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CLINICAL REPORT

Development of takotsubo cardiomyopathy with severe pulmonary edema before a cesarean section

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Abstract Takotsubo cardiomyopathy is an acute syndrome involving apical ballooning and consequent dysfunction of the left ventricle. Most cases of left ventricular dysfunction resolve within 1 month. We present the case of a 40-year-old woman who developed severe heart failure caused by takotsubo cardiomyopathy with severe left ventricular dysfunction during the perinatal period. Because of the presence of multiple myomas, she was scheduled to undergo a cesarean section under general anesthesia. However, after induction of general anesthesia, she had to be awakened because of the presence of a difficult airway. Because she exhibited insufficient oxygenation, she was transferred to the emergency center. Upon hospital admission, she expectorated large amounts of pink sputum, indicating severe pulmonary edema. Cesarean section was performed immediately. Echocardiography revealed severe left ventricular dysfunction. Full recovery of cardiac function required almost 1 month, after which she was discharged from the hospital without further complications. This is the first reported case of takotsubo cardiomyopathy induced by a failed intubation during a scheduled cesarean section. Takotsubo cardiomyopathy usually shows a good prognosis, but if this myopathy develops during the perinatal period, it can worsen because of excessive preload following the termination of feto-placental circulation.

Keywords Takotsubo cardiomyopathy · Peripartum cardiomyopathy · Cesarean section · Anesthesia

Introduction

Takotsubo cardiomyopathy is an acute stress-induced cardiomyopathy presenting as left ventricular apical ballooning. Postmenopausal women are particularly susceptible to this myopathy, which is generally associated with a good prognosis and rapid convalescence [1]. Cases in which takotsubo cardiomyopathy develops during a cesarean section are very rare [2, 3]. In the present article, we describe the case of a patient with takotsubo cardiomyopathy that was probably induced by failed intubation at the time of cesarean section. Although the condition was initially suspected to be peripartum cardiomyopathy caused by severe left ventricular dysfunction, echocardiography and the therapeutic course subsequently indicated the presence of takotsubo cardiomyopathy.

Case

A 40-year-old woman (weight 69.3 kg, height 160 cm) was scheduled to undergo an elective cesarean section because of multiple myomas at 37 weeks and 3 days of gestation. Her family and clinical histories were unremarkable.

General anesthesia was performed by an obstetrician. After administering thiopental (300 mg), suxamethonium chloride (40 mg), and diazepam (4 mg), the obstetrician tried to intubate the patient with a Macintosh laryngoscope.

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Because of a difficult airway, he did not succeed, and the patient had to be awakened from anesthesia and transferred to our emergency center. On admission to the hospital, she expectorated large amounts of pink sputum. When she came to our hospital, her level of consciousness was almost clear (E4V4M5); blood pressure 110/70 mmHg; heart rate 140 beats per minute (bpm); and percutaneous oxygen saturation (SpO_2), 90 % under 10 l/min oxygen administered via a recovery mask. The fetal heart rate was more than 100 bpm, and no bradycardia was observed. Blood gas analysis indicated mild hypoxia, as well as the following conditions: pH, 7.320; pCO_2, 36.4 mmHg; pO_2, 65.5 mmHg; HCO_3, 18.2 mmol/l; tCO_2(P)c, 19.3 mmol/l; and ABEc, −6.7 mmol/l. Chest radiography indicated pulmonary congestion and a high cardiothoracic ratio of 58 %, and chest computed tomography indicated severe lung edema with pulmonary congestion. The possibility of pulmonary thromboembolism and amniotic embolism was excluded, and no ST segment elevation was observed on an electrocardiogram.

