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Case Report

Subarachnoid haemorrhage following spinal anaesthesia– A dreaded complication

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ABSTRACT

We present a female patient posted for fixation of fracture femur under subarachnoid block. After spinal anaesthesia she developed hypotension and cardiac arrest. Subarachnoid haemorrhage was diagnosed by CT scan of head when she did not regain the consciousness. Inspite of all the efforts she had a fatal outcome. We discuss the probable causes of subarachnoid haemorrhage after a simple procedure like spinal anaesthesia with an unusual presentation.

1. Introduction

Spinal anaesthesia is generally a safe & effective procedure. Subarachnoid haemorrhage has been reported as a complication of lumbar spinal anaesthesia. Though rare it is a life threatening complication which requires urgent diagnosis and therapy. Eighty five percent of cases of atraumatic subarachnoid haemorrhage result from a ruptured aneurysm.[1] The prevalence of aneurysms in the general population is roughly 2-5% greater in those with family history of Ehler-Danlos or polycystic kidney disease.[2] We report here a case of acute subarachnoid haemorrhage following subarachnoid block though diagnosed early the patient had an adverse outcome.

2. Case Report

A 60-year old female victim of fall presented to the emergency. The patient had sustained a fracture of shaft of femur. She was haemodynamically stable and systemic examination revealed no other abnormal findings. There was history of focal epilepsy for which she was well controlled on regular treatment. The Computerised tomography (CT) scan of the head showed granuloma, old infarcts and brain atrophy as shown in Figure 1 done in the pre-operative period as advised by neurophysician. Any active intervention was ruled out. All the laboratory investigations were within normal limits but the Electrocardiograph (ECG) showed inferior wall ischaemia. However the Echocardiography done showed no abnormality and no active intervention was required from cardiology point of view. The patient was posted for cannulated compression screw (CCS) for fracture of femur after 5 days. In the operation theatre the patient was fully conscious and oriented to time, place and person. Pulse, Blood pressure, Respiratory rate were within normal limits.

Inside the operation theatre monitoring included 5 lead ECG, Non-invasive blood pressure (NIBP) and Oxygen saturation (SPO2). After adequate preloading spinal anaesthesia was given at the level of L3-4 space using a 26G Quinke's spinal needle taking standard aseptic precautions in the sitting position. At the first attempt clear Cerebrospinal fluid(CSF) was obtained & 3ml of 0.5% Bupivacaine (Heavy) was injected. After 10minute sensory level of T6 was achieved and the patient was handed over to surgeon for cleaning and draping. After about 10minute interval patient complained of sudden headache,

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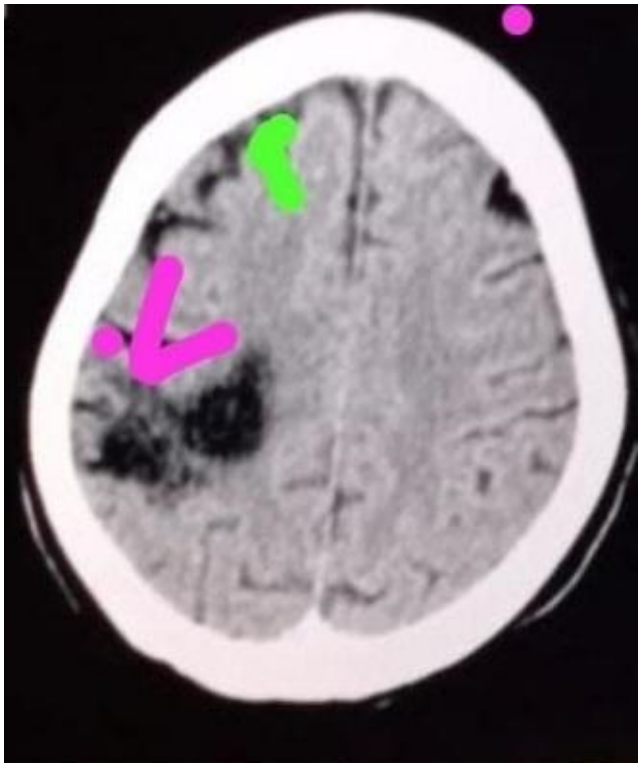

Fig. 1: Showing old cerebral infarct and atrophy of right cerebral hemishpere

nausea and monitor showed systolic BP of 70mm Hg. Immediately Inj. Mephenteramine 6mg was injected and the blood pressure came back to 110/76mm Hg after some time. The patient again developed hypotension with severe bradycardia followed by cardiac arrest. She was given inj Atropine 0.6mg three times and inj Epinephrine 1mg. The cardiopulmonary resuscitation(CPR) was immediately started after intubating the patient. After some time the patient developed spontaneous breaths along with normal vitals but the level of consciousness was not appropriate, suspecting it to be some neurological event (as patient was known case of epilepsy), the surgery was postponded for time being. The patient was shifted to the intensive care unit. The blood sugar levels, arterial blood gas analysis and serum electrolytes level were within normal limits. The patient was subjected to CT scan of the head as she did not regain the consciousnesss, which showed subarachnoid haemorrhage with midline shift as shown in Figure 2. The patient was shifted to a higher centre for neurosurgical intervention but she succumbed to death before she could be operated upon.

