Lumbar Plexus Block Causing Symptoms Mimicking Brain Death

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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) 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 milliampere. 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. Cardiopulmonary 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]. Central nervous system toxicity can occur at lower
doses than cardiovascular toxicity [3]. 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 [4]. 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 [5]. In
addition, a preganglionic sympathetic blockade caused by
ropivacaine may result in severe cardiovascular depression
requiring at times cardiorespiratory support [6]. Similar to
our patient, two cases of brainstem anesthesia were reported;
the first case occurred after a retrobulbar block with
bupivacaine [7]. The second case of brainstem anesthesia
followed a suboccipital craniotomy under generous cervical
field block with bupivacaine [8,9]. 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 [10,11]. 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 [12,13,14].
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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