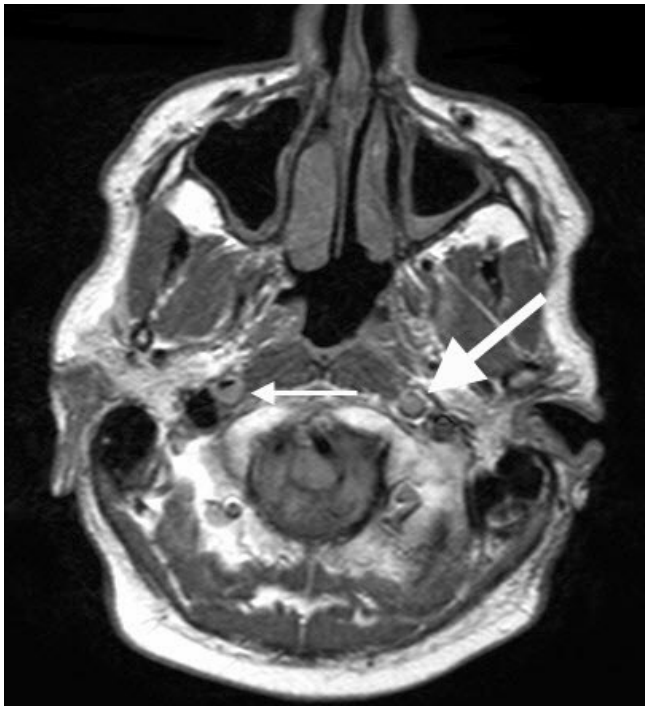

Figure 3: Axial T1 Weighted MRI spin echo sequence at foramen magnum level. Left internal carotid artery shows complete loss of flow void suggesting complete occlusion (THICK arrow). The right internal carotid artery shows a small lumen surrounded by intramural haematoma (THIN arrow).

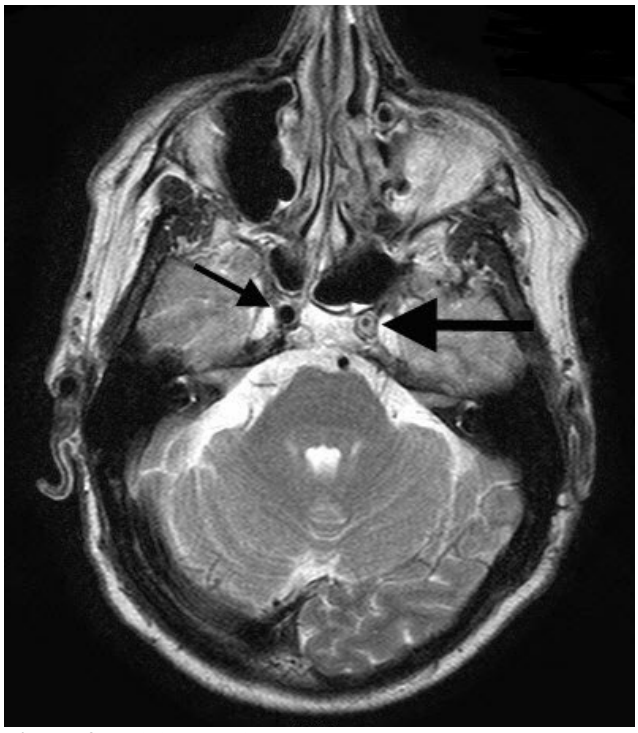

Figure 4: Axial T2 Weighted MRI at skull base shows normal right internal carotid artery flow (THIN arrow) The left internal carotid artery flow remains narrow (THICK arrow).

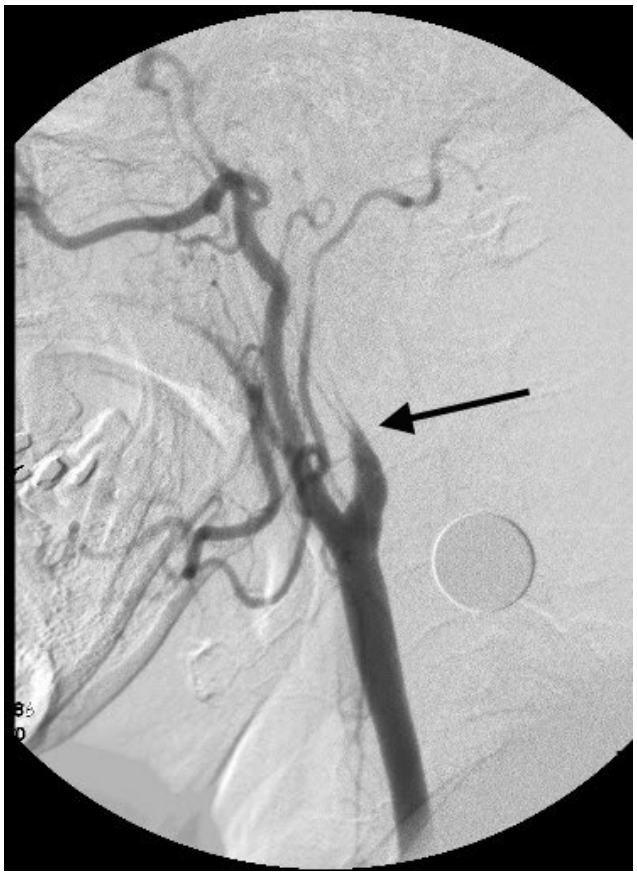

Figure 5: Catheter digital subtracted angiogram (lateral view) showing tapered flame shaped lumen of the left internal carotid artery consistent with dissection and total occlusion.

