Vigilance in detecting traumatic blunt neck injuries: A case report

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Abstract

There has been an increase in the number of Motor Vehicle Accidents (MVA) in Malaysia throughout the years. Although blunt neck injury is uncommon, it is associated with severe, permanent neurological deficit with risk of mortality. This case is a classical presentation of a young male involved in a MVA who sustained head and neck injuries of varying severity. After a short symptom free interval, the patient started to develop neurological signs. Presenting signs and symptoms include Horner's syndrome, dysphasia, hemiparesis, obtundation or monoparesis. A computed tomography (CT) scan of brain must be done and if the findings showed that there is no intracranial bleeding (ICB), high suspicions with further evaluation should be done. Confirmation can be obtained by Doppler ultrasonography, magnetic resonance imaging, magnetic resonance angiography (MRA), CT angiography (CTA) or catheter angiography to rule out carotid artery injury.

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Case Report

The patient is a 21-year old Malay male post-MVA at 7am while riding his motorcycle to work. Directly after the accident he went to the nearest hospital for a checkup. He was well with Glasgow Coma Scale (GCS) of 15/15. He was discharged and went home after being given head injury advice. Later in the afternoon he became aphasic with jerky movement of his right hand. At 8pm his mother found him unconscious at home. He was brought to the hospital with GCS of 11/15 (E4V1M6) with equal pupils. Physical examinations were unremarkable. There were no obvious external injuries and no carotid bruit heard. A CT scan of the brain was done and showed no intracranial bleeding. He was admitted to the ward for observation. The following morning his GCS worsened [10/15 (E4V1M5)] and he developed right hemiparesis with Babinski reflex over his right foot which was equivocal. A CT scan of his brain was repeated and it showed acute left middle cranial artery (MCA) infarct. The following day CT angiography showed evolving left MCA infarct with new acute left anterior cranial artery (ACA) infarcted with increased mass effect, cerebral edema and obstructive hydrocephalus. These features are likely to represent left internal carotid artery dissection (ICD) and are possibly complicated by thromboembolism. Antithrombotic medication, tablet aspirin 150mg daily with tablet clopidogrel 75mg daily was commenced and the patient was referred to a neurosurgical centre and a left decompressive craniectomy was done. Conventional angiogram showed dissection of left internal carotid artery. Unfortunately, the patient died after a few days.

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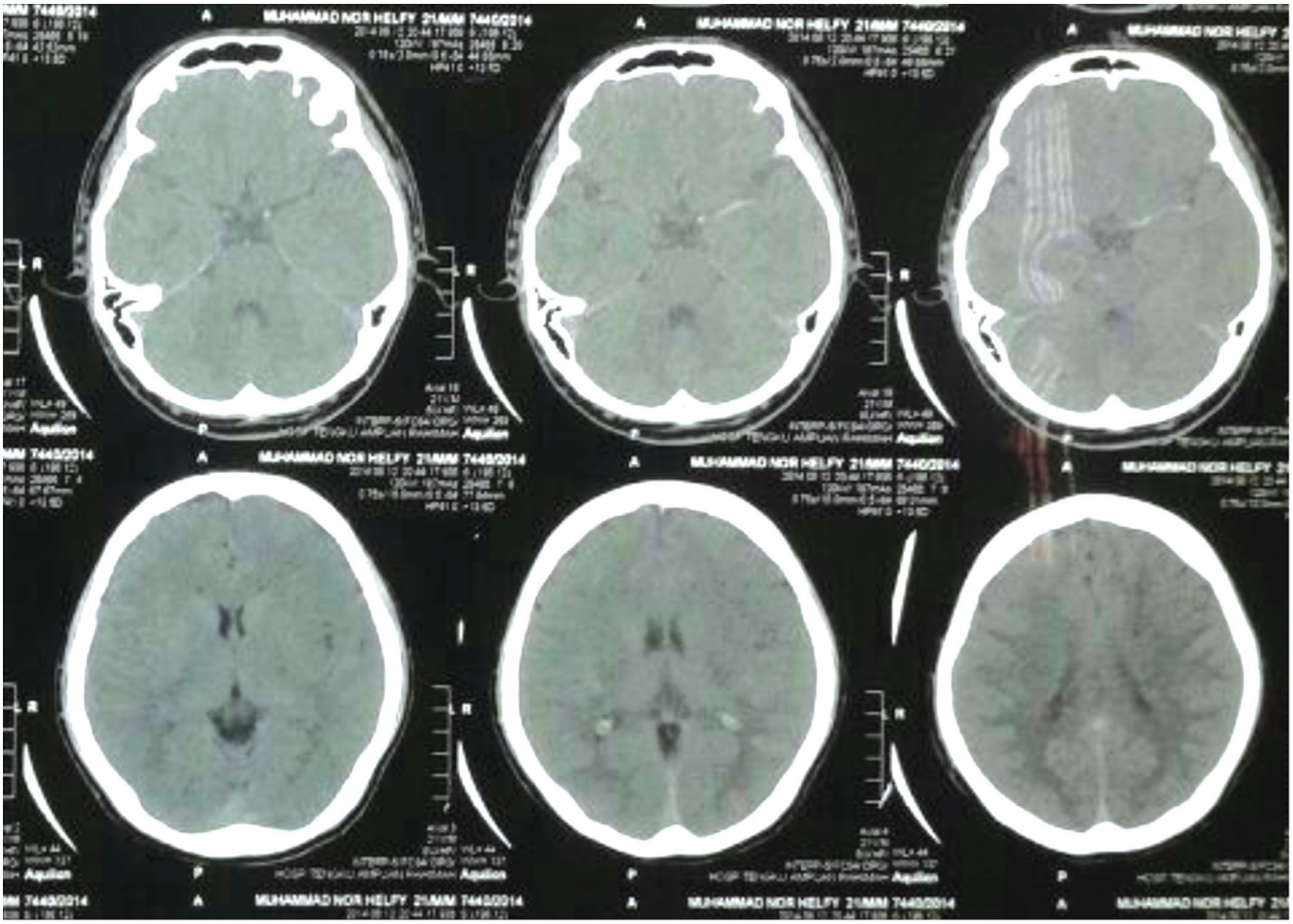

