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CASE REPORT

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Stress cardiomyopathy should be considered with unexpected hypotension in pregnant women

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Abstract

Stress-induced cardiomyopathy is expected that with the wide application of critical ultrasound, ICU doctors will find more cases, particularly among pregnant patients. Patients can actually survive the acute period of excessive stress, and their cardiac function can recover as long as effective treatment measures are taken.

KEYWORDS

bedside ultrasonography, hypotension, pregnant women, Takotsubo cardiomyopathy

1 | INTRODUCTION

China's universal two-child policy has prompted older Chinese women, who might face high-risk pregnancies, to have a second child. Treating pregnant women with severe obstetric diseases and rescuing those in critical conditions has become the top priority of perinatal medical doctors and the multidisciplinary team of specialists including intensivists, obstetricians, anesthesiologists, hematologists, and nephrologists. With only a few exceptions, most of these patients may face risks such as postpartum hemorrhage, hemorrhagic shock, heart failure, and pulmonary edema that needs a multidisciplinary treatment program. The outcome of the treatments reflects the quality of care a hospital is able to deliver.[1] As a center for critical maternal treatment in Beijing's Chaoyang district, the surgical intensive care unit (SICU) has successfully treated a number of patients with severe obstetric diseases. Among them, two have experienced a change in conditions, as presented below.

2 | MATERIAL AND METHODS

2.1 | Case 1

At 10:00 on 5 February 5 2017, a 38-year-old female complained of oliguria for 10 hours postdelivery and was transferred to the China-Japan Friendship Hospital (CJFH). The patient had been bleeding since a vaginal delivery at 16:45 on 4 February 4 2017. She had received poor conservative treatment where a hysterectomy was performed at 22:00, with cumulative bleeding of 2700 mL and a minimum hemoglobin of 49/L. Postsurgery, the patient was transferred to CJFH for anuria and hypoxemia. The results of her physical examination before her admission were as follows.

T 37.4 C, HR 82 beats/min, RR 20 times/min, BP 136/78 mm Hg, and SpO2 92% (mask oxygen inhalation 8 L/min). Without any significant wet rales, both lungs had low respiratory sounds. ECG showed sinus rhythm, V1-V6 lead T wave inversion. Myocardial enzymes were normal and troponin negative.

Because of her hypoxemia and persistent anuria, she received a bedside ultrasonography upon admission to the hospital. Echocardiography showed that the left ventricular apical wall motion was significantly reduced and spherical changed, and left ventricular systolic function was reduced, with an ejection fraction (EF) of about 40% (Video S1). The patient received symptomatic treatment with noninvasive positive pressure ventilation, blood pressure control, continuous renal replacement therapy (CRRT), and reduced fluid intake. Five days later, echocardiography showed that the wall motion disorder was significantly alleviated, EF up to 60%, T wave inversion of the ECG gradually improved, and urine volume increased to 800-1000 mL/d. We concluded that the case was stress-induced cardiomyopathy (Figure S1).

2.2 | Case 2

At 19:30 on 17 March 2017, a 32-year-old female with amniotic fluid embolism was transferred to the SICU of CJFH. The patient had complained of postpartum hemorrhage since the delivery of a baby girl at 10:30 on 17 March 2017. Accompanied by a decrease in blood pressure, she had experienced persistent vaginal bleeding, which amounted to 1200 mL/h postdelivery. Symptomatic treatment of antishock, blood transfusion, tracheal intubation, and mechanical ventilation was administered to the patient immediately. After uterine artery embolization at 15:00, her vaginal bleeding gradually stopped and her circulation stabilized. However, the patient's oxygenation persisted poorly, so she was transferred to CJFH where she had a physical examination before admission with the following results.

Sedative state, T 37.6 C, HR 93 beats/min, RR 15 times/min, BP 142/98 mm Hg, SpO_2 90%, and moist rales were heard in both lower lungs. Blood gas analysis: PH 7.228, $PaCO_2$ 26.6 mm Hg, PaO_2 58.9 mm Hg, HCO_3^- 20.2 mmol/L, BE −5.4 mmol/L, Lac 2.3 mmol/L (FiO_2 100%, PEEP 10 cm HO_2), TNI 2.733 ng/mL, and BNP 190.9 pg/mL.

In the ICU, she received a bedside ultrasonography, which aimed to identify the cause of hypoxia. Bedside echocardiography revealed, she had left ventricular segmental dyskinesia, manifested by marked reduction in basal wall motion, enhancement of apical motion, and the ejection fraction of 40% (Video S2). Both sides of her lungs were B-lines. Electrocardiogram did not find any obvious abnormalities. We considered the case to be a stress-induced cardiomyopathy (Figure S2), associated with pulmonary edema caused by cardiac insufficiency. The patient received continuous infusion of tolasemide and continuous mechanical ventilation therapy, in an effort to recover her cardiac function. After that, the levels of TNI and BNP gradually decreased, and the oxygenation gradually improved. Four days later, the endotracheal intubation was removed, and echocardiography showed that the left ventricular segmental dyskinesia improved significantly, with an EF of 50%. Seven days later, when an echocardiography of her left ventricular with an EF of 60% did not find obvious segmental dyskinesia, the patient was discharged from the hospital.

