South Afr J Anaesth Analg. 2023;29(2):73-76
https://doi.org/10.36303/SAJAA.2954

South Afr J Anaesth Analg
ISSN 2220-1181 EISSN 2220-1173

CASE REPORT

Perioperative haemodynamic instability caused by Takotsubo cardiomyopathy

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Takotsubo cardiomyopathy is characterised by the acute onset of a distinctive pattern of left ventricular regional wall abnormalities. This acute cardiomyopathy is commonly triggered by emotional or physiological stressors, as may occur in the perioperative period. We present a case of perioperative Takotsubo cardiomyopathy complicated by cardiac arrhythmias and haemodynamic instability. We discuss the interplay of pre-existing patient risk factors and the physiological triggers that culminated in the development of Takotsubo cardiomyopathy in this patient. The importance of correct diagnosis and management is highlighted, with special reference to the value of point-of-care echocardiography.

Keywords: Takotsubo cardiomyopathy, perioperative, point-of-care echocardiography

Introduction

Takotsubo cardiomyopathy was first described in a Japanese case series.[1] The patients typically presented with chest pain and ECG patterns consistent with an acute coronary syndrome; yet no angiographic evidence of coronary artery occlusion was demonstrated. Contrast ventriculography revealed an unusual pattern of regional wall motion abnormalities, creating the appearance of a narrow left ventricular (LV) base combined with outward 'ballooning' of the apex during systole.[1] This appearance resembled the Japanese clay pot used to catch octopi called a 'Takotsubo'. Case reports classically identified a preceding emotional or physiological stressor. This led to the eponymous labels of "broken heart syndrome" and "stress cardiomyopathy".[2]

Takotsubo cardiomyopathy is currently a well-recognised acquired cardiomyopathy with variable features. Interestingly, the pathophysiology is not entirely understood.[2] The cardiomyopathy is usually transient, most patients recovering fully within days to weeks; however, complications occurring in the acute phase may be life-threatening.[3] In 2005, a case report describing the occurrence of Takotsubo in the perioperative period was published.[4]

Further perioperative cases have since been documented.[5] Anaesthetists need to be aware of this perioperative complication, as correct diagnosis and management are crucial for good outcomes.

Case report

A 69-year-old woman with breast carcinoma was scheduled for wide local excision. She had a background medical history of chronic hypertension, type II diabetes mellitus, and hypercholesterolaemia; managed on amlodipine, metformin and simvastatin respectively. Glucose control was sub-optimal, ranging between 7.6–15.8 mmol/L in the 72 hours preoperatively in the ward. Her oncological treatment included anastrozole (anti-oestrogen therapy) and anthracycline neoadjuvant chemotherapy. The anthracycline treatment had been completed six weeks prior to the surgery, with no residual side effects. The patient had no history of smoking, and had NYHA grade II function with no restriction of activities of daily living. Four months prior to this surgery, she had undergone uneventful breast biopsy under general anaesthetic. Preoperative urea, creatinine, electrolytes and full blood count were within normal limits. The preoperative chest radiograph was unremarkable. Her ECG revealed a sinus rhythm of 76 beats per minute (bpm), complete right bundle branch block and a QTc of 472 ms.

Pre-induction vitals included a blood pressure (BP) of 145/90 mmHg and heart rate of 60 bpm.

Induction of anaesthesia was accomplished with fentanyl 200 ug, propofol 60 mg and rocuronium 30 mg. Tracheal intubation was uneventful. Analgesia consisted of ketamine 15 mg, paracetamol 1 g and morphine 4 mg. The wide local excision proceeded without complication and was completed in 45 minutes. However, immediately after tracheal extubation, the patient developed laryngospasm with stridor and a decrease in oxygen saturation. This did not respond to FiO2 100% and positive pressure mask ventilation. Immediate tracheal reintubation was facilitated by etomidate 10 mg and suxamethonium 100 mg. Her oxygen saturation reached a nadir of 82% during this event.

