Dominant Fronto-temporal Lobectomy for Refractory Intracranial Hypertension following an Acute Arterial Ischemic Stroke in a Child

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Abstract

Fronto-temporal lobectomy for refractory intracranial hypertension following an acute arterial ischemic stroke in a child is rarely performed following failed conventional measures including decompressive craniectomy. We present a case of a 10-year-old child who presented with acute ischemic stroke with intractable cerebral edema and failed conventional measures including decompressive craniectomy and had significant neurological recovery following frontotemporal lobectomy.

Keywords

- frontotemporal lobectomy
- stroke in a child
- decompressive craniectomy
- acute ischemic stroke
- refractory intracranial hypertension.

Introduction

Acute ischemic stroke (AIS) in a child is a rare medical emergency with an incidence of 2 to 3 per 100,000.[1] In 7% to 20% of patients with large hemispherical strokes, deterioration has been associated with secondary cerebral edema.[2] Intractable intracranial hypertension and transtentorial herniation after AIS are rare and associated with extremely poor outcomes. In some patients, conventional modalities such as the evacuation of intracranial hematoma, osmotic diuresis, cerebrospinal fluid drainage, hyperventilation, and barbiturate-induced coma fail to control intracranial hypertension. The survival and functional outcomes in these patients are grave, with a mortality of up to 86%.[3] Here in, we are reporting a 10-year-old young boy who had benefitted from frontotemporal lobectomy for AIS with intractable cerebral edema following failed conventional measures including decompressive craniectomy.

Case Report

A 10-year-old right-handed boy presented to our hospital with a sudden deviation of mouth to one side while at school followed by right-sided hemiparesis. A non-contrast computed tomography (NCCT) done at a local hospital was suggestive of a well-developed left middle cerebral artery (MCA) territory infarct involving the anterior division of the left MCA. MRA (magnetic resonance angiography) showed focal segment narrowing and decreased signal in left MCA branches. He presented to us after 7 hours of the onset of symptoms (►**Fig. 1A,B**).

At presentation, he was irritable, and dysphasic with spontaneous eye opening, right upper motor neuron

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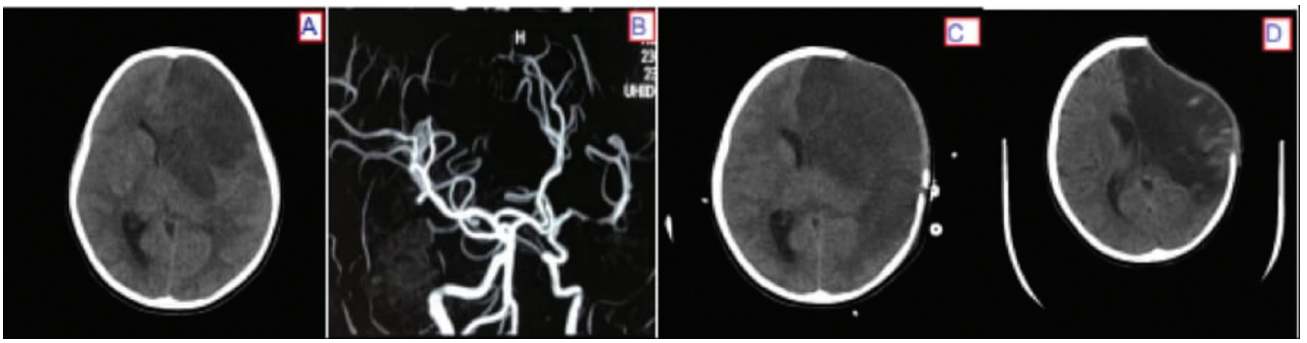

Fig. 1 Axial NCCT head showing left frontotemporal infarct (A), MR angio brain showing left MCA proximal stenosed segment (B), axial post-op CT head (C), and post-op axial CT (D).

(UMN) facial paresis, and right hemiparesis with the power of grade 0/5 in the upper limb and of 3/5 in the lower limb. He was started on aspirin and managed conservatively. Over the next 24 to 48 hours, he had deterioration in the sensorium with pupillary asymmetry and hypotension requiring ventilatory support and the use of vasoactive agents to maintain cerebral perfusion pressure. All neuroprotective strategies were undertaken, including sedation, hyperosmolar therapy with hypertonic 3% saline infusion, adequate analgesia, and maintenance of normocarbia, normothermia, and normoglycemia. NCCT head showed an increase in left MCA infarct with midline shift. Despite escalating antiedema measures, his condition worsened.

He was kept adequately ventilated and sedated. Antiedema measures were maximized but he continued to deteriorate, neurologically as well as hemodynamically. The requirements for vasoactive agents continued to worsen.

The pupils became asymmetric and the optic nerve sheath diameter (ONSD) measured was 0.66 cm bilaterally, suggestive of raised intracranial pressure. ONSD is used as a surrogate for direct intracranial pressure measurement. He underwent left fronto-temporo-parietal decompressive craniectomy with lax duroplasty. Intraoperatively, the brain was tense, pale in color, and had feeble pulsations.