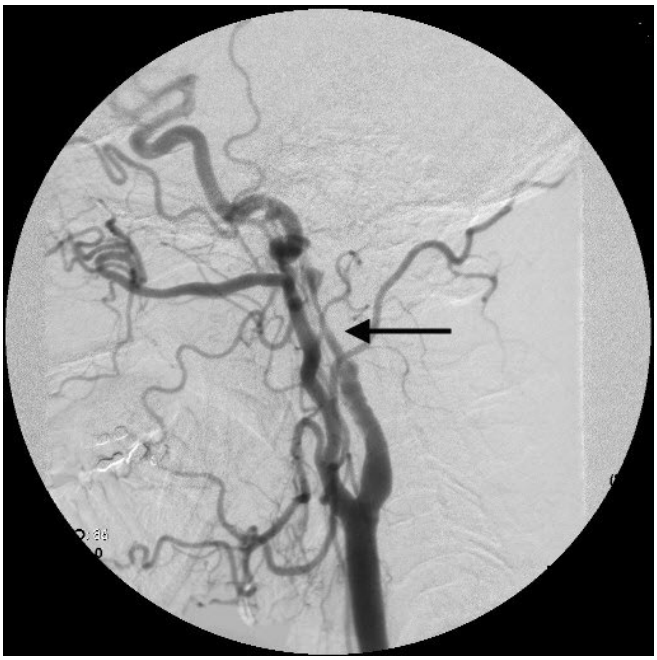

Figure 6: Catheter digital subtracted angiogram (lateral view) showing marked irregularity and narrowing of the right cervical internal carotid artery reflecting a non-occlusive dissection, with approximately 80% narrowing in parts.

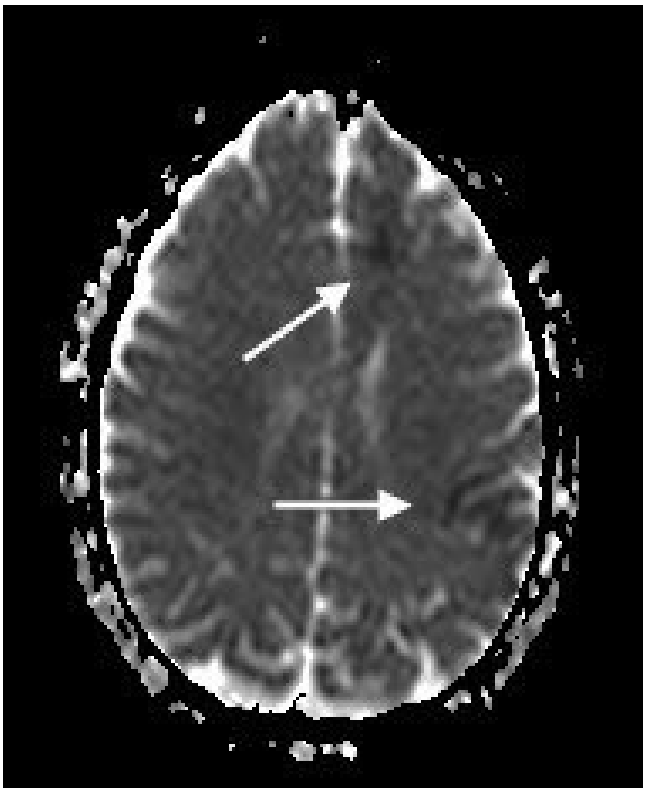

Figure 8: Apparent diffusion coefficient (ADC map) showing similar focal changes (dark) as seen in figure 7 indicating acute infarction.

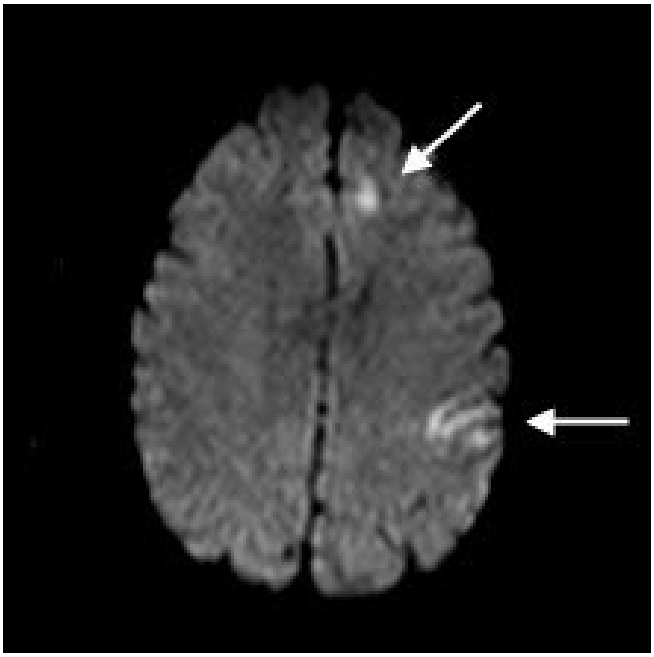

Figure 7: Diffusion Weighted Images show areas of high signal in the left parietal and frontal regions signifying infarcted matter.

KEYWORDS

Carotid dissection; screening; CTA; MRA

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