ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26feb250

Transient Bulbar-Like Neuromuscular Manifestations Following Spinal Anaesthesia with Hyperbaric Bupivacaine: A Case Report

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Publication Date: 2026/02/10

Abstract: Spinal anaesthesia using hyperbaric bupivacaine is widely regarded as a safe and reliable technique when administered within recommended dose ranges. While complications such as high spinal anaesthesia and local anaesthetic systemic toxicity are well described, atypical neurological manifestations without associated hemodynamic instability are rarely reported. We describe a case of a young adult male who developed transient bulbar-like neuromuscular manifestations following spinal anaesthesia with a standard dose of hyperbaric bupivacaine. After a rapid onset of complete lower limb motor block and mid-thoracic sensory blockade, the patient exhibited subtle lower limb fasciculations and subsequently developed tongue fasciculations in the absence of hypotension, bradycardia, respiratory compromise, or altered consciousness. The symptoms persisted into the early postoperative period and resolved spontaneously without intervention or residual neurological deficit. This case highlights an unusual presentation suggestive of abortive neurotoxicity following intrathecal bupivacaine administration and underscores the importance of recognising atypical, self-limiting neurological events during spinal anaesthesia.

Keywords: *Spinal Anesthesia; Bupivacaine; Neurotoxicity Syndromes; Fasciculation; Neuraxial Anesthesia.*

How to Cite: Dr. Ajit Kumar; Dr. Saif Ali Chishti; Dr. Radheshyam; Dr. Vigneshwar (2026) Transient Bulbar-Like Neuromuscular Manifestations Following Spinal Anaesthesia with Hyperbaric Bupivacaine: A Case Report. *International Journal of Innovative Science and Research Technology*, 11(2), 172-175. https://doi.org/10.38124/ijisrt/26feb250

I. INTRODUCTION

Spinal anaesthesia using hyperbaric bupivacaine is a commonly employed regional anaesthetic technique for lower limb surgery owing to its rapid onset, predictable block characteristics, and favourable safety profile when administered within recommended dose ranges. The therapeutic window for intrathecal bupivacaine is broad, with evidence demonstrating adequate spinal anaesthesia at doses ranging from approximately 4 to 15 mg, depending on patient positioning and surgical context.[1-3] Serious neurological complications are uncommon, and recognised adverse effects of spinal anaesthesia include hypotension and bradycardia secondary to sympathetic blockade. In contrast, rarer complications include high or total spinal anaesthesia and local anaesthetic systemic toxicity.

The extent and clinical effects of spinal anaesthesia are influenced by multiple factors beyond the absolute dose of local anaesthetic, including cerebrospinal fluid dynamics, baricity of the solution, patient positioning, and individual anatomical variability. These considerations have led to an increasing emphasis on evidence-based, patient-specific dosing strategies, including height-adjusted approaches, to optimise block characteristics and minimise adverse effects.[1] Transient neurological manifestations that do not conform to classical patterns of high spinal anaesthesia or extensive sensory blockade are infrequently reported and may present diagnostic uncertainty in the intraoperative setting.

We report a case of transient bulbar-like neuromuscular manifestations following spinal anaesthesia with hyperbaric bupivacaine to highlight this uncommon presentation and discuss its clinical relevance.

II. CASE REPORT

- *Patient Information*

A young adult male with an anterior cruciate ligament (ACL) tear in the right knee was scheduled for arthroscopic ACL repair. He weighed 63 kg and was 170 cm tall.

- *Pre-anaesthetic Evaluation*

Pre-anaesthetic evaluation was performed the day before surgery. The patient was classified as American Society of Anesthesiologists (ASA) physical status I. He had

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26feb250

no known comorbid illnesses, was not receiving any regular medications, and had no history of drug allergies or previous anaesthetic exposure. There was no history suggestive of anxiety or panic disorder. General physical and systemic examination, including neurological assessment, was unremarkable. Routine laboratory investigations were within normal limits. The patient was advised standard preoperative fasting and planned for spinal anaesthesia.

➢ *Anaesthesia in the Operating Theatre*

On arrival in the operating theatre, standard ASA monitors were applied. Baseline hemodynamic parameters were within normal limits, and oxygen saturation was normal while breathing room air. Intravenous access was secured, and an initial normal saline infusion was initiated. The patient was calm, cooperative, and oriented.

