Multimodality Neuromonitoring and Decompressive Hemicraniectomy After Subarachnoid Hemorrhage

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Abstract

Background and Methods We report the case of a young woman with delayed cerebral infarction and intracranial hypertension following subarachnoid hemorrhage requiring hemicraniectomy, who underwent multimodality neuro-monitoring of the contralateral hemisphere before and after craniectomy.

Results Intracranial hypertension was preceded by signs of ischemia and impaired brain metabolism diagnosed through cerebral microdialysis and PbtO2 monitoring, as well as a decrease in cerebral perfusion pressure (CPP) to <40 mmHg despite increasing vasopressor requirements. We describe how a comprehensive multimodality neuromonitoring approach was utilized to inform the decision to perform an early decompressive hemicraniectomy. Post-operatively, CPP and intracranial pressure (ICP) normalized, and the patient was weaned off all pressors within hours. The modified Rankin score at 3 and 12 months was 5.

Conclusions Delayed rescue hemicraniectomy can be life-saving after poor grade SAH. The role of multimodality brain monitoring for determining the optimal timing of hemicraniectomy deserves further study.

Keywords Intracranial monitoring · Multimodality brain monitoring · Decompressive craniectomy · Microdialysis · Brain oxygen

Introduction

Trials have shown that some patients with malignant MCA infarctions may benefit from early hemicraniectomy [1–3]. However, the role of decompressive hemicraniectomy (DH) in the treatment of other types of severe acute brain injury remains controversial [2]. DH is performed rarely as a salvage procedure for intractable intracranial hypertension in poor-grade SAH. Often the difficulty lies in determining when to perform the operation, and in severely brain-injured patients with limited neurological exams, this decision is even more challenging [1]. The advent of advanced intracranial neuromonitoring techniques shows promise in providing clinicians with additional resources to determine the ideal time to perform craniectomy, before irreversible damage occurs. However, no systematic protocol exists to utilize the data provided by advanced multimodal neuromonitoring in the context of decompressive craniectomy.

Case Report

A 26-year-old healthy woman without significant past medical history, was found collapsed in her home. Upon arrival to the hospital she was unconscious and unresponsive to stimulus. She was emergently intubated, and a CT
was performed which revealed diffuse cisternal and sylvian fissure subarachnoid hemorrhage (Fig. 1). CTA demonstrated a 6 by 4.5 mm left ophthalmic aneurysm. The patient received mannitol, amicar, fosphenytoin, decadron, and nimodipine. On admission to the neurological intensive care unit (NICU), Hunt and Hess grade and modified Fisher scales were 5 and 3, respectively, and the GCS was 4. The APACHE II score was 19. After external ventricular drain placement, cerebral angiography and successful coil embolization of the left ophthalmic aneurysm was performed.

Upon return to the NICU, the patient was stabilized and multimodality neuromonitoring was instituted (SAH day 2). A LICOX IM3™ (Integra Neurosciences™, Plainsborough, NJ), triple lumen bolt was introduced into the right frontal parenchyma, anterior to the coronal suture, through which an intracranial pressure (ICP) monitor (Ventrix NL960-V™ Integra Neurosciences™, Plainsborough, NJ), a brain tissue oxygen (PbtO2) probe (LICOX CCLSB,™ Integra™) and a microdialysis catheter (MD) (CMA/Microdialysis™, North Chelmsford, MA) were each placed, respectively. The MD catheter was perfused at 0.3 ml/min with a microinfusion pump (CMA/Microdialysis™). Samples for bedside analysis of glucose, pyruvate, and lactate were collected every 60 min.

Initial ICP was 12 mmHg, PbtO2 was 29 mmHg, lactate-to-pyruvate ratio (LPR) was 28, intracerebral glucose was 0.42 mmol/l, and cerebral perfusion pressure was maintained greater than 60 mmHg (Fig. 2). However, over the course of SAH day 3 CPP fell below 60 mmHg despite increasing vasopressor therapy. The LPR increased to greater than 40 and glucose decreased to 0.22 mmol/l over this same period (Fig. 2). The next day (SAH day 4) the LPR was sustained in the 40–50s, PbtO2 dropped to less than 25 mmHg, and the patient developed an ICP elevation to greater than 30 mmHg treated with standard medical therapy of CSF drainage, hyperventilation, head elevation, sedation and boluses of mannitol, and 23.4% normal saline.

