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Epidural Blood Patch in the Treatment of Spontaneous Intracranial Hypotension

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Abstract

Spontaneous intracranial hypotension (SIH) can occur due to sub-dural haematoma which may be secondary to an unrecognized cerebrospinal fluid (CSF) leak. Here, we report a case of spontaneous intracranial hypotension post-surgery who was treated with epidural blood patch. The patient had initially undergone a bilateral fronto-parietal burrhole and evacuation of hematoma. Subsequently, he was readmitted 10 days later with worsening of consciousness and a possible unrecognized CSF leak causing low intracranial pressure was suspected. Epidural blood patch (EBP) has emerged as the most important nonsurgical treatment for spontaneous CSF leaks. We administered the lumbar epidural blood patch twice within a time span of 5 days after which a considerable improvement was seen in his sensorium.

Keywords: Epidural blood patch, Subdural haematoma, Subdural hygroma, CSF leak, Spontaneous intracranial hypotension

Introduction

Spontaneous intracranial hypotension (SIH) is known to produce serious complications like sub-dural haematoma (SDH) which may occur secondary to an unrecognized cerebrospinal fluid (CSF) leak. This leak leads to a loss of CSF volume which supports the brain and spinal cord. SIH usually presents with orthostatic headache caused by low cerebrospinal fluid (CSF) pressure of spontaneous origin which may be accompanied by neck stiffness and subjective hearing symptoms. Headache associated with SIH generally remits after normalization of CSF pressure. The severity of the patient's symptoms usually determines the management of SIH. Although some patients may respond to conservative treatments like bed rest, analgesia, adequate hydration, acetazolamide, intravenous caffeine, oral theophylline and oral corticosteroids, epidural blood patch (EBP) has been regarded as the mainstay of therapy for SIH. EBP provides symptomatic relief in a majority of the cases regardless of the site of the leak. Here, we report a case of spontaneous intracranial hypotension post-surgery who was treated with epidural blood patch.

Case presentation

A sixty one year old male presented with 2 days history of headache, altered sensorium and a Glasgow coma score (GCS) of 13/15. Magnetic resonance imaging revealed bilateral sub-dural hematoma along the frontal and parietal convexities of the brain. The haematoma on the right and left sides measured 7 mm and 13 mm, respectively with no midline shift. He underwent bilateral fronto-parietal burrhole and evacuation of hematoma following which his neurological condition improved and was discharged after 5 days with a GCS of 15/15. He was readmitted 10 days later with worsening of consciousness (GCS - 7/15). Computed tomography (CT) of brain revealed bilateral frontoparietal sub-dural hygroma measuring 17 mm and 14 mm along left and right parietal convexities. A bilateral burrhole hygroma evacuation was done under general anaesthesia. Post-procedure, he was mechanically ventilated due to low GCS. Antiedema and neuroprotective measures were instituted over the next 24 hours. He was gradually weaned off mechanical ventlation and extubated the following day. Forty-eight hours later, his level of consciousness started deteriorating again. In view of worsening sensorium (GCS - 9/15), a repeat CT brain revealed features of low intracranial pressure (effacement of cortical sulci and squashed cisterns), enhancement of the pachymeninges, engorgement of venous structures and pituitary hyperaemia. A possible unrecognized CSF leak causing low intracranial pressure was suspected. EBP was considered as the next line of management wherein 20 ml and 10 ml of autologous blood was injected into L3/L4 and T12/L1 space, respectively under strict aseptic conditions. This was done because injecting a large volume at a single intervertebral level may cause discomfort and cramping. Hence, we presumed that the above volumes of EBP administered at T12/L1 (10 ml) and L3/L4 (20 ml) may create a blood patch sufficient to cover the CSF leak without producing any discomfort for the patient. This is also evident from the fact that the patient's sensorium improved after administering the EBP and was shifted to the ward. After 3 days of performing the EBP, patient became obtunded with a GCS – 10/15. A repeat EBP was performed with 20 ml of autologous blood injected into L3/4 space following which his GCS improved. He was then discharged without any further relapses.