Three minutes after re-intubation, the patient developed a monomorphic ventricular tachycardia (VT). During the VT, systolic BP was 190 mmHg. Amiodarone (300 mg over 5 minutes) restored sinus rhythm (90 bpm) and her BP decreased to 118/70 mmHg.

The patient was again extubated. She was awake, responsive and pain-free. However, upon transfer to her ward bed, asystole occurred. After two minutes of CPR, return of spontaneous circulation occurred. ECG now revealed complete heart block, with a ventricular escape rhythm of 30 bpm. The patient was persistently hypotensive and showed little clinical response to atropine, adrenaline (50 ug boluses and infusion

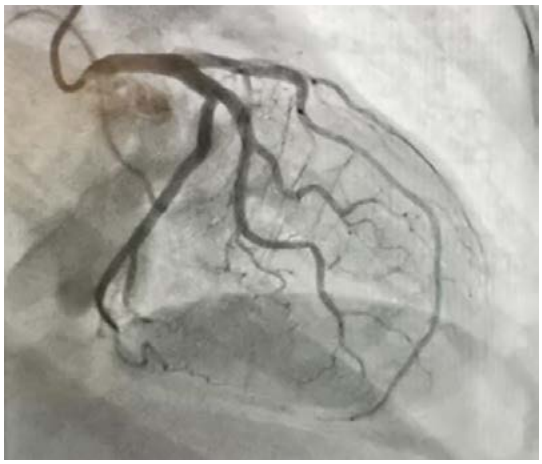

Figure 1: Coronary angiogram showing unobstructed coronary arteries

0.2 ug/kg/minute) as well as external cardiac pacing. Transvenous endocardial right ventricular pacing via a right internal jugular 8.5 French introducer sheath was initiated. The addition of vasopressin to the adrenaline infusion was required to maintain systolic BP ≥ 80 mmHg.

The attending physicians suspected myocardial ischaemia as cause for the arrhythmias and LV dysfunction. However, transfer to the angiography suite and immediate coronary angiography revealed unobstructed coronary arteries (Figure 1). Repeat 12-lead ECG showed return of sinus rhythm and 1 mm ST elevation in leads V5 and V6. Transthoracic echocardiography showed multiple regional wall motion abnormalities (RWMAs) with LV basal hyperkinesis and 'ballooning' of the apical segments; a classic Takotsubo pattern (Figure 2). Left ventricular outflow tract (LVOT) obstruction was not present.

The patient was managed further in the intensive care unit. Initial Hs-Trop T (taken five hours after initial event) was 385 ng/L and decreased to 74 ng/L within 24 hours. The adrenaline and vasopressin were substituted with dobutamine and phenylephrine infusions. These infusions were weaned over the following 48 hours. The patient regained full consciousness and the trachea was extubated the next day. One week after the event, echocardiography showed good LV function (ejection fraction 50%) and complete resolution of the wall motion abnormalities (Figure 3).

