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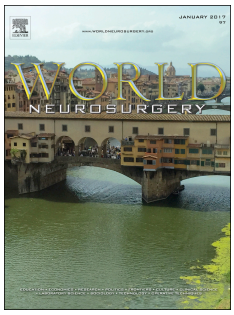

Intracranial hypotension following traumatic brain injury: a diagnostic and therapeutic challenge

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Intracranial hypotension following traumatic brain injury: a diagnostic and therapeutic challenge

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Abstract

Background

Intracranial hypotension (IH) is a recognised cause of coma, however, the diagnosis is often challenging, especially in patients with superimposed traumatic brain injury (TBI).

Case report

We report a case of a 67-year-old patient who became comatose following evacuation of bilateral acute subdural haematomas with concurrent respiratory failure. Imaging and intraparenchymal intracranial pressure (ICP) monitoring confirmed secondary IH. She was managed with an epidural blood patch, and a 72 hours period in the trendelenberg position guided by ICP monitoring and clinical assessment. She subsequently made an excellent neurological recovery from an initial Glasgow coma scale (GCS) of 3 to a GCS of 15.

Conclusion

A diagnosis of secondary IH can easily be missed in patients who have suffered a primary brain injury. In patients with a poor neurological recovery, clinicians should rule out secondary IH as a potential cause as immediate treatment can lead to a profound clinical improvement.

Key words: Intracranial hypotension, trauma, ICP monitor, trendelenberg.

Introduction

Spontaneous intracranial hypotension (SIH) was first described by the German physician Shaltenbrand in 1938 and is characterised by reduced cerebrospinal fluid (CSF) pressure [8]. This may be due to a CSF leak, most commonly in the spine, or over drainage in patients with internal shunts. SIH can be diagnosed by measuring the opening CSF pressure with a lumbar puncture, typically less than 6 mmHg, or by characteristic features on magnetic resonance imaging (MRI) studies.

SIH most commonly presents with low-pressure postural headaches, nausea, vomiting, and neck pain. In severe SIH, patients can present with a poor GCS or in some cases, coma. While mild SIH can be managed with bed rest and hydration, in more severe cases, placing the patient in the trendelenburg position can result in a profound neurological improvement.[1] In patients who do not improve with non-invasive treatment modalities, epidural blood patching has been described with good outcomes.[2]

Schievink reported a misdiagnosis of SIH in up to 94% of patients, despite it being an established cause of coma [9]. The diagnostic challenge of intracranial hypotension (IH), primary or secondary, may be due to the wide variety of clinical presentations associated with the pathology, especially in patients who have suffered an underlying traumatic brain injury (TBI). Despite typically following a relatively benign course, the presentation of IH can mimic more serious neurological conditions, presenting with acute quadriplegia with cerebellar haemorrhage [10], and fronto-temporal dementia.[4]

We report a 67-year-old patient who sustained a head injury whilst on clopidogrel who developed bilateral acute subdural haemorrhages but remained neurologically stable. She developed IH induced coma following surgery. The aim of this case report is to raise awareness of secondary IH as a diagnosis in patients with TBI.

Case Report

A 67-year-old female patient was admitted onto the neurosurgical ward following a fall and head trauma. She initially complained of headache, episodes of confusion, and vomiting. However, her GCS remained 15 with no focal neurological deficit, and she had no signs of post-traumatic CSF rhinorrhea or otorrhea. She gave no history of trauma or pain in her spine. She underwent a computed tomography (CT) scan of her head that showed bilateral acute subdural haematomas (Figure 1). She was admitted into the high dependency bay on the neurosurgical ward for close neurological monitoring.

Three days later after initial conservative management, she became drowsy and dropped her GCS by 2 points. Clinical examination revealed pyrexia, and urine dipstick revealed a urinary infection, for which she received antibiotic treatment. She also underwent an interval CT head that revealed worsening of the mass effect of the haematomas. She subsequently underwent bilateral mini-craniotomies and evacuation of the haematomas and bilateral subdural drains were left in situ. Her immediate postoperative GCS was 15 with no focal neurological deficits. However, 4 hours later she was hypoxic and she de-saturated to 80%. Her neurological status deteriorated to a GCS of 5 (E1V1M3). Her pupils remained reactive to light. She suffered no postoperative complication related to her surgery.