3. Discussion

Cranial hypotension and long lasting cerebrospinal fluid (CSF) loss may distract cerebral bridging veins that can easily rupture and lead to acute or chronic subdural or subarachnoid haematoma.[3] Bradycardia & cardiac arrest

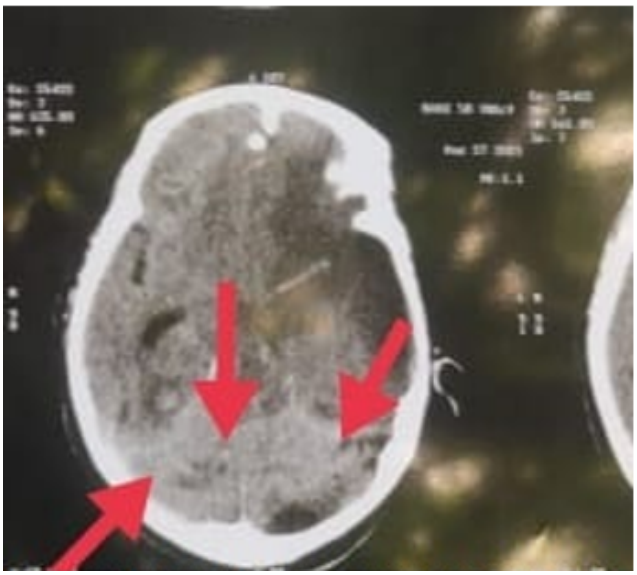

Fig. 2: Showing subarachnoid bleed

are the most worrisome complications related to spinal anaesthesia. The influence of cardio-acceleration fibres originating between T1-4 plays a crucial role in maintaining blood pressure & heart rate according to the level of anaesthesia induced by spinal block, depleted vascular volume or insufficient replacement with fluids & the presence of deep sedation is considered a risk factor for bradycardia and cardiac arrest. Surgical intervention may also trigger bradycardia and cardiac arrest by vagal discharge or embolization. Early administration of epinephrine is important in case of severe bradycardia especially in unresponsive cases to atropine and ephedrine that should be administered previously.[4]

In our case the patient was given three shots of atropine after which a dose of epinephrine was given along with cardiac massage which was successful. The cause for bradycardia and cardiac arrest was probably higher level of spinal anaesthesia achieved leading to blockade of cardio-accelerator fibres. Although the spontaneous breaths were there after sometime along with normal vitals the level of consciousness was poor. Suspecting it to be a neurological insult the CT head was done which revealed a subarachnoid haemorrhage. There were old changes of cerebral infarct and brain atrophy.

Spontaneous subarachnoid haemorrhage is a rare event, ruptured intracranial aneurysm being the most common cause (51-80%), followed by hypertensive disorders [10-15%] and arterio-venous malformations (5-10%). Rare causes include meningeal infection, tumors and blood dyscrasias.[5,6] In 5-10% of spontaneous SAH, no causative lesion can be identified.[6,7] Computed tomography is a highly effective method of detecting subarachnoid blood if performed early after aneurysmal rupture, being 95 to 98%

positive when lumbar puncture is positive.[6]

Atrophy in specific brain areas correlates with poor neuropsychological outcome after subarachnoid haemorrhage.[8]

Loss of CSF from puncture site with a subsequent decrease in cerebrospinal fluid(CSF) pressure and an increase in transmural wall tension of the vessels might be predisposing factors for the rupture of a pre-existing cerebral aneurysm.[9] Although the CT scan head done showed the presence of subarachnoid haemorrhage the aneurysm detection requires a CTangiogram which could not be done in our patient. The sensitivity of CTA is 92.3% for aneurysm <4 mm and in contrast to pathologies where the size of lesion correlates with the severity of disease (i.e pulmonary embolism), a small ruptured cerebral aneurysm can still lead to significant morbidity and mortality.[10]

Aneurysmal pathophysiology has been theorized to involve congenital weakness in the vessel wall or degenerative changes resulting in destruction of elasticity of the vessel wall at points of high turbulence such as bifurcations.[11] The pre-existing findings of the brain in addition to the subarachnoid bleed in the patient probably lead to rapid deterioration with mortality in our patient.

4. Conclusion

It is important for the anaesthesilogists to recognise the complications of subarachnoid block promptly to avoid any delay in treatment and fatal consequences.

Any patient who doesn't regain consciousness after high spinal anaesthesia or witnessed cardiac arrest in operation theatre should be subjected to neurological screening. Prompt intervention by the neurophysician and neurosurgeon could save the life of a patient.

5. Source of Funding

None.

6. Conflict of Interest

The authors declare no conflict of interest.

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