Case report

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Undiagnosed and Untreated Postdural Puncture Headache Following Myomectomy under Subarachnoid Block: A Case Report

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Abstract:

Postdural puncture headache can be a complication of either unintentional or intentional dural puncture. Although the incidence is low, it could arise from subarachnoid block. This case report describes the management of a 6-month history of severe unrelenting, intractable headache with accompanying photophobia following a myomectomy performed under spinal anaesthesia. Twenty millilitres of autologous blood was taken from the patient and aseptically administered into the epidural space. Following this, a transient tonic-clonic seizure with loss of consciousness was observed but was immediately controlled. This case report highlights the possible complications that could follow the administration of an autologous epidural blood patch.

Keywords: epidural blood patch, postdural puncture headache, seizure, spinal anaesthesia

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Introduction

Postdural puncture headache (PDPH) is a major complication that can accompany a subarachnoid block or an accidental dural puncture (ADP). PDPH is easily described by the International Classification of Headache Disorder as a headache occurring within 5 days of an intentional or unintentional lumbar puncture. It is aggravated by standing or sitting and relieved by lying down[1]. This type of headache occurs more frequently in women and has a strong relationship with significant loss of cerebrospinal fluid (CSF) through dural tears. The incidence of PDPH varies from 6 to 40% depending on age, gender, and needle size[2,3].

Both modifying and non-modifying factors can influence the incidence of PDPH. Some of the modifying factors include needle size, needle design, bevel direction, stylet replacement, operator experience, and number of LP attempts, while the non-modifying factors include gender and age. The most commonly used spinal needle size is 25G, which may be pencil point (atraumatic) or bevelled (traumatic). The traumatic needle type is more frequently associated with PDPH.

The goal of this study was to highlight an undiagnosed and untreated PDPH that reduced the quality of life in a patient who had a myomectomy 6 months before presentation in our facility.

Case report

Following the approval of the hospital's Ethics and Research Board, we report the case of a 49-year-old female who was seen in the outpatient department with a 6-month history of severe, persistent headache, insomnia, dizziness, and weakness. The headache began on the first post-operative day after a myomectomy under spinal anesthesia at another hospital, but the surgeon reassured her about its transient nature. However, it persisted and became more severe, leading to her return to the hospital after her initial discharge. The headache continued to worsen, resulting in complete incapacitation of the patient, who was unable to leave her bedroom and developed severe photophobia.

Examination revealed an ill and anxious-looking patient who was unable to rise from bed due to a severe headache on attempting to do so. She weighed 85 kg and measured 168cm in height (BMI=30.1). Vital signs: Pulse: 110/min, BP: 146/81 mmHg, Temp: 37.1 ^{o}C, Resp: 20/min, SpO_2: 98%.

There was no pallor, fever, or icterus, hydration was satisfactory, and there was no peripheral edema. She was conscious, alert, and oriented in time, place and person with a Glasgow Coma Score of 15. Cranial nerves could not be properly assessed due to the patient's extreme discomfort and photophobia. Deep tendon reflexes were globally normal and power was grade 5 in all limbs.

She had been on over-the-counter medications, including diclofenac 50mg, paracetamol 1 gram every 8 hours for the headache with no relief. Her quality of life continued to depreciate as she was unable to return to work and could not carry out regular home chores. History suggested that she was prostrate throughout a 24-hour period in order to secure some relief from the headache, which was worse when standing or sitting down. The patient rated the headache as severe (7-9/10 on the Numeric Pain Rating Scale (NPRS). She was not a diagnosed hypertensive or diabetic, and there was no history of head trauma. Other causes of headaches such as hypertensive emergencies, acute hydrocephalus, dural sinus thrombosis, intracranial haemorrhage, acute angle-closure glaucoma, medication overuse headache, space-occupying lesions, tumours, abscesses, cysts, and others were excluded. An ophthalmologist's consultation was sought to exclude some of these differentials. Findings from fundoscopy were essentially within reference ranges.

On arrival at our facility, her baseline vital signs, pulse, blood pressure, peripheral oxygen saturation, and

electrocardiographic tracing were essentially normal. A request for a blood film showed normal cells and there were no malaria parasites observed. Further investigations, including Brain computerized tomogram (CT) (in the figure below), showed the cerebral parenchyma to be normal in morphology and density with normal grey-white matter differentiation. There was no intra- or extra-axial mass lesion seen and the sulci and gyri were within normal limits. The ventricular system was normal and the cerebellum and brain stem were also unremarkable. Following a normal study of all investigations, a diagnosis of PDPH was made. The patient was then prepared for an elective epidural blood patch in the operation room after consent for the procedure was obtained.

