Posterior Circulation Approach for Anterior Circulation Thrombectomy in a Patient with Dysgenetic Internal Carotid Artery

INTRODUCTION

Hyperacute ischemic stroke from emergent large vessel occlusion (ELVO) is increasingly being treated with mechanical thrombectomy, which leads to significantly improved recovery compared to other treatment options.[1] However, establishing access to intracranial proximal occluded arteries within the time window can be a challenge at times. Apart from age-related and atherosclerotic changes, conditions like fibromuscular dysplasia in the neck vessels producing vessel narrowing, which are common difficulties in the access, congenital anomalies may significantly hinder intracranial arterial access in rare cases for which the interventionist may need to rethink and improvise the standard approach.

Dysgenesis of internal carotid artery (ICA) is a developmental anomaly comprising agenesis, aplasia, or hypoplasia of the artery. Its prevalence is less than 0.01% of the population.[2‑5] Although the diagnosis can be established by identification of narrow ipsilateral bony carotid canal of the petrous temporal bone,[6] which confidently differentiates this rare congenital anomaly from collapsed ICA due to intracranial terminal carotid occlusion, it may present a significant problem during the procedure for thrombectomy as access is not feasible. Interestingly, the usually prominent embryonic collaterals in these individuals can be utilized as alternative conduits in these scenarios. We describe a rare and interesting case of mechanical thrombectomy in the left proximal middle cerebral artery (MCA) occlusion in a patient with a hypoplastic variant of ipsilateral internal carotid artery (ICA) dysgenesis, which rendered the access difficult. To add to the problem, the right ICA also showed stenosis at the origin from calcific plaques with relatively reduced distal caliber, making access via anterior communicating artery (ACOM) challenging.

CASE REPORT

A female in her 80s presented with acute onset right‑sided weakness and confusion for 2 h after the onset of symptoms. She had no significant past medical history. On examination, she had right hemiparesis, loss of speech, and the National Institute of Health Stroke Scale (NIHSS) score was 19. CT Brain showed [Figure 1] early ischemic signs in the left frontal lobe involving the MCA distribution and showed hyperdensity involving the left proximal MCA. CT Angiogram neck and brain showed [Figure 2] left MCA occlusion in distal M1/proximal M2 segment. It showed ICA with very thin caliber from the origin and was not visible after the paraclinoid segment. There was a prominent left posterior communicating artery (PCOM) visualized on both sides with both the vertebral artery (VA) showing prominent flow. In addition to the presence of emergent large vessel occlusion (ELVO), the systolic blood pressure at presentation was above 200 mm Hg despite intravenous labetalol and hence thrombolysis was avoided, and she was taken up for mechanical thrombectomy under anesthesia.

The procedure was performed under general anesthesia in a Biplanar cath lab, and an 8F short sheath was placed in the
Figure 1: CT brain and CT Angiogram Neck and brain at presentation (a) Coronal reconstructed unenhanced CT Brain; arrow shows hyperdense left MCA suggesting thrombus. (b) Axial unenhanced CT Brain arrow shows obscured left insular rim suggestive early ischemic changes in the left MCA territory. (c) Axial Maximal intensity projections of CT angiogram brain, arrow shows left proximal MCA occlusion at M1 segment. (d) Axial Maximal intensity projection of CT angiogram brain, arrow shows a paucity of blood vessels in left MCA territory. (e) Bone window axial image of the CT angiogram brain, arrow shows narrow bony canal of left ICA, suggesting hypoplastic left ICA. (f) Maximal intensity projection of CT angiogram brain axial image arrow shows prominent left PCOM artery which contributed to left-sided anterior circulation.

