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Case Report

Anesthetic Management of Cesarean Delivery in a Parturient with Nutcracker Syndrome: A Case Report

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Abstract: Nutcracker syndrome (NCS) is a rare vascular disorder caused by compression of the left renal vein, and pregnancy may aggravate its clinical manifestations. We report the anesthetic management of a 37-year-old primigravida with pre-existing NCS who underwent elective cesarean delivery at term. Spinal anesthesia was performed with continuous arterial pressure monitoring and preparation for potential bleeding. Transient hypotension following spinal anesthesia was promptly treated with a vasopressor, and the subsequent perioperative course was uneventful. This case suggests that spinal anesthesia may be a feasible option for cesarean delivery in parturients with stable NCS when accompanied by careful hemodynamic management and appropriate preparation for potential complications.

Keywords: Nutcracker Syndrome, Pregnancy, Cesarean Delivery, Spinal Anesthesia; Hematuria, Hemodynamic Management.

INTRODUCTION

Nutcracker syndrome (NCS) is a rare vascular disorder caused by compression of the left renal vein (LRV), most commonly between the superior mesenteric artery and the abdominal aorta, resulting in renal venous hypertension [1]. Clinical manifestations include hematuria, left flank pain, orthostatic proteinuria, and symptoms of pelvic venous congestion [1, 2].

Pregnancy may aggravate NCS because pregnancy-related physiological and anatomical changes can increase LRV compression and renal venous hypertension [3, 4]. However, NCS during pregnancy is rarely reported, and its clinical course and management remain poorly defined [3-5].

Neuraxial anesthesia is generally preferred for cesarean delivery, although spinal anesthesia may cause hypotension through sympathetic blockade and reduced venous return [6]. We report the successful anesthetic management of a 37-year-old primigravida with pre-existing NCS who underwent elective cesarean delivery under spinal anesthesia.

CASE REPORT

A 37-year-old primigravida with a history of NCS was scheduled for elective cesarean delivery at 39 weeks of gestation because of advanced maternal age. Five years previously, microscopic hematuria had been incidentally detected during a routine health examination. Further evaluation, including computed tomography angiography, demonstrated compression of the left renal vein between the superior mesenteric artery and the abdominal aorta, leading to the diagnosis of NCS. She was subsequently managed conservatively by the nephrology department with an angiotensin-converting enzyme inhibitor and aspirin for approximately 6 months. Thereafter, her clinical condition remained stable, and she continued aspirin therapy without requiring surgical or endovascular intervention.

During pregnancy, the degree of hematuria remained similar to that before pregnancy, without apparent exacerbation of NCS-related symptoms. Preoperative laboratory investigations revealed a hemoglobin concentration of 12.1 g/dL, hematocrit of 34.9%, and platelet count of 243,000/mm³. Blood urea nitrogen and serum creatinine concentrations were 7.6 and 0.43 mg/dL, respectively. Urinalysis demonstrated protein 1+ and blood 1+. The coagulation profile, including bleeding time and prothrombin time, was within normal limits. Serum antinuclear antibodies were absent. Renal ultrasonography showed normal kidneys, and cystoscopic examination demonstrated healthy urothelium without an identifiable source of urinary tract bleeding.

Considering the potential for perioperative hemodynamic changes and bleeding associated with NCS, careful anesthetic planning was undertaken. On arrival in the operating room, standard monitoring with electrocardiography, pulse oximetry, and noninvasive blood pressure monitoring was initiated. A catheter was placed in the right radial artery for continuous invasive arterial blood pressure monitoring, and an additional peripheral intravenous line was secured. A cell salvage system was also prepared in anticipation of possible significant intraoperative bleeding.

After administration of 500 mL of Hartmann's solution, spinal anesthesia was performed with the patient in the left lateral decubitus position using a midline approach. A 25-gauge spinal needle was advanced into the subarachnoid space, and 8.5 mg of 0.5% hyperbaric bupivacaine combined with fentanyl 15 μg was administered intrathecally. Following spinal anesthesia, the patient was positioned supine with left lateral uterine displacement to minimize aortocaval compression.

Shortly after spinal anesthesia, arterial blood pressure decreased to 80/54 mmHg. Phenylephrine 100 μg was administered intravenously, after which the blood pressure promptly increased to 135/81 mmHg. No additional vasopressor administration was required, and the patient remained hemodynamically stable throughout the remainder of the procedure.

The cesarean delivery was completed uneventfully. The duration of surgery was approximately 60 min, and the total anesthetic time was 75 min. Estimated blood loss was approximately 500 mL. A total of 1,400 mL of crystalloid solution was administered intraoperatively, and urine output was 120 mL. Neither allogeneic blood transfusion nor reinfusion of cell-salvaged blood was required.

The postoperative course was uneventful. Postoperative hemoglobin and hematocrit were 10.2 g/dL and 30.1%, respectively. Renal function remained clinically stable, and no anesthetic or surgical complications occurred. Hematuria gradually resolved. At the 4-week postpartum follow-up, the patient remained clinically well and had experienced no further episodes of hematuria.