Ringer's lactate 1000 ml was administered intravenously over 20 minutes with further fluid therapy based on patient need. Her vital signs were measured and recorded and thereafter measured every 5 minutes until the end of the procedure. She received a prophylactic intravenous antibiotic (ceftriaxone 1 gram).

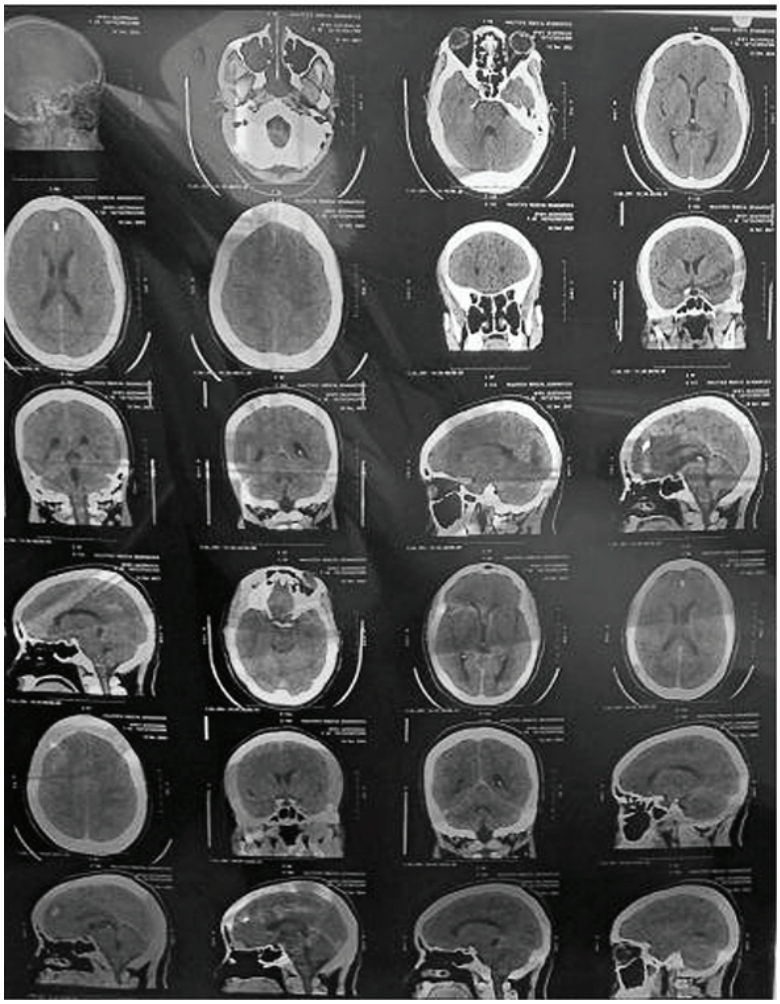

Figure 1 Computerized tomogram of the patient's brain

The patient was positioned sitting with her legs resting on a stool. Her back was cleaned with povidone iodine in alcohol and covered with a sterile drape. The entry point of the Tuohy needle was infiltrated with 3 ml of 1% lidocaine at the L2/L3 intervertebral space. An 18-G Tuohy needle was introduced through the L2-L3 interspace into the epidural space using the loss-of-resistance to air technique with the Ogan's slingshot epidural syringe[4].

With the assistance of a second anaesthetist, 20ml of the patient's blood was withdrawn from her antecubital vein under aseptic conditions and handed over to the administrator of the patch. The blood was injected at the rate of 0.5 ml/min into the epidural space, pausing after every 5 ml of injected autologous blood to assess any discomfort while continuous monitoring was undertaken on the multiparameter monitors. The Tuohy needle was withdrawn after injecting the blood into the epidural space, and a light dressing was placed over the puncture site. While the index patient was being returned to the supine position, she became unconscious and was observed to be having seizures. While her blood pressure was slightly higher than the baseline values, she developed severe bradycardia with a pulse rate of 45 beats per minute from 68 beats per minute. Her respiration remained spontaneous but fairly regular throughout this period. The seizure was aborted with diazepam 5 mg, atropine 1mg and oxygen supplementation administered for about 15 minutes. Following this, she returned to consciousness within 15 minutes and maintained a normal pulse rate, blood pressure and peripheral saturation of oxygen. Through verbal contact, the patient confirmed resolution of the headache and was later returned to the ward for further monitoring of vital signs. She was counseled to lie still in bed for 48 hours after the epidural blood patch. In the post-patch period, she rated the pain as zero on the NPRS.

