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Case Report: An Acute Onset of Intracranial Subdural Haematoma 2 Days after Spinal Anesthesia

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Introduction: Spinal anesthesia (SA) is commonly used in surgery, but serious complications such as intracranial subdural hemorrhage (ICSH) are uncommon. A subdural hematoma (SDH) is a collection of blood below the dura's inner layer but outside of the brain and arachnoid membrane. Spinal anesthesia is a type of regional neuraxial anesthesia involving the injection of a local anesthetic or opioid into the subarachnoid area, typically through a fine needle, typically 9 cm in length. If necessary track with capnography. A preoperative opioid is usually helpful in relieving the pain associated with needle insertion, although local anesthetics are also appropriate. The sitting posture is optimal anatomically, but it can lead to vasovagal syncope in highly sedated patients. The most common form of the intracranial traumatic mass lesion is a subdural hematoma. It's usually caused by a heavy enough head injury to burst blood vessels. That could drive pooled blood on the brain. Age, drugs that thin out the blood, and alcohol abuse increase the risk. ICSH incidence after SA was recorded as being 1 in 500, 000. Thirty-five cases were reported during literature review, and most had a chronic course linked to post-dural puncture headache (PDPH). We record an acute onset case of ICSH 2 days after SA, with no trauma history.

Case Study: A 21-year-old young male presented with fistula in ano, and was scheduled for an elective surgery in our hospital. The patient belonged to American society of Anesthesiologists physical status1 with no comorbid conditions. The patient had no history of any pre-existing brain pathology. The anesthetic procedure was completed with ease in a single attempt with a lumbar puncture at the L2/3 level using 25 gauge Quincke needle; there was a free flow of clear cerebrospinal fluid and 0.5%heavy bupivacaine injected. T12 level of the block was achieved and surgery completed uneventfully. On 2nd postoperative day, pt suddenly developed a severe headache and 2 hours later he developed generalized tonic-clonic seizure and became unconscious. The patient was immediately intubated and administered intravenous phenytoin and mannitol as advised by a neurologist. Computed tomography (CT) scan was obtained which showed right subdural hematoma (SDH) with midline shift of 1.3 cm. Patient was shifted to the operation theatre where craniotomy with evacuation of hematoma was done. Post craniotomy pt was shifted to neurointensive care unit and after 48 hours of ventilator support, was extubated. On the third postoperative day, non-contrast CT was done, which showed a complete resolution of SDH. Pt was discharged on 7post operative day with left residual hemiparesis. He was discharged on Ryles tube feed indwelling Foley's catheter. Fifteen days later his left residual hemiparesis resolved and his Glasgow coma scale became 15. Sixty days later the patient came back to our hospital for duroplasty which was done and he was discharged after 3 days.

There are very little data on the consequences of SA-induced ICSH. Since symptomatology frequently imitates PDPH, a number of these are frequently identified missed. According to the literature, its prevalence ranges from 1/500,000 to 1/1,000,000.[4,5] A Swedish retrospective study published in 2004 and covering a decade (1990–1999) mentions 127 cases of SA-related neurological complications, of which the risk was assessed at 1/20,000–1/30,000, including two cases of SA-ICSH. A French study recorded 4 of these cases of SA-ICSH in a medium-size center over a span of 9 years.

SA-ICSH has physiopathogenic mechanisms that are poorly understood. A CSF leak at the arachnoid tear stage would likely cause a reduction in intraspinal and intracranial pressure. The resulting dynamic alteration of the CSF flow results in a relative ventricular collapse and central nervous system rostrocaudal movement. As a result, responsive meningeal structures or meningeal pain receptors are stretched along the cranial nerve and bridging-veins that link the encephalon with the meninges and drain into the dural sinuses.

Electron Microscopic data on human bridging veins reveal the circumferential arrangement of collagen fibers, thin walls of variable thickness, and arachnoid trabecules lack of external reinforcement. This all makes the subdural portion of the vein more vulnerable than the subarachnoid part thereof. Such features clarify the laceration of the veins and the subdural position of the resulting hematomas which can develop in various ways, acute or chronic. Cerebral SDH is a serious SA complication that must be held in mind and which can mimic PDPH.

The history of lumbar puncture, prolonged non-postural PDPH, and neurological symptom progression should be treated as a warning sign of an intracranial hematoma and prompt diagnosis and treatment of the same.

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