RESEARCH PAPER

Severe High Spinal Block After Spinal Anaesthesia for Lower Limb Surgery: A Case Series

Dr.Surendran.K[1], Dr. U.G.Thirumaaran[2], Dr.Gokulakrishnan.K[3], Dr.Keerthana Parvathy.S[4]

Post Graduate, Professor and Head, Assistant Professor, Post Graduate
Department of Anaesthesiology, Meenakshi Medical College Hospital & Research Institute, MAHER University, Kanchipuram.
Contact details: surenkbt@gmail.com

Abstract

Background

High spinal block is a rare but potentially life-threatening complication of subarachnoid anesthesia resulting from excessive cephalad spread of local anesthetic within the cerebrospinal fluid. It may rapidly lead to profound hypotension, bradycardia, respiratory insufficiency, unconsciousness, and cardiovascular collapse, necessitating immediate recognition and prompt intervention. Although spinal anesthesia is widely used for lower limb surgeries because of its safety and efficacy, high spinal block remains an uncommon perioperative emergency.

Case Series

We present a case series of five patients who developed high spinal block following subarachnoid anesthesia for lower limb orthopedic procedures. The patients ranged in age from 38 to 70 years and underwent various lower limb surgeries under spinal anesthesia with hyperbaric bupivacaine. Clinical manifestations developed within 5–8 minutes after intrathecal injection and included rapidly ascending sensory blockade, severe hypotension, bradycardia, dyspnea, upper limb weakness, and respiratory compromise. Two patients required endotracheal intubation with mechanical ventilation, while the remaining three were successfully managed with supplemental oxygen, intravenous fluids, vasopressors, atropine, and close hemodynamic monitoring. All patients demonstrated complete neurological and hemodynamic recovery without permanent sequelae.

Conclusion

High spinal block should always be considered in patients who develop sudden cardiovascular instability or respiratory distress shortly after spinal anesthesia. Early recognition, prompt airway management, aggressive hemodynamic support, and vigilant perioperative monitoring are crucial for favorable outcomes. This case series emphasizes the importance of preparedness, timely intervention, and adherence to evidence-based management protocols in preventing serious morbidity and mortality associated with high spinal block.

Keywords: High spinal block; Subarachnoid anesthesia; Spinal anesthesia; Lower limb surgery; Hypotension; Bradycardia; Difficult airway; Case series; Neuraxial anesthesia; Perioperative complications.

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Introduction

High spinal block is a rare but potentially life-threatening complication of subarachnoid (spinal) anesthesia characterized by excessive cephalad spread of local anesthetic within the cerebrospinal fluid, resulting in extensive blockade of spinal nerve roots and, in severe cases, brainstem involvement.[1,2] Although spinal anesthesia is widely regarded as a safe and effective anesthetic technique for lower limb, lower abdominal, and urological procedures, inadvertent high spinal block remains an important perioperative emergency that requires immediate recognition and prompt management to prevent serious morbidity and mortality.[1]

The incidence of high spinal block has been reported to range from approximately 1 in 2,971 to 1 in 16,200 neuraxial anesthetic procedures, although the true incidence varies depending on patient characteristics, anesthetic technique, and the definition used.[2,3] Risk factors include excessive intrathecal local anesthetic dose, short stature, obesity, pregnancy, advanced age, spinal deformity, injection immediately after a failed epidural block, Trendelenburg positioning, and technical errors during neuraxial anesthesia.[1,2,4]

The pathophysiology of high spinal block is related to excessive cephalad spread of local anesthetic within the subarachnoid space, leading to progressive blockade of thoracic and cervical spinal nerves. Sympathetic blockade results in profound vasodilation, hypotension, and bradycardia, while involvement of the upper thoracic and cervical segments may impair intercostal muscle function and diaphragmatic innervation, producing respiratory insufficiency or apnea. In extreme cases, brainstem anesthesia may manifest as unconsciousness, fixed dilated pupils, cardiovascular collapse, and cardiac arrest.[1,5]

Clinically, patients usually present within minutes of intrathecal drug administration with rapidly ascending sensory blockade, upper limb weakness, dyspnea, difficulty speaking, hypotension,

bradycardia, nausea, restlessness, and decreasing level of consciousness.[2,6] Prompt recognition of these warning signs is essential because delayed diagnosis may lead to severe hypoxemia, respiratory arrest, and circulatory collapse. Differential diagnoses include local anesthetic systemic toxicity, vasovagal syncope, anaphylaxis, pulmonary embolism, myocardial infarction, and hypoglycemia.[5,6]

