

Preoperative anxiety can cause convulsion and severe hypotension immediately after spinal anaesthesia for caesarean delivery: a case report

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Abstract

Preoperative anxiety in patients under spinal anaesthesia may cause serious complications. We report a case of combined transient convulsion and severe hypotension immediately after spinal anaesthesia for caesarean delivery in a patient who presented with severe preoperative anxiety. Our patient's consciousness and blood pressure recovered normally without any sequelae. However, preoperative anxiety can induce such complications, particularly in patients under regional anaesthesia. Therefore, early detection and deliberate management for preoperative anxiety are required for the patient's safety and satisfaction.

Keywords

Caesarean delivery, hyperventilation, hypotension, preoperative anxiety, spinal anaesthesia, convulsion, pregnancy

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Introduction

The advance of regional anaesthesia techniques in caesarean delivery has decreased the obstetric mortality rate and improved patients' safety.¹ However, regional anaesthesia may cause serious complications,

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such as cardiovascular collapse or seizures.² Furthermore, because patients under regional anaesthesia are alert, their anxiety can be a critical problem in the perioperative period. We report a case of a woman who presented with sudden convulsions and severe hypotension following spinal anaesthesia for caesarean delivery.

Case report

A 38-year-old woman (158 cm, 72 kg), at 37+1 weeks of gestational age was scheduled for caesarean delivery. The patient had previously received caesarean delivery because of preeclampsia. In this pregnancy, the patient had been diagnosed with pregnancy-induced hypertension and was prescribed aspirin 100 mg once a day for prevention of preeclampsia for 12 to 36 weeks. Blood pressure was not well controlled during the week leading up to surgery and was associated with overall discomfort. Therefore, the surgery was performed 2 weeks earlier than the planned date. A preoperative laboratory test, including Na^+ , K^+ , Ca^{2+} , and Mg^{2+} , and chest radiography were all within the normal range, and electrocardiography showed sinus tachycardia.

In the operating room, the patient was monitored with three-lead electrocardiography, pulse oximetry, and non-invasive blood pressure monitoring. The initial blood pressure and heart rate were 140/80 mmHg and 122 beats per minute (bpm), respectively. Peripheral oxygen saturation was 99%, with 2 L/minute of oxygen supplied via nasal prongs. Before induction of anaesthesia, the patient had rapid, shallow breaths and appeared to be extremely anxious. However, considering effects on the foetus, no anxiolytics were used, and only verbal comment was provided. Spinal anaesthesia was performed in the left lateral position at the third and fourth lumbar intervertebral space with a 25-gauge

needle. After free flow of cerebrospinal fluid was identified, 11 mg of hyperbaric bupivacaine was slowly administered intrathecally.

The patient was then placed in the supine position, and initial sensory blockade was assessed by the perception of cold sensation using an alcohol-soaked sponge and by detecting pain. Before this could be completed, the patient presented with sudden onset of convulsions in the face and bilateral upper extremities for approximately 5 seconds. She also showed a slightly decreased mentality, but did not lose consciousness. At that time, haemodynamic monitoring showed the following values: blood pressure of 55/31 mmHg, heart rate of 140 bpm, and oxygen saturation of 99% on 2 L/minute of oxygen. Immediately after detecting hypotension, 500 mL of Hartmann's solution was infused, and 60 μg of phenylephrine was administered intravenously. Despite severe hypotension, the patient did not show respiratory depression and the end-tidal carbon dioxide concentration, which was measured via a tight fitting facial mask, was 17 mmHg. After 1 minute, blood pressure was not measured, and another 100 μg of phenylephrine was administered. After 2 minutes of the second phenylephrine administration, blood pressure was 40/25 mmHg and the heart rate was 130 bpm. Arterial catheterisation was then performed at the left brachial artery for invasive blood pressure monitoring, and a further 100 μg of phenylephrine was administered intravenously. Arterial blood gas analysis was immediately performed and showed acute respiratory alkalosis with a pH of 7.543, partial pressure of arterial carbon dioxide (PaCO_2) of 25.4 mmHg, partial pressure of arterial oxygen (PaO_2) of 184.5 mmHg, and bicarbonate concentration (HCO_3^-) of 43.1 mEq/L. Arterial blood pressure improved to 100/74 mmHg, with a heart rate of 110 bpm, and peripheral oxygen saturation was 100%. By this time, the patient