Discussion

Spontaneous intracranial hypotension (SIH) has an incidence of 2-5

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cases per 100,000 patients per year. It was originally described by Schaltenbrand in 1938 [1, 2]. Some studies have reported that connective tissue disorders such as Marfan syndrome, Ehlers-Danlos syndrome type 2, and autosomal dominant polycystic kidney disease play a significant role in causing SIH. SIH has also been reported in patients with a serious but rare complications like SDH [3]. Important magnetic resonance imaging (MRI) features of SIH include sub-dural fluid collections, enhancement of the pachymeninges, engorgement of venous structures, pituitary hyperaemia and sagging of the brain (mnemonic – SEEPS). Other features which can be identified in MRI include descent of the cerebellar tonsils, effacement of the basal cisterns, bowing of the optic chiasm and flattening of the pons. The patient reported in this study had all the above mentioned MRI features suggestive of SIH. The most characteristic brain MRI finding in SIH is diffuse pachymeningeal enhancement which is caused by an increase in venous blood volume secondary to a loss of CSF pressure [4]. This may result in a negative intracranial pressure gradient that may damage the cerebral venous endothelial lining and lead to sub-dural haematoma by producing tears and finally rupturing the bridging veins in the dural border cell layer. Secondly, the loss of CSF volume may reduce CSF absorption into the cerebral venous sinuses, resulting in increased blood viscosity in the venous compartment [5]. However, any acute changes in the level of consciousness is an emergency indication of surgical evacuation of SDH [6]. Our patient also had sub-dural haematoma which was evacuated when he was admitted the first time. Spontaneous intracranial hypotension is generally a diagnosis of exclusion. SIH may be precipitated by events such as recent operative procedures, lumbar puncture or traumatic dural tear and other medical conditions (severe systemic infection, diabetic coma, administration of hypertonic solution, hyperpnoea) [3]. Once a diagnosis of SIH is made, the site of CSF leak must be identified and for this purpose radionuclide cisternography is considered to be the most accurate method [7]. Even with advanced imaging, the CSF leak can only be localized in 50% of cases. The CSF leak can also be localised noninvasively using MR myelography, which shows pachymeningeal enhancement, extra-dural fluid extravasation extending to the paraspinal soft tissues, and engorgement of epidural venous plexuses [8]. The primary goal of SIH therapy is to stop the CSF leak and increase CSF volume [9]. Conservative management strategies to curtail the CSF leak include strict bed rest, plenty of caffeine intake, avoiding sitting upright analgesics. High intake of salt, oral, or IV hydration help in restoring the volume of the CSF, which in turn alleviates the symptoms [10]. Conservative management strategies are generally observed to have a low efficacy (approximately 7%) in treating the symptoms of SIH [11]. If conservative methods fail to mitigate SIH within 1 to 2 weeks or if the headache is associated with debilitating features, EBP is the first line of therapy. EBP may be successful in 70% of cases (range 33-90%) [12, 13]. In our patient, it is possible that the patient developed SDH subsequent to SIH because once the SDH was evacuated, patient developed worsening of symptoms. Lumbar EBP can be repeated because of its low risk of severe complications [14]. Larger volumes of EBP provides greater spread of clot (five to nine spinal segments) achieving a greater mass effect and a higher probability of successful treatment. Clotted blood causes mechanical tamponade, promoting dural sealing. It relieves symptoms by compressing the lumbar cisterns, increasing CSF pressure, reducing traction on brain structures causing immediate pain relief. EBP can be very rewarding in medically-refractory cases of intracranial hypotension.

Conclusion

Chronic low intracranial pressure is invariably due to unrecognized spontaneous CSF leak intracranially or otherwise. Epidural blood patch (EBP) has emerged as the most important nonsurgical treatment for spontaneous cerebrospinal fluid (CSF) leaks. If lumbar EBP fails to provide relief, it can be repeated because of its low risk of severe complications similar to our patient where we administered the lumbar epidural blood patch twice within a time span of 5 days after which a considerable improvement was seen in his sensorium.

Clinical message

This case report discusses the effectiveness of epidural blood patch for the treatment of Spontaneous intracranial hypotension.

Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his/her consent for his/her images and other clinical information to be reported in the Journal. The patient understands that his/her name and initials will not be published, and due efforts will be made to conceal his/her identity, but anonymity cannot be guaranteed.

Conflict of interest: Nil **Source of support:** None

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