She was intubated and ventilated. A chest radiograph revealed a total 'white out' of her left lung and she received an emergency bronchoscopy. She subsequently made a good respiratory recovery. She was slightly hypernatraemic at the time but this was treated successfully without any improvement to her neurological status. Blood cultures were sent and were negative. Despite the management of her medical comorbidities, her neurological status remained poor, with a GCS of 3, and reactive pupils. She underwent a brain MRI, which revealed inferior transtentorial herniation with significant compression of the midbrain and upper brainstem (Figure 2 C). Intracranial hypotension was suggested as a possible cause. An intraparenchymal pressure monitor (Integra® Camino®) (ICP) was inserted with an opening pressure of -4 mm Hg. She received a blind epidural blood patch at the level of L4/5 in the lumbar spine, and was placed in the trendelenberg position (initially 15 degrees head down) guided by ICP readings that were maintained between 10-15 mm Hg. Her GCS improved from 4 to 9 in the first 48 hours, and then to 14 by day 7 of trendelenberg positioning. As her neurological status improved and her ICP increased, she was gradually nursed in the supine position initially, then at 30 degrees, and finally 45 degrees. Once clinically stable, she was transferred to the ward where she continued her neurological rehabilitation and was discharged with good mobility. CT scan of her head before discharge demonstrated radiological improvement that correlated with the patient's clinical condition (Figure 3).

Discussion

Intracranial hypotension is an established neurological diagnosis with an incidence on of 5 per 100,000[2]. However, the actual figures are much higher due to misdiagnosis [9]. IH can present with a wide variety of symptoms and signs. Classically, patients with IH present with an orthostatic headache that starts after sitting or standing, which improves on lying supine. Other cardinal clinical features include nausea and vomiting, visual disturbances, and cranial nerve palsies[6]. Although rare, a decline in cognition has also been reported in patients with SIH[14]. The wide variety of symptoms associated with IH may contribute to the diagnostic challenge. In addition, the diagnosis may be further clouded by concurrent TBI or medical complications. In the present case, our patient developed severe respiratory failure requiring intubation and ventilation as well as an emergency bronchoscopy. A CT head (Figure 2 A) did not explain the drop in her GCS initially and it was thought to be due to respiratory compromise. However, the patient's neurological status was poor with a GCS of 3. Given that the CT scan of her head did not show high pressure, features a potential intracranial hypotension was postulated as a possible diagnosis. She underwent a prognostic MRI head (Figure 2 B) after her medical comorbidities had resolved. This revealed slumping of the brainstem and crowding of the foramen magnum. These features supported our theory of IH. Whilst we do not propose performing routine MRI scans at early stages post TBI, it may be worth considering early MRI scans in patients with unexplained poor neurological status, especially the elderly population, regardless of concurrent medical complications, to rule out IH.

Brain imaging studies is the most helpful non-invasive method of diagnosing IH. Radiological findings on MRI studies have been well described in the literature. In 2012, Loya et al reported MRI studies providing the definitive diagnosis of SIH in 90% of cases reviewed [6]. Typical radiological features of SIH on MRI studies can be classified as qualitative and quantitative signs. The most common qualitative sign is pachymengeal enhancement [5]. Other qualitative signs include increased blood volume within the venous system, most commonly dilatation of the inferior intercavernous sinus [1], and diffuse brain

swelling [7]. Quantitative signs include a mamillopontine distance of less than 5.5mm with a pontomesencephalic angle of 50 degrees or less [11]. These radiological features are due to a 'sagging' effect of the brain onto the cranial floor, leading to crowding within these areas.

Once diagnosed, patients may be managed conservatively or medically, depending on the severity of their symptoms. In patients who have failed conservative management, epidural blood patching (EBP) is a definitive method of managing intracranial hypotension. The efficacy of EBP has been well described in the literature[12]. First suggested by Thorsen in 1947[13], the mechanism of EBP is postulated to be due to a tamponade effect. Another method of treating SIH is placing the patient in trendelenberg position. Ferrante et al. reported treating SIH with a combination of EBP and trendelenberg position with a success rate of approximately 99% [3].

