An unusual reason for severe bradycardia leading to cardiac arrest during general anaesthesia: A case report

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Background. Takotsubo cardiomyopathy also known as transient ballooning syndrome is an increasingly reported phenomenon characterized by acute reversible apical or midventricular dysfunction. This stress-induced cardiomyopathy mimics myocardial infarction, but without significant coronary artery disease, and rarely presents in perioperative period.

Methods and Results. We report a case of postmenopausal woman scheduled to undergo elective cholecystectomy, with no history of coronary artery disease. She presented perioperatively with Takotsubo cardiomyopathy by unique manifestation- asystoly. This uncommon cause of cardiac arrest during anaesthesia was possibly induced by preoperative emotional stress. There was full recovery thanks to intensive management. In Takotsubo cardiomyopathy related cardiogenic shock we used the calcium sensitiser levosimendan successfully.

Conclusion. Takotsubo cardiomyopathy has an excellent long-term prognosis and nearly all patients have full recovery of left ventricular function. We emphasize the importance of heavy premedication by stress compromised patients and the need of sufficiently deep anaesthesia and analgesia during surgeries.

Key words: Takotsubo cardiomyopathy, cardiac arrest, levosimendan, general anaesthesia, severe bradycardia

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INTRODUCTION

The incidence of Takotsubo cardiomyopathy is estimated to be 1% to 2% of patients with acute myocardial infarction^{1,2}. Takotsubo cardiomyopathy (TTC), also called stress-induced cardiomyopathy or transient left ventricular ballooning syndrome, is a clinical entity first described in 1990 by Sato et al. in Japan. This syndrome is characterized by transient systolic dysfunction of apical³ and/or mid segments of left ventricle that mimics myocardial infarction (MI), but with absence of significant coronary artery disease. It could manifest as typical ischemic chest symptoms: elevated ST segment on electrocardiogram and elevated cardiac disease markers. TTC predominantly affects postmenopausal women exposed to emotional or physical stress. The treatment of Takotsubo cardiomyopathy is generally supportive and long-term prognosis is generally promising with normalization of wall motion abnormalities within weeks¹. Although infrequent, recurrence of the syndrome has been reported².

CASE REPORT

History

62-year-old woman with a history of rheumatoid arthritis treated with prednisone (2.5 mg per week) was scheduled for elective cholecystectomy due to cholelithiasis. The patient had no history of coronary disease. She suffered from anxiety concerning the surgery and working under great pressure in the preoperative period.

Anaesthetic Management

We administered midazolam 7.5 mg orally 12 h and 1 h before surgery. We induced general anaesthesia using sufentanil, propofol and cis-atracurium and performed orotracheal intubation.

We maintained anaesthesia with sevoflurane (0.8 MAC) and sufentanil. After the induction, patient's blood pressure was 110/70 with a heart rate of 80 bpm. Severe bradycardia and sudden cardiac arrest- asystoly occurred ten min after the beginning of the surgery. We begun chest compressions at a rate of 100 min⁻¹ immediately (ECR guidelines 2005) and administered atropine 3 mg, epinephrine 1 mg and amiodarone 300 mg total intravenously during 10 min lasting resuscitation. We induced mild therapeutic hypothermia (by administration of 4 °C

saline 2 L), regarding to ILCOR recommendations, and hemodynamic support by norepinephrine administration ($0.01\text{--}0.02\text{ mg}\cdot\text{kg}^{-1}\cdot\text{h}^{-1}$) after restoring a hemodynamically significant rhythm. The electrocardiogram shown a significant ST segment elevation in the lead II. We cancelled the elective surgery without cholecystectomy due to a suspected myocardial infarction.

Postanaesthetic Management

We admitted the critically ill patient to the intensive care unit due to the signs of cardiogenic shock. Immediate transthoracic echocardiography detected akinesia of the mid portion and normokinesia of the distal and basal portions of the left ventricle. The coronary angiography finding was without critical narrowing. The ventriculography showed severe systolic dysfunction of the mid segment of the left ventricle and the left ventricular ejection fraction was 26% (Fig. 1). The CT pulmonary angiogram and the 12-lead ECG were normal. The Troponin T was significantly elevated ($0.12\text{ }\mu\text{g}\cdot\text{L}^{-1}$ 1 h after the cardiac arrest, $0.97\text{ }\mu\text{g}\cdot\text{L}^{-1}$ 8 h after the cardiac arrest).

Because of patient's heart failure and hemodynamic instability, we used hemodynamic monitoring system (Vigileo, Edwards Lifesciences, USA) to coordinate the following hemodynamic support with norepinephrine and dobutamine administration. After 24-h mild therapeutic hypothermia ($33\text{--}34\text{ }^{\circ}\text{C}$), active rewarming was started. We used levosimendan the following day ($12.5\text{ mg}\cdot 24\text{ h}^{-1}$), which led to reduction in norepinephrine and dobutamine dose. The clinical condition of the patient improved over the next 5 days and we extubated her and weaned her for the vasopressors successfully. On the 6th day we transferred the patient to the coronary care unit for the following intensive and diagnostic care. Within 11 days the echocardiographic findings resolved and the patient was without any neurological deficit.