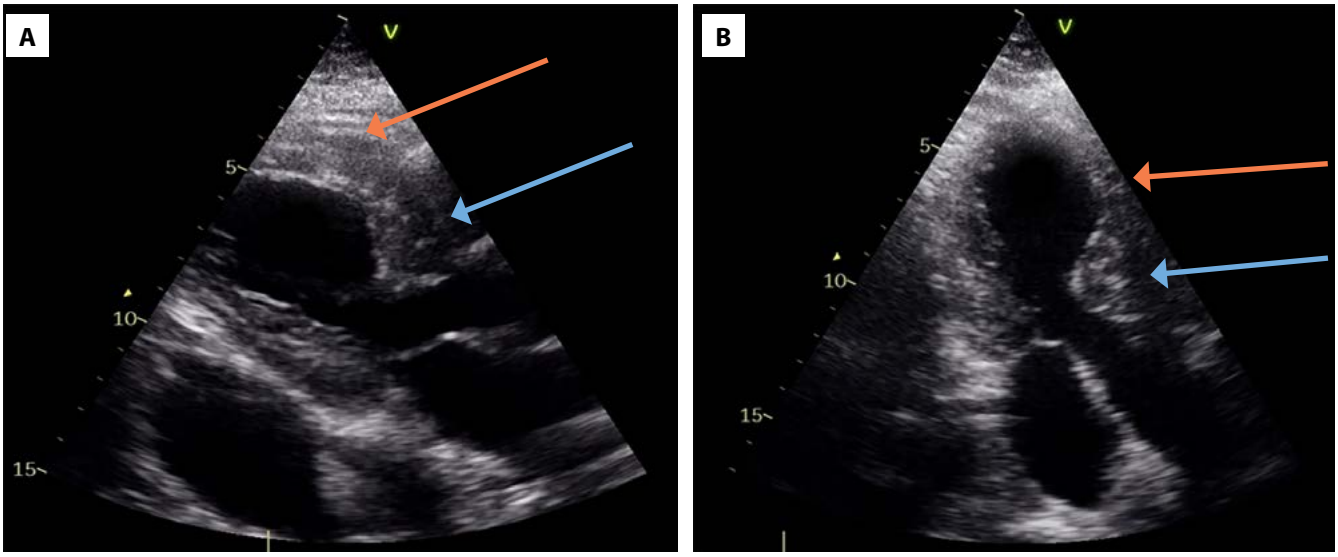

Figure 2: Cardiac echo (2A – parasternal long axis view; 2B – apical 3 chamber view) showing the narrow LV base (blue arrow) and outward bulging of the mid and apical LV segments (orange arrow) at end-systole

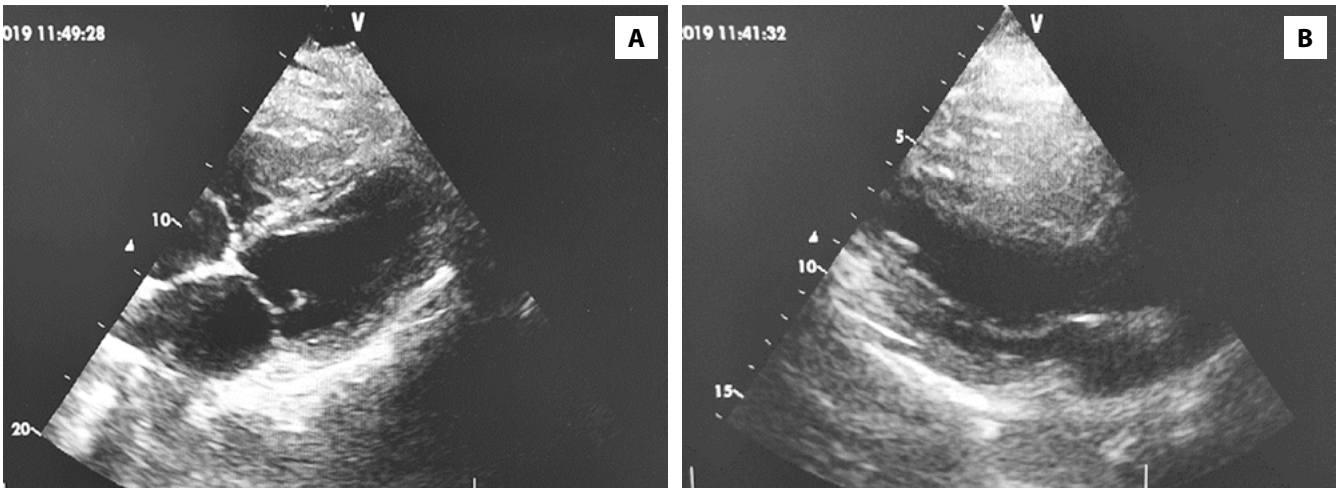

Figure 3: Cardiac echo (3A – subcostal view; 3B – parasternal long axis view) showing a normal end-systolic LV view due to recovery of the wall motion abnormalities

