Emergency decompressive craniectomy for fulminating infectious encephalitis

Report of two cases

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In patients who develop fulminant cerebral edema and elevated intracranial pressures, viral encephalitis can result in devastating neurological and cognitive sequelae despite antiviral therapy. The benefits of decompressive craniectomy, if any, in this group of patients are unclear. In this manuscript, the authors report their experience with 2 patients who presented with herpes simplex virus requiring surgical decompression resulting in excellent neurocognitive outcomes. They also review the literature on decompressive craniectomy in patients with fulminating infectious encephalitis.

Four published articles consisting of 13 patients were identified in which the authors had reported their experience with decompressive craniectomy for fulminant infectious encephalitis. Herpes simplex virus was confirmed in 6 cases, Mycoplasma pneumoniae in 2, and an unidentified viral infection in 5 others. All patients developed clinical signs of brainstem dysfunction and underwent surgical decompression resulting in good (Glasgow Outcome Scale [GOS] Score 4) or excellent (GOS Score 5) functional recoveries.

The authors conclude that infectious encephalitis is a neurosurgical disease in cases in which there is clinical and imaging evidence of brainstem compression. Surgical decompression results in excellent recovery of functional independence in both children and adults despite early clinical signs of brainstem dysfunction.

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Key Words • decompressive craniectomy • herpes simplex virus • infectious encephalitis

Infectious encephalitis can result in devastating neurological sequelae caused by edematous and hemorrhagic necrotizing cerebral lesions characteristic of these viral illnesses. The reported morbidity and mortality rates can be as high as 70% without medical treatment, but they can be reduced to 30% when treated with antiviral agents. Some physicians advocate aggressive medical management in cases of severe cerebral edema including ICP monitoring, external ventricular drainage, steroids, hyperosmolar therapy, and hyperventilation; however, in the absence of any randomized clinical trials, the utility of surgical intervention remains uncertain.

In this manuscript, we review the contemporary literature on decompressive craniectomy for the treatment of infectious encephalitis and report our experience with 2 patients with HSV encephalitis who underwent decompressive hemicraniectomy, anterior temporal lobectomy, and dural augmentation for fulminant cerebral edema and early brainstem dysfunction.

Case Reports

Our experience with infectious encephalitis includes 2 patients with HSV who required emergency decompressive hemicraniectomy, anterior temporal lobectomy, and dural augmentation after presenting with fever and headache. Despite a brief interlude of aggressive medical ICP therapy, both patients subsequently experienced a rapid deterioration in their neurological condition (< 48 hours after admission) due to temporal lobe edema and uncal herniation (ICP > 20 cm H₂O) (Fig. 1). In both cases, we resected the anterior 4 cm of the temporal lobe and placed an artificial dural graft substitute (DuraGen) over the cortical surface prior to closing the scalp. The temporal lobectomies were performed in both patients to decompress the temporal fossa and midbrain because the lobes were very edematous and herniating rapidly out of the craniectomy site at the time of surgery. The 6-month follow-up in both of these...
patients was excellent (GOS Score 5), suggesting that decompressive craniectomy for infectious encephalitis is beneficial in patients with early signs of brainstem dysfunction.

**Discussion**

Using the Medline search engine, we searched the National Library of Medicine database for reports and clinical studies in which authors evaluated the efficacy of decompressive craniectomy in patients with infectious encephalitis. The search was performed with the dates “1965 to present” and the keywords, “encephalitis” and “craniectomy,” “hemicraniectomy,” “herniation,” “operation,” or “surgery.”

We identified 4 reports (13 patients) in which the authors discussed the usefulness of decompressive craniectomy for fulminant infectious encephalitis (Table 1). There were 11 adults and 2 children (< 18 years old). Herpes simplex virus was confirmed in 6 patients, *Mycoplasma pneumoniae* in 2, and an unidentified viral infection in 5 others. All patients developed clinical signs of brainstem compression from uncal or transtentorial herniation due to frontal or temporal lobe edema confirmed on CT scanning of the brain. A nonsurgical trial of ICP management failed in 10 of the patients before they underwent decompressive craniectomy; it was not specifically mentioned whether the other 3 patients underwent medical ICP management prior to craniectomy. All patients made good (GOS Score 4) or excellent (GOS Score 5) functional recoveries; in those case reports in which a GOS score was not provided, a score was assigned based on the long-term follow-up examination reported by the authors.

Fulminating cerebral edema, resulting in rapid neurological deterioration, can occur in patients with infectious encephalitis despite aggressive nonsurgical management of ICP. In a series published by Barnett et al., 1 half (5 of 10) of the patients died of cerebral edema and transtentorial herniation despite medical management with hyperosmolar therapy (serum osmolarity > 295 mEq/L). This study suggests that medical management of elevated ICP in fulminating cases of infectious encephalitis is inadequate and surgical decompression may be necessary.

We identified 4 case reports in the literature supporting craniectomy in patients with fulminating infectious encephalitis resulting in good or excellent long-term neurological outcomes (Table 1). However, no clinical trials were identified in this literature search, and therefore treatment guidelines regarding the benefits, timing, and long-term neurocognitive outcomes for decompressive craniectomy cannot be defined for patients with fulminating infectious encephalitis. Evidence supporting decompressive craniectomy for cerebral edema and herniation syndrome comes from ischemic stroke and traumatic brain injury studies in which early decompressive craniectomy on the side of the injury clearly is beneficial to survival and neurological outcome.

