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CASE REPORT

Myocardial Dysfunction and Stress-Induced Cardiomyopathy Induced by Septic Shock After Hip Arthroplasty

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SUMMARY

Background: **Stress-induced cardiomyopathy, also known as Takotsubo cardiomyopathy or apical ballooning syndrome, is a disease of acute reversible myocardial injury characterized by transient localized systolic left ventricular insufficiency.**
Methods: **Appropriate laboratory tests are carried out, Next Generation Sequencing (NGS), Echocardiography.**
Results: **Postoperative echocardiography showed myocardial dysplasia of the left ventricle and abnormal wall motion of the left ventricular phases. NGS: Burkholderia cepacia onionis, human herpesvirus type 6B, and human polyomavirus type 2. The patient's condition improved after aggressive anti-infective and symptomatic treatment.**
Conclusions: **Because of the difficulty in distinguishing acute coronary syndromes from stress-induced cardiomyopathy, we should be on the lookout for stress-induced cardiomyopathy in any patient who develops a severe physical or emotional reaction during the perioperative period, with electrocardiographic abnormalities and refractory hypotension. Prompt and effective treatment can reverse myocardial damage.**
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KEYWORDS

stress-induced cardiomyopathy, septic shock, perioperative period

CASE REPORT

Stress-induced cardiomyopathy, first proposed by Japanese scholars and characterized by a morphology similar to a pot used to catch octopus in Japan, is also known as Takotsubo syndrome, broken heart syndrome, and other names [1]. It is an acute reversible myocardial injury disease characterized by transient localized systolic left ventricular insufficiency. Clinical manifestations include acute chest pain, dyspnea, and electrocardiographic changes. Its clinical manifestations are difficult to distinguish from acute coronary syndromes. The diagnosis can be clarified by left ventriculography and coronary angiography.

The patient is an elderly woman with no underlying disease. On July 15, 2024, she was admitted to the Depart-