Postoperatively, for the next 2 days, he was continued on ventilatory support, sedation, and anti-edema measures. A CT head done showed a persistent mass effect (►**Fig. 1C**). Although pupillary asymmetry improved and ONSD decreased to 0.45 cm, he remained hemodynamically unstable, requiring high doses of noradrenaline and titrated hypertonic saline infusions (with serum osmolality of 338 mOsm/kg and serum Na 156 meq/L).

On day 4, he again showed signs of worsening with bradycardia and pupillary asymmetry with increasing ONSD (0.68 cm). CT head done was suggestive of worsening left MCA territory infarct and midline shift. A decision of left frontotemporal lobectomy as a life-saving measure to control intracranial pressure was made. The parents were counseled and risks of speech and memory disturbance following dominant lobe lobectomy were explained. He then underwent re-exploration of the left fronto-temporo-parietal flap, opening of dura followed by frontal and temporal lobectomy. Intraoperatively, the brain was tense with no pulsations. Landmarks for frontal lobectomy included posterior corticectomy limit of 4 cm from frontal pole, inferiorly involving middle frontal gyri, and medially involving flax. Partial temporal lobectomy involved, a posterior corticectomy incision at the level of middle temporal gyri, 4 cm from the temporal tip on the dominant side. Medial temporal resection extended up to the temporal horn and inferiorly exposing tentorium respecting the arachnoid layer. The resection was limited at the point where brain tissue was felt to be lax on inspection considering the lobectomy landmarks. Following decompression, the brain was fairly lax with persistent bulge with feeble pulsations.

After 3 days of the second surgery, his requirement for ventilatory support decreased. His blood pressure improved and noradrenaline was gradually tapered off. His pupils became equal in size and his ONSD was 0.39 cm. His sensorium gradually improved and had spontaneous eye opening and started localizing to pain on the right. Tracheotomy was performed during his hospital stay and was gradually weaned off from ventilator support on day 7 post-surgery.

His pro-thrombotic workup, which included protein C and S, homocysteine, factor V Leiden mutation, anti-phospholipid antibody, prothrombin gene, and antithrombin were found to be negative. Echocardiography and doppler studies of neck vessels were normal. The blood and urine cultures were sterile. The anti-neutrophil cytoplasmic antibody (ANCA) and antinuclear antibodies (ANA) were negative. Though a definitive cause of focal narrowing could not be identified, vasculitis idiopathic or infective could most likely be the etiology.

On follow-up at 2 months, he was alert, obeying simple commands with improving right hemiplegia. Cranioplasty using an autologous bone graft was done after 3 months of discharge. After 4 months, he was dysphasic, able to walk with support with right hemiparesis of grade 3/5. He could do most of his day-to-day activities. His follow-up NCCT head prior to cranioplasty procedure showed lax flap with decreased mass effect (►**Fig. 1D**). Extended Glasgow outcome scale (eGOS) at 3 and 6 months post-surgery were 5 and 6, respectively.

Discussion

Standard therapeutic options for patients with non-traumatic intracranial hypertension include evacuation of

intracranial mass lesions, hyperosmolar therapy, drainage of cerebrospinal fluid, hyperventilation, and barbiturate-induced coma. Some patients develop intractable intracranial hypertension that does not respond to any of these interventions. The survival and functional outcomes in this group are poor. Eisenberg et al reported a mortality rate of 86% in patients with intractable intracranial hypertension who failed to respond to conventional treatment.[3] Decompressive craniectomy has been shown to have favorable outcomes in 62% of such pediatric patients with refractory intracranial hypertension in a meta-analysis.[4]

A more aggressive approach for refractory intracranial pressure is brain lobectomy. The experience with brain resections is very limited, and a few publications include only a small number of cases of traumatic brain injury in adults.[5–8] Nussbaum et al reported 10 patients with temporal lobectomy for posttraumatic injury with a survival of 70% with acceptable functional outcomes.[5] In their study Lee et al from Taiwan showed a mortality of 56% and 8% in the two groups undergoing craniectomy and debridement and craniectomy and temporal lobectomy, respectively.[6] The mean GOS (Glasgow outcome scale) was 2.2 ± 0.4 and 4.0 ± 0.4 after 24 to 36 months of follow-up, respectively. The authors concluded that aggressive lobectomy had better survival and functional outcomes.

Oncel et al reviewed 183 patients over 13 years who underwent lobectomies, frontal, temporal, or combined.[8] Forty-eight percent of the 133 survivors had good functional outcomes and 51.9% had poor functional outcomes (including 15% with a persistent vegetative state).

Partial lobectomy for a patient with intractable intracranial hypertension not responding to decompressive craniectomy should be considered as an option in severe refractory intracranial hypertension. Our patient improved well after craniectomy, followed by frontotemporal lobectomy and has recovered well on follow-up.

Conclusion

Severe refractory intracranial hypertension secondary to acute ischemic stroke is a challenging condition. Partial lobectomy should be considered in the extreme case where all routine measures including surgical decompressive craniectomy fail to control intracranial hypertension.

Conflict of Interest
None declared.

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