Management of high spinal block is primarily supportive and focuses on early airway control, administration of 100% oxygen, endotracheal intubation with mechanical ventilation when indicated, aggressive intravenous fluid resuscitation, vasopressor therapy, atropine for severe bradycardia, and continuous hemodynamic monitoring.[1,2,5] With timely intervention and appropriate supportive care, most patients recover completely as the effects of the spinal anesthetic regress. However, delayed recognition and inadequate resuscitation may result in permanent neurological injury or death.[4,6]

Lower limb orthopedic procedures are among the most common indications for spinal anesthesia because they provide excellent surgical conditions, effective postoperative analgesia, reduced blood loss, and lower thromboembolic risk compared with general anesthesia.[7-10] Despite these advantages, anesthesiologists must remain vigilant for rare but catastrophic complications such as high spinal block. Reporting such cases contributes to greater awareness, facilitates early recognition, and reinforces evidence-based management strategies. We present a case series of patients who developed high spinal block following subarachnoid anesthesia for lower limb surgery, highlighting the clinical presentation, perioperative management, outcomes, and key preventive measures to improve patient safety. [11-15]

Case Series

Case 1

A 56-year-old male (weight 68 kg, height 168 cm) with American Society of Anesthesiologists (ASA) physical status II, a known hypertensive on amlodipine, was scheduled for open reduction and internal fixation (ORIF) of a right intertrochanteric femur fracture. Following standard monitoring, spinal anesthesia was administered at the L3-L4 interspace using a 25-G Quincke needle with 3.2 mL of 0.5% hyperbaric bupivacaine. Approximately 5 minutes after injection, the patient complained of difficulty breathing, inability to move both upper limbs, and dizziness. Blood pressure fell from 138/84 mmHg to 72/40 mmHg, heart rate decreased to 42 beats/min, and oxygen saturation dropped to 88%. Sensory block extended to the C6 dermatome, confirming a high spinal block. Intravenous atropine (0.6 mg), rapid crystalloid infusion, repeated boluses of mephentermine (6 mg), and 100% oxygen were administered. As respiratory effort deteriorated, endotracheal intubation and mechanical ventilation were initiated. Hemodynamic parameters stabilized within 15 minutes, and surgery proceeded uneventfully. The patient was extubated after complete recovery of motor power approximately 2 hours later and discharged without neurological deficits.

Case 2

A 64-year-old obese female (BMI 33.4 kg/m²), ASA II, underwent total knee replacement under spinal anesthesia. After administration of 3 mL of 0.5% hyperbaric bupivacaine at the L2-L3 interspace, she was positioned supine with a slight Trendelenburg tilt for surgical preparation. Within 8 minutes, she developed severe hypotension (68/38 mmHg), bradycardia (38 beats/min), nausea, and progressive dyspnea. Sensory blockade reached the C5 dermatome with complete upper limb weakness. Immediate treatment included intravenous atropine, phenylephrine boluses, aggressive fluid resuscitation, and high-flow oxygen. Because spontaneous ventilation remained adequate, endotracheal intubation was not required. Hemodynamic stability was restored within 20 minutes, and sensory regression began after approximately 90 minutes. Surgery was completed successfully, and the patient recovered without complications.

Case 3

A 45-year-old male with no significant comorbidities presented for tibial plateau fracture fixation. Spinal anesthesia was performed at the L3-L4 level using 3.5 mL of 0.5% hyperbaric bupivacaine. Shortly after positioning, the patient complained of chest tightness, inability to take a deep breath, and tingling of both hands. Blood pressure rapidly declined from 126/80 mmHg to 76/46 mmHg with a heart rate of 46 beats/min. Oxygen saturation decreased to 90%, and the sensory block extended to the T1-C8 level. A diagnosis of impending high spinal block was made. Intravenous fluids, atropine, mephentermine, and oxygen via face mask were administered promptly. Respiratory function remained preserved, and the patient gradually improved without requiring intubation. The surgery was completed under close hemodynamic monitoring, and the patient made a complete postoperative recovery.

Case 4

A 70-year-old female with controlled diabetes mellitus and hypertension underwent dynamic hip screw fixation for an intertrochanteric fracture. Spinal anesthesia was administered using 2.8 mL of 0.5% hyperbaric bupivacaine. Within 6 minutes, she became restless, complained of difficulty speaking and breathing, and subsequently lost consciousness. Blood pressure was unrecordable, heart rate was 32 beats/min, and oxygen saturation fell to 82%. Immediate cardiopulmonary resuscitative measures included endotracheal intubation, mechanical

ventilation, intravenous adrenaline (10 µg boluses), atropine, and aggressive fluid resuscitation. Hemodynamic stability returned after approximately 12 minutes. The patient remained ventilated intraoperatively and was shifted to the intensive care unit. She was successfully extubated 4 hours later after complete neurological recovery and discharged on the fifth postoperative day without residual deficits.