Discussion

Diagnosis

The diagnosis of Takotsubo cardiomyopathy may be challenging as the clinical presentation and ECG changes may be indistinguishable from acute coronary syndrome (ACS).[2] Cardiac troponins are elevated in most cases, but peak levels are usually lower than those seen in myocardial infarction.[6] Coronary angiography usually reveals unobstructed coronary arteries. Even if coronary lesions are present, the RWMAs are not limited to the diseased coronary artery supply territory.[3] An emotional or physical stressor has been found to precede the onset of symptoms in 70% of cases.[3]

The varied clinical presentation has led to non-uniform diagnostic criteria. The most recent diagnostic guidelines, the InterTAK Diagnostic Criteria, were developed by an international expert consensus committee.[2] These guidelines are summarised in Table I. The differential diagnosis may include myocarditis, cardiomyopathies, pulmonary embolism, anaphylaxis, and coronary artery vasospasm. Other non-cardiac causes of central chest pain, such as oesophageal spasm, may also be considered. Cardiac MRI is recommended as a useful tool in confirming the diagnosis of Takotsubo,[3] albeit costly and challenging in a haemodynamically unstable patient.

The perioperative setting presents further diagnostic challenges as anaesthesia may mask symptoms and have a physiological impact on the haemodynamic presentation. The value of focussed anaesthetist-operated echocardiography in haemodynamically unstable patients is increasingly being recognised. The diagnosis of Takotsubo requires cardiac imaging and point-of-care cardiac echo in the perioperative setting may provide the opportunity for rapid diagnosis. Lung ultrasound was not performed in this case, but may also have been informative in demonstrating lung congestion.

Table I: The InterTAK Diagnostic Criteria

1. Transient LV dysfunction with regional wall motion abnormalities, usually extending beyond a single epicardial vessel supply territory. Apical ballooning may be present. Right ventricular dysfunction may be present.
2. An emotional or physical trigger may precede the onset.
3. Neurological disorders (e.g. subarachnoid haemorrhage) or pheochromocytoma may serve as triggers.
4. New ECG changes (e.g. ST-segment elevation or depression, T-wave inversion, QTc prolongation) are usually present.
5. Cardiac biomarker levels are usually moderately elevated. Significant elevation of brain natriuretic peptide is common.
6. Significant coronary artery disease does not preclude the diagnosis.
7. Patients have no evidence of infectious myocarditis.
8. Postmenopausal women are usually affected.

Adapted from InterTAK Diagnostic Criteria[2]

Pathophysiology

The pathophysiology of Takotsubo is not completely understood. Evidence suggests that (endogenous or exogenous) sympathetic stimulation plays a key role in the pathogenesis; however, the exact mechanism of the resultant wall motion abnormalities is unknown.[2] It is hypothesised that the RWMAs are caused by myocardial microcirculatory and endothelial dysfunction, and/or direct catecholamine-induced toxicity of cardiac myocytes.[7] The latter hypothesis is supported by the observation that the LV apex has a higher beta-receptor density rendering it more vulnerable to high levels of catecholamines. Catecholamine-induced LVOT obstruction may contribute to the clinical picture.[2]

It is also unclear as to why certain individuals develop Takotsubo while others, exposed to similar catecholamine surges, do not. Several risk factors have been identified that may result in higher susceptibility to the condition. The preponderance of Takotsubo cardiomyopathy in postmenopausal females suggests that decreased oestrogen levels may be a pathogenetic factor. Links between Takotsubo and psychiatric conditions, particularly depression and anxiety, have been demonstrated.[2] Some patients have multiple episodes of Takotsubo and a familial preponderance has also been described.[8] This risk of recurrence is of importance for anaesthetists. Patients with a history of Takotsubo cardiomyopathy may again present for surgery and it is crucial to minimise potential perioperative triggers that may cause a recurrence.