Figure 1: Initial CT Brain showing no ICB

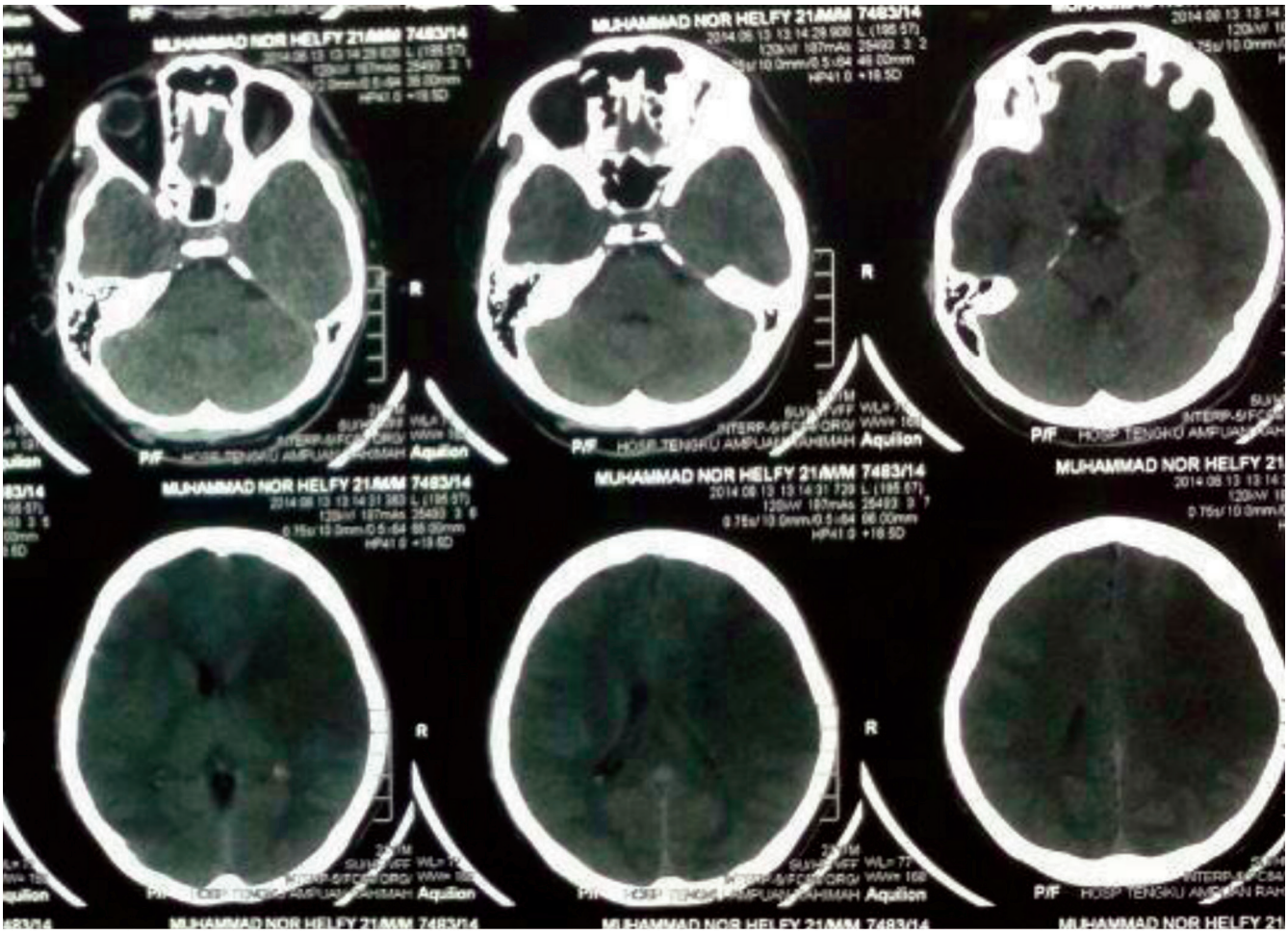

Figure 2: Subsequent CT Brain showing left MCA Infarct

Discussion

Dissection implies a tear in the wall of a major artery with intrusion of blood within the layers of an arterial wall causing intramural hematoma. This causes stenosis of the lumen when blood collects between the intima and media or an aneurysmal dilatation of the artery when the hematoma predominantly involves the media and adventitia[1]. Three basic mechanisms of injury are by extreme hyperextension and rotation, direct blow to the vessel and vessel laceration by adjacent bone fracture[2]. ICD can be further classified into intracranial and extracranial, where extracranial is more common. Commonly ICD occurs 2cm distally to ICA. Only 6-10% have immediate symptoms within the first hour whereas most of them occur within 24 hours (57-73%); a lower percentage of 17-35% will present after 24 hours[3]. General examination would be beneficial if carotid bruit is present with external injury. However injury might be occult. Once neurological findings have been detected such as Horner's syndrome, dysphasia or hemiparesis, we should be more vigilant about other possible injuries. The common cause of stroke is arterial thrombosis resulting in permanent neurological deficits. The grading scale by Biffl et.al.4 holds prognostic value for future risk of stroke. The stroke risk and mortality (%/%) for each grade are as shown in Table 1.

Table 1: Grading scale (Biffl *et. al.*, 1999)[4]

Injury Grade	Description
I	Luminal irregularity or dissection with <25% luminal narrowing
II	Dissection or intramural haematoma with ≥25% luminal narrowing
III	Pseudoaneurysm
IV	Occlusion
V	Transection with free extravasation

Early detections are very important and to avoid further delay, a screening protocol can be used such as the Memphis or Denver criteria as shown in the Table 2. Patients with these criteria should be further investigated.

