The effect of the first EBP waned and a second EBP was performed about 42 days later. A larger blood volume (35 ml) was injected, and the patient was maintained in a head-down position for 20 min. Within a couple of hours, he became more alert and there was marked improvement in his social behaviour. This improvement was not as marked as that following the first EBP and lasted for <1 month. A repeat MRI of the brain did not show any CSF leak.

At the request of the neurologist and the family, a third and final EBP was performed about 5 months later. Twenty millilitres of blood were injected into the epidural space. This resulted in improvement of symptoms, both social and cognitive, albeit short-lived.

HHB is responsible for the most severe form of apathy. Diagnosis is mainly clinical. The most characteristic features on MRI are brainstem swelling and sagging of the brain in the absence of meningeal enhancement.<sup>2</sup> EBP resolves symptoms in patients with HHB<sup>2</sup> by elevating the epidural and CSF pressures. The most likely mechanism causing symptoms in HHB is spontaneous intracranial hypotension secondary to CSF leak.<sup>3</sup>

After consideration of the limited literature with regard to treatment of the condition and the potential benefits of an EBP, we decided to proceed. It was unfortunate that no CSF leak was identified, as it could then have been amenable to radiological or surgical intervention for sustained relief of symptoms. We performed the EBP in the lumbar inter-space, as it is technically easier, safer, and can accommodate a larger volume of blood. The patient required a repeat EBP after a month in view of the non-sustained effect of the previous blood patch. No complications, attributed to the epidural, were seen in our patient during the follow-up.

## **Conflict of interest**

None declared.

J. A. Khan D. McAtamney P. A. Farling\* *Belfast, UK* \**E-mail: peter.farling@dnet.co.uk* 

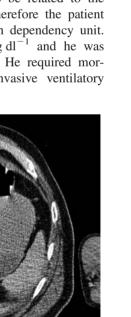
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## Liver trauma secondary to ultrasound-guided transversus abdominis plane block

Editor—The use of ultrasound for placement of transversus abdominis plane (TAP) block has recently been described for a number of surgical procedures.<sup>1–3</sup> Liver trauma after TAP block using the landmark technique has been described, but there are no documented complications with ultrasound-guided placement.<sup>4</sup> We report a case of ultrasound-guided TAP block causing peritonitis in a patient undergoing an inguinal hernia repair.

A 61-yr-old male with an unremarkable past medical history other than a 47 pack year history of smoking was admitted with a strangulated inguinal hernia. This was reduced under simple analgesia and the patient subsequently taken to theatre for repair of both this hernia and an incidental umbilical hernia. After induction of general anaesthesia, bilateral TAP blocks were performed by a consultant anaesthetist using a 50 mm 22 G regional block needle, a SonoSite iLook portable ultrasound with a 10-5 MHz linear probe and a dose of bupivacaine 0.375% (20 ml) given on each side. The operation proceeded uneventfully and the patient was discharged back to the general surgical ward. That evening, he then complained of right upper quadrant pain and subsequent examination revealed obvious peritonitis together with hypoxia, hypotension, and tachycardia. Blood gas analysis revealed a metabolic acidosis and after fluid resuscitation a CT abdomen (Fig. 1) was performed which revealed peri-hepatic fluid suggestive of blood secondary to liver injury. This was presumed to be related to the TAP block performed that day and therefore the patient was managed conservatively on a high dependency unit. His Hb decreased from 16.5 to  $8.8 \text{ g dl}^{-1}$  and he was therefore transfused 2 units of blood. He required morphine PCA for analgesia and non-invasive ventilatory



45 Fig 1 Peri-hepatic fluid suggestive of blood secondary to liver injury.

support with continuous positive airway pressure (CPAP) for 2 days. He was discharged home 7 days after his operation.

This is the first reported case of an ultrasound-guided TAP block causing a significant postoperative complication and highlights the potential for iatrogenic injury even when ultrasound is used. The injury here was likely due to a failure to accurately image the entire needle during the right-sided needle placement, resulting in excessive depth of penetration. The iLook machine is marketed for use in vascular access procedures and superficial imaging and as such may not produce images of sufficient quality to be used in this setting. Any practitioner using ultrasound for this type of block should be adequately trained in its use and be fully aware of the potential for complications.

## **Conflict of interest**

None declared.

P. Lancaster\* M. Chadwick *Manchester, UK* \**E-mail: plancaster@doctors.org.uk* 

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## Gluteal compartment syndrome presenting with features of iatrogenic epidural haematoma

Editor—The debate about whether epidural analgesia can delay the diagnosis of compartment syndrome has recently been reviewed.<sup>1</sup> An alternative, infrequently considered interaction between compartment syndromes and epidural analgesia occurs when the symptoms of compartment syndrome are incorrectly attributed to epidural complications and diagnosis of the true pathology is delayed. We present a case where the diagnosis of gluteal compartment syndrome was delayed due to the erroneous investigation of epidural haematoma.

A 58-yr-old male was admitted electively for elective right common femoral endarterectomy, patch profundaplasty, and above-knee femoro-popliteal bypass. He was morbidly obese, BMI 41 kg m<sup>-2</sup>, but was otherwise generally well. The anaesthetic plan was to perform the surgery under combined epidural and spinal anaesthesia. After establishing epidural cannulation, it proved impossible to safely identify the subarachnoid space and the anaesthetic management was converted to general anaesthesia with an epidural infusion.

The 5 h operation was uneventful and the epidural was working satisfactorily in the immediate postoperative period. He later developed symptoms of paralysis and loss of sensation of the right leg and was reviewed by an anaesthetist and a vascular surgeon who found that tone, muscle power, and reflexes were reduced or absent throughout the right leg. Sensation was absent throughout the sciatic nerve distribution and reduced in the femoral nerve distribution. Perianal sensation and anal tone were normal with no loss of bowel or bladder control. There was no evidence of vascular compromise in the affected leg or of compartment syndrome in the thigh and calf. The patient described mild right hip pain on the affected side which was poorly localized, which was unaffected by palpation or passive hip movement but increased by active movement.

It was felt essential to exclude an epidural haematoma, and following assessment, the neurosurgeons arranged magnetic resonance imaging of the spine which was normal. Plain X-rays of the pelvis and right hip were also normal.

On further review, the pain required increasing analgesia and had progressed to being exacerbated by passive movement. It was now localized to the right buttock which was tender to palpation. Gluteal compartment syndrome with sciatic nerve compression was suspected and the patient taken to theatre for measurement of buttock compartment pressures and emergency fasciotomy.

The fascial pressures were measured at 54 mm Hg on the right (affected side) and 5 mm Hg on the contralateral side (normal range: below 10–12 mm Hg). An emergency fasciotomy was performed around 23 h after the initial symptoms. The fascial compartments of gluteus maximus and medius were both tense and upon decompression, the muscle was oedematous but viable. Motor and sensory function returned rapidly after operation, and the patient made an uneventful recovery and was discharged 8 days later.

Gluteal compartment syndrome is rare and many anaesthetists may be unaware of its existence. It is usually caused by direct trauma or prolonged immobilization,<sup>2</sup> but has been reported after surgery where obesity was felt to be a risk factor.<sup>3–5</sup> It can have devastating consequences causing permanent paralysis of the limb, renal failure, and death due to rhabdomyolysis. Treatment must be aggressive and delivered early, certainly <8 h after the onset of