COMPLETE FLACCID PARALYSIS FOLLOWING SPINAL ANAESTHESIA – A CASE REPORT

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SUMMARY

Neurological complications after a well-conducted spinal anaesthesia though reported, are rare. Various lesions as aseptic meningitis, anterior spinal artery syndrome have been documented. A number of mechanisms for the same have been reported. We report a case with complete flaccid paralysis following spinal anaesthesia of which the exact cause could not be detected.

Keywords : Spinal anaesthesia, Neurological complications.

Introduction

Complications following well-conducted spinal anaesthesia are rare¹. Neurologic complications as anterior spinal artery syndrome, transverse myelitis, chronic arachnoiditis, cauda equina syndrome following spinal and epidural anaesthesia have been documented².

Though in most of the cases, the exact etiology cannot be detected, various theories such as direct trauma, chemical irritation, and sepsis have been put forth³. We report a case of aseptic meningitis leading to paraplegia following a spinal anaesthesia.

Case Report

An 18-year-old girl presented to us with acute pain in right iliac fossa since 8 hours with no other significant bowel, bladder and menstrual complaints. She had a pulse of 98min⁻¹ blood pressure of 120/80mmHg. On abdominal palpation, she had rebound tenderness in right iliac fossa. Plain radiograph of the chest and the abdomen were normal while ultrasonography of the abdomen was suggestive of acute appendicitis. The patient was posted for emergency appendicectomy. Under strict aseptic precautions, using a no. 23G spinal needle with the patient in left lateral position, 3.4ml of 0.5% preservative free bupivacaine was injected in the L3-4 interspace in the midline after confirming entry by free and clear flow of cerebrospinal fluid (CSF). Patient developed a sensory blockage upto T10 dermatomal level. The operation lasted for half an hour and the intraoperative course was uneventful with patient maintaining all vital parameters within the normal range when acutely inflamed appendix was removed.

Patient had sensory blockade up to T-10 level before shifting from the table and postoperatively the patient was given head low position as a routine protocol. Eight hours after the surgery, patient was haemodynamically stable while she complained of inability to move both lower limbs with no bowel and bladder control. She had no fever and neurologic examination revealed flaccid paralysis up to T10 level. It was initially thought that the spinal anaesthesia has lasted for a long time and hence patient was kept under close observation. Twenty-four hours after the surgery, patient had similar clinical features with extensor plantar reflexes. The neurologic opinion was sought when a diagnostic lumbar puncture was advised. After fundoscopic examination, diagnostic lumbar puncture was done which aspirated a clear CSF. The CSF examination was normal with normal proteins, no leucocytosis. The CSF culture also did not grow any bacteria. Patient was subjected for magnetic resonance imaging, which was also totally normal. With exclusion of all organic lesions, a diagnosis of aseptic or viral meningitis was done. In the mean time, she was on broad-spectrum antibiotics, analgesics and was started on oral feeds. Patient was offered conservative treatment, however patient wanted to go home and was discharged against medical advice. Though a lot of postal and personal communication was attempted, she was lost to follow-up.
Discussion

Neurologic complications though rare have been documented following spinal and epidural anaesthesia. Usubiaga et al have reported serious neurologic complications as anterior spinal artery syndrome, transverse myelitis, cauda equina syndrome with an incidence of one in 10,000 cases following either spinal or epidural analgesia. However these neurologic lesions can occur spontaneously in absence of anaesthesia or following general anaesthesia.

In most of these cases the exact etiological cause cannot be detected. However, various mechanisms have been reported as:

1. Direct trauma - usually associated with multiple attempts with trauma to spinal cord or nerve roots. Symptoms are usually in the distribution of sciatic nerve and present with numbness, weakness or pain in the legs. Murray et al have reviewed 201 cases of lower limb paralysis due to injury to sacral plexus and have reported that bilateral lesions do not occur. Hematomas can occur leading to compressive symptoms leading to paraplegia.