A head CT was obtained which revealed interval evolution of large infarction in the left frontoparietal region with increased mass effect, global cerebral edema and near-complete effacement of the left lateral ventricle (Fig. 1). Given the increasingly refractory ICP elevations, decreased CPP, increasing vasopressor requirements, and persistent evidence of biochemical ischemia, the decision was made to immediately perform a left fronto-parieto-temporal decompressive hemicraniectomy on SAH day 4. Post-operatively, ICP was maintained below 25 mmHg, and CPP increased to above 60 mmHg (Fig. 2). Vasopressor requirements immediately decreased, and all vasopressor medications were weaned to off by 6 h post-operatively. LPR remained stable though elevated for the remainder of the monitoring period, while intracerebral glucose levels rose to >1.4 mmol/l (Fig. 2). Unfortunately, the PbtO2 catheter malfunctioned and immediate post-operative values could not be obtained. Re-establishment of the probe on SAH day 6 demonstrated a gradual return to PbtO2 levels above 20 mmHg. The neuromonitoring bundle was discontinued on SAH day 8. She subsequently underwent placement of a ventriculoperitoneal shunt, and approximately 1 month after admission was transferred to a subacute nursing facility with a GCS of 6. Three- and 12-month modified Rankin score were both 5.

**Discussion**

This case report describes the temporal changes in multimodality monitoring parameters preceding and following decompressive hemicraniectomy performed in a poor-grade SAH patient with delayed development of infarcts and intracranial hypertension.

The decrease in CPP to less than 60 mmHg was coincident with an increase in the LPR >40, which preceded by several hours the development of refractory intracranial hypertension.

![Fig. 1 Head CT on admission (left); on post-bleed day 4 (middle) prior to hemicraniectomy, demonstrating evolving left frontal infarct, worsening global cerebral edema with increased right shift and near-complete effacement of the left lateral ventricle, and right frontal neuromonitoring device; on post-bleed day 7 (right) demonstrating decompression of left hemisphere](image-url)
hypertension and decrease in PbtO2 to less than 20 mmHg (Fig. 2). The patient also developed progressive neurogenic hypotension, with decreasing MAP despite increasing doses of multiple vasopressors (Fig. 2). Interestingly, after hemicraniectomy was performed all pressors were weaned off in a matter of hours, suggesting that relieving brainstem compression led to a restoration of normal basal sympathetic vascular tone.

Several studies have evaluated the role of decompressive hemicraniectomy in poor-grade subarachnoid hemorrhage, both for decompression of large associated hematomas as well as for delayed cerebral infarction and vasospasm [4–7]. One study found that for intractable intracranial hypertension in SAH, long-term outcome was better for patients who underwent early decompression within the first 48 h of SAH [6], which underscores the need to characterize trends in brain pathophysiology preceding ICP crisis, so that DH may be undertaken as early as possible. Comprehensive studies of hemicraniectomy evaluating the role of multimodality monitoring are also lacking [8–10]. In a retrospective study of 20 patients with SAH, ischemia as diagnosed by low PbtO2 levels (<10 mmHg) preceded the occurrence of herniation requiring craniectomy, although no definitive conclusions could be drawn regarding the use of this modality [11]. MD has been studied more extensively in relation to ischemic stroke and traumatic brain injury [12,13], with one study of 34 patients with MCA infarction showing that a decrease in CPP to <50–60 mmHg, with a decrease in PbtO2 to <10 mmHg, and a significantly higher LPR occurred in 17 patients who experienced a malignant course and herniation [14]. A PbtO2 value of <10 mmHg has been previously shown to be threshold for significant tissue hypoxia as well [15]. There is increasing evidence for the relevance of high ICP and its relation to poor outcome in SAH [16]. However, the relationship between ICP and metabolic parameters remains less clear. In a study of 25 patients with TBI, Belli et al. [17] showed that an abnormal LPR (>25) could predict an ICP rise above normal levels in 89% of cases. The authors concluded that biochemical changes reflected in cerebral microdialysis can occur before intracranial hypertension [17]. Relative change in monitoring parameters can also be of use in detecting adverse events. Unterberg et al. [18] found a 20% increase from baseline in both LPR and glycerol concentration preceded the onset of delayed ischemic neurological deficit in 17 of 18 SAH patients by a mean interval of 11 h.