➢ *Anaesthetic Technique*

Spinal anaesthesia was planned as the sole anaesthetic technique. With the patient in the sitting position, the back was prepared and draped under strict aseptic precautions. A total of 2.5 mL of 0.5% hyperbaric bupivacaine (12.5 mg) was drawn into a 3-mL syringe. Subarachnoid block was performed at the L4–L5 interspace using a 26-gauge Quincke spinal needle. Free flow of cerebrospinal fluid was confirmed, and the local anaesthetic was injected intrathecally at an approximate rate of 0.1 mL/s. No intrathecal adjuvants were used. The patient was positioned supine immediately after completion of the injection.

➢ *Intraoperative Events*

Approximately two minutes after intrathecal drug administration, motor blockade progressed rapidly to complete lower limb paralysis consistent with a Bromage score of 3. Sensory blockade was assessed to be up to approximately the T8 dermatome, with motor block corresponding to around the T10 level. Hemodynamic parameters and oxygen saturation remained stable throughout, without hypotension, bradycardia, or desaturation, and the patient continued to breathe comfortably on room air.

During the surgical team's positioning of the operative limb, subtle fasciculation-like movements were observed around the knee joint. At this time, lower limb motor power was absent, and vital parameters remained unchanged.

Shortly thereafter, the patient complained of a brief sensation of difficulty breathing, despite being observed to be breathing adequately. This episode was transient, lasting approximately 20–25 seconds, and resolved spontaneously without intervention. The patient remained conscious, calm, and cooperative throughout, with stable hemodynamic parameters and oxygen saturation.

Following resolution of this transient episode, the patient was asked to open his mouth and protrude his tongue as part of routine clinical reassurance. At this point, frank tongue fasciculations were observed. There were no associated changes in oxygen saturation, blood pressure, heart rate, or level of consciousness. No intravenous benzodiazepines or opioids were administered at any time.

Subsequently, in accordance with institutional orthopaedic protocol, tranexamic acid 1 g was added to the second ongoing infusion of normal saline, with approximately 60–70% of the volume remaining. Ondansetron 4 mg was administered intravenously. Surgery proceeded uneventfully and was completed within approximately 90 minutes.

➢ *Post-anaesthesia Care and Follow-up*

Postoperatively, the patient was monitored in the post- anaesthesia care unit for 45 minutes. During this period, tongue fasciculations persisted, while all other parameters remained within normal limits. Recorded vital signs included a blood pressure of 132/80 mmHg and a heart rate of 72 beats per minute. Oxygen saturation remained 100% on room air.

No additional neurological or systemic abnormalities were noted.

On reassessment later that evening, the patient demonstrated complete recovery of lower limb motor power, with full resolution of tongue fasciculations. Follow-up examination on the following day revealed no neurological deficits. The postoperative course remained uneventful, and the patient was discharged on postoperative day 2.

➢ *Differential Diagnosis Considered*

High spinal anaesthesia was considered unlikely given the limited sensory spread, preserved hemodynamic stability, absence of respiratory compromise, and maintained level of consciousness. Anxiety-related dyspnoea was considered but did not adequately explain the objectively observed and persistent tongue fasciculations. Local anaesthetic systemic toxicity was considered unlikely due to the absence of central nervous system excitation, seizures, cardiovascular instability, or progression of symptoms. An accidental subdural or dual-compartment block was considered; however, the rapid onset of dense motor blockade, a well- demarcated sensory level, the absence of delayed or progressive cephalad spread, and the absence of cranial nerve palsies or altered consciousness made this unlikely. Tranexamic acid–related neurotoxicity was considered unlikely, as the drug was administered only after the onset of neurological manifestations. Intrathecal opioid-related effects were excluded, as no intrathecal adjuvants were used.

III. DISCUSSION

Hyperbaric bupivacaine remains the most commonly used local anaesthetic for spinal anaesthesia in lower limb surgery because of its predictable onset and block characteristics when administered within recommended dose ranges. Standard intrathecal doses for adult patients typically range from 10 to 15 mg, with recent literature increasingly emphasising tailoring the dose to patient height and body habitus rather than relying on a fixed volume [1–3].

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Studies evaluating height-adjusted dosing have demonstrated that doses in the range of 10–13 mg reliably achieve adequate sensory block for lower limb surgery while minimising excessive cephalad spread and hemodynamic instability [2,3]. In the present case, a dose of 12.5 mg (2.5 mL of 0.5% hyperbaric bupivacaine) was administered, which lies well within the recommended and commonly studied range and cannot be considered excessive [1–3].