2. Drug factors: Subarachnoid reactions to local anesthetics are a function of the histotoxic properties inherent in the local anesthetic. Agents as chloroprocaine are acidic in nature. These low pH solutions directly irritate nerve roots and may also cause vasospasm. These are also hyperosmolar and may damage nerve roots due to osmotic effects. Further, some of these agents have preservatives, alcohol, acacia, strychnine that can further damage the spinal cord. Not only the preservatives, reports of neurological complications due to contaminants such as talcum powder of the surgical gloves, bits of cotton of the wrapped syringes, and even the ions of the metallic syringes have also been reported. Entry of contaminants as formaldehyde, phenol while these ampoules being stored can also lead to neurological problems. Some of these anesthetic agents are added with epinephrine or other additives to achieve the desired effect. These additives may induce vasospasm and lead to ischemic injury or thrombosis of the spinal artery. Some drugs as chloroprocaine and bupivacaine have been reported to cause vasoconstriction in gravid uterine vessels. Winkleman et al have reported chronic arachnoiditis in 11 patients, which was attributed to the detergent solutions due to incomplete rinsing of the syringes. Bergner et al have reported 6 cases to have been an undetected chemical contaminant of the spinal solutions. Moore had stated local anesthetic solution to be the cause of these neurological sequelae when used in large volumes and higher concentration. However, a decreased incidence of neurological complication has been attributed to the use of lower concentration of the local anesthetic agents without any preservatives.

3. Management related factors: Hypotension following epidural and spinal anaesthesia has been the most common cause of spinal cord damage due to ischemia. Bromage et al have reported 4 cases of spinal cord infarction due to prolonged hypotension following epidural anaesthesia. Hypotension may cause spinal cord ischemia in the watershed areas or may lead to thrombosis of anterior spinal artery in a preexisting atherosclerotic artery. The posture of the patient can further add to hypotension with caval compression. Ditzler and Mcliver have reported a case of flaccid paraplegia following hypotension during general anaesthesia, which has been attributed to anterior spinal artery thrombosis.

4. Preexisting neurologic lesions. The coincidental occurrence of paralysis following spinal anaesthesia has been reported. Marinacci and Courville have found that out of 482 patients with neurologic complaints following spinal anaesthesia, in 478 patients concurrent and totally unrelated lesions caused it.

Various neurologic lesions as aseptic meningitis, anterior spinal artery thrombosis, chronic arachnoiditis, cauda equina syndrome have been documented following spinal or epidural anaesthesia. Patients with aseptic meningitis usually have fever, headache, nuchal rigidity, photophobia and CSF shows increased pressure with raised polymorphonuclear cells with negative cultures. In this condition, the symptoms start within 24 hours and recovery is spontaneous within several days to weeks. Patients with cauda equina syndrome have onset of the symptoms immediately after the effects of the spinal anaesthesia are worn off and present with urinary and fecal incontinence, localised sensory loss in the perineal area with varying degrees of lower limb weakness. These symptoms may be permanent or may show gradual regression over weeks or months.

Adhesive arachnoiditis present with gradual progressive weakness and sensory loss of lower limbs occurring weeks to months after the spinal anaesthesia, which may progress to complete paraplegia and death in severe cases.
As prevention is better than cure, care should be taken to use appropriate anesthetic agents in proper dosage and concentration. Before administering the drug the syringes should be adequately rinsed to remove any of the contaminant. A proper knowledge of the anatomy of the spinal and the epidural space is essential. A thorough monitoring of the patient during the intraoperative period is necessary to prevent hypotension of any cause. Most important is to have a good preoperative history and rule out any contraindications.

If neurologic complication is suspected after the spinal or epidural anaesthesia, a complete history and a thorough neurological examination by an unbiased expert neurologist may be necessary. Diagnostic lumbar puncture is essential, however in adhesive arachnoiditis, it may be difficult or may be impossible to obtain CSF, but Queckenstedt’s test is positive\(^7\). Electromyography has an added advantage in differentiating neurological disease of extradural origin from intradural origin\(^7\). As spinal anaesthesia cannot give rise to extradural lesions, an identification of such lesion makes it coincidental appearance with spinal anaesthesia.

Myelography was previously the investigation of choice as it was used to detect the level of subarachnoid block. But it has a disadvantage that it is invasive and has a further chance of aggravating the inflammatory response of the meninges and complicates adhesive arachnoiditis\(^7\). Recently, magnetic resonance imaging (MRI) gives excellent visualisation of the spinal cord and the nerve roots without any introduction of any contrast. Hence MRI is at present the investigation of choice.

Our patient was subjected to preservative free spinal anaesthesia with complete aseptic conditions. Patient developed flaccid paralysis below the dermatomal level of T10. The exact etiology could not be detected, as all investigations including the CSF and MRI were normal.

Reference