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Case Report

Headache after dural puncture subarachnoid hemorrhage posing as meningitis

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ABSTRACT

Headache after neuraxial blocks is a common complication. This case discussion highlights an important diagnostic dilemma when a sentinel subarachnoid hemorrhage mimics meningitis after spinal anesthesia. A patient presented with features of meningismus after spinal anesthesia. To confirm diagnosis, a second dural tap was done which resulted in a frank sub-arachnoid hemorrhage. This correspondence highlight importance of early vascular imaging when a patient presents with atypical headache after a dural puncture.

Keywords: Meningitis, Post dural puncture headache, Precipitating factors

INTRODUCTION

Headache after neuraxial blocks is a common complication.[1] This case discussion highlights an important diagnostic dilemma when dealing with an atypical headache after spinal anesthesia. Differential diagnosis of headache after dural puncture includes post dural puncture headache (PDPH), migraine, drug induced headache, meningitis as well as some rare complications like sub arachnoid hemorrhage, sinus thromboses etc.[2] Ironically, often, a second dural puncture is needed to confirm diagnosis of a complication of a previous puncture. This second procedure, may now trigger a catastrophe as we noted in this case.

CASE REPORT

A 16 years old male, American Society of Anesthesiologists risk grade 1, was operated for right sided inguinal hernia repair. Spinal anaesthesia was administered taking all aseptic precautions, using a 25-gauge Quinke's spinal needle at L3-L4 space in single attempt. After confirming free flow of cerebrospinal fluid, 2.5ml of 0.5% bupivacaine heavy was administered. Intraoperatively, hemodynamics was monitored and maintained. Patient was discharged on 3rd post op day (POD) without any complaints. On POD 5, patient presented to the surgeons with complaints of moderate fronto-temporal headache which was postural in nature. The patient was referred to anaesthesia department where patient was counseled regarding post dural puncture headache (PDPH) and advised rest, NSAIDS and plenty of oral fluids. After 2 days, on POD 7, patient came with increased severity of headache and nausea. Patient was admitted and neck rigidity was noted on admission. A non-contrast CT brain revealed no abnormalities. Diagnostic lumbar puncture was done to rule out meningitis. The report mentioned a total cell count of 1000.00 cells/microliter (polymorphs 52% and lymphocytes 48%, occasional erythrocytes). CSF glucose level was 52mg/dl when patient's serum glucose level was 128mg/dl and protein levels were 57.3 mg/dl. Appearance of CSF was clear but on culture, it grew Acinetobacter. A presumptive diagnosis of meningitis was made and patient started on intravenous antibiotics, dexamethasone and inj. mannitol on which he showed improvement in symptoms. Patient requested discharge

against medical advice after two days. By this time, he had shown symptomatic improvement and he was advised daily attendance in the hospital and thrice daily intravenous Inj. Ceftriaxone 2 gm. Patient did not turn up for the next two days. When he next presented to us, patient had stopped the antibiotics and was in an altered sensorium with rigidity of whole body and vomiting. An urgent CT scan was done which showed frank subarachnoid hemorrhage with mild hydrocephalus. MRI was also done for the patient which was suggestive of features of vasospasm. Patient was kept in ICU started on nimodipine and managed as a SAH. Condition of the patient stabilized and once symptomatic improvement was evident, patient was shifted out of the high dependency unit by day 7 after second admission. The patient could be discharged from hospital after three weeks' stay at the hospital, with no neurological deficit.

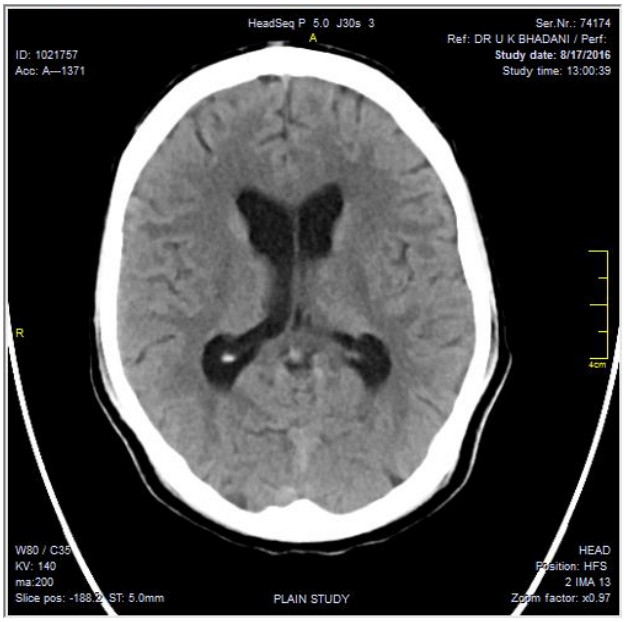

FIGURE 1: CT scan of patient with subarachnoid haemorrhage after dural puncture at the time of discharge without neurological sequelae.

