



SUBARACHANOID HEMORRHAGE FOLLOWING SPINAL ANAESTHESIA IN CASE OF UNKNOWN CEREBRAL ANEURYSM

Anaesthesiology

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ABSTRACT

The overall incidence of subarachnoid hemorrhage (SAH) is approximately 9/100 000 person-years and in which incidence in women is 1.24 times higher than in men. The exact incidence of SAH after spinal anesthesia is not known, though few cases are reported in the literature. The usual presentation of SAH following subarachnoid block (SAB) is after one or two weeks and most of the cases have history of multiple pricks during SAB with headache. We report a case of SAH following an SAB for total abdominal hysterectomy, presenting within nine hours after SAB with atypical symptoms of hypertension and generalized tonic clonic convulsions. Non contrast CT and MRI showed parafalcine grade I subarachnoid hemorrhage with vasogenic edema and venous infarcts. Subsequently it was found that patient had been suggested to undergo clipping for cerebral aneurysm ten years back. Early diagnosis with aggressive management prevented further complications in our case. Subarachnoid hemorrhage following lumbar puncture can present within 24 hour with atypical presentation. High suspicion index and further management without much hemodynamic variability can prevent fatal complications.

KEYWORDS

Subarachnoid Hemorrhage, Spinal Anaesthesia, Cerebral Aneurysm, Early Diagnosis

INTRODUCTION

The overall incidence of subarachnoid hemorrhage (SAH) is approximately 9 per 100 000 person-years and in that incidence in women is 1.24 times higher than in men [1]. Though spontaneous SAH is a rare event, ruptured intracranial aneurysms being the main cause (51–80%), followed by hypertensive disease (10–15%) and arteriovenous malformations (AVM) (5–10%)[2,3]. The actual incidence of SAH following spinal anesthesia is unknown as the SAH with small hematomas are asymptomatic and remain unreported. The incidence of spontaneous SAH is estimated to be five-fold more in parturient patients compared with the non-pregnant population due to physiological changes in pregnancy but data is not reliable as it is drawn from reported cases only[4]. The literature regarding SAH after spinal anesthesia in non pregnant patients is very limited. Also the usual presentation is after one or two weeks later or following multiple pricks for spinal anesthesia [5]. We are reporting a case, who presented with atypical signs and symptoms of subarachnoid hemorrhage within 24 following uneventful spinal anesthesia.

CASE REPORT

A 40 year old patient, American society of Anesthesiologist (ASA) physical status II with fibroid uterus was planned for elective total abdominal hysterectomy. Patient was known case of hypothyroidism for 5 years, on tablet eltroxin 100 mcg OD and currently euthyroid. There was no other significant history. All investigations were within normal limits. Standard ASA fasting and monitoring protocols were followed. Under all aseptic precautions, spinal anesthesia was given in L3-4 interspace with 26 G quincke needle in single attempt in sitting position with 16.5 mg of 0.5% bupivacaine (heavy) with 30 mcg of clonidine, total volume of 3.5 ml. Maximum sensory level achieved was T4. Intraoperative all vitals were stable. The fall in mean arterial blood pressure (MAP) was never more than 20% of baseline. Patient was started on paracetamol infusion for post operative pain relief. Almost 9 hours following time of SAB, patient had one episode of generalized tonic clonic convulsions (GTCS) and was managed with lorazepam 4 mg IV and phenytoin 10 mg/kg IV. In postictal phase patient was hemodynamically stable with no sign symptoms of aspiration with Glasgow coma scale (GCS) of 15/15. There was no focal neurological deficit other than the presence of post seizure aura. Complete blood count (CBC), serum electrolytes, coagulation profile were sent which were within normal limits. Following this patient was planned for CT brain. During this period, patient was monitored for neurological deterioration and vital signs. The blood pressure started rising and from 130/90 reached to 160/120 within one hour. Labetolol infusion at 10mg/hour was started but there was persistent rise in blood pressure, reaching to 180/150. Following this patient developed one

more episode of GTCS, which responded to lorazepam. NTG infusion at 5mcg/Kg/min was started and patient was shifted for CT scan with all drug infusions and monitors. Non contrast CT scan showed grade I unilateral parafalcine subarachnoid hemorrhage. Thereafter patient was started on nifedipine and phenobarbitone and MRI was advised. MRI brain showed vasogenic edema with venous infarcts and SAH with posterior reversible encephalopathy syndrome. While disclosing the report to patient's attendants, it was found that she had been diagnosed with cerebral aneurysm 10 years back. Patient was asymptomatic and no treatment was taken for that. After 5 days of conservative management she was referred to neurosurgeon for clipping and further management.

DISCUSSION

Rupture of intracranial aneurysm is the most common cause of spontaneous SAH[2]. Transmural pressure (TMP) of an aneurysm changes depending on MAP and intracerebral pressure (ICP). Extremes of changes on either side can lead to rupture of aneurysm[5]. The leakage of CSF following SAB is usually of a minor degree and asymptomatic. Loss of CSF due to repeated dural punctures can cause dangerous reduction in ICP, leading to rupture of aneurysm[5]. The leakage of CSF through dural hole continues over a period of time which leads to significant increase in TMP, manifesting as SAH from 48 hours to three weeks [6].

Our patient without having history of multiple dural punctures manifested within unusual short time of 9 hours.

Previously weakened vessels can rupture with small blood pressure changes even before autoregulation becomes effective. In our patient the return of sympathetic tone causing rise in MAP could have resulted into rupture of weakened vessel.

The classic presentation of aneurysmal SAH is that of a very severe headache associated with neck stiffness, photophobia, nausea, vomiting, and often with transient loss of consciousness. [7] But our patient presented with seizure and hypertension not headache. The possibilities for this presentation could be hypertensive encephalopathy, electrolyte disturbances or any kind of intracranial pathology. Serum electrolytes were within normal limits and rise in blood pressure was noticed after first episode of seizures. Also around 3-26% patients can present as seizure on onset of aneurysm rupture. [8] Cerebral pathologies like ischemia and hemorrhage are almost always accompanied by increase in blood pressure of at least 10% due to alteration of the self-regulatory mechanisms induced by vasoactive substance release at the injury site. [9] In our patient sudden rise of

blood pressure may be secondary to rise in intracranial pressure. Non-contrast CT within early hours of injury was helpful in diagnosis of SAH.

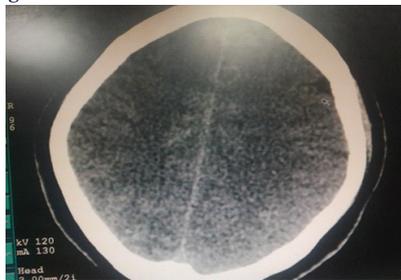
As with this case the presence of cerebral aneurysm was missed during preoperative assessment. Whether this was due to deliberate secrecy on patient's part or failure of anesthesia team is not certain. The principal aim when dealing with intracranial aneurysms is to avoid increase in TMP, maintaining adequate CPP to prevent ischemia, while also controlling excessively high blood pressure to prevent aneurysm rupture and further bleeding. General anesthesia should be given avoiding major fluctuations in blood pressures.

There is no absolute contraindication for giving regional anesthesia. [10]

Epidural anesthesia may be given with expert hands. In our case timely diagnosis and aggressive management prevented further complications.

In conclusion subarachnoid hemorrhage following lumbar puncture can present within 24 hour with atypical presentation. High suspicion index and further management without much hemodynamic variability can prevent fatal complications.

NCCT SCAN showing grade 1 parafalcine subarachnoid hemorrhage



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