Table 2: Screening protocols using Denver and Memphis criteria (Arthurs *et al.*, 2008)[2]

Denver Criteria	Memphis criteria
Signs/symptoms	Cervical spine fracture
Arterial hemorrhage or expanding hematoma	Neurological exam not explained by brain imaging
Cervical bruit	Horner's syndrome
Focal neurological deficit	Le Forte II or III fracture pattern
Neurological exam inconsistent with head CT findings	Basilar skull fracture with involvement of the carotid canal
Stroke on follow-up head CT	Neck soft tissue injury (seatbelt sign or hanging or hematoma)

Selective digital subtraction angiography (DSA) is the gold standard for screening patients with suspected blunt neck injury[2]. The limitations of this procedure are that it is invasive with technical limitations and it carries a small risk of stroke. Alternative modalities include Duplex scanning which was compared against cerebral angiography by Guzman[5]. Evaluation of carotid artery stenosis is limited when evaluating lesions with <60% stenosis and when small intimal tears or non-occlusive dissections cannot be identified. MRA is an option as a noninvasive modality as it can show images in this anatomic region, projections of vessel, and is able to assess intracranial for signs of stroke. The main limitation of this modality is that it is time consuming and thus is not suitable in trauma cases. CTA offers advantages over DSA. It is noninvasive and results can be obtained within 5 minutes. It also captures 3-dimensional images of the vessel wall and has a sensitivity of 95% and specificity of 93% for detecting carotid artery stenosis >50%. Several observational and retrospective studies (Denver and Memphis) have compared CTA and MRA to conventional angiography[2]. Using DSA as the "gold standard," CTA has a sensitivity of 47–68% and specificity of 67–99%. MRA sensitivity is 50–75% and specificity is 67–100%6. Both study groups concluded that CTA and MRA are appealing but should only be used when angiography is not available.