The present case illustrates how comprehensive multimodality neuromonitoring can be employed to inform the
decision to perform a life-saving decompressive hemicraniectomy on a poor-grade comatose SAH patient with limited to no neurological exam. It is in this sub-group of SAH patients that such an approach offers perhaps the greatest promise, precisely because the clinical exam is so often absent or unreliable [19, 20]. In this case, a decrease in CPP to <50, neurogenic hypotension reflected by increasing norepinephrine and vasopressin requirements, a >20% increase in the LPR to >40, and persistently low cerebral glucose (<0.7 mmol/l) all contributed to a poor physiological profile (Fig. 2) that motivated the decision to perform immediate surgical decompression at the first sign of increased intracranial pressure. Following the procedure, ICP was stabilized, all pressors were weaned to off within hours, CPP normalized and intracerebral glucose levels increased. LPR, which demonstrated an increasing trend prior to DH, stabilized. While LPR levels remained elevated after DH, it should be noted that initial LPR values were also elevated (>30), likely reflecting the insult from the initial aneurysm rupture. In this case, the relative change of this parameter over time was perhaps more informative than the absolute values.

The location of the neuromonitoring bundle relative to injury is a source of ongoing debate [21]. Nordstrom et al. [12] studied the impact of different levels of CPP on brain metabolism in 50 TBI patients using MD catheters placed in the vicinity of evacuated hematomas or contusions (tissue at risk), and in non-lesioned frontal cortex. A CPP <50 mmHg was associated with a significantly higher average MD lactate and MD-LPR in the tissue at risk only, not in the non-lesioned tissue [12]. In this patient, the bundle was placed contralateral to the side of infarct and hemicraniectomy, yet elevations in LPR were still noted well in advance of ICP elevation (Fig. 2). It could be argued that the persistently elevated LPR values are reflective of the probe being placed in non-viable, infarcted brain tissue. However, in this patient the contralateral, non-infarcted hemisphere remained viable (Fig. 2), which suggests that the markers of metabolic distress recorded by the monitoring bundle were indicative of a more global cerebral dysfunction and injury. The optimal placement of neuromonitoring probes relative to injury is not known, although some evidence exists for placing the probes in the penumbra surrounding a focal lesion [21]. However, this risks inadvertent placement directly into a clot. In this patient, placement into the contralateral, non-dominant hemisphere was chosen to avoid this potential complication. Studies describing neurochemical changes in the hemisphere contralateral to infarction are lacking. In Dohmen et al.’s study of 34 patients with MCA infarction, all catheters were placed ipsilateral to the stroked territory [14]. One case report of a fatal MCA stroke described neurochemical alterations in the noninfarcted hemisphere, noting a particular sharp rise in glutamate and glycerine which commenced hours before pupillary disturbances occurred and a marked increase in ICP was measured [22]. However, decompressive craniectomy was not performed in this report.

Much of the difficulty in utilizing neuromonitoring data effectively lies in determining the complex inter-relationships between the various modalities. For instance, in a recent study of 4 children suffering from traumatic head injury, rises in ICP were variably accompanied by decreases in brain oxygen levels, and low absolute brain oxygen levels during the first 24 hours did not necessarily correlate with outcome [23]. Relative changes in multimodality parameters, rather than establishing absolute cutoffs, may offer more clinical utility on an individual patient basis. As illustrated in the present report, we believe the integration of monitoring modalities—to create a “physiological profile” for each patient by which relative changes in multiple variables over time can be interpreted and acted upon—will be an important paradigm in future studies. Randomized studies are needed to determine the role of advanced multimodality monitoring techniques in relation to decompressive hemicraniectomy.

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References