

High intracranial pressure, brain herniation and death in cerebral venous thrombosis

Sir, the study by Canhao and colleagues made the important point that the most frequent cause of death in patients with cerebral venous thrombosis (CVT) is transtentorial herniation and that these patients may potentially have benefited from decompressive hemicraniectomy [1]. We would like to corroborate this argument by providing evidence for a rise in intracranial pressure (ICP) preceding brain herniation and death in a patient with CVT.

A 29 year old pregnant woman who presented with confusion and vomiting was admitted to a district general hospital. A right sided weakness developed within two days. A CT brain scan showed a left temporal haemorrhage without mass effect. Her GCS dropped to 8/15 she was intubated and transferred to the neurocritical care unit at the National Hospital. A repeat CT demonstrated worsening of the haemorrhage with obliteration of the 3rd and 4th ventricles and the diagnosis of a CVT was confirmed by MRV. At this point the left pupil became fixed. An ICP bolt was inserted (opening pressure 50 mmHg, Figure 1). ICP targeted management (propofol, fentanyl, midazolam, ventilation to maintain a pCO₂ 4.0 – 4.5kPa and i.v. norepinephrine to main blood pressure) and anticoagulation with intravenous heparin were started. Because an ICP <20 mmHg could not be maintained, paralysis and moderate hypothermia were initiated. Despite these measures ICP continued to rise. A treatment trial with thiopentone to lower ICP failed. Both pupils became fixed and dilated on day 6 on ICU. Several intractable peaks of ICP (>60 mmHg) were followed by development of diabetes insidipus necessitating treatment with DDAVP. At 8:00 AM on the 7th day periods of ventricular tachycardia and flimmer started to appear leading to severe haemodynamic compromise and elevated ICP. The clinical diagnosis of brain stem herniation was made and treatment was de-escalated after an informed discussion with the family took place.

Mortality in CVT has decreased over the last decades from 30-50% to about 4.3% in the acute phase [1,2,3]. Anecdotelly decompressive hemicraniectomy

has been performed successfully in patients in whom medical treatment failed [4]. It is of note that all three patients treated by Stefani and colleagues already showed signs of brain herniation at time of operation. The authors pointed out that indications for surgical intervention are almost completely lacking. The decision to proceed with surgery implies that treatment (heparin) needs to be discontinued. In an individual case this may be a difficult decision, because of the arguably beneficial effect (small sample sizes and large confidence intervals) [5]. Furthermore there is no guide towards the best timing for surgical intervention. Fixed and dilated pupils may be too late a sign and repeated brain imaging is logistically difficult in the critically ill patient. Continuous ICP and ABP monitoring as performed in the present case provides important data at the bedside. There is a need to investigate whether decompressive hemicraniectomy would be of benefit in those patients in whom medical management of ICP fails.

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Figure 1 ICP (diamonds), MAP (squares) and CPP (dots) over 5 days preceding brain herniation and death.