ment of Orthopaedics of our hospital with a 10-year history of pain in her right hip. The ECG (Figure 1) was suggestive of VI-V4 T-alterations and V4-V6 U waves, and the cardiac ultrasound was generally normal. On July 18, 2024 she underwent a right total hip replacement under lumbar general anesthesia. During the operation, 500 mL of blood was lost, 300 mL of plasma was transfused, 3 U of suspended red blood cells were transfused, 500 mL of autologous blood was transfused, the blood pressure was maintained at 130/90 mmHg, and the arterial partial pressure of oxygen was 99.6 %. The patient returned to the ward after the operation. At 18:12 the patient had a decrease in blood pressure and complained of dyspnea, and the blood pressure did not increase significantly after fluid rehydration. ECG (Figure 2): QRs: I + II + III < 1.5 mV; V5V6 < 1 mV; ST: II III aVF V1 - V6 downshift > 0.05 mV; T: II III aVF V1 - V6 negative and positive bidirectional. At 20:00 she was transferred to the ICU. On July 19, 2024, echocardiography suggested a diffuse reduction in the amplitude of the left ventricular wall motion movement, left ventricular ejection fraction of 30%, mild bicuspid, and tricuspid regurgitation, and reduced left heart function. Bedside chest X-ray suggested: exudative lesions in both lungs. Blood routine: RBC 3.97 x 10^{12}/L, WBC 21 x 10^{9}/L, PLT 180 x 10^{9}/L, LYM 1.35 x 10^{9}/L. The patient had an infection and was given tigecycline 50 mg 2/day for anti-infection. Coagulation series: D-dimer 6,137 ng/mL plasma prothrombinogen time 17.2 seconds, PT% 59 %, PT-INR 1.37, low molecular heparin calcium injection 1 mL 2/day subcutaneous anticoagulation was given. Blood biochemistry: albumin 34.5 g/L, aspartate transferase 64 U/L, creatine kinase 1,057 U/L, creatine kinase isoenzyme 61 U/L, direct bilirubin 8.4 μmol/L, indirect bilirubin 18.4 μmol/L, total bilirubin 26.8 μmol/L, and lactate dehydrogenase 356 U/L. Blood gas analysis: PH 7.34, PCO2 24.6 mmHg, PO2 67.8 mmHg, cLac 7.8 mmol/L, ABE-11 mmol/L P/F 183 mmHg, diagnosis of metabolic acidosis. Cardiac enzymes: creatine kinase 475 U/L, creatine kinase isoenzyme 26 U/L, high-sensitivity troponin 1,659.5 ng/L, diagnosed as cardiogenic shock, given levosimendan injection 12.5 mg intravenous pumping, isosorbide mononitrate 20 mg 1/day pumping to expand the tube. The patient's cardiac function was extremely poor and her condition was critical, and she was given aortic balloon counterpulsation (IABP) and tracheal intubation for adjunctive therapy. On July 20, 2024, the patient had an invasive blood pressure of 91/51 mmHg (norepinephrine 1.1 μg/kg/minute) under adjunctive therapy with persistent IABP, while the patient had intermittent hyperthermia, and anti-infective therapy was added with meropenem 1 g 3/day. BNP: 34,887.5 pg/mL and T-waves were seen on the EKG leading to the administration of clopidogrel 75 mg 1/day for antiplatelet aggregation. On July 23, 2024, we added aspirin 0.1 g 1/day antiplatelet drug therapy, metoprolol 12.5 mg 2/day nasally to control ventricular rate, spironolactone 20 mg 1/day nasally to prevent ventricular remodeling, furosemide 20 mg nasally 1/day for diuresis, and cyclophosphate adenosine glucosamine 90 mg 1/day IV to improve cardiac function. On July 24, 2024, Beijing experts were invited for consultation, and the patient was considered to have stress-induced cardiomyopathy, at the same time, ivabradine 2.5 mg 2/day nasal feeding was added to control the ventricular rate. On July 26, 2024, the patient's peripheral blood NGS results were returned: onion Burkholderia cepacia, human herpesvirus type 6B, and human polyomavirus type 2, and the diagnosis was: infectious shock. Discontinuation of ticlopidine. On July 30, 2024, blood routine: RBC 3.97 x 10^{12}/L, WBC 16 x 10^{9}/L, PLT 118 x 10^{9}/L, LYM 1.35 x 10^{9}/L, ceftazidime avibactam discontinued and replaced with cefoperazone sulbactam 3 g 2/day. On July 31, 2024, coronary CTA: Coronary atherosclerosis with a coronary artery score of 51.26 suggesting mild calcification, further confirming the diagnosis of stress-induced cardiomyopathy. Cardiac enzymes: creatine kinase 96 U/L, creatine kinase isoenzyme 14 U/L, high-sensitivity troponin I 29.7 ng/L. Blood gas analysis PH 7.446, PCO2 32.3 mmHg, PO2 115 mmHg, cLac 1.3 mmol/L, ABE -1.2 mmol/L, P/F 395 mmHg. On August 2, 2024, an echocardiogram: a slightly low amplitude of motion of the left ventricular anterior wall and apical portion of the heart, mild bicuspid and tricuspid regurgitation, and a normal low left ventricular ejection fraction; the patient's cardiac function improved after the above treatments, and then she was transferred to orthopedics for further treatment.

DISCUSSION

Takotsubo cardiomyopathy was first reported in Japan in the early 1990s [1] and named after its characteristic ventriculography features. This disease primarily affects postmenopausal women experiencing acute physical or emotional stress [2]. Based on the underlying etiology, stress-induced cardiomyopathies are usually categorized into two groups. The first category is associated with emotional distress, which leads to a surge of catecholamines, causing cardiac insufficiency. The second category is associated with physiopathologic distress secondary to a disease entity (including pneumonia, severe acute asthma, or anaphylaxis), where the catecholamine surge is a symptom secondary to the underlying disease process that has secondarily induced this unique cardiomyopathy. Both types of stress-induced cardiomyopathy are accompanied by the release of large amounts of epinephrine and norepinephrine, which are considered to be the main mediators of cardiomyopathy. Catecholamines temporarily destroy the cardiac microvasculature, leading to myocardial dysfunction [3]. The mechanism of sepsis-induced cardiomyopathy is different from the classical emotional or physiological stress-induced Takotsubo cardiomyopathy and is mainly related to systemic inflammatory response, microcirculatory disorders, and metabolic disturbances, rather than a sim-