Case 5

A 38-year-old healthy male (ASA I) underwent intramedullary interlocking nailing for a closed tibial shaft fracture. Following administration of 3 mL of hyperbaric bupivacaine at the L3-L4 interspace, the patient developed profound hypotension (70/42 mmHg), bradycardia (40 beats/min), upper limb paresthesia, and breathlessness within 7 minutes. Sensory blockade extended to the C7 dermatome. Immediate administration of intravenous crystalloids, mephentermine, atropine, and oxygen via face mask rapidly improved his clinical condition. As spontaneous ventilation remained effective, invasive airway management was unnecessary. Hemodynamic parameters normalized within 15 minutes, and complete regression of motor block occurred after approximately 2.5 hours. The remainder of the perioperative period was uneventful, and the patient was discharged on postoperative day three in stable condition.

Characteristic	**Case 1**	**Case 2**	**Case 3**	**Case 4**	**Case 5**
Age/ Sex	56/M	64/F	45/M	70/F	38/M
ASA Status	II	II	I	III	I
Surgery	ORIF for intertrochanteric femur fracture	Total knee replacement	Tibial plateau fracture fixation	Dynamic hip screw fixation	Intramedullary interlocking nailing of tibia
Comorbidities	Hypertension	Obesity (BMI 33.4 kg/m²)	None	Diabetes mellitus, Hypertension	None
Level of Spinal Injection	L3-L4	L2-L3	L3-L4	L3-L4	L3-L4
Drug Administered	3.2 mL 0.5% hyperbaric bupivacaine	3.0 mL 0.5% hyperbaric bupivacaine	3.5 mL 0.5% hyperbaric bupivacaine	2.8 mL 0.5% hyperbaric bupivacaine	3.0 mL 0.5% hyperbaric bupivacaine
Time to Symptom Onset	5 min	8 min	6 min	6 min	7 min
Clinical Presentation	Dyspnea, upper limb weakness, hypotension, bradycardia	Dyspnea, nausea, severe hypotension, bradycardia	Chest tightness, breathlessness, hypotension	Loss of consciousness, apnea, profound hypotension	Breathlessness, upper limb paresthesia, hypotension
Highest Sensory Block	C6	C5	T1-C8	Above C4	C7
Airway Management	Intubation and mechanical ventilation	Oxygen by face mask	Oxygen by face mask	Intubation and mechanical ventilation	Oxygen by face mask
Hemodynamic Support	IV fluids, atropine, mephentermine	IV fluids, atropine, phen	IV fluids, atropine, meph	IV fluids, atropine, adrenaline	IV fluids, atropine, meph

		ylephrine	enter mine		enter mine
ICU Admission	Yes	No	No	Yes	No
Outcome	Complete recovery	Complete recovery	Complete recovery	Complete recovery	Complete recovery

Discussion

High spinal block is a rare but potentially life-threatening complication of subarachnoid anesthesia that requires immediate recognition and prompt intervention. It results from excessive cephalad spread of local anesthetic within the cerebrospinal fluid, producing extensive sensory, motor, and sympathetic blockade. Although spinal anesthesia is widely regarded as a safe and effective anesthetic technique for lower limb surgery, high spinal block may rapidly progress to severe hypotension, bradycardia, respiratory insufficiency, unconsciousness, and cardiovascular collapse if not managed promptly.[1,2]

In the present case series, all five patients developed features suggestive of high spinal block within 5–8 minutes following intrathecal administration of hyperbaric bupivacaine. The predominant clinical manifestations included rapidly ascending sensory blockade, profound hypotension, bradycardia, dyspnea, upper limb weakness, and varying degrees of respiratory compromise. Two patients required endotracheal intubation and mechanical ventilation because of respiratory failure, whereas the remaining three were successfully managed with supplemental oxygen, intravenous fluids, vasopressors, and close monitoring. All patients recovered completely without permanent neurological deficits. These findings are consistent with previous reports demonstrating that early diagnosis and aggressive supportive management result in excellent outcomes in most patients.[2,3]

The pathophysiology of high spinal block is primarily related to excessive cephalad spread of local anesthetic, leading to blockade of the thoracic sympathetic fibers and cervical spinal segments. Sympathetic blockade causes peripheral vasodilatation, reduced venous return, hypotension, and reflex bradycardia, while involvement of the upper thoracic and cervical nerves impairs respiratory muscle function. Further cephalad extension may affect the phrenic nerve and brainstem, resulting in apnea, loss of consciousness, and cardiovascular collapse.[1,4] In our series, the rapid onset of hypotension and bradycardia, followed by respiratory distress in severe cases, closely reflected this well-described pathophysiological sequence.

