Postoperative Takotsubo cardiomyopathy triggered by intraoperative fluid overload and acute hypertensive crisis

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Abstract

The Takotsubo cardiomyopathy is a rare haemodynamic dysfunction, only recently reported perioperatively. While the diagnostic criteria have been established and the outcome is known as favorable, the pathophysiological mechanisms are not entirely understood. Here we present the case of a patient scheduled for laparoscopic hysterectomy and adnexectomy, who early postoperatively developed a Takotsubo cardiomyopathy supposedly triggered by an acute hypertensive crisis due to intraoperative fluid overload.

Keywords: Takotsubo cardiomyopathy, surgery, fluid overload, hypertension

Introduction

Takotsubo cardiomyopathy (TC) or transient left ventricular apical ballooning is a relatively rare clinical entity, only recently reported in the anaesthetic literature. While Dote et al. [1] were first to describe this syndrome in Japanese patients, its perioperative occurrence was first reported in the Japanese anaesthesia literature by Mizutani et al. [2] and Takigawa et al. [3] and in the English anaesthesia literature by Gavish et al. [4] and Lentschener et al. [5] in 2006.

Since then, numerous case reports have been published, each focusing on the possible etiology of the perioperative TC.

We present a case of early postoperative TC, supposedly triggered by fluid overload and acute hypertensive crisis. To date, this has not been described as a possible etiology of TC.

Case report

The patient’s consent was obtained for the scientific use and publication of this case report.

A 54 year old patient, weighing 51 kg, with a BMI of 22.7 kg/m² was scheduled for an elective laparoscopic hysterectomy and adnexectomy for a fibromatous uterus. Her medical history revealed an acute thyroiditis many years before, treated with hormonal replacement that was stopped a few years later and allergy to penicillin and a non steroidal anti-inflammatory drug. She had no cardiac or other systemic disease and had good functional capacity. Her preoperative ECG was normal.

Standard anaesthesia monitoring was employed throughout surgery. Anaesthesia was induced with fentanyl, propofol and cis-atracurium and maintained with sevoflurane and aliquots of fentanyl when deemed necessary. A mixture of 40% oxygen in air was administered and volume controlled ventilation was tailored to maintain an EtCO₂ between 32-36 mmHg. The CO₂ pneumoperitoneum insufflation pressure was 14 mmHg.
The operation lasted 135 min and the first half of it was without incident. As during the first hour the patient was anuric and hypovolemia was presumed, several boluses of lactated Ringer’s solution were administered; up to a total volume of 2000 mL over two hours (the last volume of 1000 mL was administered over 45 minutes). Mild intraoperative tachycardia (heart rate up to 95 bpm) was interpreted as surgical pain and was treated with repeated boluses of fentanyl up to a total dose of 16 μg/kg. Following this, despite the large doses of fentanyl and despite increasing the concentration of sevoflurane, the patient’s blood pressure was 190/115 mmHg and remained high until the end of surgery.

Throughout the whole procedure, the patient’s oxyhemoglobin saturation (SpO₂) was 100% and the EtCO₂ was 30-31 mmHg. Surgical blood loss was minimal and total urinary output was 100 mL.

At the end of the procedure, with the effect of the muscle relaxants reversed, and the patient awake with adequate motor strength, her trachea was extubated. Approximately 30 minutes following extubation, in the recovery room, while breathing oxygen spontaneously through a face mask, the patient’s SpO₂ gradually dropped to 88% thus, high flow oxygen through a non rebreathing mask with reservoir was administered. The arterial blood gas analysis showed a PaO₂ of 54 mmHg, a PaCO₂ of 43 mmHg and a pH of 7.31. Sixty minutes after her arrival in the PACU, the patient’s blood pressure dropped from 155/84 to 100/68 and her heart rate increased to 117 bpm. With the suspicion of myocardial ischemia/infarction, a 12-lead ECG was performed which showed nonspecific T-wave changes in lead V2 and a prolonged QT segment (QTc 490 ms). Troponin I was 1.2 ng/mL (the normal value in our hospital is up to 0.05 ng/mL).