Cardiac magnetic resonance imaging wasn't performed because of the patient's claustrophobia.

We confirmed TTC based on the clinical and laboratory findings, typical echocardiography and no severe findings on coronary angiography.

DISCUSSION

There have been no reports of cardiac arrest due to TTC during anaesthesia so far. We have experienced asystoly as a unique manifestation of TTC in perioperative period. A diagnosis of Takotsubo syndrome was postulated based on the clinical and laboratory findings, typical echocardiography and no severe findings on coronary angiography.

Perioperative state is known to induce stress in patients. Triggering factors of TTC could be preoperative psychological stress, anxiety concerning surgery, induction of anaesthesia, laryngoscopy, hemodynamic instability, postoperative pain or sepsis. The goal of preoperative management of TTC is to reduce, or better to avoid emotional and physical stress. Establishing a good relationship

Fig. 1. A good contractile function of the apex and the base of the left ventricle and a severe midventricular dysfunction documented on the ventriculogram. Borderlines of the affected segments marked by the arrows.

with the patient and administering pre-operative anxiolytic drugs are two measures that could readily be taken. For induction of anaesthesia, the patient may benefit from a dose-dependent myocardial depression caused by inhalation of anaesthetic agents. Direct laryngoscopy should be brief to minimize activation of sympathetic nervous system, reintubation might cause a catecholamine surge with the consequent development of TTC. Sympathetic hyperactivity is common in the perioperative period, therefore sufficiently deep anaesthesia and analgesia during surgery may avoid surges in catecholamine levels in this potentially life-threatening but rapidly reversible syndrome^{4,5}.

We could also consider other diagnostic options leading to the similar clinical manifestation, such as prolonged vascular spasm or thrombotic occlusion of the coronary artery. However, we can support our assertion with a strong evidence. The patient expressed an extreme level of psychological stress before the surgical procedure - this is a high risk factor of the TTC development. We have performed the coronary angiography within 2 h after the cardiac arrest with a result of no alteration to the TIMI flow in all coronary vessels whatsoever. The result of the coronarography and the specific localization of the contractility dysfunction of the left ventricle, atypical for a local ischemia due to a vessel occlusion, exclude ischemic etiology with a very high probability. We found a medium-high elevation of the Troponin T cardiac marker, which is also typical for TTC. The typical symptom of the significant chest pain cannot be discussed, as we assume that the acute phase of TTC developed rapidly during the first minutes of the surgical procedure when the patient was already under a general anaesthesia. If the patient was conscious, she would probably report to a physician about

her incoming symptoms. Most of all, the diagnosis of TTC is strongly supported by the results and the course of multiple echocardiography examinations. In the beginning, that is within minutes and hours after CPR, we could see the severe alteration to the left ventricular systolic function with the very low ejection fraction - under 20% - and the mid-ventricular localization of the local hypokinesis. The following days, we observed the slow left ventricular function restitution. Finally, almost two months after the cardiac arrest, we found the completely normal function of the left ventricle, with the ejection fraction of 60%. We could only hesitate about the reasons of the prolonged restitution of the left ventricular systolic function - a little longer than in most referred cases of TTC. We can only speculate that this could have happened due to the extreme global ischemia of cardiomyocytes during the cardiopulmonary resuscitation.

There is no evidence of a positive role of levosimendan administration during TTC attack. In our case it led to the reduction of norepinephrine and dobutamine dose and stabilization of the patient's circulation. In spite of increasing number of severe cases of TTC with a successful use of this drug cited in recent publications⁶, we believe that the possibility of routine application of levosimendan in patients with TTC still needs further broad research.

Recurrence of this syndrome is low, with an incidence of <10% of patients^{1,2}. There is no evidence to support any specific management strategy or drug therapy, however, administering higher doses of anxiolytic⁶ drugs earlier before surgery might be beneficial. The optimal delay of the elective surgery after an occurrence of TTC is unclear. There is no evidence whether regional anaesthesia is better than general⁷. However, regional anaesthesia with sufficient sedation may represent a good option for patients with TTC undergoing surgical procedures.

CONCLUSION

The phenomenon of Takotsubo cardiomyopathy occurs more and more frequently. We believe that this new nosological entity will be soon more widely recognized.

Specific therapy of TTC has not yet been established, the treatment is generally supportive. For a patient with TTC complicated by cardiogenic shock, levosimendan, as a non-catecholamine inotropic substance, seems to be a good choice of treatment. Using exogenous catecholamines as inotropic support (e.g. norepinephrine, dobutamine, epinephrine) may be counter-productive, as they could maintain the pathogenetic mechanism of TTC.

Furthermore, this case emphasizes the importance of being aware of uncommon causes of cardiac arrest during anaesthesia.

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