Acute respiratory failure due to thyroid storm developing immediately after delivery

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Introduction
In pregnant women, acute respiratory failure is rare, with a rate of only 0.1%. The reported causes of this condition include pulmonary edema, community-acquired pneumonia, aspiration, pulmonary embolism, asthma exacerbation, amniotic fluid embolism, and venous air embolism [1]. However, our literature search yielded very few reports of thyroid storm causing respiratory failure immediately after delivery [2]. We herein describe our experience with a case in which thyroid storm caused acute pulmonary edema which, in turn, led to respiratory failure.

Case Presentation
The patient was a 41-year-old woman, gravida 1, para 1. She had no history of hyperthyroidism. Her pre-pregnancy height and weight were 159 cm and 69 kg, respectively. She had received prenatal checkups at another hospital since the early stage of pregnancy. Her blood pressure was 142/80 mmHg at 12 weeks of gestation and 158/90 mmHg at 19 weeks. She had experienced palpitations and fatigue but had not sought treatment for these symptoms. Subsequently, her blood pressure was 140–150/80–90 mmHg, and a qualitative urine protein test was negative. Moreover, her weight decreased by 8 kg through the 23rd gestational week and then increased by 7 kg between 23 and 36 weeks.

Meanwhile, although ultrasonography revealed mild fetal growth restriction, there were no other fetal abnormalities.

At 36 weeks and 5 days of gestation, she was admitted due to premature rupture of membranes. Labor started spontaneously and a male infant weighing 1994 g was delivered transvaginally with Apgar scores of 8 and 9 at 1 and 5 min, respectively, at 36 weeks and 6 days of gestation. The delivery lasted for 3 h 14 min, and the volume of blood loss was 150 g. During the period from the start of labor pain until delivery, her blood pressure was 140/70–90 mmHg and pulse was 90–110/min, with no signs of dyspnea. Immediately after delivery, acute dyspnea occurred, prompting the initiation of oxygen administration. While her systolic blood pressure had decreased to 80 mmHg at 9 min after delivery, oxygen saturation (SpO₂) dropped to 50%. In addition, disturbance of consciousness manifested. Thus, pulmonary embolism was suspected, and she was emergently transferred to our hospital.

Key Clinical Message
Acute respiratory failure occurs in less than 0.1% of pregnancies. Thyroid storm should be included in the differential diagnosis of possible causes of acute respiratory failure occurring immediately after delivery, and delivery is a high risk factor for thyroid storm in pregnant women with thyrotoxicosis.

Keywords
Acute respiratory failure, lung edema, postpartum, thyroid storm.
On arrival, even after administration of 10 L of oxygen, she still showed disturbance of consciousness, with blood pressure 129/90 mmHg, pulse 170/min, and SpO₂ 93%. Because pulmonary embolism was suspected, a CT (computed tomography) scan was performed, demonstrating infiltrative shadows in both lung fields. Pulmonary edema was thereby diagnosed. There were no abnormalities on a CT scan of the head. Based on the diagnosis of respiratory failure due to acute pulmonary edema, she was intratracheally intubated and managed in the ICU (intensive care unit).

After admission to the ICU, her temperature was 38.1°C, pulse 120–140/min, and BNP (brain natriuretic protein) 128.7 pg/mL. Echocardiography showed a reduction in cardiac contraction, although the image was not clear due to tachycardia. Treatment with dobutamine at a dose of 3 mg/kg/min was started. Echocardiography performed on the third postpartum day showed a mild reduction in contractile function of the entire wall with an ejection fraction of 48%.

Moreover, she presented with left exophthalmos. Although no thyroid enlargement was detected by palpation, cervical echography revealed swelling of both lobes of the thyroid gland.

The blood tests performed on admission to our hospital showed a TRAb (thyroid-stimulating hormone receptor antibody) level of 7.3 IU/L (reference value, 0–1.9 IU/L), a TSH (thyroid-stimulating hormone) level of <0.005 μIU/mL (reference value, 0.5–5.0 μIU/mL), a FT (free thyroxine) 3 level of 5.57 pg/mL (reference value, 2.3–4.3 pg/mL), and an FT4 level of 2.59 ng/dL (reference value, 0.9–1.7 ng/dL), indicating hyperthyroidism. Respiratory failure due to thyroid storm was thereby diagnosed.

The patient was diagnosed with Graves’ disease and started on treatment with iodide (50 mg/day), hydrocortisone (500 mg/day), carperitide (0.01 µg/kg/min), and propranolol (30 mg/day) on postpartum day 3, and PTU (propylthiouracil, 300 mg/day) on postpartum day 4. Carperitide was discontinued, on postpartum day 10, because the respiratory discomfort symptoms had disappeared and there were no signs of pulmonary edema on a chest X-ray. As her general condition steadily improved and thyroid function essentially normalized, the patient was discharged on postpartum day 18.

**Discussion**

This case highlights the following two important clinical issues: thyroid storm should be included in the differential diagnosis of possible causes of acute respiratory failure occurring immediately after delivery, and delivery is a high risk factor for thyroid storm in pregnant women with thyrotoxicosis.

Above all, thyroid storm should be included as one of the possible causative factors when making a differential diagnosis of acute respiratory failure occurring immediately after delivery. Thyroid storm is a rare, life-threatening condition characterized by severe clinical manifestations of thyrotoxicosis [3].

There are no universally accepted criteria or validated clinical tools for diagnosing thyroid storm. Our patient presented with tachycardia, heart failure, fever, and neurological symptoms in addition to hyperthyroidism. Using the scoring system of Burch and Wartofsky [4] (clinical criteria for the identification of thyroid storm), the score was 90, leading to a diagnosis of thyroid storm.

Although the most commonly recognized causes of respiratory failure occurring immediately after delivery include pulmonary embolism and amniotic fluid embolism, thyroid storm, which can cause acute respiratory failure as was seen in our present case, should be considered among the differential diagnoses.

Second, delivery is a high risk factor for thyroid storm in pregnant women with thyrotoxicosis. Thyroid storm is often precipitated by surgery, trauma, delivery, infection, etc., in patients with untreated or poorly controlled hyperthyroidism [5]. Hyperthyroidism occurs in 0.1–0.4% of pregnant women [6]. Although the incidence of thyroid storm is low in those with thyrotoxicosis, mortality from thyroid storm in affected patients is high at 20–30% [5, 7]. Because of diminished peripheral vascular resistance and increased cardiac output during pregnancy as compared to nonpregnant periods, heart failure is more likely to occur during pregnancy. Heart failure reportedly occurs in 10% of pregnant women with thyrotoxicosis [8]. After delivery, compression of the vena cava by the uterus resolves, and venous return thus increases [9]. In our case, it was assumed that increased venous return immediately after delivery led to an increase in cardiac load which rapidly exacerbated heart failure caused by thyrotoxicosis before delivery, resulting in the development of pulmonary edema and, consequently, acute respiratory failure.

The patient had symptoms suggestive of hyperthyroidism, such as weight loss, hypertension, and proptosis, during pregnancy. Had she been properly treated for hyperthyroidism during pregnancy, the life-threatening respiratory failure described herein would likely not have developed. This patient’s course highlights the importance of careful history taking and monitoring the general conditions of pregnant women.

Thus, in pregnant women with thyrotoxicosis, thyroid storm can be precipitated by delivery, leading to acute respiratory failure. Because these patients are at high risk for such an outcome, it is important not to miss any of the symptoms suggestive of hyperthyroidism during pregnancy.
Conflict of Interest

The authors have no conflicts of interest to declare.

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