DISCUSSION

Leakage of cerebrospinal fluid (CSF) following lumbar puncture is usually of little clinical significance and seldom gives rise to any symptoms. In literature, PDPH has been described to begin typically between days 1 and 7, though we also came across a report for onset of PDPH on day 12.[1] It is postulated that a potentially continual source of CSF leak leads to low CSF pressure with pulling on the dura and bridging veins, causing post dural puncture headache (PDPH).[2] If this headache is left untreated, the shearing forces can lead to venous tears and acute or chronic subdural hematomas. Such patients with prolonged PDPH who become treatment resistant lose the postural aggravation typical of PDPH. Neurological symptoms and signs of intracranial hemorrhage then develop. Spontaneous SAH is a rare event, ruptured intracranial aneurysm being the main cause (51-80%) followed by hypertensive diseases (10-15%) and arteriovenous malformations (5-10%).[3] In about 10% of spontaneous SAHs, no causative lesion can be identified even on an angiogram. Very few reports describe intracranial hemorrhages after dural puncture and most of these are spinal subdural hematomas.[4,5] Authors have postulated that low CSF pressure exacerbated by a second dural puncture can trigger intracranial hemorrhage even without PDPH. A decrease in intracranial pressure could cause an increase in transmural pressures across the arterial wall, facilitating a rupture of any concomitant vascular malformation.

In our patient, lumbar puncture for providing spinal anaesthesia and a second diagnostic lumbar puncture, led to repeated decreases in CSF pressure and hence repeated increases in transmural pressure and wall stress of a probably pre-existing vascular malformation. CSF exam after first lumbar puncture showed no xanthochromia although it takes time to develop after SAH and it is possible that our CSF was sampled too soon after the onset of headache.

Small sentinel SAH may have erythrocytes in the CSF. Distinguishing a traumatic lumbar puncture from SAH is sometimes problematic. There are no accepted numbers of erythrocytes in the CSF that distinguish the two although authors believe subarachnoid hemorrhage should be considered when there are more than 400 cells per mm^3. A case report of an acute headache due to hemorrhage of an aneurysm found 3467 per mm^3 erythrocytes in the CSF.[2] The total cell count in our patient was 1000 per mm.[3]

Sequence of events as we believe in our patient were as follows. Post spinal anesthesia, due to reasons which have been dealt earlier, there was a small SAH. Sentinel signs were present as headache with which he presented to us. CT scan failed to pick up or missed the early SAH and did not reveal significant intracranial pathology. A second lumbar puncture was done to rule out meningitis. Now the CSF and clinical picture mimicked meningitis. Subarachnoid CSF findings may mimic those of meningitis as was seen in a case report earlier.[2]

A second lumbar puncture done to rule out meningitis, then precipitated a frank SAH with clear sign and symptoms. SAH was then diagnosed on CT scan. After the second dural tap, patient deteriorated due to CSF leak leading to fall in ICP which caused increase in transmural pressure across what we believe may have been a pre-existing undiagnosed vascular malformation, resulting in its rupture. Though the catastrophe was life threatening, we could manage this patient and he was discharged from hospital without any neurological sequelae. The 3rd CT scan done at time of discharge showed significantly resolved subarachnoid hemorrhage as may be seen in figure.

CONCLUSION

In conclusion, this case highlights possibility of a subarachnoid hemorrhage as an atypical cause of post spinal headache. Sentinel or a small SAH which mimics PDPH and meningitis forces us to go for a second lumbar puncture which, as we have seen in our case may be catastrophic. High index of suspicion and early vascular imaging may be warranted in patients with atypical headaches or altered consciousness after lumbar puncture.

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