NEUROLOGICAL DEFICIT FOLLOWING COMBINED SPINAL-EPIDURAL ANESTHESIA FOR KNEE ARTHROPLASTY

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Abstract

A healthy man developed cauda equina syndrome after uneventful combined spinal and epidural anesthesia. No pre-existing neurologic disorder was recorded. There was no pain or paresthesia during needle placement, drug injection or catheter insertion. The sensory levels were improved within a few days following the deficit but little improvement on motor power but not on sphincter tone. Local anesthesia neurotoxicity was thought to be the leading cause of neurologic deficit in our case.

Introduction

Regional anesthesia for lower limb surgeries is generally held to be inherently safe. Spinal and epidural blocks are therefore used widely, with the more recently introduced combined spinal and epidural technique gaining popularity. One of the rare complications is cauda equina syndrome. Cauda equina syndrome (CES) is a serious neurologic disorder that is caused by damage of the conus medullaris or the spinal nerve roots comprising cauda equina. It is associated with varying degrees of signs and symptoms including loss of bowel and bladder function, insensate perineal areas and lower extremity muscle weakness[1,2]. Permanent neurological complications like CES after central neuraxial blockades occur significantly more often after spinal blockage with especially intrathecal administration of lidocain[3]. We reported a case in which regional anesthesia using combined spinal-epidural anesthesia set was associated with cauda equina syndrome postoperatively.

Case Report

A 83-yr-old male, 160 cm, 59 kg, BMI 23kg/m^2, ASA physical status I patient underwent elective left total knee arthroplasty. History of right shoulder surgery under general anesthesia 10 years ago was without complication. No history of pre-existing neurologic disorder reported. Drug history included use of NSAID for treatment of knee arthralgia. Physical examination and all laboratory studies were within normal range. The operation was planned under combined spinal epidural anesthesia. No premedication was given before the operation. The patient was connected to standard monitor and 18-G intravenous cannula was inserted. Consequently, he was given 250 ml RL solution immediately prior to anesthetic for prehydration. With the patient in the sitting position, the lumbar area was disinfected with a solution of 10% povidone-iodine (Alphadine solution; Manufactured by Riyadh Pharma, Riyadh, Saudi). After removing excess moisture from the disinfected site, a 18-G Tuohy needle (B/Braun® Combined Spinal/Epidural Minipack with 27-G Pencil Point Needle and 20-G catheter) was used to identify the L_3-L_4 epidural space by

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using a loss of resistance to air technique via midline approach. A 27-G pencil point spinal needle was placed through the Tuohy needle into the subarachnoid space with the needle through needle technique. After flow of clear cerebrospinal fluid, the patient received 10 mg 0.5% hyperbaric bupivacaine and 25 μg fentanyl (2.5 mL) combination intrathecally. The spinal needle was removed and an epidural catheter was inserted 5 cm into the epidural space. Aspiration test revealed clear fluid came out from catheter then catheter removed. Another attempt was done at L_4-L_5 by using same technique; epidural catheter inserted 5 cm into epidural space. After withdrawal of the Tuohy needle, the patient was placed supine. The urinary bladder was catheterized. There was no pain or paresthesia during needle placement or drug injection. Twenty minutes after bupivacaine administration, the sensory level block was bilateral, symmetric, and caudal to T10, as assessed using the pinprick method. The 165-min surgical procedure was conducted uneventfully with the patient in the supine position. Throughout the procedure, his arterial blood pressure remained normal. At the end of the surgery, the patient was transported to the postanesthesia care unit (PACU), sensorial block regressed to L_1, and Bromage score was 3 (Unable to flex knees, but with free movement of feet), then epidural infusion started with mixture of bupivacaine 0.0625% with fentanyl 12 μg/mL at rate 5 ml/hour. He was discharged from the PACU to the ward. Low molecular weight heparin was given subcutaneously as a prophylaxis for thromboembolia. In second post OP day surgery the Bromage score was still 3. Pain management team discontinued the epidural infusion and decided to remove the catheter the next day at morning according to coagulation profile result and they hold clexan dose to be restarted 4 hours after catheter removal. They started him on Tylenol 1g orally q 6h. One day later, neurological examination revealed loss of sensation under the knees, weakness of lower extremities more on the left side and sphincters incontinence. After neurology consultations, a lumbosacral magnetic resonance imaging (MRI) was performed and revealed no intra spinal collection, no spinal stenosis and no compression on the cauda equina nerve roots with diffuse degenerative changes and diffuse degenerative disc disease. Nerve conduction study (NCS) and electromyogram (EMG) showed severe axonal type neuropathy, neuroradiculopathy on the lower limb, sparing the sensory fibers. Cauda equina syndrome was diagnosed and an initial dose of dexamethasone 10 mg was started intravenously followed by 4 mg intravenous dose every six hours. The neurologist and physiotherapist follow up the patient and he was improving slowly as the motor power became better with intact sensation. One month postoperatively, the patient underwent a debridement of a wound abscess (staph A) at the surgical site under general anesthesia. Few days later, the patient deteriorated and developed pre-renal azotemia, with jerky movements of the upper limbs and trunk along with neck stiffness, fever, shivering, confusion (GCS 10/15) and shortness of breath. The patient was transferred to the surgical ICU with severe septicemia and ARDS. He developed multiple organ failure and later had cardiac arrest and expired after 68 days from the procedure.

