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CASE STUDY

PROLONGED HYPOTENSION AND POSTURAL HEADACHE AFTER SPINAL ANESTHESIA, INTRACRANIAL HYPOTENSION SIMILAR CLINICAL PICTURE

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ABSTRACT

We aimed to present a 24 years old male patient that had similarities with intracranial hypotension accompanied headache, syncope attacks, persistent and prolonged hypotension (both ortostatic and supine position) after spinal anesthesia for varicosele operation, relieved by epidural blood patch on twentieth day. The cerebrospinal fluid leakage has to be kept in mind after prolonged, persistent hypotension with headache and syncope attacks.

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INTRODUCTION

Although spinal anesthesia is an easy, fast applicable regionel anesthesia method, its early frequent complication is hypotension where as late complication is headache (Bernards, 2009; Park *et al.*, 2011). Hypotension is an inevitable complication of spinal anaesthesia that occurs when the sympathetic chain becomes blocked (Mokri, Bahram, 2015). Cranial hypotension and long-lasting CSF is the reason of headache. We aimed to present a case with prolonged hypotension after spinal anesthesia.

Case report

A 24 year old 170 cm 72 kg ASA I male patient was scheduled for varicocele operation. Preoperative laboratory tests including complete blood count and coagulation parameters were within normal limits. The patient was consented for spinal anaesthesia. Oxygen saturation, non-invasive arterial blood pressure and heart rate were continuously monitored in the operating room. On the first attempt, spinal anesthesia was

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performed with the patient in sitting position with a 27-gauge Quincke type spinal needle at the L4- L5 interspace using 15 mg of 0.5% heavy marcaine. There was no blood or araesthesia during insertion of the needle and he had adequate block. He had uneventful intraoperative course during the 95 min. surgery. On the fourth postoperative day the patient experienced postdural headache accompanied with nause and hypotension attacks with diplopia. The patient was admitted to the hospital. Arterial sistolic blood pressure was 70-80 mmhg and diastolic was 30-40 mmhg. Hypotension continued even during the supine position. Syncope attacks were seen when he stood up. Cardiological examination revealed no pathology. Postoperative laboratory tests were normal. Intracranial hypotension was thought due to his having postdural headache, vision problems and nause. A magnetic resonance (MR) cranial and lumbar detected normal. There was no history explaining this situation. CSF pressure couldn't be measured. Conservative theraphy of cafein, teofilin bed rest intravenous hydration were started. His complaints remained for 20 days. We decided to perform epidural blood patch with neurosurgeons. After written consent epidural blood patch was performed through L4-5 interspace in the operating room. Arterial blood pressure increased dramatically to 115-125 mmhg and diastolic 75-85 mmhg following the epidural patch

in an hour. Headache was subsided. A week later he was discharged fully recovered.

DISCUSSION

Spinal anesthesia has several advantages, including spared spontaneous respiration, low cost, reduced risk for pulmonary aspiration secondary to vomiting in patients whose stomach is full, elimination of intubation, minimal disruption of blood chemistry, reduced hemorrhaging during surgery, and earlier return of intestinal motility (Bernards, 2009; Park et al., 2011). Earlier complication is hypotension (Mokri, Bahram, 2015). In a study Hartmann and friends performed hypotension incidence was 5%-77 (Hartmann et al., 2002). First reason for hypotension is arteriel-venous vasodilatation and spinal sympathetic nerve blockage. The primary physiologic alterations are decreased preload and cardiac volume, which combine with bradycardia to reduce arterial blood pressure and cardiac output. Mild hypotension or bradycardia may be treated with volume expansion, ephedrine, or atropine (Critchley, 1996; Mercier et al., 2013; Volk, 2010). The extraordinary part of our case even with the volume hydration hypotension persisted with syncope attacks untill performing the epidural blood patch on the postoperative 20th day. Diplopia, headache, nause were the components of intracranial hypotension as seen in our case and we didn't come upon a case in literature up to date as our's. The predominant symptom of this syndrome is postural headache of acute onset, which is slight when the patient is lying down but rapidly worsens when the patient sits or stands. Its incidence is now about 5 per 100 000 of the population (Gordon et al., 2009). Other symptoms include neck pain and stiffness, diplopia, nausea, vomiting, vertigo, tinnitus, impaired hearing, convulsions, and cognitive abnormalities. The syndrome is due to leakage of CSF from a tear in the dura. The mechanism of its aetiology can, therefore, be compared with the headache that often follows a lumbar puncture (Kim et al., 2012; Urbach, 2014). Intracranial hypotension may lead to cortical vein thrombosis, possibly due to a decline in the venous blood flow and this in turn may cause the vessel wall to rupture with resulting subarachnoid haemorrhage (Gordon, 2009; Urbach, 2014). In our case, cranial and lumbar MR showed no pathology. We assume that leakage of CSF was the reason of our patient's prolonged hypotension. Treatment of acute symptoms consist of hydration, bed rest, cafein and steroids. If this fails, an epidural blood patch or a percutaneous injection of fibrin glue can be used to seal the leak, although

sometimes open surgery is needed. Epidural blood patch is controversial but in our case the symptoms dramatically subsided.

Conclusion

Spontaneous prolonged hypotension may be an uncommon but a treatable cause of spinal anaesthesia. The sooner correct diagnosis can be made appropriate treatment can be started. Due to the leakage of CSF epidural blood patch must be on mind as a treatment.

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