Case Report

Differentiating the headache of cerebral venous thrombosis from post-dural puncture: A headache for anaesthesiologists

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ABSTRACT

Cerebral venous thrombosis (CVT) is a rare complication of lumbar puncture. Occasionally, the clinical picture of CVT may mimic post-dural puncture headache (PDPH) resulting in delayed diagnosis. A case of PDPH progressing to CVT is presented and the pathophysiology, diagnostic challenges and management options discussed in this article.

Key words: Blood patch, cerebral venous thrombosis, post-dural puncture headache

INTRODUCTION

Post-dural puncture headache (PDPH) has a benign course with most of the patients recovering completely with or without autologous epidural blood patch (AEBP). Very rarely PDPH can progress to cerebral venous thrombosis (CVT), especially in patients with hypercoagulable states.[1] The clinical presentation of CVT sometime mimics PDPH resulting in delayed diagnosis.[2] We report here a case of PDPH progressing to CVT, causing dilemma in diagnosis.

CASE REPORT

A 33-year-old male patient, American Society of Anesthesiologists physical status 1, underwent fissurectomy under spinal anaesthesia with a 25-gauge Quincke spinal needle (BD, Becton Dickinson S. A), in a single attempt. His post-operative period was uneventful, and he was discharged the next day. The patient returned on the fifth post-operative day with complaints of headache, which had started on the second post-operative day. The headache was present all over the head and was not associated with nausea, vomiting or any other neurological signs. It increased in intensity on assuming erect posture and was relieved on assuming supine position.

A diagnosis of PDPH was made, and AEBP was planned in the operation theatre. In the theatre, after establishing standard monitors, under all aseptic precautions and patient in lateral position, epidural space (at L_2–L_3) was identified with loss of resistance to air. Autologous blood (20 ml) was drawn aseptically from the left cephalic vein and was injected into epidural space after confirming negative aspiration. At the end of the epidural injection, the patient became apnoeic and unresponsive. Immediately, the position of the patient was changed supine and bag-mask ventilation was initiated. Within minutes, he became

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fully conscious and responsive, with no neurological deficit.

On evaluation over the next 24 h, it was found that the patient had only partial relief of headache. Next day, his headache increased in intensity and was orthostatic in nature. Although he had no other neurological symptoms, due to the transient unresponsiveness that followed the first AEBP, we did not attempt a second AEBP and decided to evaluate him with magnetic resonance imaging (MRI) of the brain.

Imaging revealed acute thrombosis of entire superior sagittal sinus and bilateral major cortical veins with features of increased intracranial pressure (ICP) (effacement of parietal lobe sulci) [Figure 1a and b]. Fundus examination revealed papilloedema. Therefore, the patient was started on low molecular weight heparin 40 mg twice daily. After 2 days, warfarin 3 mg was added to achieve a target International Normalised Ratio of 2–3. Repeat MRI brain showed resolving signs of CVT. There was complete resolution of the headache. Protein C, Protein S and antithrombin III levels were within normal limits.

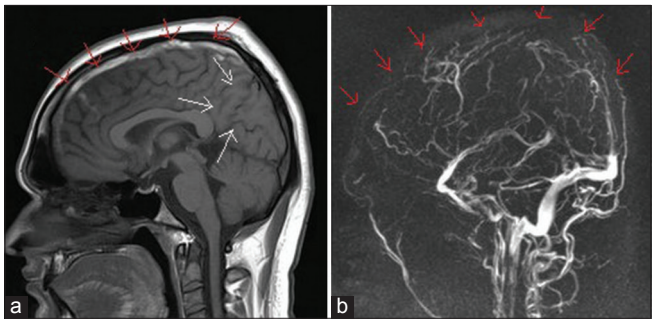

Figure 1: (a) Sagittal T1-images show absence of flow void with hyperintense signal in superior sagittal sinus (b) magnetic resonance imaging venography confirms superior sagittal sinus thrombosis

DISCUSSION

The clinical manifestations and severity of CVT are highly variable and are often confused with the relatively benign PDPH [Table 1]. With the progression of PDPH to CVT, the headache usually loses its postural relationship and becomes continuous. However, even this may be variable as the change in the pattern of headache occurred only in 40–78% of patients in different surgical settings.[1] Therefore, until features of raised ICP (seizures, papilloedema) supervene, it may be difficult to differentiate between the two conditions based on the pattern of headache alone.

Although AEBP is one of the treatment modalities in PDPH, it may be harmful if administered to patients with CVT, as it may further increase the ICP.[2,5] The intrathecal and epidural spaces become non-compliant with the loss of CSF following LP.[6] Hence, injection of AEBP can lead to raised ICP causing transient cord compression, as probably happened in our patient, who became transiently unresponsive after the AEBP.

Table 1: Comparison of post-dural puncture headache and cerebral venous thrombosis

Features	PDPH	CVT
Risk factors	Young women particularly during pregnancy, past history of PDPH, LP using larger size needles[3]	Pregnancy, oral contraceptive use, prothrombotic conditions like protein C and S deficiency, factor V Leiden mutation, etc.[2]
Pathophysiology	Lowering of CSF pressure causes traction on the pain sensitive intracranial structures causing headache Loss of CSF produces a compensatory intracerebral venodilatation (Monro-Kellie doctrine), producing headache[3]	CSF volume and pressure are reduced after LP, resulting in venous volume expansion (Monro-Kellie doctrine).[1] Also, the blood flow velocity can be reduced in the sinuses after LP[4] predisposing to CVT
Symptoms	Headaches the major complaint. It is exacerbated by adoption of upright posture and relieved by lying down. Pain is more over the frontal and occipital areas radiating to neck and shoulders. Other symptoms include nausea, vomiting, hearing loss, tinnitus, vertigo, dizziness and visual disturbances[3]	The main complaint is headache. Other signs include papilloedema, focal neurological deficit, seizures, coma.[2] With the progression of PDPH to CVT, the headache loses its postural relationship and becomes continuous. Rarely persistent orthostatic headache can be seen[1]
Investigations	Clinical diagnosis with history of dural puncture. In doubtful cases, diagnostic LP may show low CSF opening pressure, rise in CSF protein and lymphocyte count[3]	MRI of the brain with venography. Protein C, Protein S and antithrombin III levels to rule out hypercoagulable conditions
Management	Supportive therapy such as supine posture, rehydration, acetaminophen, non-steroidal anti-inflammatory drugs, opioids, and anti-emetics. Epidural blood patch, caffeine, triptans, abdominal binder[3]	Anticoagulation, ICP reducing measures

PDPH – Post-dural puncture headache; CVT – Cerebral venous thrombosis; LP – Lumbar puncture; CSF – Cerebrospinal fluid; MRI – Magnetic resonance imaging; ICP – Intracranial pressure

CONCLUSION

The recognition and differentiation of CVT from PDPH is a clinically daunting process and both have radically different treatment options. Although the inadvertent use of AEBP did not affect the clinical outcome in our patient, the procedure may be implicated in case of development of neurological deficits.

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Conflicts of interest

There are no conflicts of interest.

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