Most patients have an excellent prognosis, as the transient LV dysfunction resolves within days to weeks. However, the condition is associated with an in-hospital mortality of ± 4.2%, due to cardiogenic shock, pulmonary oedema, and/or cardiac arrest.[9]

Perioperative Takotsubo cardiomyopathy

Catecholamine surges frequently occur during perioperative events such as airway manipulation, surgical stimulation, and acute painful stimuli. Inadvertent intravenous injection of adrenaline during procedures has also triggered the development of perioperative Takotsubo cardiomyopathy.[5] A recent systematic review identified 102 perioperative reports of Takotsubo cardiomyopathy.[5] This review indicated that perioperative Takotsubo cardiomyopathy occurred most often postoperatively. It nevertheless could present at any time from before induction to a few days postoperatively. Interestingly, these authors also identified that a significant number of cases occurred during regional anaesthesia.[6]

Common presenting perioperative signs included ST-T wave changes, hypotension, cardiac failure, arrhythmias and/or cardiac arrest. The hypotension may be due to cardiogenic shock, LVOT obstruction, or combinations thereof. The 'classic' apical ballooning pattern was observed in 84% of cases.[5] An extensive literature search revealed only one previously reported perioperative case originating from South Africa. In that instance, Takotsubo occurred on the first postoperative day, also presenting with cardiogenic shock.[10]

Our patient had multiple risk factors for being susceptible to Takotsubo. Her age, postmenopausal state, significant

illness (breast malignancy) and recent chemotherapy have all been demonstrated to be risk factors.[2] Anastrazole (anti-oestrogen therapy) has also been associated with Takotsubo cardiomyopathy.[11] The perioperative triggers in our case were likely the catecholamine surges associated with laryngospasm-induced hypoxaemia and the hypertensive response to emergency re-intubation. Indeed, hypoxia due to upper airway obstruction has been documented previously to trigger Takotsubo cardiomyopathy.[12,13] The exogenous adrenaline administration in our index case may have aggravated the situation. As demonstrated in this case, Takotsubo may also present with arrhythmias. Ventricular tachycardia has been shown to occur in 3% of cases.[3] The 3rd-degree heart block observed in our patient has been noted to occur in approximately 5% of reported perioperative cases.

Management

Definitive management guidelines are lacking and are largely based on physiological principles and previous case data.[3] Management depends on the presenting symptoms and their severity. Our case presented with ventricular tachycardia followed by a 3rd-degree heart block, which required temporary transvenous pacing. Cardiogenic shock associated with Takotsubo cardiomyopathy is difficult to manage because the use of inotropes acting primarily at adrenergic receptors may be problematic, considering the role of catecholamines in the pathogenesis of the condition, and the risk of aggravating the LVOT obstruction.[14,15] Indeed, our patient responded poorly to adrenaline. Non-catecholamine inotropic agents, levosimendan and phosphodiesterase inhibitors (e.g. milrinone), have previously been used with success.[14] LVOT obstruction may respond favourably to vasopressors (e.g. phenylephrine and/or vasopressin), and intravenous volume expansion.[14] Other therapeutic modalities include mechanical assist devices such as an intra-aortic balloon pump, LV assist devices, or ECMO.[14] Indeed, mechanical support is an appropriate bridge in light of the transient nature of Takotsubo cardiomyopathy.

Conclusion

Takotsubo cardiomyopathy is an unusual cause of perioperative haemodynamic instability. Severe cardiac arrhythmias, conduction defects and features of cardiac failure may be present. The ability to correctly diagnose perioperative Takotsubo timeously is clearly beneficial in aiding prompt and correct management. Without correct diagnosis, the management of hypotension with catecholamines could be ineffective and possibly even aggravate the condition. The anaesthetist who is proficient in echocardiography clearly is well positioned to diagnose this uncommon cause of perioperative haemodynamic instability timeously. Fortunately, if the patient can be bridged, the cardiomyopathy is reversible in most cases. It is our hope that this case report will raise awareness of this infrequent, but potentially life-threatening disorder.

Conflict of interest

The authors declare no conflict of interest.

Funding source

No funding source to be declared.

Ethical approval

Ethics approval was obtained from the Stellenbosch University Human Research Ethics Committee (Ref: C22/06/016).

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