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Spinal Anesthesia Leading to Permanent Neurologic Damage due to Neurotoxicity of Hyperbaric 0.5% Bupivacaine

SCORE

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01/02/2015

Key words : spinal block, cauda equina syndrome, complication, anesthetic technique, hyperbaric 0.5% bupivacaine, permanent injury. Contact: c.denis@student.uclouvain.be

OBSERVATION

A 66-year-old ASA II male patient with past medical history of high blood pressure, hypothyroidism, COPD (GOLD 2), chronic temporomandibular joint disorder, and a body-mass index of 27 kg/m² was scheduled to undergo endovenous laser therapy under spinal anesthesia (SA). His past anesthetic history consisted of a successful SA with 2.2 ml of hyperbaric 0.5% bupivacaine for a knee arthroscopy 3 years earlier. The recorded site of injection was at the L3/L4 level, using a 25G Braun® needle. No complications were reported. Patient current medications included tiotropium bromide (anticholinergic), association of salmeterol xinafoate (long acting $beta_2$ agonist) and flutocasone propionate (inhaled corticosteroids), acetaminophen, tramadol, N-acetylcysteine, and L-thyroxin.

For the here concerned intervention, SA was performed in the sitting position, the patient receiving supplementary oxygen through a face mask. An aseptic technique was applied. The level of needle insertion was estimated to be the L4/L5 interspace. Five mL of 2% lidocaine was injected subcutaneously. Two attempts were needed to reach the subarachnoid space, using a 25G Braun needle through an introducer. Cerebrospinal fluid (CSF) outflow was objectified through the needle, and CSF swirl within the syringe was noticed before injection of the anesthetic solution. It consisted in 2.2 mL of a mixture containing 8.8 mg of bupivacaine and 2.2 µg of sufentanil. No paresthesia occurred during needle insertion or local anesthetic agents (LA) injection. The patient did not experience any pain. No blood backflow was occurred. The patient was promptly placed in the supine position. After having assessed that sensory level was reaching T10, the patient was prepped and draped for surgery. The anesthetic technique was almost identical to the one used for the previous SA, which was uneventful. There were no unexpected events during surgery, and the patient was discharged 5 hours later when usual criteria were met, including ability to eat and drink, walk alone, and urinate.

Eight days later, the patient saw his general practitioner (GP) to remove surgical stitches. GP did not notice any abnormalities, except for high blood pressure. The patient did not complain about his health status.

Twelve days after surgery, the patient was admitted in our emergency department because of nausea, vomiting and disabling headache for a few days. He appeared to the physician as slightly confused. Vital parameters showed temperature of 37.4°C. Arterial and venous blood samples showed sodium and potassium levels in the normal range and a metabolic alkalosis with insufficient respiratory compensation. No biological signs consistent with meningitis or an inflammatory process were noted. The cerebral CT showed no acute injury. The emergency physician managed the case by

discontinuing tramadol and correcting electrolyte disorders. The patient stayed one night in the hospital and was discharged the next morning with good recovery from vomiting.

Six days later (twenty days after surgery), the patient came back to our emergency department complaining of **sensitivity loss in the perineum area**, as well as **bladder** and **bowel dysfunction** since the surgical procedure. On examination, we found anesthesia of peri-anal, scrotal and sacral regions, balance disorders, and lower limb paresis. Achilles and anal reflexes were absent. Palpation of the lower abdomen revealed distended bladder due to urinary retention. A diagnosis of cauda equina syndrome (CES) was made by the neurologist and the neurosurgeon. The lumbosacral MRI excluded signs of any compressive mechanism and the myelographic MR imaging did not bring out signs consistent with arachnoiditis or other inflammatory or infectious processes. Blood analysis did not show any abnormalities. Corticotherapy was initiated using decreasing doses of methylprednisolone. A urologist placed an indwelling suprapubic catheter. The mechanism of the CES was postulated to emerge from LA toxicity.

The patient spent 4 months in our rehabilitation ward, with intensive physiotherapy and psychosocial support, during which his status gradually improved. He first recovered from bowel dysfunction and was able to dispense the indwelling suprapubic catheter after 3 months. He benefited from a monthly neurological follow-up, with several electromyoneurography (EMNG) examinations. Upon discharge, the clinical picture had resolved except for the persistence of a lower limb paresis.

A six months neurologic follow-up showed improvement of EMNG signs and a stable neurologic status with a persistent motor deficit of both lower limbs.

DISCUSSION

Etiology. As direct neurotoxicity of LA is a diagnosis of exclusion, other causes had to be excluded. Indeed, the diagnosis was initially overlooked, but deeper patient questioning confirmed that the symptoms had begun on the day of SA. The initial clinical signs were first denied by the patient in the hope of a prompt recovery. Absence of complaints to his GP and emergency physician was also linked to embarrassment and shame about bowel and urinary dysfunction. Medical imaging (MRI and CT) excluded a space-occupying lesion such as hematoma, tumor, abscess, prolapsed disc, or spinal stenosis. Surgical causes or positioning of the patient were not incriminated. Direct trauma to the spinal cord or intraneural injection were unlikely, insofar as the patient did not experience any pain or paresthesia at the time of SA. A possible aseptic meningitis was not documented. The only ambiguous sign of an infectious etiology was a rise in temperature up to 37.4 °C on the twelfth day post operatively. No lumbar puncture was performed at that time. The most likely causative agent in

our case could be neurotoxicity. Even if we had very little argument, an infectious etiology cannot totally be excluded.

Regarding the **clinical outcome**, the incidence of **permanent injury** (defined by Cook and colleagues as symptoms persisting for more than six months) following SA is between 0.9 and 2.2 per 100 000 [1] . Among these cases, if we consider only those caused by infection and/or LA toxicity, as the case we reported, the incidence is even lower.

Comment [BV1]: This is not documented. Please omit.

Regarding the **clinical picture** (CES) and the presumed **etiologic mechanism** (toxic or infectious), a Swedish survey of 1.26 million SA performed between 1990 and 1999 showed an incidence of CES from toxic origin of 18/ 1 260 000 (1.4 per 100 000), either by direct neurotoxicity or thought to be related to a compression by a LA volume-effect. Among these 18 patients, six received 0.5 % hyperbaric bupivacaine [2]. Other patients had received either 5 % hyperbaric lidocaine or 0.5 % isobaric bupivacaine. During this period, the overall use of bupivacaine for SA was approximately 2.4 times the one of hyperbaric bupivacaine. Five % hyperbaric lidocaine is more neurotoxic than 0.5 % hyperbaric bupivacaine [3] [4]. Given these facts, the incidence of CES after 0.5 % bupivacaine is much lower than 1.4 per 100 000.

Neither the patient nor the technique seemed to present any identifiable **risk factors** like pre-existing pathology of the spine, technical difficulties, lidocaine use, or lithotomy positioning during surgery [5].

It has to be kept in mind that permanent neurologic deficits like CES are described after both general and regional anesthesia. Most cases are multifactorial and their etiology is sometimes difficult to elucidate [6]. Permanent neurologic deficits after atraumatic SA rarely occur in patients who have received 0.5 % hyperbaric bupivacaine [7].

CONCLUSION

In our patient, although it remains unclear, the most likely cause of CES following an uneventful SA is LA neurotoxicity. The patient received 0.5 % hyperbaric bupivacaine, which is rarely associated with direct neurotoxicity. CES is a very rare but dramatic complication of SA, particularly if causing a permanent injury as in our case. Delayed diagnosis could reflect inadequate post operative surveillance.

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