

Case Report

Decompressive craniectomy in herpes simplex encephalitis: a case report

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ABSTRACT

Herpes Simplex Encephalitis is the commonest form of sporadic encephalitis. Availability of effective antiviral therapy viz Acyclovir has significantly reduced the mortality of Herpes Simplex Encephalitis. Elevated intracranial pressure resulting in herniation syndromes continues to be an important cause of mortality. Antiviral therapy and medical measures for managing raised intracranial pressure including osmotic diuretics, careful usage of steroids and controlled hyperventilation continue to be the cornerstones in management of these patients. Authors present a 38-year-old male patient with Cerebrospinal fluid Meningo-encephalitic panel positivity for herpes simplex virus 1 and bilateral temporal lobe lesions with secondary decline due to impending herniation syndrome despite osmotic diuretics and steroids with patient survival and complete recovery following decompressive hemicraniectomy.

Keywords: Encephalitis, Hemicraniectomy, Herpes simplex

INTRODUCTION

Herpes simplex encephalitis is the commonest form of sporadic encephalitis world-wide accounting for 10-20% of all cases of encephalitis. Herpes Simplex Virus-1 encephalitis is caused by Herpes simplex virus in 90% of the cases with 10% being caused by Herpes Simplex virus-2.

Mortality from HSV encephalitis has reduced from 70% to 5-15% after the availability of effective anti-viral treatment.¹ Mortality despite effective anti-viral therapy may result from fulminant cerebral edema with increased intracranial pressure resulting in herniation syndrome.² Herniation syndromes are commonly seen in patients with large areas of unilateral disease, those with bilateral disease or in patients with intracranial hematoma

complicating the encephalitic zone. Decompressive hemicraniectomy is still not an established modality of management for these patients.

Authors report a case of HSV encephalitis with bilateral hemispherical involvement with impending herniation syndrome who was managed with decompressive hemicraniectomy.

CASE REPORT

A previously healthy, 38-year-old male, developed 2 days of fever, headache followed by development of a confused state within 12 hours of onset of fever and rapid decline of sensorium over the next 48 hours. Examination at admission revealed a stuporose patient with flexor response in all 4 limbs and extensor plantars with a

temperature of 100°F. MRI Brain (Figure 1) revealed evidence of right mesial temporal, bilateral insular, anterior basifrontal and cingulate gyrus areas of restricted diffusion with hyperintense FLAIR signal without any enhancement.

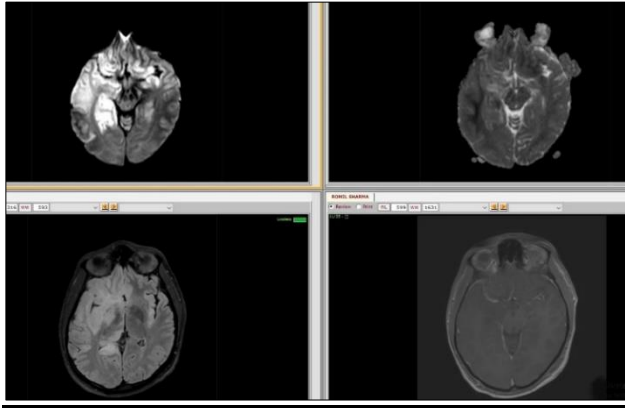


Figure 1: Right mesial temporal, bilateral insular, anterior basifrontal and cingulate gyrus areas of restricted diffusion with hyperintense FLAIR signal without any enhancement.

MR venogram study did not reveal any evidence of sinus venous thrombosis. CSF examination revealed protein 151.6, sugar 89 with 80 cells (100% lymphocytes). Meningo-encephalitis PCR panel was positive for HSV-1. Patient was managed with intravenous acyclovir, osmotic diuretics, anticonvulsants and other supportive measures. Patient developed secondary decline in neurological status on Day 4 with central neurogenic hyperventilation and decline in motor responses following which patient was electively intubated and ventilated and intravenous dexamethasone was added. CT scan (Figure 2) revealed hypodensity in right (>left) temporo-parietal region, insular cortex, basifrontal lobes with 4mm midline shift to the left.

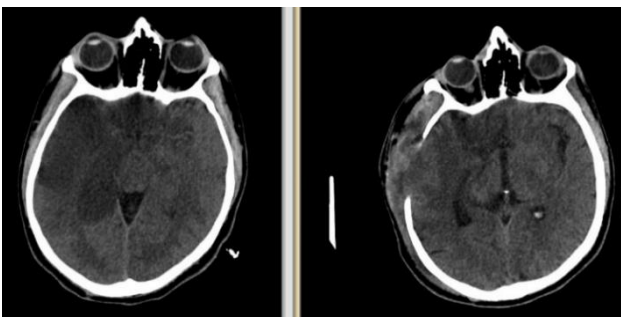


Figure 2: Hypodensity in right (>left) temporo-parietal region, insular cortex, basifrontal lobes with 4mm midline shift to the left (pre-op and post-op).