An emergency cesarean section was performed under general anesthesia, which was induced by propofol (200 mg) and rocuronium (100 mg). Although slight laryngeal edema was noted, intubation could be performed easily. General anesthesia was maintained through administration of 1.5–0.8 % sevoflurane and remifentanil (0.15–0.3 μg kg^{-1} min^{-1}). SpO_2 was maintained at ∼94–96 % under the following settings: FIO_2, 0.8; positive end-expiratory pressure (PEEP), 10 cmH_2O; and pressure-support (PS), 15 cmH_2O. Intermittent administration of the hypertensive drugs ephedrine hydrochloride (total amount, 24 mg) and phenylephrine hydrochloride (total amount, 0.5 mg) was needed. In addition, continuous administration of dopamine hydrochloride at 2–8 μg kg^{-1} min^{-1} was required. The patient's blood pressure was maintained at ∼100/50 mmHg. In total, 550 ml crystalloid was infused intraoperatively, 100 ml urine was produced, and 825 ml fluid was lost, including blood and amniotic fluid. The patient was transferred to the intensive care unit immediately after the cesarean section. The infant's 1- and 5-min Apgar scores were 1 and 5, respectively. The baby was mechanically ventilated for 1 day. He was discharged from our hospital with his mother without complications.

When the patient was admitted to the intensive care unit, her blood pressure was 120/80 mmHg and her heart rate was 100–110 bpm. On echocardiography performed after the cesarean section, left ventricular wall motion indicated diffuse hypokinesis, and the left ventricle was dilated. The ejection fraction, as measured using a modified Simpson's method, was 24 % (Fig. 1). We first diagnosed the patient with peripartum cardiomyopathy. Continuous administration of carperitide (0.1 μg kg^{-1} min^{-1}), isosorbide dinitrate (2 mg/h), dobutamine hydrochloride (8 μg kg^{-1} min^{-1}), and noradrenalin (0.02 μg kg^{-1} min^{-1}) was initiated for the treatment of cardiac failure. Heparin sodium (1,000 IU/day) was administered 5 days after the cesarean section to prevent thrombus formation resulting from low cardiac function. After diuresis, oxygenation was improved, and the patient could be extubated 2 days after the cesarean section.

One week after the cesarean section, cardiac function had not improved, and echocardiographic examination indicated that ejection fractions were only ∼30–40 %. Echocardiographic findings also revealed apical ballooning, which is indicative of takotsubo cardiomyopathy. Because the patient still had severe heart failure and because takotsubo cardiomyopathy was suspected, she was orally administered torasemide (4 mg/day), spironolactone (25 mg/day), imidapril

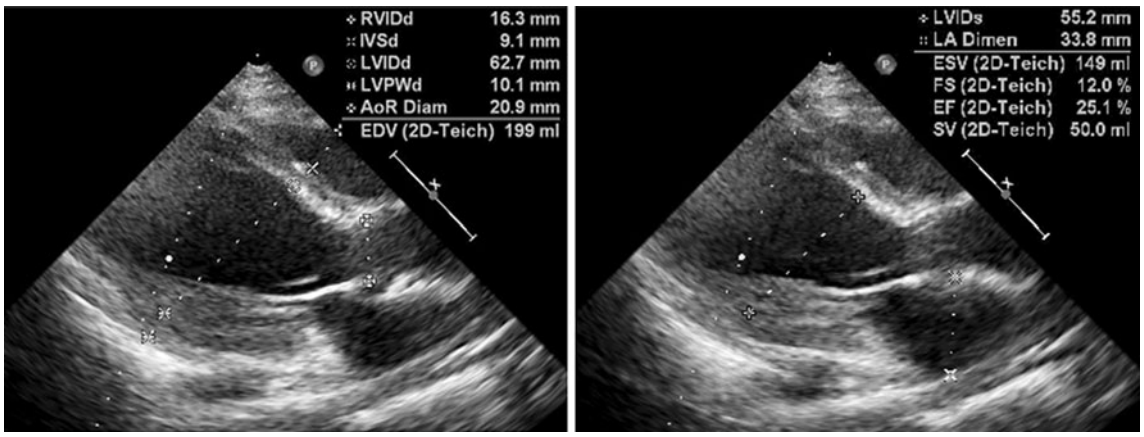

Fig. 1 Echocardiographic findings after cesarean section. The left ventricular end-diastolic dimension (*LVDd*) was 62.7 mm. Left ventricular dilatation was observed, and left ventricular wall motion showed severe hypokinesis. Left arterial dimension was 33.8 mm; it was not dilated. The posterior left ventricular wall thickness (*PWT*) was 10.1 mm. The percent fractional shortening (*% FS*) was 12 %. The ejection fraction was 25.1 %. As measured by a modified Simpson's method, the ejection fraction was 24 %, which indicated dilated cardiomyopathy-like movement

