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Transient Hypercapnia-Induced Loss of Consciousness Following Carbetocin Administration During Cesarean Delivery: A Case Report

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Abstract **Case Report**

Carbetocin, a long-acting oxytocin analogue, is widely used to prevent uterine atony. While its safety profile is well-established, its physiological impact can be amplified by a patient's emotional and metabolic state. We report a case of a 31-year-old parturient undergoing elective cesarean section for twins who experienced a tonic seizure after carbetocin administration. Despite a standard 1-minute intravenous injection, the patient exhibited unresponsive rigidity followed by a seizure. Arterial blood gas analysis (ABGA) revealed acute respiratory acidosis with normal electrolytes. The seizure was attributed to the synergistic effect of carbetocin-induced respiratory depression, spinal anesthesia-related intercostal muscle paralysis, and increased metabolic CO2 production from severe anxiety regarding fetal distress. The patient recovered completely within 5 minutes after low-dose propofol administration and ventilatory support. This case emphasizes the need for vigilant respiratory monitoring following carbetocin administration, especially in anxious parturients.

Keywords: Carbetocin, Hypercapnia, Seizure, Cesarean delivery, Spinal anesthesia, Respiratory acidosis.

INTRODUCTION

Cesarean delivery is one of the most performed surgical procedures worldwide, and neuraxial anesthesia is widely regarded as the preferred anesthetic technique due to its favorable maternal and neonatal outcomes [1,2]. Despite its safety profile, cesarean delivery is associated with a range of intraoperative complications, including hemodynamic instability, respiratory compromise, and, less commonly, acute neurologic events [1,3]. Among these, the occurrence of seizure or seizure-like activity during cesarean delivery represents a rare but potentially life-threatening condition that necessitates immediate evaluation and management [3,4].

In obstetric patients, eclampsia is often the primary consideration when seizure occurs; however, a broad differential diagnosis must be explored [5]. Potential etiologies include venous air embolism, amniotic fluid embolism, local anesthetic systemic toxicity, metabolic disturbances, and primary epileptic disorders [6–8]. Each of these conditions has distinct pathophysiological mechanisms and management strategies, making early recognition crucial for optimal patient outcomes [4,8].

Here, we describe a case of intraoperative seizure-like activity occurring during cesarean delivery under neuraxial anesthesia after carbetocin administration.

CASE REPORT

The Institutional Review Board of Presbyterian Medical Center approved this study and waived the requirement for informed consent owing to the retrospective review of medical records (IRB No. PMC 2026-06-017).

A 31-year-old woman (height, 155.5 cm; weight, 65.1 kg; body mass index, 26.92 kg/m²) was scheduled for an elective Cesarean delivery. She was at 38 weeks of gestation with a dichorionic diamniotic twin pregnancy complicated by intrauterine growth restriction. The patient had no significant past medical history or medication history, except for a prior uterine polypectomy. Preoperative investigations, including electrocardiography, chest radiography, and routine laboratory tests, yielded unremarkable findings. On the day of surgery, no premedication was administered, and baseline vital signs were within normal limits. Upon arrival in the operating room, standard monitoring

devices—including noninvasive blood pressure, electrocardiogram, pulse oximetry, and patient state index—were applied. Initial vital signs were stable, with a blood pressure (BP) of 157/77 mmHg, a heart rate (HR) of 70 beats/min, and an oxygen saturation (SpO2) of 97% on room air. Spinal anesthesia was successfully performed without technical difficulty, and a sensory block level of T6 was achieved. The patient remained hemodynamically stable without any subjective complaints until delivery. Approximately 5 minutes after the start of surgery, a uterine incision was made. The first neonate was delivered in a breech presentation with a true umbilical cord knot. The neonate exhibited generalized cyanosis and poor crying, with a 1-minute Apgar score of 7. Blood-tinged amniotic fluid was noted, and placenta accreta was identified. Two minutes later, the second neonate was delivered in a vertex presentation, cried vigorously, and appeared well, with a 1-minute Apgar score of 9. The placenta of the first twin was not completely delivered owing to the placenta accreta. Following the complete delivery of the second placenta, carbetocin (Duratocin®) 100 μg was administered intravenously. At that time, maternal BP was 98/48 mmHg. Approximately 10 minutes post-delivery (8 minutes after carbetocin administration), the patient's SpO2 decreased to 94%, and the end-tidal CO2 waveform was not detected. Upon immediate physical assessment, the patient was found to be in a state of generalized tonic seizure with unresponsiveness and muscle rigidity followed by upward eye deviation and loss of consciousness. Hemodynamic parameters remained stable. Airway management with assisted ventilation using 100% oxygen was immediately initiated. After approximately 3 minutes of assisted mask ventilation, an arterial blood gas analysis (ABGA) was performed. The ABGA revealed consistent with acute respiratory acidosis without significant metabolic compensation [table 1].

