A Rare Case of Traumatic Middle Cerebral Artery Thrombosis Without Internal Carotid Artery Dissection

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Introduction

Trauma is a well-recognised cause of middle cerebral artery (MCA) infarction. Most cases are attributable to dissection of the internal carotid arteries (ICA). Here we describe a rare case of localised M1/M2 thrombosis.

Case Description

A 59 year old male presented with head and neck injuries as a result of blunt head trauma. He was agitated with an oral degloving injury and a deep neck laceration. His Glasgow Coma Scale (GCS) was 12 and he was intubated and ventilated and managed in the intensive care unit (ICU).

Emergency imaging of his head and neck demonstrated skull fractures and extensive soft tissue swelling. There was no evidence of vascular injury or dissection, but calcified plaque was identified at the left carotid bifurcation.

On day 2 of his admission his sedation was weaned and he was noted to be agitated with a dense right hemiparesis. Magnetic resonance (MR) imaging revealed an extensive area of acute infarction in the left MCA territory with associated mass effect. MR Angiography demonstrated partial occlusion of the left MCA at the M1-M2 junction.

The patient was transferred for neurosurgical and stroke care and underwent emergency decompressive hemi craniectomy. He required a long period of inpatient rehabilitation. Residual deficits include severe articulatory and functional dyspraxia, right inattention, reduced upper limb function and spasticity.

MRI Head showing Left MCA infarction (B1000r)

Timeline of Hospital Admission

MRA reconstruction showing occlusion at Left MCA M2 branch with no evidence of dissection of ICA

Discussion

Literature review finds similar case reports of traumatic CMA infarction without internal carotid artery dissection. Proposed mechanisms include localised vessel dissection. Case reports have described microscopic rupture of the intima and elastic lamina at the site of the occlusive thrombus on post-mortem examination (1). One group proposed that this localised vessel dissection could be secondary to the impact of rotational forces applied to the proximal M1 segment where it has a firm connection with the skull base (2). Other theories have included cerebral vasospasm. One small study demonstrated delayed cerebral arterial spasm with transcranial Doppler ultrasound, cerebral blood flow, and angiography in 8/30 patients following closed head injury (3). We propose a third possible mechanism in our patient of embolic infarction, with emboli originating from the Left ICA plaque as a result of direct external trauma.

Conclusion

This case demonstrates the importance of monitoring for stroke syndromes after blunt head trauma.

References

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