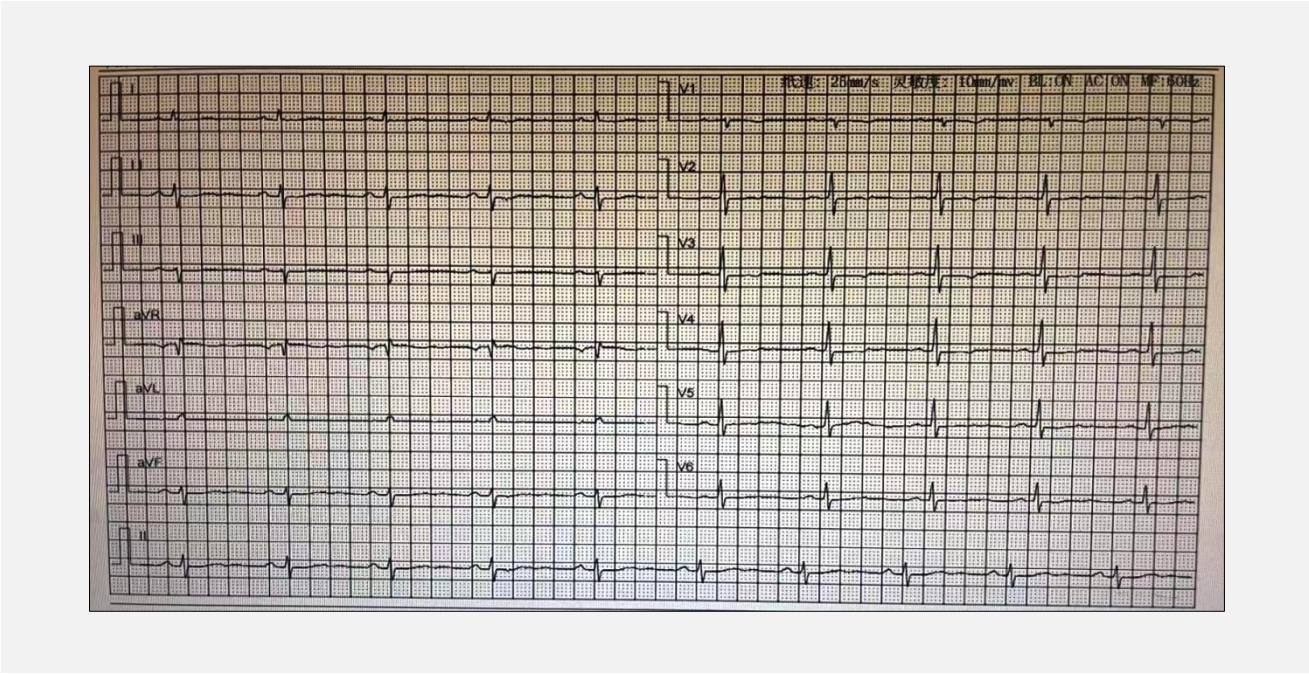

Figure 1. ECG suggests the presence of T-wave changes and visible U-waves.