A chest X-ray revealed pulmonary congestion and a transthoracic echocardiography performed by a cardiologist revealed a normal right ventricle, left ventricular apical akinesia, left ventricular diastolic dysfunction and an ejection fraction of 35%.

After two hours in the PACU, given the patient’s worsening dyspnea and hypoxemia, her trachea was reintubated and synchronized intermittent mandatory ventilation with 5 cmH₂O PEEP was started.

Following this, cardiac catheterization was performed showing normal coronaries. Left ventriculography showed systolic apical ballooning with severe akynesia-dyskinesia of the apex and mild basal hypercontractility, suggestive for TC – Figures 1 and 2.

The patient was transferred to the ICU, where she was treated with furosemide and mechanical ventilation. Low dose norepinephrine was required for hemodynamic support. Twenty four hours later, her oxygenation improved, her trachea was extubated and treatment with ACE inhibitors was initiated. At this point, the echocardiography still showed left ventricular apical, mid-septal and anterior wall movement abnormalities. Left ventricular ejection fraction this time was 46%. Treatment with beta-blockers was started along with thromboprophylaxis. A repeated cardiac control three months after patient’s discharge from the hospital revealed a normal echocardiography with a left ventricular ejection fraction of 58%, thus, reinforcing the diagnosis of postoperative transient apical ballooning – Takotsubo cardiomyopathy.
Discussion

Our patient suffered from TC that was presumably triggered by fluid overload and an acute hypertensive crisis during surgery.

TC is a clinical entity characterized by transient left ventricular dysfunction, with apical akinesia/dyskinesis causing a peculiar echocardiographic and ventriculography picture, new electrocardiographic changes (T-wave as well as QT and PR interval changes) that can mimic acute myocardial infarction and a minimal increase in cardiac enzymes in the absence of coronary occlusion [6, 7].

For the diagnosis of TC, all four proposed Mayo criteria must be met including the three mentioned above, as well as the absence of concurrent pathology such as intracranial bleeding, pheochromocytoma and myocarditis or hypertrophic cardiomyopathy [7]. All these diagnostic criteria were fulfilled in our case.

TC is more frequently encountered in women and its prevalence remains uncertain. Psychological and physical stress, including surgery, are its known triggers. Hypoxemia, anxiety, pain and other stressful triggers have been implicated [4, 8-10].

Why, under the same circumstances, do some patients develop TC, while others do not, has not yet been determined. One theory [7, 11] claims that individual differences in regional sympathetic nerves distribution throughout the left ventricle may be responsible for the inter-patient variability. This may explain the dynamic mid-ventricular obstruction [12] with basal hypercontractility and apical ischemia as a response to a stressful condition causing catecholamine surge and “flooding” of the catecholamine receptors in the left ventricle.

The treatment of TC is mainly supportive, with diuretics, ACE inhibitors, beta blockers and if necessary mechanical ventilation and even intra-aortic balloon pump to sustain blood pressure and organ perfusion [13].

The clinical spectrum of TC still presents a myriad of problematic questions regarding its incidence and the understanding of its etiology and management, which to date remains unanswered. One such conflicting issue is why only certain patients develop TC, while others do not. Also controversial is the use of inotropic drugs which may be detrimental by worsening the dynamic obstruction of the left ventricle, a condition that responds well to treatment with beta blockers. However, clinical judgement may dictate the use of inotropic agents and avoiding beta blockers in situations where the left ventricular contraction is severely compromised and complicated with cardiogenic shock and frank pulmonary edema as was the case with our patient. Some authors believe that an intra-aortic balloon pump would be a safer choice under these circumstances [13].

Despite the often “cloudy” clinical picture, the outcome of TC in the majority of cases is favourable as was also the case with our patient.