**Conclusions**

Infectious encephalitis is a neurosurgical disease in cases in which there is clinical and imaging evidence of early brainstem compression. We strongly recommend decompressive craniectomy for patients with fulminating infectious encephalitis in whom a brief trial of aggressive medical ICP management and external ventricular drainage has failed. Excellent neurocognitive recovery can be expected in these patients after surgical decompression despite early signs of brainstem dysfunction.
TABLE 1
Summary of cases identified in the literature that underwent decompressive craniectomy for brainstem dysfunction due to fulminant infectious encephalitis

<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>Age (yrs), Sex</th>
<th>Infectious Origin</th>
<th>Clinical Findings</th>
<th>CT Findings</th>
<th>Medical ICP Management</th>
<th>Op</th>
<th>Long-Term Outcome (GOS Score)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schwab et al., 1997</td>
<td>32, F</td>
<td>unknown</td>
<td>early: rt hemiparesis late: lt BFDP, ext</td>
<td>lt hemispheric edema</td>
<td>yes</td>
<td>lt C&amp;D</td>
<td>short-term memory loss (4)</td>
</tr>
<tr>
<td></td>
<td>29, F</td>
<td>unknown</td>
<td>early: rt hemiparesis late: BFDP, ext</td>
<td>lt hemispheric edema, midline shift</td>
<td>yes</td>
<td>lt C&amp;D</td>
<td>mild rt hemiparesis (4)</td>
</tr>
<tr>
<td></td>
<td>39, M</td>
<td>unknown</td>
<td>early: rt hemiparesis late: PERRL, no corneal reflex, flaccid</td>
<td>lt hemispheric edema</td>
<td>yes</td>
<td>lt C&amp;D</td>
<td>(5)</td>
</tr>
<tr>
<td>Ebel et al., 1999</td>
<td>0.7, F</td>
<td>HSV</td>
<td>early: somnolence late: lt GTC, lt hemiparesis</td>
<td>—</td>
<td></td>
<td>lt C + ATL</td>
<td>(5)</td>
</tr>
<tr>
<td>Taferner et al., 2001</td>
<td>42, M</td>
<td>HSV</td>
<td>early: headaches late: anisocoria, coma-tose</td>
<td>rt temporal lobe edema, hemorrhagic conversion</td>
<td>yes</td>
<td>rt C&amp;D, evacuation of temporal lobe IPH</td>
<td>minor neurocognitive deficits (4)</td>
</tr>
<tr>
<td></td>
<td>25, F</td>
<td>HSV</td>
<td>obtunded</td>
<td>rt frontotemporal lobe edema, transtentorial herniation</td>
<td>yes</td>
<td>rt C&amp;D</td>
<td>completed university studies (5)</td>
</tr>
<tr>
<td></td>
<td>28, M</td>
<td>Mycoplasma pneumoniae</td>
<td>FP</td>
<td>rt hemispheric edema, 10-mm midline shift &amp; transtentorial herniation</td>
<td>yes</td>
<td>rt C&amp;D</td>
<td>mild lt hemiparesis, resumed work as a car salesman (4)</td>
</tr>
<tr>
<td></td>
<td>17, M</td>
<td>unknown</td>
<td>CN III palsy, coma-tose, tetraparesis</td>
<td>diffuse bilat brain edema, hemorrhagic conversion, transtentorial herniation</td>
<td>yes</td>
<td>bilat C&amp;D</td>
<td>no cognitive deficits; permanent paraplegia required him to get a different job (4)</td>
</tr>
<tr>
<td>Yan 2002</td>
<td>48, F</td>
<td>HSV</td>
<td>early: severe HA late: rt hemiparesis, anisocoria</td>
<td>lt temporal lobe edema, hemorrhagic conversion</td>
<td>—</td>
<td>lt C&amp;D + ATL</td>
<td>postop stutter (4–5)</td>
</tr>
<tr>
<td></td>
<td>37, F</td>
<td>HSV</td>
<td>early: HA, memory loss, personality change late: anisocoria, GCS score dec (14–9)</td>
<td>lt temporal lobe edema, hemorrhagic conversion, &amp; brainstem compression</td>
<td>—</td>
<td>lt C&amp;D + ATL</td>
<td>(5)</td>
</tr>
</tbody>
</table>

* ATL = anterior temporal lobectomy; BFDP = bilaterally fixed and dilated pupils; CN = cranial nerve; C&D = craniectomy and duraplasty; dec = decrease; early = symptoms and neurological examination at presentation; ext = extensor decerebrate posturing; FP = flexion posturing on neurological examination; GCS = Glasgow Coma Scale; GTC = generalized tonic–clonic seizure; HA = headache; IPH = intraparenchymal hemorrhage; late = delayed symptoms and neurological examination; NDP = nonreactive dilated pupil; PERRL = pupils equal round reactive to light; — = not discussed.

References

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