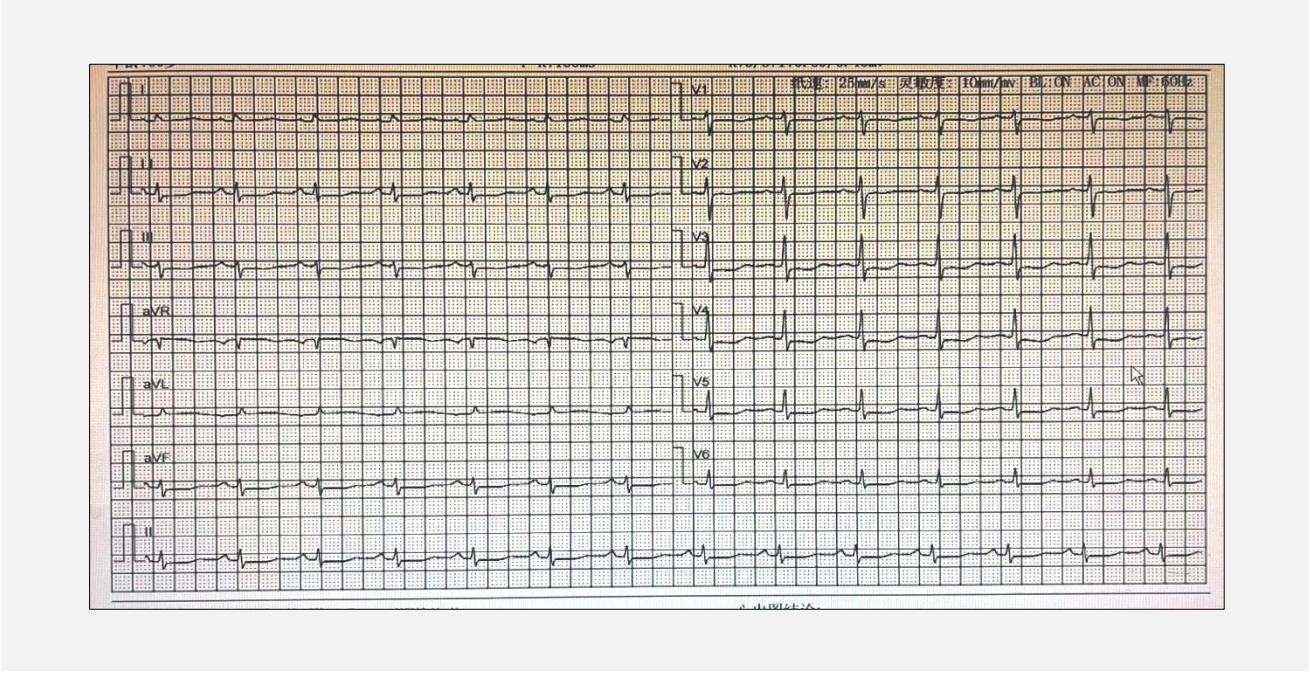

Figure 2. ECG suggests the presence of ST-T changes.

ple catecholamine surge. Infection-induced cytokine storm (e.g., TNF-α, IL-6) can directly inhibit myocardial contractility and lead to microvascular endothelial injury, thrombosis, and myocardial ischemia. Meanwhile, metabolic disorders of the body (e.g., lactic acid accumulation, and impaired energy metabolism) further impair cardiac function in sepsis [4]. Secondly, it has been shown that sepsis-induced myocardial depression

is associated with myocardial edema, which is not associated with catecholamine toxicity or tissue ischemia [5]. Stress-induced cardiomyopathy is usually manifested on the electrocardiogram, cardiac enzymes, and echocardiogram. The first symptoms and abnormalities on the ECG are indistinguishable from acute coronary syndrome (ACS) [6]. Therefore, Takotsubo cardiomyopathy is an important differential diagnosis of ACS, and when the diagnosis is suspected, coronary angiography or coronary CTA is required. The first symptom in most patients with stress-induced cardiomyopathy is chest pain and dyspnea, and the most common ECG abnormalities are ST-segment elevation and T-wave inversion. Echocardiography usually shows hypokinesia or inability to move the mid and apical segments of the left ventricle with preserved systolic function of the basal segments [7]. Coronary angiography usually shows normal coronary arteries or only mild luminal narrowing [8]. In stress-induced cardiomyopathy, the use of catecholamines such as norepinephrine require caution. In conjunction with our patient, who suffered from persistent hypotension, fever, and high inflammatory markers after hip arthroplasty, which can be due to infection leading to decreased vascular tone, norepinephrine was used to correct the intractable hypotension, and at the same time, after aggressive anti-infective and symptomatic treatment, the patient's cardiac function returned to normal.

CONCLUSION

Currently, the understanding of stress-induced cardiomyopathy is relatively new, and the challenge of accurately diagnosing and treating this disease in the perioperative period arises. In this case, we need to be alert to the possibility of stress-induced cardiomyopathy in any patient who develops severe physical or emotional reactions with ECG abnormalities and intractable hypotension in the perioperative period, and we need to observe the patient for signs of infection to avoid sepsis-associated stress-induced cardiomyopathy, and when the patient is unfortunate enough to have an infection, aggressive anti-infective therapy is key.

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Ethical Approval:
This study was approved by the ethics committee of North China University of Science and Technology Affiliated Hospital. All procedures performed in the studies were in accordance with the ethical standards. Informed consent was obtained.

Declaration of Interest:
No conflicts of interest.

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