Conclusion

IH is not an uncommon neurological presentation that carries a fairly good prognosis. However, in the presence of an underlying TBI and medical complications, the diagnosis can be delayed. Clinicians should rule out secondary IH as a potential cause for poor neurological status in TBI patients, as immediate and prolonged treatment can lead to a profound clinical improvement.

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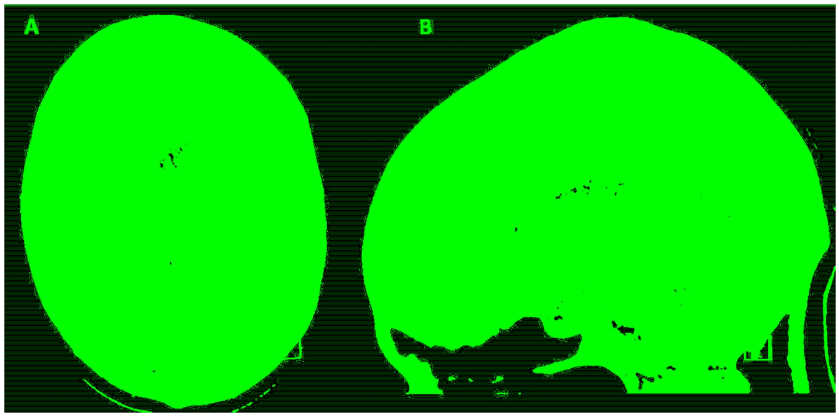
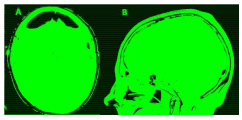
Figure legends

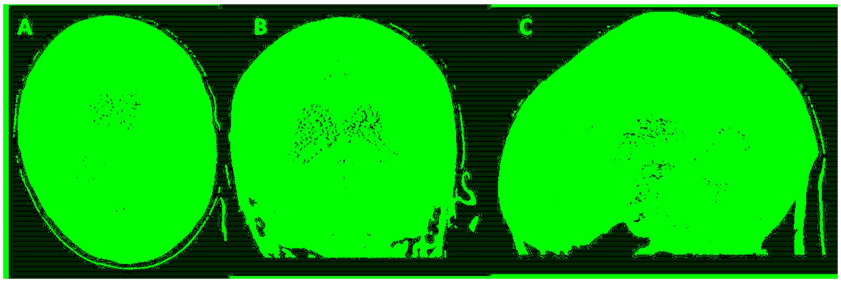
Figure 1 A Axial CT image demonstrates bilateral acute subdural haemorrhages (arrows) with mass effect. B A sagittal CT image shows patent foramen magnum and no inferior trans-tentorial herniation.

Figure 2 A Post-craniotomy (day 1 after patient deterioration due to chest infection/aspiration) axial CT images shows evacuated acute subdural haemorrhage and pneumocephalus. B Sagittal T2WI MRI reveals crowding at foramen magnum with inferior trans-tentorial herniation.

Figure 3 A Axial CT, B Coronal CT and C Sagittal CT head images demonstrate resolution of supra-tentorial mass effect and inferior trans-tentorial herniation.







Highlights summary

- Intracranial hypotension continues to be a misdiagnosed neurosurgical condition.
- The diagnosis of secondary intracranial hypotension is challenging, especially if superimposed by traumatic brain injury.
- Consider early MRI scans in patients with unexplained poor neurological status, regardless of potential concurrent medical complications, to rule out IH.
- Placing IH patients in the trendelenberg position can lead to an impressive and profound neurological recovery.

Abbreviations

CSF	Cerebrospinal fluid
CT	Computed tomography
EBP	Epidural blood patching
GCS	Glasgow coma scale
ICP	Intracranial pressure
IH	Intracranial hypotension
MRI	Magnetic resonance imaging
SIH	Spontaneous intracranial hypotension
TBI	Traumatic brain injury