Table 1: Results of Arterial Blood Gas Analysis (ABGA) and electolyte

Parameter	Patient Value	Reference Range
pH	7.25	7.35 - 7.45
PaCO2 (mmHg)	54	35.0 - 45.0
PaO2 (mmHg)	452	83.0 - 108.0
HCO3- (mmol/L)	23.7	21.0 - 27.0
Base Excess (mmol/L)	-1.8	-2.0 - +2.0
SaO2 (%)	100	
Na+ (mmol/L)	137	135-145
Cl- (mmol/L)	107	98-107
K+ (mmol/L)	3.8	3.5-4.5

The patient remained unresponsive with persistent respiratory distress. Because no clinical improvement was observed 8 minutes after the onset of the event, intravenous propofol of 50 mg was administered for neural stabilization and seizure control. Within 5 minutes, the seizure activity ceased, and the patient gradually regained clear consciousness (alert state, Glasgow Coma Scale 15) along with spontaneous respiration. During this recovery period, SpO2 remained stable at 99–100%, and hemodynamic parameters were maintained with a systolic BP of 90–100 mmHg and a mean arterial pressure of 64–72 mmHg.

Following the recovery of consciousness, the sensory block level was reassessed and found to have extended to T4. The patient exhibited retrograde amnesia regarding the event but presented no neurological deficits or sequelae. In the post-anesthesia care unit, her body temperature was 36.6°C. Although no shivering or chills were reported, mild generalized tremors were observed without postictal symptoms. Vital signs remained stable. The tremors gradually resolved within approximately 1 hour, and the patient was transferred to the general ward after 1 hour and 25 minutes of observation. Subsequently, no further neurological deficits, myalgia, or respiratory complications were observed. Routine postoperative laboratory evaluations and electroencephalography (EEG) showed no significant abnormalities, and she was discharged on schedule.

DISCUSSION

In this case, a 31-year-old parturient undergoing elective twin cesarean section experienced a sudden loss of consciousness accompanied by tonic seizure-like activity and severe respiratory acidosis following intravenous carbetocin administration. While intraoperative seizures during neuraxial anesthesia immediately raise suspicion for eclampsia, local anesthetic systemic toxicity (LAST), or amniotic fluid embolism (AFE), the prompt recovery without neurological sequelae, coupled with the rapid onset of hypercapnia, suggests a unique multifactorial etiology. We hypothesize that this transient neurological event was driven by acute hypercapnia resulting from a synergistic interaction between carbetocin-induced chest tightness, high spinal block, and extreme maternal anxiety, culminating in hypercapnic encephalopathy.

Carbetocin, a long-acting oxytocin analogue, is widely recommended for the prevention of postpartum hemorrhage due to its sustained uterotonic efficacy [9].

However, its administration is frequently associated with systemic side effects, particularly transient hypotension, tachycardia, and respiratory discomfort such as chest tightness or dyspnea [10]. Oxytocin receptors present in the cardiovascular and respiratory systems can induce vasodilation and transient bronchospasm or chest oppression when stimulated rapidly [10,11]. Although these side effects are usually self-limiting, their clinical impact can be substantially amplified when superimposed on preexisting physiological or psychological vulnerabilities. A key contributing factor in this patient was the cephalad extension of the spinal sensory block to the T4 level. While high spinal anesthesia ensures adequate surgical analgesia, high thoracic sensory blockade (T4–T6) suppresses motor nerve supply to the intercostal muscles, impairing chest wall expansion and reducing vital capacity [12]. Although diaphragmatic innervation via the phrenic nerve (C3–C5) remains intact, patients with a high sensory block rely almost exclusively on abdominal breathing. This mechanical limitation diminishes the patient's ability to compensate for sudden increases in metabolic demand or respiratory resistance [12,13]. Furthermore, maternal emotional distress plays a critical role in altering intraoperative respiratory dynamics. In this case, the delivery of the first twin was complicated by breech presentation, a true umbilical cord knot, generalized cyanosis, and blood-tinged amniotic fluid indicative of fetal distress. The acute psychological stress arising from concern over the neonate's well-being undoubtedly triggered a severe surge in catecholamines and metabolic rate [14]. Sudden anxiety often leads to subconscious breath-holding or erratic, shallow breathing patterns, leading to acute carbon dioxide retention, especially when thoracic compliance is already compromised by a T4 sensory block [14,15]. When carbetocin was administered, the sudden onset of carbetocin-induced chest tightness acted as a trigger. Compounded by intercostal muscle paresis from the T4 spinal block, the patient was unable to overcome the sensation of respiratory oppression. This led to hypoventilation, severe hypercapnia, and acute respiratory acidosis. Rapidly rising arterial carbon dioxide ($PaCO_2$) causes profound cerebrovascular dilation and transient intracellular central nervous system acidosis, which can acutely depress cortical function and trigger hypercapnic encephalopathy [16,17]. Hypercapnic encephalopathy classically presents with loss of consciousness, motor irritability, myoclonus, and tonic muscle rigidity mimicking epileptic seizures [17,18]. The absence of prior seizure history, quick resolution following ventilation and low-dose propofol, and normal postoperative EEG further support hypercapnic encephalopathy rather than a primary epileptic event or LAST.

Conclusion

This case highlights that even standard doses of carbetocin can precipitate life-threatening transient hypercapnia and neurological collapse when combined with a high spinal block and high maternal anxiety regarding neonatal distress. Clinicians must maintain a high index of suspicion for respiratory depression following uterotonic administration, ensure vigilant respiratory monitoring beyond simple pulse oximetry (such as capnography), and provide early ventilatory support to prevent hypercapnic neurological events.

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