Case Report

Delayed Presentation of Post-traumatic Internal Carotid Artery Occlusion “Case Report”

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Abstract

A case of delayed presentation of post-traumatic internal carotid artery occlusion is reported. This rare entity can easily be misdiagnosed because presenting symptoms can be delayed for a long time after trauma. This report aims to highlight the diagnostic clues and treatment options for clinical practitioners.

Keywords: Carotid artery, occlusion

INTRODUCTION

Thrombosis after blunt injury of the internal carotid artery (ICA) is a rare entity, which occurs more often in young individuals. Signs and symptoms of ICA occlusion after trauma may develop hours to weeks. Delayed presentation of this condition can make the diagnose difficult. We present a case of a young man with an ICA occlusion whose presenting symptoms were delayed two weeks time after trauma.

CASE PRESENTATION

A 19 year-old man admitted to our clinic unconscious following epileptic seizure with a history of a blunt trauma to the neck in a basketball game two weeks ago. On examination his Glasgow coma scale (GCS) was 13/15. The first computed tomography (CT) scan was performed immediately and it was normal. Within 30 minutes he developed right hemiplegia and facial paralysis. Repeated CT scan, cranial magnetic resonance imaging (MRI) and carotid artery color doppler ultrasonography were normal. After 6 hours his unconscious recovered (GCS:15), but aphasia was examined. We first thought this condition as Todd paralysis. In the follow up period MRI was performed two days after the onset of symptoms and revealed a large left parietal infarct (Fig.1). On magnetic resonance angiography (MRA) left intracranial ICA wasn't visualised (Fig. 2). Conventional angiography (CA) showed the left ICA occlusion at the level of cavernous segment (Fig. 3). The patient was treated nonsurgically with nadroparine calcium 0.6
ml twice daily, subcutaneously. Physiotherapy was performed apart from the second day. Follow up MRA was performed on the 15th day and showed that the left distal middle cerebral artery was still occluded with adequate collateral circulation via the circle of Willis (Fig. 4). On the 20th day he was discharged with a mild hemiparesia and dysphasia.

**Figure 1:** Axial T2 MRI shows left parietal infarct.

**Figure 2:** MRA demonstrates occlusion of the left internal carotid artery.

**Figure 3:** Conventional left internal carotid angiography demonstrates the absence of flow in the artery. (a) oblique image, (b) town image.

**Figure 4:** Follow up MRA shows still occluded left distal middle cerebral artery with adequate collateral circulation via the circle of Willis.
DISCUSSION

Presentation of ICA occlusion following trauma is highly variable. The time interval between the blunt trauma and the onset of symptoms may vary from 1 hour to as long as 10 years (2). Remote symptoms are invariably related to embolisation from clot formation within a dissection of carotid artery. Neurological deficits occur when there is a thrombosis with lack of collateral flow, or immobilisation from the site of thrombosis or extension of the thrombosis (5).

Diagnosis is made when a high index of suspicion, based on mechanism of injury or symptoms. Physical examination can yield no diagnostic signs about this illness. Magnetic resonance angiography (MRA) can accurately demonstrate carotid occlusion with a high sensitivity and specificity (3). Arteriography is considered the gold standard and should be carried out in patients who develop neurological symptoms that can't be explained by the findings of the brain CT scan after trauma (3).

In our case, despite mimicking symptoms of Todd paralysis and negative findings of CT scan above mentioned tests were insistently performed to clarify the cause of condition.

The treatment recommended for these patients who develop ischaemic symptoms is the early anticoagulation. Because of the high bleeding complication rates we used low molecular weight heparin (LMWH). We think it is useful with its excellent anticoagulating properties and lower bleeding complication profile. Surgical treatment carries high morbidity and mortality. It should be reserved for the minority of patient with deteriorating symptoms despite receiving the best medical treatment (4).

Majority of patients maintain good neurological function with conservative treatment. This may be explained by the absence of atherosclerosis and a good collateral circulation as these patients are young (1).

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