The treatment of ICD can either be by medication or surgical intervention. Treatment is to prevent thrombus formation of injured endothelial surface and avoid artery to artery embolism. Anticoagulants and antiplatelet agents are commonly used in the treatment of ICD. Fabian *et.al.* reported improved neurological outcome with early use of antithrombotic therapy[7]. Analysis revealed the benefits of heparin in decreasing rate of neurological deterioration after symptoms developed and decreasing the rate of new neurological events. Heparin therapy showed a dramatic reduction in neurological morbidity and mortality as compared to previously reported rates, as high as 58% and 31% reduction respectively. However, by using anticoagulation, Biffl *et al.* reported the outcome in eight patients with grade IV injuries whereby four of the patients who did not commence with anticoagulation suffered severe neurological deficit while the remaining four patients treated with heparin immediately upon diagnosis survived without deficit[4]. There have been no data to establish the optimal duration of anticoagulation. However, 3 – 6 months is probably suitable in most cases. Antiplatelet agents can be considered as an acceptable alternative when anticoagulation is contraindicated.

For surgical intervention, its requirement is determined by the thrombogenicity of injured carotid artery, state of collateral circulation to brain, presence of expanding haematoma or worsening neurological symptoms despite anticoagulation. Extracranial-intracranial bypass is useful for re-vascularisation. The preferred method is excision of the damaged arterial segment with reconstruction of the ICA to preserve flow. Another intervention is endovascular stenting. Current indications for stent placement include enlarging pseudoaneurysms and dissections that progress and threaten to limit flow despite full anticoagulation. It also prevents late complications. Two important issues for consideration are firstly, acute injured ICA should not be manipulated within 48 – 72 hours due to the risk of stroke from manipulation of catheters in acute injured vessel. The recommendation is to wait 7 days before attempting to stent. Secondly, endovascular stents in traumatised arteries should be treated with full systemic anticoagulation to prevent stent occlusion resulting in stroke. Recent evidence suggests that anti-platelet therapy alone may be adequate to prevent thrombotic complications for carotid artery stents (Singh *et al* 2004)[8].

Long-term follow up demonstrates that anti-thrombotic therapy prevents cerebral infarction; antiplatelet therapy and anticoagulation are equally effective; and carotid stents appear to be safe and effective for lesions that develop pseudoaneurysms or extensive dissections. Edward *et.al.* followed up patients treated

with antithrombotic, antiplatelet and anticoagulation with mean of 34.4 months and endovascular stenting with mean of 29.7 months[9].

Conclusion

Blunt neck injury is a rare complication of trauma but associated with high morbidity and mortality. ICD should be considered in patients after severe trauma presenting with localising signs and normal CT scan. The heterogeneous clinical pictures associated with ICD often leads to delay in diagnosis and treatment. The condition may be asymptomatic or result in minor symptoms, stroke, or even death. It would be beneficial if a screening protocol is followed as it gives a guide towards highly suspicious patients for further evaluation. Early treatment should be started once diagnosis is established.

REFERENCES

1. Thanvi B, Munshi SK, Dawson SL, Robinson TG - Carotid and vertebral artery dissection syndromes. Postgrad Med J 2005; 81: 383-8.
2. Arthurs ZM, Starnes BW. Blunt carotid and vertebral artery injuries. Interventional Radiol Trauma 2008; 39: 1232–41.
3. Greenberg MS. Handbook of Neurosurgery 7th Edition, pp 1499-1500.
4. Biffl WL, Moore EE, Offner PJ, Brega KE, Franciose RJ, Burch JM. Blunt carotid arterial injuries: implications of a new grading scale. J Trauma 1999; 47: 845-53.
5. Guzman R. Controversies in cerebrovascular disease 4. Appropriate imaging before carotid endarterectomy. Can J Surg 1998; 41(3): 218-23.
6. Miller PR, Fabian TC, Croce MA, Cagiannos C, Williams JS, Vang M, et al. Prospective screening for blunt cerebrovascular injuries: analysis of diagnostic modalities and outcomes. Ann Surg 2002; 236(3): 386-93 discussion 393-5.
7. Fabian TC, Patton JH Jr, Croce MA, Minard G, Kudsk KA, Pritchard FE. Blunt Carotid Injury: Importance of early diagnosis and anticoagulant therapy. Ann Surg 1996; 223(5): 513-25.
8. Singh RR, Barry MC, Irelanda A, Hayes DB. Current Diagnosis and Management of Blunt Internal Carotid Artery Injury: European J Vascular Endovascular Surg 2004; 27(6): 577–84.
9. Edwards NM, Fabian TC, Claridge JA, Timmons SD, Fischer PE, Croce MA. Antithrombotic Therapy and Endovascular Stents are Effective Treatment for Blunt Carotid Injuries: Results from Longterm Followup. J Am Coll Surg 2007; 204(5): 1007-13.