

Traumatic Middle Cerebral Artery Occlusion : Case Report and Review of Pathogenesis

R.J. Mobbs, K.N. Chandran

Department of Neuroscience
The Canberra Hospital, Clinical School of the University of Sydney
Woden 2606, ACT, Australia.

Summary

Traumatic occlusion of the middle cerebral artery (MCA) is a rare cause of cerebral infarct. We describe a case of MCA occlusion following blunt head trauma. The literature reports some 65 cases of MCA occlusion following non-penetrating blunt trauma to the head. Arterial dissection, cerebral vasospasm and thrombosis have been some of the theories discussed in the pathogenesis of this condition. We review the pathogenesis based on anatomy of the M1 segment.

Key words : Traumatic occlusion, MCA, Intimal dissection.

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Introduction

Traumatic occlusion of the middle cerebral artery (MCA) is a rare cause of cerebral infarction. Our research finds a total of 65 cases described in the literature. MCA infarct resulting in death of the patient was encountered in 18 of the reported cases, with a mortality rate of 27%. Arterial dissection is one form of traumatic vascular injury, and from observations reported in the literature, the vertebro-basilar arteries are the most frequently involved, followed by the carotid arteries and their branches.¹ Dissection is usually followed by intramural bleeding with the formation of a haematoma in the dissected space.

Case Report

A 15 year old right hand dominant male riding a
Correspondence to : Dr. R.J. Mobbs, 15 Soudan St, Randwick
2031 NSW, Australia.

bicycle fell and hit the road on his left forehead. He was wearing a helmet. He sustained a brief loss of consciousness. On presentation to the emergency department, he became confused, disoriented and combative. Initial head CT and biochemical evaluation was normal. 12 hours following presentation, he was noted to have left sided hemiparesis and was not obeying commands. A Camino (110-4B) intracranial pressure monitor was inserted via a right frontal approach. Initial ICP measurements were between 5-10 mmHg. On day 2, the CT scan noted some mild oedema in the right MCA territory. Cerebral angiography was performed which demonstrated occlusion of the right MCA, several mm from its origin (Fig 1). Other intra and extra cranial vessels were normal. CT on day 4 demonstrated a right MCA infarct (Fig 2). ICP readings of 15-22 mmHg were recorded between 24-72 hours post injury. Cerebral hypertension was medically managed, and the Camino monitor was removed on day 5, following 24 hours of normal ICP

Table I

Traumatic MCA Occlusion : Reports with Autopsy Findings

Author	Age and sex	Injury	Angiographic findings	Autopsy findings
DeVeer and Browder ¹³	42 Male	Fall 7 meters onto head. LOC 20 mins	Not done.	Dissection at origin of M1.
Dratz and Woodhall ²	21 Female	Fall from bicycle. Semicomatose.	Not done.	Dissection of ICA, ACA and proximal 3 cm of MCA.
Linell and Tom ³	58 Male	Fall from a standing height. No LOC.	Not done.	Dissection of proximal 5 mm of MCA.
Verbiest and Calliauw ⁴	67 Male	Ejected from automobile. Brief LOC.	Not done.	Thrombosis of MCA. Precise location not documented.
Duman and Stephens ¹⁴	21 Male	Fall while water skiing. No LOC.	Occlusion of MCA at M1 segment.	Dissection at origin of M1.
Hollin et al ⁵	54 Female	Automobile accident. Coma	Occlusion of MCA at M1 and M2 segment.	Patent MCA (20 days post injury).
Jacques et al ⁶	40 Male	Motorcycle accident. LOC 15 minutes.	Occlusion of MCA at origin of M1.	Emboli in all branches of MCA Mural thrombus in proximal ICA.
Dujovny et al ⁷	62 Male	Fall from horse . carriage. Coma	M1 occlusion.	Not discussed in paper.
Roessmann and Miller ¹⁵	Day 3 following birth.	Failed forceps . delivery & emergency cesarean section	Not done.	Thrombosis at M1 segment 8mm from origin.
Mizutani et al ⁸	19 Male	Fall while water-skiing. No LOC.	Spasm of M1 and M2 with occlusion of distal branches of M2.	Dissection of M1 and M2 segments.
Schmitt and Miltner ¹	19 Male	Repeated kicks to the head.	Not done.	Dissection at origin of M1.
Kojimahara et al ¹⁶	16 Male	Motorcycle accident. Semicomatose.	Tapering occlusion of the ICA at the level of C3.	Dissection at the origin of M1.
Sharif et al ⁹	17 Male	Go-cart accident. Low speed. No LOC.	Occlusion of M1 and A1 segments.	Dissection of M1 segment.
Adams and Trevenen ¹⁰	12 Female	Hit head on coffee table. No LOC.	Not done.	Dissection of M1 segment.
Rutherford et al ¹¹	11 Male	Dive into empty swimming pool. Coma.	Not done.	Dissection of MCA from origin to trifurcation.
	3 Male	Fall off a tractor. GCS 4 on admission.	Not done.	Dissection of MCA from origin to trifurcation.
	17 Female	Fall from a horse.	Not done.	Dissection within distal segment of M1.
De Caro et al ¹²	25 Female	Motor vehicle accident. No LOC.	Not done.	Dissection of MCA 5 mm from origin.