Figure 2: Digital subtraction angiogram during the thrombectomy (a) Lateral Neck acquisition of left CCA injection, arrow shows hypoplastic left ICA uniformly thin starting from the origin. (b) Lateral Head acquisition of left CCA injection, arrow shows uniformly thin hypoplastic left ICA terminating in the left ophthalmic artery. (c) Left VA injection, arrow shows cut off at the level of left proximal MCA with opacification of left intracranial ICA via left PCOM. (d) Injection via Sophia 6F catheter with the tip at the basilar bifurcation, arrow shows the abrupt cut off at left proximal MCA, double arrow shows solitaire stent placed in the occluded segment. (e) Left vertebral artery injection post thrombectomy, double arrow shows recanalized M1 segment of the left MCA and single arrow shows recanalized left middle cerebral artery bifurcation. (f) Left vertebral artery injection post thrombectomy, single arrow shows filling of distal branches of the left middle cerebral artery with TICI3 recanalization.
right common femoral artery (CFA). A 6F Neuron max long sheath over 6F neuron select SIM curve catheter (Penumbra, Inc., Alameda, CA, USA), and a 0.035 Radifocus standard guidewire (Terumo) was placed in the left mid common carotid artery (CCA), and an angiogram was performed. It confirmed that the left ICA was of uniform thin caliber starting from the origin, the bone window showed a narrow left bony carotid canal which confirmed that we were dealing with a congenitally hypoplastic left ICA and not a collapsed artery secondary to distal occlusion. Realizing this, we immediately changed our approach and placed the 6F neuron max long sheath using the same technique in the left VA at the distal V2 segment. A 6F Sophia plus aspiration catheter (Microvention Costa Rica SRL, Alajuela, Costa Rica) over Radifocus 0.035 standard guidewire was placed initially to the left VA up to the V3 segment. Through this, we advanced a Headway 27 microcatheter (Microvention, Inc., Tustin, CA, USA) over 0.014 traxcess microguidewire (Microvention, Inc., Tustin, CA, USA), sequentially via the basilar artery (BA), the P1 segment of the left posterior cerebral artery (PCA), the left PCOM, the left ICA, and then to left MCA. This circuitous route [Figure 3] allowed us to bypass the dysgenetic left ICA and provided direct access to the thrombus. A Solitaire Platinum Revascularization device 4 × 20 mm (Micro Therapeutics, Inc., Irvine, CA, USA) was deployed in the occluded segment along with continued aspiration via the Sophia plus catheter using an Aspiration pump (Penumbra INC., Alameda, CA, USA). We performed two passes and achieved recanalization of the occluded segment which scored 3 in the Thrombolysis in Cerebral Infarction (TICI) scale. The result was achieved at 4 h and 30 min after the symptom onset, and the puncture to recanalization time was 75 min. The patient improved to NIHSS 11 at 6 h postprocedure, and follow-up CT at 36 days showed minimal cortical damage [Figure 4]. Her modified Rankin Scale (mRS) at 30 days was 3.

**Discussion**

Dysgenetic ICA is usually asymptomatic as intracranial circulation is taken over by embryonic collaterals. They may
be via the Circle of Willis via ACOM or PCOM (as in our case), or via enlargement of the external carotid artery (ECA) to ICA anastomosis across the skull base.[7]

Narrow ICA is a hindrance for intervention with no direct access to anterior circulation arteries. However, the congenital variant must be differentiated from the collapse of the ipsilateral proximal ICA, secondary to a major intracranial occlusion. This is because in secondary collapse, the intervention route can still be through the collapsed carotid; but in the congenital variant, the artery will not be of sufficient caliber to allow safe passage of the aspiration catheter and the stent.

After visualizing the narrow-left ICA starting from the origin and continuing without any change of caliber, we suspected arterial dysgenesis, confirmed by CT Angiogram bone window. Also in an angiogram, the ICA was uniformly thin smooth caliber from the origin which is seen in the dysgenetic artery, whereas in pathological narrowing it is not usually uniformly thin. It led to approach the MCA occlusion via posterior circulation route as CT angiogram showed good caliber of bilateral VA and a prominent left PCOM. The other potential route although more technically challenging, could have been approaching via the right ICA and then across the ACOM reaching left anterior circulation. However, the right ICA also showed stenosis at the origin of calcific plaques, making access via ACOM challenging. Hence, the left VA–BA–PCA P1 segment – PCOM – ICA – MCA route appeared to be less technically challenging due to good caliber of vessels and less challenging vascular angles.

After a literature search, we found only a single case report which demonstrated a similar approach in an individual with dysgenetic ipsilateral ICA.[9] In a different case series, Ozdemir et al.[10] performed three thrombectomies using a similar route in patients with tandem carotid-middle cerebral occlusions. Hui et al.[10] reported a similar approach in one patient to recanalize MCA with age indeterminate occluded cervical ICA. Hence, an innovative approach in the cathlab may save lives in ELVO.

**Conclusion**

Carotid dysgenesis may mimic carotid collapse secondary to occlusion, and other conditions of carotid narrowing can be differentiated by the narrow bony carotid canal in CT scan and by uniform and smooth caliber in the angiogram. Endovascular access to anterior circulation is tricky when the carotid is dysgenetic. In dysgenetic carotid, anterior circulation thrombectomy can be performed by using embryonic collaterals.

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**Conflicts of interest**

There are no conflicts of interest.

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