was recovered and completely alert. After the patient's haemodynamic status was stabilised, we rechecked the level of the sensory blockade and achieved a T4 level of sensory block. After discussion with the surgeon, the operation finally began, and a female neonate who weighed 3350 g was delivered within 4 minutes following the initial skin incision. Despite severe maternal hypotension, the neonate was healthy, and the Apgar score was 7 at 1 minute and 9 at 5 minutes after birth. Maternal arterial blood gas analysis that was performed 10 minutes after foetal delivery showed a compensated respiratory alkalosis with a pH of 7.414, PaCO₂ of 26.4 mmHg, PaO₂ of 207.7 mmHg, HCO₃⁻ of 16.5 mEq/L, and base excess of -6.6 mEq/L. The blood glucose level was 97 mg/dL. We replaced the nasal prongs with a partial re-breathing mask, but arterial blood gas analysis after 20 minutes showed a pH of 7.425, PaCO₂ of 25.2 mmHg, PaO₂ of 257.2 mmHg, HCO₃⁻ of 16.2 mEq/L, and base excess of -6.6 mEq/L. After the operation was completed, the patient was transferred to the post-anaesthesia care unit. After caesarean delivery, a consultation for neurological evaluation of the transient convulsions was provided, but no specific cause was found. The mother and neonate were subsequently discharged after 4 days without any complications.

Because this is a case report, ethical permission from the Institutional Review Board was not mandatory. The patient provided verbal consent for publication of this report.

Discussion

In the present case, the patient presented with a combination of convulsions and severe hypotension. There may be several possible causes for sudden onset of convulsions with slightly decreased mentality. First, a patient's preoperative anxiety can

cause symptoms, such as transient convulsion or syncope. Respiratory alkalosis induced by hyperventilation can cause dizziness, syncope, or even convulsive disorders.³ Burden et al.⁴ reported hyperventilation-induced unconsciousness during labour of a healthy woman. Our patient showed severe preoperative anxiety before induction of anaesthesia, but no anxiolytic was provided, except for some verbal support. Preoperative anxiety may be incrementally aggravated during an entire period of induction of regional anaesthesia and surgical preparation. Because our patient did not show complete loss of consciousness, we suspected that the anxiety induced hyperventilation, which caused the convulsions, rather than any other causes of seizure disorder. Second, cerebral hypoperfusion resulting from systemic hypotension may cause neurological symptoms. Severe hypotension following spinal anaesthesia may explain the decrease in mentality experienced by the patient.⁵ Aortocaval compression after a change in position from the lateral decubitus to supine position can aggravate post-spinal hypotension, thereby causing deterioration of consciousness due to secondary cerebral hypotension following spinal anaesthesia in caesarean delivery.⁶ Third, neurotoxicity of bupivacaine might have been a cause of the convulsions.⁷ However, in the present case, the brief duration of the episode and the other clinical symptoms (e.g., hyperventilation and tachycardia) decreased the possibility of bupivacaine neurotoxicity. Although the patient had a history of preeclampsia, she only experienced pregnancy-induced hypertension during this pregnancy, and we followed her up and provided continuous management. Furthermore, in the present case, convulsion was accompanied by hypotension, which offered less relation with the convulsion caused by eclampsia.

Severe hypotension, combined with convulsion in the present case, can occur in spinal anaesthesia for caesarean delivery because of a significant decrease in systemic vascular resistance via sympathetic blockade, even in healthy parturients.⁸ In pre-eclampsia, persistent vasoconstriction also depletes intravascular volume status and may cause possible left ventricular dysfunction.⁹ Furthermore, the use of high-dose local anaesthetics, preoperative hypovolemia, and aortocaval compression by the foetus can be associated with severe hypotension after spinal anaesthesia in parturients.^{10,11} A prophylactic low-dose phenylephrine infusion, fluid preloading, and the use of a lower extremity compression device may reduce the incidence of severe hypotension in spinal anaesthesia.^{5,12} In the present case, we used 11 mg of bupivacaine for spinal anaesthesia. Although this might be considered as a relatively high dose because an adequate level of anaesthesia could be achieved with only 7 to 8 mg of bupivacaine, preoperative anxiety also contributed to the severe hypotension. In patients with increased preoperative anxiety, sympathetic activity is exaggerated. Therefore, blockade of such increased sympathetic activity can cause marked hypotension after induction of spinal anaesthesia.¹³ In addition to the sympathetic blockade, intravascular volume depletion also contributes to severe hypotension, and sufficient intravenous hydration before spinal anaesthesia would provide a better outcome.

Preoperative anxiety may cause severe complications associated with the central nervous system or cardiovascular system. Unfortunately, in the present case, preoperative anxiety was not assessed, and was not clearly correlated with complications. However, by assessing preoperative anxiety with a validated method, we can provide adequate anxiety control before surgery and also provide better anaesthesia.¹⁴

A single dose of midazolam or fentanyl before caesarean delivery can lower the patient's anxiety and have no adverse neonatal effects.^{15,16} Even listening to music before surgery may be helpful for reducing stress-related physiological reactions.¹⁷ Careful consideration for the patients' anxiety is required in patients under regional anaesthesia compared with those under general anaesthesia.

Declaration of conflicting interest

The authors declare that there is no conflict of interest.

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