Although stress seems to be the most frequent trigger for the development of TC, a stressful event is not always evident [3, 14] as happened in our case, where preoperative anxiety and intra and postoperative pain were not observed. Our patient received midazolam and was calm preoperatively. Furthermore, she received a quite large dose of fentanyl (16 μg/kg), thus, it seems unlikely that she suffered from severe pain during surgery.

We suggest that intraoperative fluid overload and the acute hypertensive crisis might have triggered the TC in our patient. Our patient received a volume of lactated Ringer’s solution of 20 mL/kg over a 40 minute period and a total of 40 mL/kg in two hours, while she had no apparent preoperative fluid deficit and the surgery was associated with minimal blood loss.

Fluid overload has been found to have detrimental cardiac effects in patients undergoing noncardiac surgeries [15]. Myocardial ischemia, cardiac arrhythmias and heart failure were more frequent in patients who received liberal fluid treatment perioperatively, as compared to those receiving more restrictive fluid regimes. This may be true even in patients without preoperative cardiac pathology since pneumoperitoneum by itself may significantly decrease cardiac output [16] and by this, may limit the ability of the heart to cope with volume overload. In the absence of painful stimuli, the acute intraoperative hypertensive crisis encountered in our patient towards the end of surgery, refractory to fentanyl treatment and deepening of anaesthesia with sevoflurane, might have heralded the presence of fluid overload.

In dogs, acute volume overload has produced an increase in heart rate, left ventricular peak systolic pressure and left ventricular end diastolic pressure [17].

We speculate that in our case, the fluid overload and the acute hypertensive crisis caused an increase in left ventricular end diastolic volume and pressure and decreased coronary perfusion pressure which together with an increased myocardial oxygen consumption, might have caused global or regional myocardial ischemia, left ventricular dysfunction and TC. We cannot explain why fluid overload and hypertension presumably triggered TC in our patient and did not in many other patients suffering from fluid overload. This question however remains critical and to date unanswered with all the other stressful triggers of TC as well.

Although we presume that fluid overload was the culprit, we cannot rule-out the possibility that carbon
dioxide peritoneal insufflation (carboperitoneum) during laparoscopy might have been the trigger of TC. Anderson et al. [18] found increased catecholamine blood levels, five minutes after peritoneal insufflation with CO₂. This scenario however seems to be less conceivable, considering that our patient’s EtCO₂ was low (30-31 mmHg) throughout the whole procedure. Furthermore, it would be expected to occur earlier, after the initial CO₂ insufflation. In this regard, the large dose of fentanyl given to our patient might have been beneficial since as previously demonstrated, a high dose of opiates (remifentanil) can depress the release of epinephrine (though not that of norepinephrine) during CO₂ induced pneumoperitoneum [19].

**Conclusion**

We presented a case of Takotsubo cardiomyopathy presumably triggered by intraoperative fluid overload and acute hypertensive crisis. Awareness to the possible perioperative occurrence of TC is essential since it can be misdiagnosed as an acute myocardial infarction.

**Conflict of interest**

Nothing to declare

**References**


Cardiomiopatie Takotsubo declanșată de supraîncărcare volemică și de criza acută hipertensivă

**Rezumat**

Cardiomiopatia Takotsubo este o disfuncție hemo-dinamică rareori întâlnită, de curând semnalată în perioada periopeatorie. Deși criteriile pentru diagnostic au fost stabilite iar evoluția este considerată ca fiind favorabilă, mecanismul fiziopatologic este încă insuficient cunoscut. Prezentăm cazul unei paciente operate laparoscopic pentru histerectomy și anexectomie, care a dezvoltat imediat postoperator o cardiomiopatie Takotsubo, declanșată cel mai probabil de o criză hipertensivă acută consecutivă unei supraîncărcări lichidiene intraoperațiorii.

**Cuvinte cheie:** cardiomiopatie Takotsubo, chirurgie, supraîncărcare volemică, hipertensiune