The block characteristics observed in this case—rapid onset of dense lower limb motor blockade (Bromage score 3) with a stable and well-demarcated sensory level limited to approximately the T8 dermatome—are consistent with a typical intrathecal block profile. Notably, there was no progression of sensory blockade, no hemodynamic instability, and no alteration in consciousness, effectively excluding high or total spinal anaesthesia [2,3]. The stability of the sensory level over time further argues against excessive cephalad spread.

The most distinctive feature of this case was the presence of tongue fasciculations occurring in the absence of hypotension, hypoxemia, altered sensorium, or other features suggestive of classical local anaesthetic systemic toxicity. The term “bulbar-like” is used descriptively in this report to denote transient involvement of a bulbar-innervated muscle group, evidenced by tongue fasciculations, in the absence of cranial nerve dysfunction, dysarthria, dysphagia, altered consciousness, or respiratory compromise. No features of true bulbar palsy were present. Fasciculations are not characteristic of anxiety-related phenomena or high spinal anaesthesia and suggest transient neuronal irritability rather than conduction block [4]. Classical systemic toxicity typically manifests with central nervous system excitation, seizures, or cardiovascular compromise, none of which were observed in this patient.

Accidental subdural block was considered, as atypical neurological manifestations with preserved hemodynamic stability are often attributed to this entity. However, subdural block is classically associated with delayed onset, patchy and disproportionately extensive sensory blockade, minimal or delayed motor involvement, and progressive symptom evolution [5]. In contrast, this case demonstrated a rapid onset of dense motor blockade, a predictable and limited sensory distribution, and the absence of delayed progression, all of which make subdural injection unlikely. A mixed or dual- compartment (intrathecal–subdural) block was also considered, particularly given the standard-to-upper-range dose used. However, mixed blocks are typically characterised by evolving or delayed neurological findings attributable to subdural spread, which were not observed in this case [5]. The stability of the block characteristics and the self-limiting nature of the fasciculations argue against significant subdural involvement.The patient's brief subjective sensation of difficulty breathing was not accompanied by hypoxemia, hemodynamic instability, or objective respiratory compromise and resolved spontaneously within seconds. In the absence of physiological deterioration, such transient dyspnoea perception during spinal anaesthesia is most plausibly explained by altered thoracic sensory or perceptual input due to neuraxial blockade, rather than actual ventilatory impairment. Preservation of oxygen saturation on room air and normal consciousness in this case supports a sensory-perceptual explanation rather than respiratory dysfunction or evolving high spinal anaesthesia.

Taken together, the findings in this case are most consistent with an atypical, self-limiting neurological manifestation following intrathecal bupivacaine administration, best described as an abortive or incomplete neurotoxic phenomenon [4]. The absence of progression, rapid spontaneous resolution, and lack of residual neurological deficit suggest involvement at the mild end of a neurotoxic spectrum. Such presentations may be under-recognised, as symptoms can be transient and resolve without intervention.

IV. CLINICAL IMPLICATIONS

This case highlights several important clinical considerations. First, atypical neurological manifestations can occur following spinal anaesthesia even when evidence-based doses and accepted techniques are used [1–3]. Second, preserved hemodynamic stability, limited sensory spread, and absence of progression are key features that distinguish these presentations from high spinal anaesthesia or subdural block[5]. Third, not all neurological symptoms during neuraxial anaesthesia represent anxiety or mandate aggressive intervention; structured clinical assessment with close observation is often appropriate. Awareness of abortive neurotoxic manifestations may help anesthesiologists avoid unnecessary escalation of care while ensuring patient safety through vigilant monitoring [4].

V. CONCLUSIONS

This case illustrates an unusual and self-limiting presentation of transient bulbar-like neuromuscular manifestations following spinal anaesthesia with an evidence-based intrathecal dose of hyperbaric bupivacaine. Despite the rapid onset of dense motor blockade, preserved hemodynamic stability, a stable sensory level, and the absence of progressive neurological deterioration, this presentation clearly distinguished itself from high spinal anaesthesia, subdural block, and classical local anaesthetic systemic toxicity. Brief subjective dyspnoea in this context likely reflected altered thoracic sensory perception rather than true respiratory compromise. The complete spontaneous recovery without intervention suggests an abortive or incomplete neurotoxic phenomenon. Recognition of such atypical yet benign neurological manifestations is essential to guide structured clinical assessment, ensure vigilant monitoring, and avoid unnecessary escalation of care while maintaining patient safety during neuraxial anaesthesia.

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26feb250

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