3 | DISCUSSION

Stress-induced cardiomyopathy, also known as takotsubo cardiomyopathy (TTC), apical globular syndrome, or sadness syndrome, was first described by Japanese scholar Dote in 1991.[2] According to its unique findings through echocardiography and left ventriculography with left ventricular end-systolic apical roundness and narrow basal part of the heart, the disease was named takotsubo cardiomyopathy. Takotsubo cardiomyopathy is usually regarded as a temporary myocardial depression induced by psychological or physical stress factors, accompanied by reversible left ventricular systolic dysfunction. The 2006 version of World Health Organization guidelines on cardiomyopathy classifies the disease as a special cardiomyopathy which could be detected and diagnosed by echocardiography.[3] The updated Mayo Clinic diagnostic criteria for TTC in 2008 has included the following symptoms: (a) reversible systolic dysfunction or abnormality of left ventricular apex and middle ventricular wall beyond the scope of single vessel supply with stress factors; (b) coronary angiography showing that the degree of coronary artery stenosis was <50%, or there was no evidence of acute plaque rupture; (c) abnormal electrocardiogram or slight elevation of myocardial enzymes. Pheochromocytoma and myocarditis were excluded from the criteria.[4] However, despite the fact that the disease is related to sympathetic nerve excitation, the exact pathophysiological mechanism is still unclear. Catecholamine is secreted in large quantities under stress; the highest density of these receptors is in the apex of the heart. This leads to severe exhaustion of the receptors, which results in the ballooning of the apex of the heart during systole, and the contraction is compensated in the basal part.[5] In addition to this typical symptom, there is another reactive stress-induced cardiomyopathy (ie, the manifestation is completely opposite to apical globular degeneration syndrome). Left ventricular basal systolic function is severely reduced with apical compensation. The clinical manifestations include chest pain, dyspnea, palpitation, syncope, nausea, vomiting, hypotension or shock, sudden ventricular fibrillation, and cardiac arrest. These symptoms are reversible; most of the left ventricular segmental systolic dysfunction recovers spontaneously within 7-10 days. Therefore, the treatment measures mainly include organ support and symptomatic treatments, using drug therapy or mechanical assistance to help patients through this period of excessive stress. The majority of this

syndrome is found in elderly women and is rarely reported in perinatal women.

Postpartum stress cardiomyopathy is rarely reported in the literature.[6] The two patients in the above-mentioned cases are perinatal women with severe postpartum hemorrhage. One had severe hypoxemia and the other suffered from anuria and hypoxemia. These symptoms persisted after hemorrhage was controlled and hemoglobin rose. The results of the echocardiographs in both patients had been recorded. In the first case, echocardiography showed that apical wall motion was decreased and spherical changed, left ventricular systolic function was decreased, and segmental motion disorder was obvious, but no changes in myocardial enzymes were found. In the second case, echocardiography showed that left ventricular basal wall motion was significantly decreased, apical motion was compensated, left ventricular systolic function was decreased, and myocardial enzymes were changed, but there was no typical dynamic change in the echocardiograph. The ventricular wall dyskinesia did not match the single coronary artery blood supply area, and the changes in myocardial enzymes were not consistent with the changes in the condition. The left ventricular function of both patients recovered rapidly, so they were diagnosed with stress-induced cardiomyopathy and reactive stress-induced cardiomyopathy. The mechanism of stress-induced cardiomyopathy in pregnant women is still unclear. The circulatory capacity of the patients increased abnormally and reached the peak state in the first 24 hours postdelivery. Nevertheless, due to the decrease in plasma colloid osmotic pressure, their slight change in hydrostatic pressure may have lead to pulmonary edema. Furthermore, the significantly increased glomerular filtration rate and effective renal plasma flow, together with hypercoagulability, enlarged their kidneys. Therefore, the patients were particularly vulnerable to acute kidney injuries induced by factors such as postpartum hemorrhage, HELLP syndrome, or acute heart failure.[7] This was recorded in case 1, with acute kidney injury and left ventricular systolic dysfunction caused by stress-induced cardiomyopathy. These pregnant women are prone to heart failure, pulmonary edema, acute kidney injury, and renal failure. The second patient developed pulmonary edema, which was related to left ventricular systolic dysfunction and increased pulmonary hydrostatic pressure caused by massive transfusion. Symptomatic support proved to be the main treatment for both patients. Although left ventricular systolic dysfunction remained, the condition was not serious enough to require mechanical assistance such as IABP, ECMO, and LVAD, as reported in previous literatures.[8] There is no definite conclusion as to whether cardiotonic drugs should be added to enhance left ventricular systole. However, cardiotonic drugs are contraindicated in patients with left ventricular outflow tract obstruction due to increased compensatory left ventricular basal contraction.[9] In these specific cases, the second patient was treated with diuretics only, and the first patient was treated with CRRT dehydration and blood pressure control, all of which were effective.

4 | CONCLUSION

Stress-induced cardiomyopathy is a very critical disease in the early stage. It may lead to hypotension, dyspnea, acute pulmonary edema, cardiogenic shock, and cardiac arrest. The disease has become commonly seen by intensivists in recent years.[10] It is expected that with the wide application of critical ultrasound, ICU doctors will find more cases, particularly among pregnant patients. Although the prognosis seems poor, once confirmed as stress-induced cardiomyopathy, patients can survive the acute period of excessive stress and their cardiac function can recover, as long as effective treatment measures are taken.

CONFLICT OF INTEREST

The authors report no conflicts of interest.

AUTHOR CONTRIBUTIONS

CL: involved in research concept and design. HW and YW: analyzed and interpreted the data. CL: wrote the article. JD: critically revised the article. CL: involved in final approval of the article.

ETHICAL APPROVAL

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

INFORMED CONSENT

Informed consent was obtained from all individual participants included in the study.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section.

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