Discussion

Local anaesthetics have been placed in the intrathecal space for approximately 100 years. Bier and Hildebrandt first performed spinal anesthesia on a single patient, themselves, and then subsequently reported on the injection of cocaine into the subarachnoid space of six patients in 1899[4]. The incidence of persistent neurological sequelae after subarachnoid anaesthesia varies between 0.01 and 0.7%[5-8]. CES was first described in four cases after continuous spinal anesthesia (CSA) in 1991[9]. Three of these cases were done with a 28 gauge catheter specifically marketed for CSA; in the fourth case, 0.5% tetracaine was administered through a standard epidural catheter. Rigler et al[9] postulated that the combination of trauma, maldistribution and a relatively high dose of local anesthetic resulted in neurotoxic injury. Most cases of CES after spinal anesthesia have been reported in association with the administration of idocaine[9,10]. Limited cephalad extension of sensory block, suggesting the restricted distribution of lidocaine in the CSF and likely to have resulted in local anesthetic toxicity, was obtained in clinical reports of CES after spinal lidocaine[9,11]. Kubina et al[12] described two cases of CES following bupivcaine with glucose injected spinally, and bupivacaine without glucose injected in a combined spinal-epidural technique. The first patient had spinal

stenosis which could explain this complication; however the explanation for CES in the second patient is uncertain and consequently speculative[12]. Similar to this case, Chabbou et al[13] reported a case with CES after spinal administration of 0.5% hyperbaric bupivacaine (12.5 mg) with no causative factor in the genesis of CES. Other potential causes of neurologic injuries associated with spinal-epidural anesthesia were not identified in our case. No flashing pain or paresthesia was noted on needle placement or bupivacaine injection, as is the case in direct needle-induced trauma to the spinal cord or intraneural injection[10]. The spinal anesthetic was performed awake, allowing the patient to respond to painful stimuli[10]. Spinal cord compression by an expanding hematoma was excluded in this case by magnetic resonance imaging[10]. There was no evidence of infection[14]. Also, the antiseptic used to clean the skin was supplied in a single-use disposable bottle and the skin was, as usual, carefully dried before skin puncture[15]. Spinal cord ischemia was also unlikely because the patient had no microvascular disease, remained hemodynamically stable, and vasoconstrictors were not administered[16]. Coincident postoperative exacerbation of a pre-existing neurologic disease was also inconsistent with the conditions of this clinical case[17]. Finally, the neurological examination was not consistent with a neurological injury from improper patient positioning or surgical trauma[18]. Infection is one of the surgical complications but there are many other risk factors in this patient that could complicate his case (e.g., prolonged hospital stay, old age, use of Foley catheter) increase stayed in the hospital, old age, Foley catheter.

In conclusion, we reported a case of cauda equina syndrome after uneventful combined spinal-epidural administration of bupivacaine. Bupivacaine neurotoxicity was suggested to be the cause for this neurologic deficit.

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