Life-threatening spontaneous intracranial hypotension responding to epidural blood patch

Editor—Following the recent case series of spontaneous intracranial hypotension (SIH) as an indication for epidural blood patch,1 we report a severe case presenting with a decreasing level of consciousness, leading to coma and requiring tracheal intubation. This patient was also treated successfully with an epidural blood patch.

A 68-year-old man presented with a 5-day history of worsening frontal headache and vomiting. His Glasgow Coma Scale, initially 14/15, deteriorated to 9/15 over the next 24 h. Brain imaging with CT scan was reported as showing increased attenuation in the basilar cisterns and the tentorium cerebelli, initially suggestive of subarachnoid haemorrhage. Subsequent review, however, identified these findings as SIH2 and led us to perform an MRI scan. By this time he was agitated and had developed apnoeic episodes, bradycardia and hypertension. He was intubated and ventilated to enable MRI scanning and facilitate further management. The MRI scan showed bilateral, 8 mm subdural fluid collections overlying the frontal lobes, with effacement of cortical sulci and basal cisterns. Post-gadolinium enhanced images showed pachymeningeal enhancement supporting a diagnosis of SIH.3 He was transferred to the Intensive Care Unit and treated with an epidural blood patch consisting of 20 ml of sterile blood injected at the L3/L4 level. Twelve hours later his sedation was stopped, to allow him to wake and be extubated. His conscious level then recovered over the next few hours to a GCS of 15/15 with complete resolution of symptoms.

The literature suggests that the breach of the dura in SIH is most often at the cervical or thoracic level.4 In our patient, the site of the dural tear was unknown. Exactly how the ‘blind’ epidural blood patch causes benefit in these cases is unclear but, as in those reported by Buguet-Brown and colleagues,1 we found it to be rapidly effective.

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Strong magnesium solution

Editor—The pain relief obtained by intra-articular magnesium sulphate after meniscectomy1 is impressive, but the concentration of magnesium is about 200 mmol litre−1 and the solution is hypertonic. The final concentration in the joint will naturally be affected by the volume of fluid residing after the surgery, and the tonicity of the solution may not be a major issue. However, the concentration of magnesium ions certainly is a problem. These concentrations are well away from normal and I would be concerned that they could cause tissue damage. Have these solutions been tested in animals, and the histological effects assessed? Perhaps the analgesia is a result of permanent nerve damage? I would strongly caution against any medical use of solutions such as these before we can be sure they do not cause damage.

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References
Abdominal surgery, morbid obesity, age, dexamethasone, diabetes mellitus and glucose metabolism

Editor—We read, with interest, the paper by Hans and colleagues showing moderately increased capillary blood glucose concentrations during and after abdominal surgery in type 2 diabetic morbidly obese patients when compared with a younger group of non-diabetic obese patients. All patients in this protocol had received a bolus of 10 mg of dexamethasone for the prophylaxis of postoperative nausea and vomiting at the induction of general anaesthesia. The peak mean glucose concentration, 2 h after the administration of dexamethasone, was 8.7 mmol litre\(^{-1}\) in the diabetic and 7.5 mmol litre\(^{-1}\) in the non-diabetic group. These are slightly higher than peak circulating blood glucose concentrations (7.0 mmol litre\(^{-1}\)) previously observed in non-diabetic, normal weight for height women undergoing abdominal hysterectomy with sevoflurane anaesthesia and who had not received dexamethasone. Some aspects of this study merit further comment.

Notwithstanding the lack of control groups and the fact that the diabetic patients had a significantly greater body mass index than non-diabetic subjects, which per se can explain the small difference in glycaemia, the manuscript by Hans does not provide certain information concerning patient characteristics and study design that are pertinent for the understanding of perioperative glucose metabolism. As hyperglycaemia depends on the magnitude of surgical tissue trauma, which is not necessarily reflected by CRP-levels obtained 24 h after surgery, it is relevant to define ‘bariatric surgery’, ‘non-bariatric laparoscopy’ and ‘non-bariatric laparotomy’. For example, did patients in the bariatric surgery group undergo open, laparoscopic Roux-en-Y gastric bypass surgery, sleeve gastrectomy, gastric banding or abdominal liposuction? The duration of preoperative fasting has been demonstrated to have an impact on insulin sensitivity and glycaemia after surgery. Therefore, it would be interesting to know whether some procedures necessitated bowel cleaning and whether preoperative fasting periods were comparable in the two groups.

As the type of analgesia has significant effects on glucose metabolism, it needs to be explained how pain was controlled after surgery. Apparently none of the patients had epidural anaesthesia or analgesia for procedures that, in many centres, are routinely performed under combined general/epidural anaesthesia. One may, therefore, appreciate a more detailed explanation of the patient selection process, that is were patients who were eligible and opted for epidural anaesthesia excluded from recruitment? In this context one would also appreciate more details about the patients’ co-morbidities and medication with a potential impact on glucose metabolism, in particular the use of \(\beta\)-adrenergic blockers or thyroid hormone replacement therapy. Did the patients intraoperatively receive vasopressors with known metabolic effects such as ephedrine, epinephrine or norepinephrine? Hypotension must have occurred frequently after premedication with 300 \(\mu\)g clonidine.

No information was provided regarding the fluid replacement strategy and transfusion practice. What was the actual blood loss? Did some patients require transfusion of packed red blood cells or fresh frozen plasma both of which contain high concentrations of glucose? It also needs to be stated which and how much crystalloid solution was administered during the study period, because, for example, the lactate content of Hartmann’s solution may affect the blood glucose levels in surgical patients. Finally, it is unknown if subcutaneous heparin was used. Heparin stimulates lipoproteinlipase \(\textit{in vivo}\) leading to an increase in free fatty acids, which subsequently impair glucose utilization by the so-called Randle mechanism. Assuming that heparin dosing was based on body weight, which was significantly greater in the diabetic group, one would expect that patients in the insulin group received more heparin.

These questions are relevant for the interpretation of small changes in glycaemia, particularly when fingerprick capillary blood glucose instead of circulating blood glucose measurements are performed; a technique that is inadequate to assess glucose metabolism during and after surgery.

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Editor—We would like to thank Drs Schricker and Carvalho for their interest in our work. Basically we agree with the majority of concerns they addressed regarding missing data...