Patient developed further decline on Day 5 with development of mild anisocoria and underwent urgent decompressive hemicraniectomy with lax duroplasty without any lobectomy. Patient showed gradual

improvement in sensorium 1-week post-decompressive hemicraniectomy and was weaned off ventilator support over the next 10 days. Patient had gradual and complete recovery of his neurological deficit including cognitive functions and underwent reconstruction cranioplasty 3 months later with NCCT Head revealing replaced cranial flap on the right with gliosis in right temporo-parietal region, bilateral insular cortex (right>left), bilateral antero-inferior frontal lobe (Figure 3).

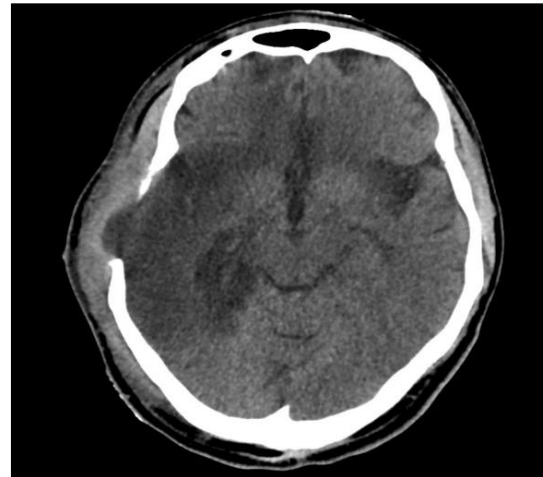


Figure 3: Replaced cranial flap on the right with gliosis in right temporo-parietal region, bilateral insular cortex (right>left), bilateral antero-inferior frontal lobe.

DISCUSSION

Skull trephination, which was a primitive form of decompressive craniectomy, was well described in the Greek era by Hippocrates.³ The first scientific reference and description of an hemicraniectomy was reported in 1896 by Charles Adrien Marcotte in his graduation thesis in medicine and surgery.⁴ The use of large decompressive craniectomy for patients with raised intracranial pressure following traumatic brain injury (TBI) was first reported by Kocher in 1901.⁵

Decompressive hemicraniectomy has been effectively used in patients with refractory intracranial hypertension following Traumatic Brain Injury (TBI) and in Strokes with malignant infarction of the MCA with high grade evidence of efficacy and has been used in certain indications with limited evidence of efficacy including vasospasm following SAH, hypertensive bleeds and cerebral venous thrombosis.⁶⁻¹¹ The evidence for decompressive hemicraniectomy in patients with fulminant increased intracranial pressure in patients with encephalitis is extremely poor and is limited to a few case reports.¹²⁻¹⁴

Acyclovir improves the outcomes achieved by HSV encephalitis and the Infectious Diseases Society of America (IDSA) recommends 2-3 weeks of acyclovir at a

dosage of 10 mg/kg/8hour.¹⁵ Aggravation of cerebral edema, formation of new intracranial hemorrhage or rapid increase in the encephalitic brain tissue may result in worsening of initial manifestation or appearance of new deficits. Medical measures to manage this increase in intracranial pressure include osmotic diuretics, steroids and hyperventilation. Some patients may deteriorate despite initiation of these medical measures as seen in our patient. Decompressive hemicraniectomy may help these patients by reducing the intracranial pressure, improving the cerebral perfusion and preventing herniation syndrome. There is lack of evidence for utilization of decompressive craniectomy in these patients of HSV encephalitis and the available evidence is limited to case reports.^{1,12-14}

Patient underwent decompressive hemicraniectomy without any lobectomy after developing life threatening impending herniation syndrome despite aggressive medical measures and recovered with no neurological deficit. It was suggested that suggest that surgical decompressive hemicraniectomy maybe considered in patients with severe HSV encephalitis who are deteriorating on account of fulminant increase in intracranial pressure despite aggressive medical measures.

CONCLUSION

Secondary decline in patients of herpes simplex encephalitis may occur on account of nonconvulsive status epilepticus, hemorrhagic transformation or fulminant increase in intracranial pressure resulting in herniation syndrome. It is important for the clinician to detect this decline in neurological status and to urgently identify the cause. Timely identification of fulminant elevation of intracranial pressure and prompt institution of medical measures is required. Patients who continue to worsen due to elevated intracranial pressure may be taken up for decompressive hemicraniectomy.

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Ethical approval: Not required

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