



The Case of a Diplopia and Visual Impairment Developing Patient after Spinal Anaesthesia

Spinal Anestezi Sonrası Diplopi ve Görme Bozukluğu Gelişen Bir Hasta Olgusu

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Dear Editor,

Blocking subarachnoid nerve roots with local anaesthetics brings about intense sensory and motor block. Although spinal anaesthesia is an very reliable method when it is applied with suitable approaches, it can still lead to back pain, severe neurological damages, and even to death. Complications like diplopia, headache, hearing loss, tinnitus, nausea, vomiting, and loss of consciousness may follow spinal anaesthesia (1). Among these complications, diplopia is very rare. In some cases, intracranial hypotension due to cerebrospinal fluid (CSF) leakage is known to give way to damages in the sixth cranial nerve which in turn leads to diplopia (2).

In this letter, we aim to present the anaesthetic management of a patient who developed diplopia and visual impairment after spinal anaesthesia.

A 27-year old male patient with no additional pathology in his medical history was taken to the operation room for a planned inguinal hernia repair. Before the operation, the non-invasive blood pressure was 135/77 mmHg while the heart rate was 75 beats/mins and SpO2 was 98%. After achieving pre-hydration with 500 mL 0.9% NaCl, we applied the spinal anaesthesia in sterile conditions in sitting position with a 22-gauge spinal needle through the L3-4 range by administering 12.5 mg 0.5% bupivacaine. Following the anaesthesia application, in the 10th minute, the sensory block level was T8-10 but in the 30th minute of the operation, the patient developed blurred vision and diplopia. At the end of the operation, now hemodynamically stable, the patient was taken to the recovery room. We administered 500 mL of colloid in the recovery room and monitored the patient for neurological symptoms, hemodynamics, and block level. The visual impairment problem recovered within an hour while the patient was neurologically and hemodynamically stable with declined sensory block. After the puncture application, due to a possible CSF leak, we sent the patient back to the service floor with suggestions of bed rest, hydration through fluid intake,

and analgesia. With no pathologies following the postoperative neurologic and ophthalmologic examinations and imaging, the patient was discharged on the second day of his hospitalisation.

The incidence of diplopia after spinal anaesthesia ranges between 1/300 and 1/8000. The incidence rate has actually fallen down as the less traumatic needles replaced formerly used spinal needles that were more likely to bring about complications. In more than 80% of patients, diplopia is reported to improve spontaneously within 2 weeks to 8 months. Although emerging intracranial hypotension is generally thought to effect all cranial nerves except for I-IX and X, cranial nerve VI is reported to be the most affected nerve due to its length in the intracranial area. The formation mechanism is described as the development of local ischemia and function failure due to stretching of nerve caused by intracranial hypotension (3).

Our patient underwent spinal anaesthesia in one sitting without any difficulty. The spinal anaesthesia related CSF loss after puncture varies depending on the thickness and type of the needle as well as the patient group. Using thinner and pencil-point spinal needles helps reduce the frequency of CSF loss after puncture. To secure the volume of CSF, patients are recommended to have bed rest and fluids to maintain hydration.

Serious complications may occur during and after spinal anaesthesia. Practitioners need to be careful when applying the required techniques. Underlining the importance of informing patients about possible risks and of monitoring patients closely in order to recognise probable temporary or permanent complications, we wanted to share our experienced with the readers.

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