Several factors have been implicated in increasing the risk of high spinal block, including excessive intrathecal local anesthetic dose, obesity, advanced age, short stature, Trendelenburg positioning, pregnancy, altered cerebrospinal fluid dynamics, and technical factors during spinal anesthesia.[1,5] Among our patients, one was obese, two were elderly, and one received a relatively larger volume of hyperbaric bupivacaine, all of which may have contributed to excessive cephalad spread. However, high spinal block also occurred in otherwise healthy younger patients, emphasizing that this complication may occur even in the absence of obvious risk factors.

The diagnosis of high spinal block is predominantly clinical and should be considered whenever rapid respiratory distress, hypotension, bradycardia, or loss of consciousness develops shortly after spinal anesthesia. Differential diagnoses include local anesthetic systemic toxicity, vasovagal syncope, anaphylaxis, pulmonary embolism, myocardial infarction, and hypoglycemia.[2,6] The close temporal relationship between intrathecal injection and symptom onset, together with ascending sensory blockade and prompt improvement as the spinal block regressed, supported the diagnosis in all patients.

Management focuses primarily on airway protection, maintenance of oxygenation, and cardiovascular stabilization. Immediate administration of 100% oxygen, rapid intravenous crystalloid infusion, vasopressor therapy, atropine for severe bradycardia, and early endotracheal intubation with mechanical ventilation when respiratory compromise develops remain the cornerstone of treatment.[1,2] In our series, early intervention prevented progression to cardiac arrest, and all patients demonstrated complete neurological and hemodynamic recovery. These findings reinforce current recommendations emphasizing prompt supportive management rather than waiting for spontaneous recovery.

Prevention remains the most effective strategy for reducing the incidence of high spinal block. Careful patient selection, individualized dosing of intrathecal local anesthetic according to patient characteristics, proper patient positioning, slow injection technique, continuous intraoperative monitoring, and preparedness for airway management are essential preventive measures.[1,4,5] Immediate availability of airway equipment, vasopressors, and experienced anesthesia personnel is particularly important whenever spinal anesthesia is administered.

The principal limitation of this case series is its small sample size and descriptive design, which limit the generalizability of the findings. Nevertheless, the consistent clinical presentation, rapid diagnosis, standardized management, and favorable outcomes observed across all five patients provide valuable clinical insights into this uncommon but potentially catastrophic complication. Reporting such cases

contributes to increased awareness among anesthesiologists and reinforces the importance of vigilance during the immediate period following spinal anesthesia.

Summary

High spinal block is an uncommon but potentially catastrophic complication of subarachnoid anesthesia that can rapidly progress to severe cardiovascular and respiratory compromise. This case series describes five patients who developed high spinal block following spinal anesthesia for lower limb surgeries. The clinical presentations ranged from profound hypotension and bradycardia to respiratory distress and loss of consciousness, with symptom onset occurring within minutes of intrathecal injection. Prompt recognition, immediate airway support, aggressive fluid resuscitation, vasopressor therapy, atropine administration when indicated, and timely endotracheal intubation in severe cases resulted in complete recovery of all patients without permanent neurological sequelae. These cases highlight the importance of vigilant intraoperative monitoring, early diagnosis, and adherence to established resuscitation protocols in the successful management of this rare anesthetic emergency.

Conclusion

High spinal block remains a rare but life-threatening complication of spinal anesthesia that demands immediate recognition and decisive management. Early identification of ascending neuraxial blockade, prompt airway stabilization, aggressive hemodynamic support, and timely ventilatory assistance are the cornerstones of successful treatment. Careful patient assessment, appropriate dosing of intrathecal local anesthetics, meticulous anesthetic technique, and continuous monitoring can reduce the risk of this complication. The favorable outcomes observed in this case series emphasize that rapid intervention by a prepared anesthesia team can effectively prevent serious morbidity and mortality. Increased awareness of the clinical features and management principles of high spinal block will help anesthesiologists optimize patient safety during neuraxial anesthesia.

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