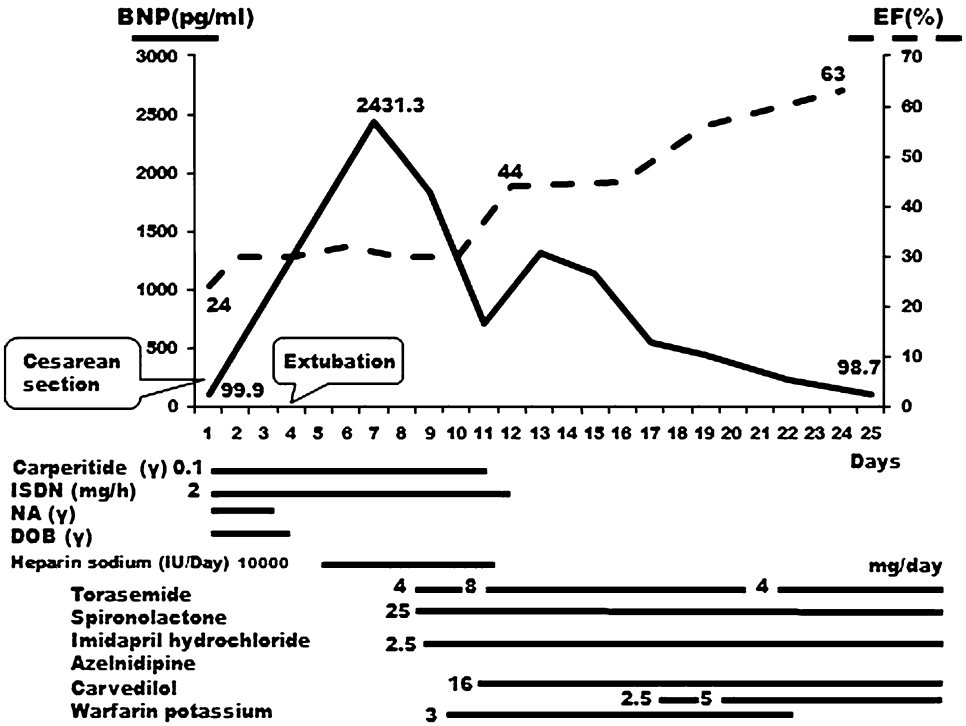

Fig. 2 The maximum level of brain natriuretic peptide (*BNP*) was 2,431.3 pg/ml immediately after admission, and gradually decreased to 98.7 pg/ml before the day of discharge. The ejection fraction was not significantly improved after the first 2 weeks; however, after 2 weeks, the ejection fraction gradually improved, and it returned to normal after 1 month

hydrochloride (2.5 mg/day), azelnidipine (16 mg/day), carvedilol (16 mg/day), and warfarin potassium (3 mg/day). Three weeks after the cesarean section, her cardiac function was restored to an almost normal level, and the patient was discharged from our center 1 month after admission. The time course of the therapy is summarized in Fig. 2.

Discussion

Takotsubo cardiomyopathy was first described in 1990 by Sato in Japan. In 2003, it was further discussed by Desmet et al. [4], and it became widely known as stress-induced cardiomyopathy or transient left ventricular apical ballooning in Europe and the U.S. In the present case, heart failure occurred during the perinatal period, and echocardiographic findings indicated severe left ventricular dysfunction (ejection fraction, 24 %), with left ventricular wall motion similar to that noted in a previous case of dilated cardiomyopathy [5]. Therefore, it was initially very difficult to distinguish between peripartum cardiomyopathy and takotsubo cardiomyopathy.

