

Lumbar Plexus Block Causing Symptoms Mimicking Brain Death

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Citation

E Bernitsas, C Grommes, K Al-Amin, A Alshekhlee. *Lumbar Plexus Block Causing Symptoms Mimicking Brain Death*. The Internet Journal of Anesthesiology. 2008 Volume 19 Number 2.

Abstract

Brainstem anesthesia presenting as transient and complete loss of brain stem reflexes along with cardiovascular compromise is an extremely rare complication of a local anesthetic block. We report a previously healthy man who became unresponsive with complete loss of brainstem reflexes (mimicking brain death) immediately following a lumbar plexus block administered for surgical repair of a hip fracture. Although the mechanism of spread of the anesthetic agent to the brain stem structures is unclear, our report should heighten the awareness of this potentially lethal complication.

INTRODUCTION

Anesthetics leak to the subdural space may cause preganglionic sympathetic blockade resulting in cardiovascular depression with subsequent cerebral dysfunction. We describe a previously healthy man who became unresponsive with complete loss of brain stem reflexes (mimicking brain death) following a block of the lumbar plexus for surgical repair of a hip fracture. We hypothesize a spinal spread of the anesthetic agent to the subdural or epidural space causing a brainstem anesthesia with transient loss of brain stem reflexes.

CASE REPORT

A 60-year-old man with a prior medical history of chronic hypertension, gout, and alcohol abuse was admitted for a hip fracture following a fall. He was treated with oxycodone (5 milligrams) for pain control. Preoperative physical and neurological examinations were normal. Surgical repair of the fracture was planned under general (twilight anesthesia) and regional anesthesia (lumbar plexus block). The former was achieved by intravenous administration of midazolam (2 milligrams) and fentanyl (100 milligrams). Vital signs and mental state were normal while patient under sedation. Regional anesthesia was performed with sterile preparation of the skin and 1% Lidocaine was subcutaneously infiltrated. Stimulating needle was inserted after connecting to nerve stimulator and quadriceps muscle twitch was obtained at 0.4 milliamperes. Negative aspiration for blood or cerebrospinal fluid was confirmed, followed by slow incremental injection of the local anesthetic compound (1% Mepivacaine, 0.5%

Ropivacaine, in 5 micrograms per milliliter) in a total volume of 40 milliliters. Immediately after the completion of the nerve block, he became unresponsive and his respiratory effort ceased. Electrocardiographic tracing showed initial sinus bradycardia followed by complete asystole, with the lowest systolic blood pressure of 82 mmHg. Cardio-pulmonary resuscitation was initiated and endotracheal intubation was performed. Atropine and phenylephrine drip were simultaneously initiated for cardiac and vasomotor support. Within less than a minute after resuscitation, the heart rate returned to sinus rhythm. Arterial oxygen saturation remained adequate (97-100%) throughout the respiratory depression and during the intubation process. Flumazenil (0.3 milligram) was given intravenously without a response. Neurological examination one hour after resuscitation showed a deep coma with non-reactive to light and dilated pupils. Brainstem reflexes including the oculocephalic, corneal, gag reflexes were absent. No spontaneous respiration was noted. Motor function was abolished with flaccid muscle tone and total areflexia. Operative plan was halted. Immediate head imaging with computerized tomography and magnetic resonance imaging scans revealed no acute pathology. Three hours later, he started showing signs of recovery with regain of consciousness and respiratory efforts. Neurological functions gradually returned to normal over the next hour except for the flaccid paralysis of the legs and a suspended sensory level at the lower thoracic segments that had completely recovered within the next 24 hours. Tracheal tube was successfully removed in the very next day and the strength in

the legs returned to normal. Full cardiac investigation including serial enzyme studies, electrocardiography and echocardiography was normal. Neuropsychological evaluation revealed no cognitive impairment. A week later, he underwent surgical repair of the hip under general anesthesia without complications.

DISCUSSION

Ropivacaine is a long-acting local anesthetic with a half life of 4 hours in the serum [1]. It is the propyl homolog of bupivacaine and mepivacaine with a similar anesthetic profile. It provides excellent pain relief with a fast recovery [2,3,4]. Central nervous system toxicity can occur at lower doses than cardiovascular toxicity [5,6]. Allergic reaction to ropivacaine was not considered in our patient because of a previous exposure to these agents for a prior surgical procedure. Two potential mechanisms could have caused a complete loss of the brainstem reflexes in our patient; first, the potential subdural or epidural spread of the local anesthetic agent. The course of neurological recovery was within the expected pharmacological duration of the injected ropivacaine. In addition, the myelopathic picture noted three hours after the injection would further point to a local mechanism resulting into secondary suppression of the brainstem reflexes. We cannot offer a convincing explanation about the mechanism of subdural versus epidural spread of the local anesthetic agents and its migration from the injection site to the brain stem structures. However, inadvertent direct intravascular placement of the needle tip could not be eliminated despite the meticulous technique. Intravascular placement of the needle tip is less likely possibility in experienced hands [7]. The second potential mechanism is the cardiovascular depression inflicted by the locally and systemically administered agents resulting in anoxic brain insult. However, the maintained systolic blood pressure and the oxygenation status, the short duration of cardiac resuscitation, and the lack of radiological evidence (on brain imaging preclude this possibility). Midazolam and fentanyl may cause mild cardiovascular depression resulting in bradycardia by inhibiting the GABAergic transmission to cardiac vagal neurons [8,9]. In addition, a preganglionic sympathetic blockade caused by ropivacaine may result in severe cardiovascular depression requiring at times cardiorespiratory support [2]. Similar to our patient, two cases of brainstem anesthesia were reported; the first case occurred after a retrobulbar block with bupivacaine [10]. The second case of brainstem anesthesia followed a suboccipital craniotomy under generous cervical

field block with bupivacaine [11]. In both cases, the proposed mechanism of brainstem anesthesia was the direct spread of the bupivacaine to the brain stem structures due to the anatomic proximity.

Complete loss of brainstem reflexes in clinical practice may pose a clinical challenge as it mimics the clinical signs of brain death. However, before declaring brain death, it is imperative to exclude other potentially reversible causes of coma, such as hypothermia and drug intoxication including alcohol, or other disorders such as locked-in state and fulminant Guillain-Barré Syndrome [12,13,14]. Sedative agents such as tricyclic antidepressants, barbiturates, and local anesthetics can cause brain stem anesthesia with subsequent loss of certain brain stem reflexes but with a relative preservation of the pupillary constriction to light [15,16,17]. Our patient had no evident hypothermia or toxic exposure prior to his procedure. Brainstem ischemia, hemorrhage and cerebral mass with brain herniation can also cause sudden loss of the brainstem reflexes. Imaging of the brain is extremely important for early intervention and treatment.

In conclusion, coma with rapid complete loss of the brainstem reflexes following lumbar plexus anesthetic administration is a possibility. Rapid mental recovery with myelopathic features may suggest a subdural or epidural spread of the anesthetic agent. Anesthetists, surgeons, and neurologists should be aware of a complete loss of the brain stem reflexes following regional lumbar blocks.

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