To do or not to do in an interesting case of stroke

Sir,

A 31-year-old male, not a known diabetic or hypertensive patient presenting with sudden onset alteration of sensorium, 8 hours before presentation which was preceded by tonic clonic movements. There was a history of dizziness and fever for the preceding three days for which he was treated with local medicines from quacks. The patient was initially admitted to a local hospital where he was intubated followed by which he was brought to us.

On initial examination, the patient looked dehydrated which was accompanied with fever, diarrhea, and hypotension. In triage, the patient was having poor Glasgow Coma Scale (GCS) (E2VtM4) status with decreased movement of left side upper and lower limbs. . He was managed initially in triage with IV fluids and antibiotic support and computed tomography (CT) scan was done in view of left hemiparesis. He had to be put on inotropic support. CT (Fig. 1, 2) brain with perfusion showed patchy hypodensity in right lentiform nucleus, external capsule, temporal and parietal cortex.

CT perfusion brain showed mismatched perfusion deficit in right external watershed zone. CT angiobrain (Fig 3) showed occlusion at right M1 segment with presence of leptomeningeal collaterals reforming MCA beyond occlusion and presence of doubtful collaterals in the basal region.

In view of the above, MRI Brain was done in which DWI (Fig 4) showed restriction in patchy areas of right lentiform nucleus, and temporal and parietal cortex.

The patient was rushed to Cath lab for mechanical thrombectomy as per standard stroke management protocol. Under general anesthesia following regular prepping and draping of the right groin, 8F short sheath was placed and Neuron Max 6F 088 was inserted through right femoral artery and parked in the right internal carotid artery (ICA). ICA injection shows occlusion / tight stenosis of M1 segment [1, 2] (Fig 5) with slow anterograde flow along with adjacent leptomeningeal collaterals. However, we advanced ACE 68 into cavernous segment. Reber microwire was negotiated beyond the occlusion site / stenotic site with least resistance and in view of this angiographic finding we concluded that there might be a presence of previous tight stenosis at M1 segment of MCA [3] with slow anterograde flow with basal collaterals suggestive of Moya Moya disease [4]. We abandoned the procedure as doing a mechanical

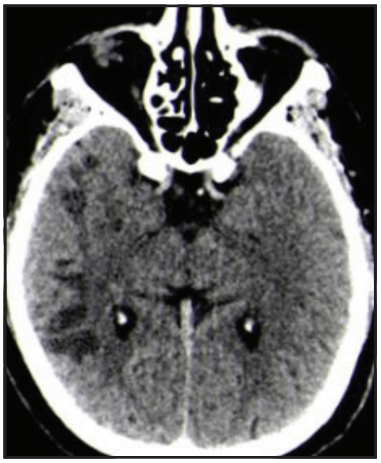

Figure 1: CT brain showing patchy infarcts in the right MCA territory.

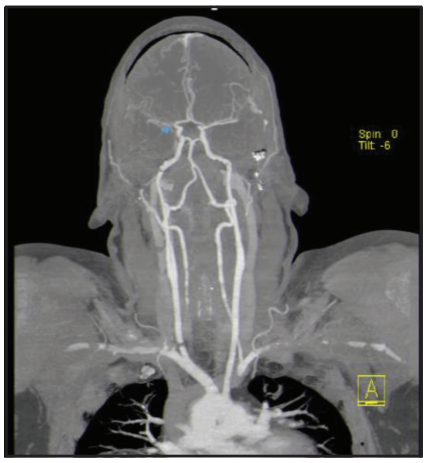

Figure 3: CT angiography showing loss of signals beyond proximal M1 segment of MCA.

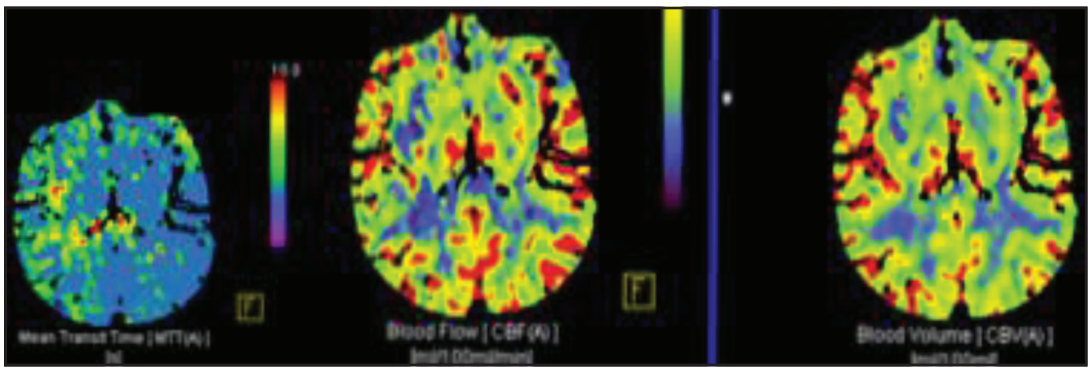

Figure 2: CT perfusion image showing patchy areas of decreased perfusion.