LOC = Loss of consciousness



Fig. 1 : DSA showing occlusion of right MCA, after its origin.

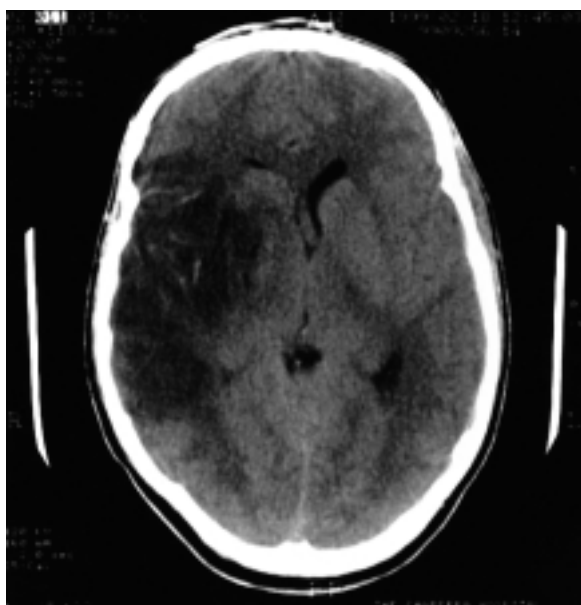


Fig. 2 : Plain CT scan showing infarct in right MCA territory.

tracings. After 6 weeks of rehabilitation, the patient was mobile with a walking stick.

Discussion

Occlusion of the MCA following blunt head trauma is a rare cause of brain infarction. 65 cases of traumatic MCA occlusion are discussed in the literature; 18 were fatal with a resulting mortality of 27.7%. Many possible pathogenic mechanisms have been discussed.¹⁻¹⁶ Review of the 18 autopsy cases (Table I) demonstrates that subintimal dissection is

Table II

Relative Distance of M1 Midpoint to Sphenoid Wing

	Midpoint left M1 segment (mm)	Midpoint right M1 segment (mm)
Shortest distance	5	3
Longest distance	26	17
Average	15.5	10.6

the most likely cause of MCA occlusion, noted in 14 out of 18 cases (78%). 2 cases (11%) involved thrombosis in the artery. There was one case of a patent M1 vessel on angiography several weeks following the event and one case from embolus. It can be concluded that the overwhelming likelihood of MCA occlusion following trauma is secondary to intimal dissection, with primary thrombosis and vasospasm as less likely causes.

Many cases were noted following minor head trauma and a relative younger age group (average 28 years). Minimal trauma was involved in our patient and he was young at 15 years. The most common site of occlusion is the origin of the M1 segment (6 out of 14 cases). This may be due to anatomical features of M1. MCA occlusion may have similar pathology as noted in acute rotational injury of the vertebral artery at the C1-C2 region, leading to intramural dissection, haemorrhage and thrombosis.¹⁷ The M1 segment proximally has a firm connection with the skull base at the carotid termination and rotational forces following blunt trauma could lead to tearing of the arterial wall. The M1 segment runs in close proximity to the posterior margin of the sphenoid wing. Impact of this vessel with the sphenoid could result in arterial dissection or intimal damage resulting in thrombosis. Review of 15 normal MRI/MRA scans of patients aged 22 to 82 years was conducted (Table II). The relative distance of the midpoint of the M1 segment to the sphenoid wing was examined to evaluate the degree of variability in distance. As the table demonstrates, the distance of midpoint MCA to sphenoid ridge varies substantially. On the left side, the closest distance was 5 mm with a maximal distance of 26 mm and an average of 15.5 mm. On the right side, the average mid point distance was 10.6 mm.

Vasospasm as an additional factor in the pathogenesis of MCA occlusion has also been discussed.^{5,9,11,18-20} Spasm following traumatic subarachnoid haemorrhage is most noticeable in the distal portion of the internal carotid artery and the proximal portion

of the MCA and ACA.²¹⁻²³ The reported incidence of intracranial arterial spasm following moderate to severe head injury is in the range of 5 to 10%.²² In a study of 350 patients with moderate to severe head injury, Suwanella²³ found narrowing of the proximal ACA and MCA in 5.1%, with spasm being found from day 1 to day 19. The role of MRA in the diagnosis of vasospasm was discussed by Yamada et al.²⁰

Based on the facts to date, the pathogenesis of traumatic MCA occlusion is primarily due to subintimal dissection and thrombosis. These lesions are due to anatomical and mechanical factors, particular to the M1 segment. In conclusion, these lesions can follow seemingly minimal trauma, be fatal in a young patient population and affect vessels with no underlying abnormality.

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