How I manage intracranial hypertension

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Why and when to manage intracranial hypertension

The detrimental effects of intracranial hypertension (HICP, high intracranial pressure) are well documented [1, 2]. HICP can cause secondary brain injury and death, and therefore, intracranial pressure (ICP) elevations should be aggressively treated.

HICP has been classically defined as an ICP > 20 mmHg, and this threshold has been considered the trigger for treatment [3]. Recent BTF guidelines have moved this threshold to 22 mmHg [4], grounded on a single-centre, retrospective study. This modification is trivial [5]. As for many other treatment options in intensive care, a single threshold is debatable. In fact, recent evidence suggests that not a single value but the time spent over the threshold is important [6]. Moreover, Guiza demonstrated that not only higher values but also prolonged exposure to values below the classical threshold are associated with negative outcomes [7]. In addition, if cerebral perfusion pressure (CPP, i.e. MAP-ICP) is critically low (< 50 mmHg), ICP is no longer a predictor for poor outcome and lower ICP values might be barely tolerated. On the contrary, ICP insults in the range 18–23 mmHg can be tolerated for a longer duration at higher CPPs. In my practice, the ICP alarm is set at 20 mmHg and low CPP alarm at 55 mmHg. This is a warning signal for nurses at the bedside. Before starting any treatments for high ICP, I consider both the intensity and duration of HICP. I am flexible with thresholds putting them in the clinical contest, considering also CPP. Short-lasting, low-intensity episodes (low ICP dose with normal CPP) are observed and not treated. On the contrary, higher ICP doses, progressively rising trends, or and HICP impacting CPP require prompt treatment.

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Figure 1 summarises the algorithm that I use in clinical practice. Before starting any ICP-directed therapies, I try to correct any reversible cause and systemic abnormality affecting intracranial volumes and causing raised ICP (see Additional file 1). I always consider the surgical option with a neurosurgeon; mass-occupying space should be promptly evacuated when indications are met, and hydrocephalus should be drained.

When I decide to administer ICP-lowering therapies, I use a “staircase” approach [1] with escalating treatment intensity (starting with low risk-benefit profiles) [8]. The first-line ICP-lowering strategies that I consider (without a priority between them) include:

- Head-up positioning (15–30°),
- Hemodynamic stability aimed to maintain an appropriate cerebral perfusion pressure (CPP 50–70 mmHg according to autoregulatory status. Increasing mean arterial pressure + 10% might be considered as a test for exploring pressure autoregulation),
- Sedation and analgesia (propofol, 4–6 mg/kg/h and opioids, fentanyl 1–4 μg/kg/h used at the lowest dose producing ICP control. Maintain CPP with vasopressors, if needed) [9],
- Mechanical ventilation to prevent hypercapnia and hypoxia (target PaCO2 at 35 mmHg, and oxygen saturation ≥ 94%),
- Normothermia; if the temperature is > 37.5 °C (internal), I start Diclofenac infusion [10],
- Crystalloids as preferred maintenance fluids [11] to maintain euvolemia and to prevent drops in plasma osmolarity. I do not use colloids or hypotonic solutions w/o glucose as maintenance fluids.

If HICP persists, I subsequently escalate to osmotic agents, mannitol (up to 0.5–1 g/kg every 4–6 h) or hypertonic saline (7.5% solution, 100 ml every 4–6 h). They have several transient mechanisms (lasting 4–6 h) mainly due to osmotic effects but also hemodilution, increased cardiac output and increased blood pressure. I prefer testing both of them (using an equimolar bolus) for evaluating their efficacy in the individual patient. Their efficacy is higher if started at an ICP > 25 mmHg [11].
When and how to escalate to upper tier therapies

I generally reserve to patients with refractory intracranial hypertension ICP-lowering strategies associated with significant side effects and potential complications as hyperventilation, metabolic suppression and decompressive craniectomy [8, 12].

Hyperventilation produces a reduction of HICP by inducing cerebral vasoconstriction and reducing cerebral blood volume [13]. The effect is short lasting and cease when the interstitial pH, alkalotic during the immediate hyperventilation phase, returns to normality. However, because of the theoretical risk of hypoperfusion, I aim to achieve mild hyperventilation, i.e. a PaCO₂ ~ 30–32 mmHg, only in patients in whom ICP remains abnormally elevated despite first- and second-line treatments, considering adding for safety a brain oxygenation monitor. I use more aggressive hyperventilation only in life-threatening cases with the risk of cerebral herniation and death.

Barbiturates have been historically used for decreasing brain metabolism and consequently cerebral blood flow/volume and therefore HICP at the cost of serious side effects including hypotension and infections. I avoid long-term administration, and I generally administer thiopentone (10 mg/kg bolus, checking its efficacy, followed by 3–8 mg/kg/h infusion) as temporary “bridge” to decompressive craniectomy (DC) in refractory cases. I prefer, as third tier therapy, DC that has a long-lasting effect on the control of refractory HICP. DC performed
without severe refractory HICP increases the rate of unfavourable neurologic outcome and should be avoided [14]. On the other hand, DC in patients with severe refractory HICP reduces mortality (22 more survivors for every 100 patients treated) [15]. At 12 months, 13/22 survivors (59%) had favourable outcomes while 9/22 (41%) were in a vegetative state or in lower severe disability. For these reasons, DC needs to wisely ponder in the context of refractory HICP and it should be undertaken timely in subjects with a potentially acceptable prognosis (i.e. before irreversible damages occurred), considering individual patient’s preferences and family’s quality of life expectations.

In conclusion, my approach to ICP-lowering strategies has a stepwise fashion associated with a continuous check of the efficacy of the therapies. This will allow me to deescalate ICP-lowering strategies as soon as possible (ICP control > 24 h). Tapering therapies (as hyperventilation and osmotic) might produce a rebound effect, and it needs to be done slowly and under ICP monitoring.

Alternatively, if the therapies are ineffective, I intensify treatments until the patients are judged salvageable. When, in more severe unsalvageable cases, everything is ineffective and DC is not an option, a wise limitation of the therapies has to be evaluated.

Additional file

Additional file 1: Summary of the remediable causes of intracranial hypertension. (DOCX 15 kb)

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