

Repeated Episodes of Respiratory Distress in an Obese Parturient After Cesarean Delivery

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A 25-yr-old obese parturient with mild asthma underwent an uneventful spinal anesthetic for primary cesarean delivery. Within 4 h after delivery, the patient twice developed acute shortness of breath, inspiratory stridor, and hypoxemia that required intubation. A battery of blood tests revealed no evidence of an allergic reaction. She had a normal echocardiogram and chest computed tomography, but her neck computed tomography showed an enlarged left thyroid lobe asymmetrically compressing the endotracheal tube cuff. We hypothesized that, after delivery, decreased maternal vascular capacitance increased central venous pressure such that venous engorgement of an undiagnosed goiter may have caused symptomatic tracheal compression.

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Although neuraxial anesthesia offers advantages for cesarean delivery,¹ it does not guarantee freedom from postoperative respiratory complications. A parturient who develops sudden shortness of breath in the first few hours after spinal anesthesia for cesarean delivery requires immediate evaluation and management to maintain adequate oxygenation and ventilation. One should also be alert to the possibility of thromboembolism or amniotic fluid embolism, the leading causes of maternal death in the United States.² Nonobstetrical causes of respiratory compromise must also be considered. Preexisting asthma should prompt diagnosis and treatment of bronchospasm. Simultaneous hypotension, bronchospasm, and respiratory distress suggest an allergic reaction or anaphylaxis.³

Thyroid gland enlargement, or goiter, has been described to cause upper airway obstruction in parturients before delivery,⁴⁻⁹ at 1 wk postpartum,¹⁰ and in association with obesity⁹ and asthma.^{6,9} We describe a case of repeated episodes of inspiratory obstruction in the first 12 h postpartum associated with increased maternal central venous pressure (CVP), physical maneuvers that increased venous engorgement of the head and neck, and a neck computed tomography (CT) scan showing tracheal compression by an undiagnosed goiter. We attempted to contact the patient to

obtain her informed consent to publish this case report; however, we were unable to reach her.

CASE DESCRIPTION

A 25-yr-old parturient, G₂ P₀₀₁₀ at 38 5/7 wk gestation presented for primary cesarean delivery secondary to uterine fibroids. She had no drug allergies or prior surgery, but her medical history was notable for obesity (weight 98.2 kg, body mass index (BMI) 36.1 kg/m²) and mild asthma (last albuterol inhaler use more than 9 mo previously). Her lung fields were clear to auscultation bilaterally and airway examination showed an inter-incisor gap >4 cm, neck flexion/extension >90 degrees, thyromental distance >6 cm and a Mallampati airway classification of 3. Routine premedications (metoclopramide 10 mg, famotidine 20 mg and cefazolin 2g IV piggy-back and sodium citrate 30 mL orally) were given with an IV fluid bolus of 500 mL lactated Ringer's solution. Spinal anesthesia with a T₄ sensory level was achieved using hyperbaric bupivacaine 11.25 mg with morphine 200 µg. Phenylephrine and ephedrine were administered to maintain her systolic blood pressure above 100 mm Hg. The cesarean delivery of a healthy male neonate was uncomplicated. Oxytocin (20 U per liter) was administered continuously IV in the operating room and in the postanesthesia care unit (PACU). Her intraoperative total IV fluids were 3000 mL, estimated blood loss was 500 mL, and urine output was 200 mL. Upon arrival to the PACU, the patient was stable (arterial blood pressure [BP] 93/37 mm Hg, heart rate [HR] 100 bpm, respiratory rate [RR] 16/min, SpO₂ 100% on room air, head elevated 30 degrees). Approximately 30 min after arrival in the PACU, ketorolac 30 mg and morphine 2 mg IV were given for pain control. In the PACU, her total IV fluids were 300 mL, urine output was 900 mL and discharge vital signs were normal (BP 100/56 mm Hg, HR 70 bpm, RR 22/min, SpO₂ 97% on room air).

The patient was transferred to the postpartum floor approximately 2.5 h after entering the PACU. Upon moving from the transport cart to her bed, the patient felt that her throat was closing; her SpO₂ was 60%-70%, BP 140/80 mm Hg, HR 130-160 bpm, and audible sounds of inspiratory obstruction were heard. Her trachea was rapidly intubated with a 7.0 ID endotracheal tube (ETT). The vocal cords showed normal movement, size, tone and color, but there was increased resistance inserting the ETT below the glottis.

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Her oxygen saturation improved, and tracheal suctioning revealed pink, frothy secretions. The initial arterial blood gas (ABG) obtained within 5 min after intubation was pH 7.17/ Pco_2 53/ Po_2 222/ HCO_3 19/ Spo_2 99.2%/fraction of inspired oxygen (Fio_2) 100%. A second ABG obtained 78 min later was pH 7.34/ Pco_2 36/ Po_2 73/ HCO_3 19/ Spo_2 94%/ Fio_2 40%. Notably, neither rales nor wheezing were evident on pulmonary auscultation. She was transferred to the medical intensive care unit (ICU), where a chest radiograph (CXR) showed only mild interstitial edema. The critical care team extubated her trachea 2 h later, noting that she was awake, alert, with a good air leak around the ETT after cuff deflation. In order to assess intravascular volume status, the ICU team elected to monitor her CVP. When placed in Trendelenberg position, the patient again developed acute shortness of breath, hypoxemia, and inspiratory obstruction. During this second hypoxic episode, she received dexamethasone 10 mg IV, diphenhydramine 50 mg IV, famotidine 20 mg IV and racemic epinephrine nebulizer treatment and eventually required reintubation with another 7.0 ID ETT. Upon laryngoscopy, the vocal cords again appeared normal; repeat ABG was pH 7.4/ Pco_2 28/ Po_2 79/ HCO_3 17/ Spo_2 95.7%/ Fio_2 60%; and repeat CXR showed worsening interstitial edema.