The exact mechanism underlying takotsubo cardiomyopathy remains unclear, although it may include myocardial stunning caused by multi-branch coronary artery spasm, catecholamine-induced cardiomyopathy, and apical ballooning from obstruction of left ventricular outflow. Postmenopausal women are most susceptible to this condition, and it is rarely observed in young women, suggesting that a decrease in estrogen is involved in the development of takotsubo cardiomyopathy [6].

Standard heart failure therapy has been indicated for takotsubo cardiomyopathy, including administration of diuretics, angiotensin-converting enzyme inhibitors, and β-adrenergic blocking agents. In cases of severe cardiac failure, where the cardiac output remains insufficient despite administration of these drugs, intraaortic balloon pumping and percutaneous cardiopulmonary support are suggested treatment options [7]. On the day the patient was transferred to the intensive care unit, we considered these options for treating the severe left ventricular dysfunction. However, we also had to consider the problems associated with anticoagulant therapy when assisted circulation is introduced. After considering the risks and benefits of therapy, we did not perform invasive therapy immediately after the cesarean section. However, in the present case, cardiac function gradually recovered, and fortunately, assisted circulation was not required. Some studies have reported the use of assisted circulation before or after cesarean section [7]. Assisted circulation should be introduced only if catecholamine or noradrenalin administration is insufficient to achieve a satisfactory cardiac output.

In the present case, the patient was initially diagnosed with peripartum cardiomyopathy because of the presence of severe left ventricular dysfunction. However, after echocardiography was performed to evaluate the time course of cardiac function recovery, we suspected that the underlying condition was takotsubo cardiomyopathy.

Considering the clinical course of this patient, we believe that the takotsubo cardiomyopathy was induced by failed intubation and that heart failure with pulmonary edema was worsened by the hemodynamic changes after cesarean section. In addition to takotsubo cardiomyopathy, negative pressure resulting from the failed induction of anesthesia may be a cause of the pulmonary edema [8]. However, postoperative echocardiographic findings indicated severe cardiac failure, so this mechanism appears unlikely in the current case. Nevertheless, this physiological stress may be a trigger of takotsubo cardiomyopathy in other cases. At the previous hospital, the obstetrician experienced a difficult airway during intubation, but in our hospital, the anesthesiologist performed good airway management without any problems. The patient did not show any outward appearance associated with a difficult airway, such as a small chin, short neck, or upper lip protrusion. In general, however, compared to a nonpregnant woman, a pregnant woman has a difficult airway [9–11]. Therefore, in a cesarean section, general anesthesia should be performed by a well-trained anesthesiologist.

If takotsubo cardiomyopathy develops before cesarean section, the heart failure would be expected to worsen because of excessive preload arising from the termination of fetoplacental circulation [12]. Although a previous report has described a case of takotsubo cardiomyopathy occurring during the induction of general anesthesia [13], the present article is the first report of takotsubo cardiomyopathy that may have been induced by failed intubation at the time of anesthesia induction. Anesthetic management for takotsubo cardiomyopathy follows that for conventional heart failure therapy, but care should be taken in the use of catecholamines [14]. In general, patients with takotsubo cardiomyopathy show good and rapid convalescence, and most patients recover within 1 month [14]. However, a few studies have reported poor clinical courses of this cardiomyopathy, including cases of cardiac rupture during the acute phase or development of pneumonitis or sepsis during therapy [15]. Therefore, it is very important that this cardiomyopathy be treated appropriately and intensively during the acute phase.

In the present report, we describe the case of a patient with takotsubo cardiomyopathy induced by failed intubation before cesarean section. Although takotsubo cardiomyopathy generally has a good prognosis, intensive heart failure therapy is still required for successful treatment in certain cases.

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