DISCUSSION

NCS results from impaired LRV outflow and consequent renal venous hypertension, which may lead to the development of collateral venous pathways [1, 2]. Hematuria, a characteristic manifestation of NCS, is thought to result from rupture of thin-walled venous collaterals into the renal collecting system [2]. The clinical severity of NCS varies considerably, and conservative management is generally appropriate for patients with mild symptoms and preserved renal function, whereas invasive treatment may be required for severe or persistent symptoms [1].

Pregnancy may influence the clinical course of NCS through substantial changes in maternal hemodynamics and abdominal vascular anatomy. Itoh *et al*., reported a patient who developed gross hematuria during the third trimester, which persisted until cesarean delivery and resolved postpartum, suggesting that pregnancy may aggravate NCS [3]. Uzu *et al*., also reported NCS presenting with hematuria during pregnancy [4]. In addition, Singh *et al*., described a pregnant woman with pre-existing NCS who developed prominent pelvic and vulvar varicosities but was successfully managed conservatively and underwent cesarean delivery with an estimated blood loss of 500 mL [5]. In contrast, our patient had only persistent microscopic hematuria without apparent worsening during pregnancy, and renal function remained preserved throughout the perioperative period.

The anesthetic management of NCS during pregnancy has rarely been described. Neuraxial anesthesia is generally preferred for cesarean delivery; however, spinal anesthesia produces sympathetic blockade, resulting in a decrease in systemic vascular resistance and venous return, and hypotension is a common consequence [6]. In term parturients, aortocaval compression by the gravid uterus may further reduce venous return. Although there is insufficient evidence to establish that patients with NCS are at greater risk of spinal anesthesia-induced hypotension, the abnormal renal and pelvic venous circulation associated with NCS raises a theoretical concern regarding tolerance to abrupt changes in venous return. Therefore, avoidance of prolonged hypotension and maintenance of adequate intravascular volume were considered important in our patient.

We administered 8.5 mg of hyperbaric bupivacaine with 15 μg of intrathecal fentanyl to limit the extent of sympathetic blockade while maintaining adequate surgical anesthesia. Dose-response studies have demonstrated that the dose of intrathecal bupivacaine influences both the adequacy of surgical

anesthesia and the incidence of hypotension or vasopressor requirement [7, 8]. Ginosar *et al*., reported ED50 and ED95 values of 7.6 and 11.2 mg, respectively, for successful cesarean delivery when hyperbaric bupivacaine was combined with intrathecal opioids [7]. More recent dose-ranging data have similarly shown an increase in phenylephrine requirement with increasing intrathecal bupivacaine doses [8]. Thus, the relatively low dose used in our patient was selected to reduce the potential magnitude of sympathectomy while providing adequate anesthesia with intrathecal fentanyl.

Despite these precautions, blood pressure transiently decreased to 80/54 mmHg shortly after spinal anesthesia. A single intravenous bolus of phenylephrine 100 µg promptly restored blood pressure to 135/81 mmHg, and no additional vasopressor was required. Left uterine displacement was maintained to minimize aortocaval compression. The rapid response to phenylephrine and subsequent stable hemodynamic course suggest that prompt recognition and treatment of spinal anesthesia-induced hypotension may permit safe neuraxial anesthesia in patients with clinically stable NCS.

Another important consideration was the possibility of increased perioperative bleeding. Chronic renal venous hypertension in NCS may lead to the development of gonadal and pelvic venous collaterals [2]. During pregnancy, these vessels may become further dilated; Singh *et al*., observed prominent left-sided pelvic veins during cesarean delivery in a patient with NCS [5]. Accordingly, an additional intravenous line was secured and a cell salvage system was prepared in our patient. Estimated blood loss was 500 mL, and neither allogeneic transfusion nor reinfusion of cell-salvaged blood was required. Postoperative hemoglobin decreased from 12.1 to 10.2 g/dL without clinical evidence of significant hemorrhage.

Preservation of adequate renal perfusion was also considered important because NCS directly affects renal venous outflow. The episode of hypotension was brief and rapidly corrected, intraoperative urine output was maintained, and no postoperative deterioration in renal function occurred. Hematuria gradually resolved after delivery and had not recurred at the 4-week postpartum follow-up. Resolution or improvement of hematuria after delivery has also been reported in previous pregnancy-associated NCS cases [3, 4], possibly reflecting reversal of pregnancy-related hemodynamic and anatomical changes.

CONCLUSION

Spinal anesthesia was successfully used for cesarean delivery in a parturient with pre-existing NCS and preserved renal function. Careful preoperative assessment, maintenance of maternal hemodynamic stability, prompt treatment of spinal anesthesia-induced hypotension, and preparation for potential bleeding may contribute to safe perioperative management. Given the limited evidence regarding anesthetic management of NCS during pregnancy, the anesthetic approach should be individualized according to disease severity, renal function, and associated venous abnormalities.

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Conflict of Interest: There in no conflict of interest.

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