Because the initial CVP was increased (11–13 mm Hg) and D-dimer was high (4.38 $\mu\text{g/mL}$), a chest and neck CT scan and echocardiogram were ordered to assess for pulmonary embolus and other possible causes for acute respiratory distress. Testing for evidence of an allergic reaction (latex, penicillin, cefazolin, tryptase, histamine, and others) was eventually negative. The CT scan excluded pulmonary embolus, but did note an enlarged 7.8 $\times$ 5.6 cm left thyroid lobe (Fig. 1). Thyroid function tests on the first postpartum day indicated apparent euthyroidism with a normal thyroid-stimulating hormone (TSH), upper normal total T_3 , and slightly below normal free T_4 (Table 1). The echocardiogram was normal, with estimated pulmonary systolic pressure 25–30 mm Hg. In the ICU, ETT cuff deflation did not reveal a leak. Diuresis of 2 L occurred over the next 2 days in association with a decreasing CVP. On the fourth postoperative day, a persistent air leak around a deflated ETT cuff allowed for successful tracheal extubation and she was discharged on the sixth postoperative day.



Figure 1. Coronal computed tomography image of the cervical region near C_6 demonstrating an enlarged left thyroid lobe (dashed line) that compresses the trachea and endotracheal tube cuff on the patient's left side. For comparison, note the walls of the endotracheal tube (arrows) and the smooth curvature of the high volume endotracheal tube cuff (dotted line) on the patient's right side.

Table 1. Thyroid Function Tests

	TSH (0.35–4.94 $\mu\text{IU/mL}$)	Total T_3 (0.8–1.7 ng/dL)	Free T_4 (0.7–1.5 ng/dL)
Postpartum day 1	1.85	1.7	0.6
Postpartum day 23	0.206		1.0
Postpartum day 24	0.099	1.3	
Postpartum day 34	0.195	1.3	1.1
Post thyroidectomy	2.649		0.9

The normal range for the test is indicated at the top of the column.

TSH = thyroid stimulating hormone.

Three weeks postpartum, our patient presented to the emergency room again noting the sudden feeling that her throat was closing. Her vital signs were hyperdynamic (BP 160/100, HR 96, RR 20, Spo_2 100% on room air), her goiter was tender to palpation, repeat thyroid function tests were consistent with hyperthyroidism (Table 1), CXR showed slight tracheal deviation to the right, and a neck radiogram showed a soft tissue density anterior to the subglottic trachea. Her dyspnea resolved spontaneously. Two months postpartum, she underwent a subtotal thyroidectomy under general anesthesia with no complications. Pathology showed a 166 g, 12.0 $\times$ 7.5 $\times$ 4.0 cm multinodular goiter with cystic, hemorrhagic, degenerative changes. Three months postpartum, her TSH returned to normal (Table 1).

DISCUSSION

Among the physiologic changes associated with term pregnancy is an increase in total blood volume of 50% that is due primarily to a progesterone-mediated increase in maternal venous capacitance.¹¹ The thyroid gland also enlarges up to 30%, with both glandular hyperplasia and vascular proliferation,¹² but goiter may not be readily appreciated on physical examination, especially in obese parturients. Spinal anesthesia-induced hypotension usually prompts administration of several liters of IV fluid and use of vasopressors to maintain desired BPs intraoperatively. In the PACU, both the progesterone and the spinal anesthetic effects on venous capacitance dissipate. The contracted uterus also contributes to autotransfusion, increasing circulating blood volume, on average, an estimated 500 mL during the first 12 to 48 h postpartum.

With the return toward normal vascular sympathetic tone and rebound reduction of maternal venous capacitance, venous return to the heart, and CVP are maximal in the first 6–12 h postpartum. The Valsalva maneuver and head-down position may contribute to an acute increase in venous congestion of the head and neck. Maximally elevated venous pressures transmitted passively to the venules of a goitrous thyroid gland could expand the glandular capsule and contribute to compression of surrounding structures, especially the trachea. Deep inspiration through a trachea with a subglottic obstruction produces postobstructive, negative-pressure pulmonary edema, consistent with the frothy sputum, and CXR findings. In contrast, laryngospasm was not evident as a source of airway obstruction during either laryngoscopy, and pulmonary edema from preeclampsia or cardiogenic causes would have been less rapid in onset and more severe (without interval spontaneous recovery).

The coronal CT image of the thyroid gland in Figure 1 showed the asymmetric enlargement of the left thyroid lobe at the level of the C₆ vertebra. The inflated ETT distal cuff also appeared asymmetrical, with a normal arched curvature to the right, but a compressed linear shadow on the left. This image indicates that the enlarged left thyroid lobe was compressing the trachea to the extent that there was little or no air cushion between the tracheal mucosa and the left side of the ETT. Tracheal deviation to the right was demonstrated on CXR 3 wk postpartum. Altogether, the thyroid gland gross pathology, low serum TSH, anterior subglottic tenderness, and clinical hyperthyroidism are consistent with postpartum thyroiditis.¹³

Perhaps the most important maternal physiologic change after delivery is the dramatic reduction in venous capacitance that increases CVP and cardiac output. Our case suggests that parturients with goiter are not only at risk for thyroid gland enlargement during pregnancy, but may also be at risk in the first 12 h postpartum, possibly due to glandular expansion due to venous engorgement. Although others have reported airway obstruction due to an expanding thyroid gland during pregnancy,^{4–7,9} to our knowledge, this is the first reported case of severe, new-onset respiratory distress due to undiagnosed goiter in the first 12 h postpartum. This case should alert anesthesiologists to the potential of maternal goiter-related tracheal compression in the immediate postpartum period.

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