About 12 hours later, she developed a severe frontal headache, severe bradycardia (pulse: 45 beats per minute) and some episodes of vomiting. She received 500 ml of intravenous 20% mannitol over one hour, and intravenous stat doses of ondansetron 8 mg and atropine 1mg. Hydrochlorothiazide 25 mg was also administered orally. Following these medications, the headache subsided. She was subsequently placed on oral pregabalin 75 mg every 12 hours, hydrochlorothiazide 25 mg daily, and paracetamol 1gram every 8 hours. She was discharged from the clinic on the third day post-treatment without any neurologic deficit after a detailed review by a neurologist. She was however, instructed to call the hospital's or the anaesthetist's phone number immediately should she develop a fever, new headache, stiff neck, tingling, weakness or numbness in her legs. The patient was seen again 14 days later and was found to be headache free. She was reviewed in the clinic a month post-procedure and found happily headache free.

Discussion

PDPH remains a nightmare to the patients who experience it, as most of the time it defies every conventional relief. There is an acute decrease in intracranial CSF pressure owing to the extraction of larger volumes of CSF, triggering meningeal vasodilatation and positional traction on intracranial structures.[5] Relief should thus aim at stopping further leakage or loss. The principle of a successful epidural blood patch (EBP) treatment could depend on either mass effect or epidural plug formation or both.

The mass effect theory suggests that an increase in the spinal compartment following an injection of blood into the epidural space causes the CSF to be displaced to the more cephalad cranial compartment. The sustained increased pressure on the thecal space for a few hours could explain the rapid resolution of the headache[6, 7].

Epidural plug theory supposes that the formation of a gelatinous plug, induced by interactions between the injected blood and procoagulant components in leaking CSF, seals off the dural tear until the natural healing process restores

the integrity of the torn dura. MRI studies have found that epidural blood adheres to the thecal sac, resulting in clot formation for 18–24 hours[3].

In the index patient, the diagnosis could have been made and treated with either the use of liberal oral fluids or caffeine and bed rest. Ignorance or lack of patient information could have contributed to the prolonged loss of cerebrospinal fluid. This CSF loss continued for 6 months, and the patient could not relate the intractable headache to CSF loss and probably out of ignorance, did not seek clinical assistance.

It is possible that either a larger spinal needle, poor needle design, or multiple attempts at dura puncture were made since the patient was moderately obese. While one study showed that multiple attempts at dura puncture were significantly associated with PDPH[5], another study found that there was no significant difference observed in the incidence of PDPH between a single shot attempt and 2 or more attempts[8].

One remarkable event that occurred during the procedure was the tonic-clonic seizures observed in our patient. Transient seizures associated with EBP are not frequently reported. Therefore, an explanation for an epidural blood patch causing seizure may be difficult to elucidate. Blood in the subarachnoid space has not been associated with seizures. However, a potential explanation could be that the brain became used to producing increased amounts of CSF in an attempt to compensate for the massive loss. When, however, the loss was stopped, there could have been an upsurge of the intracranial pressure caused by possible increased compensatory CSF overproduction. This compensatory overproduction could significantly increase the ICP, hence the seizures and severe bradycardia observed in our patient, which necessitated the administration of 500 ml 20% mannitol over one hour and a repeat dose about 12 hours later. Although treatment was empirical, a return to normal vital signs, such as blood pressure, pulse rate and regular breathing patterns, was assumed to be a successful treatment of increased ICP in this patient.

Complete seizure freedom is the ultimate goal of any anti-seizure treatment. Pregabalin is a drug which is rapidly and almost completely absorbed from the gastrointestinal tract with peak plasma concentrations within 1 hour of intake. It was used extensively in controlling pain and seizures in our patient[9]. At a dose of 75 mg, no further seizure was observed after the initial episode.

Our case report has some limitations: Firstly, we could not ascertain the cause of the PDPH from only the patient's history, and there was no other clinical information on the difficulties encountered in the conduct of anaesthesia during the myomectomy. Any of the risk factors for PDPH still have a common pathway in the clinical manifestation and possible treatment. Secondly, we could not assess the increase in intracranial pressure as we could not reach a measuring instrument immediately. The clinical features of an increased ICP were relied upon in the management of our patient.

Conclusion

Epidural blood puncture is indicated in extreme cases of refractory PDPH where other conservative methods have failed. This procedure is occasionally associated with complications that require expertise in its management. One such complication is the occurrence of short episodes of seizures. In preparation for elective EBP, the anaesthetist should be adequately prepared in order to manage this life-threatening clinical state.

Conflict of interest

The authors do not have any conflicts of interest to disclose. The patient does not want to be identified.

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