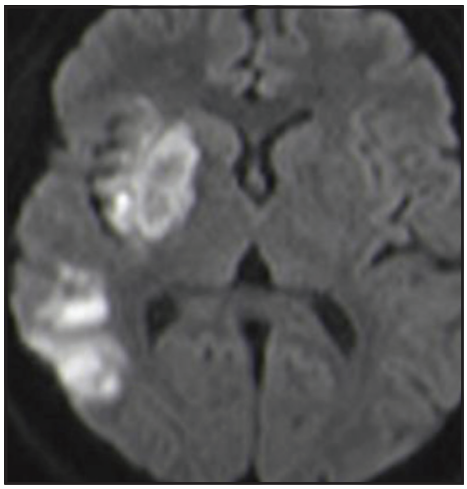

Figure 4: MRI DWI Brain showing restriction in areas of infarct.

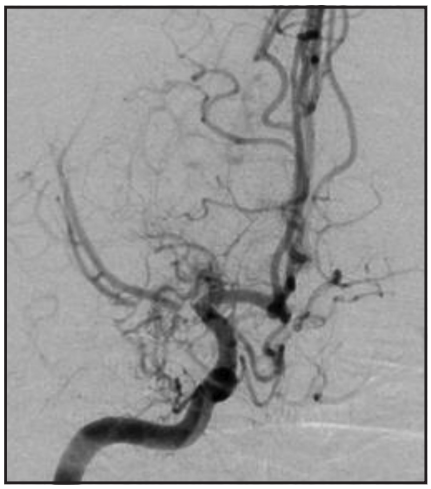

Figure 5: DSA image in right ICA injection showing right MCA M1 segment chronic occlusion with basal collaterals.

thrombectomy in an underlying vasculitic state was not justified at that time.

The patient was shifted in ICU for further conservative management. Next morning, he became E4M6Vt status after fluid management and correction of sepsis with antibiotics. No revascularisation procedure was done and the patient improved on medical management with correction of shock and maintains good mean arterial pressure.

The question in this case is whether to at all go for mechanical thrombectomy in this case. Considering the urgency of stroke management a propensity towards opening the occlusion found in CT angiography with infarcts documented in DWI was there but was abandoned in view of the chronic nature of the occlusion.

Thus, it was hypothesised that the mechanism leading to the stroke was hypotension due to sepsis. Further, which lead to hypoperfusion of right MCA territories [3] due to chronic occlusion of right M1 MCA segment [1,2]. Hence sepsis management and fluid reperfusion led to recovery of the patient.

Learning point in this case is that one should always try to assess the physiology behind the stroke for its treatment. As in this case, it led to better outcomes and might include restraining oneself from the normal protocol of stroke management. Though, this should be assessed in an individual on case by case basis.

Apratim Chatterjee[1], Anshu Mahajan[2], Vinit Banga[1], Gaurav Goel[3]

From [1]Fellow, [2]Consultant, [3]Consultant and Head of Neurointervention, Department of Neurointervention, Medanta, Medicity, Gurgaon, India.

Correspondence to: Dr. Apratim Chatterjee, Department of Neurointervention, Medanta, Medicity, Gurgaon, India. E-mail: babaiapratim@gmail.com

Received - 08 September 2019
Initial Review - 25 September 2019
Accepted - 01 October 2019

REFERENCES

1. Lhermitte F, Gautier JC, Derouesne C. Nature of occlusions of the middle cerebral artery. Neurology. 1970;20:82-8.
2. Wong KS, Li H, Lam WW, Chan YL, Kay R. Progression of middle cerebral artery occlusive disease and its relationship with further vascular events after stroke. Stroke. 2002;33:532-6.
3. Caplan LR, Wong KS, Gao S, Hennerici MG. Is Hypoperfusion an Important Cause of Strokes? If So, How? Cerebrovasc Dis. 2006;21:145-53.
4. Bang OY, Ryoo S, Kim SJ, Yoon CH, Cha J, Yeon JY, *et al*. Adult Moyamoya Disease: A burden of intracranial stenosis in East Asians? PLoS One. 2015;10:e0130663.

Funding: None; Conflict of Interest: None Stated.

How to cite this article: Chatterjee A, Mahajan A, Banga V, Goel G. To do or not to do in an interesting case of stroke. Indian J Case Reports. 2019;5(5): 505-506.

Doi: 10.32677/IJCR.2019.v05.i05.035