Clinical Journal of Gastroenterology (2019) 12:296–300
https://doi.org/10.1007/s12328-019-00967-x

CASE REPORT

A case of esophagogastroduodenoscopy induced Takotsubo cardiomyopathy with complete heart block

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Received: 4 December 2018 / Accepted: 31 January 2019 / Published online: 23 March 2019

Abstract
Takotsubo cardiomyopathy (TC) describes a reversible left ventricular dysfunction characterized by apical ballooning and basal hyperkinesis, commonly triggered by emotional or physical distress. TC associated with an esophagogastroduodenoscopy (EGD) has rarely been reported. We report a case of TC with complete heart block (CHB) in a patient receiving an EGD, who had no underlying cardiac disease, had previously tolerated both local and general anesthesia, and who had previously undergone similar endoscopic procedures without complications. The concurrence of both TC and CHB is unique in this case pertaining to a patient with no significant risk factors. The incidence, mechanism and prognosis of TC-associated arrhythmias are also reviewed.

Keywords Esophagogastroduodenoscopy complications · Takotsubo cardiomyopathy · Complete heart block · Anesthesia · Arrhythmia

Introduction

Takotsubo cardiomyopathy (TC) is a reversible cardiac dysfunction of the left ventricle (LV), characterized by mid-ventricular ballooning and basal hyperkinesis, resembling the shape of a Japanese octopus trap [1, 2]. It was first described by Sato in 1990 and has since then been increasingly recognized as a sequela of emotional or physical distress [1]. It has a predilection for postmenopausal women but can occur in any gender at any age [1]. While surgery and general anesthesia are known triggers for TC, the occurrence of TC in the setting of an endoscopic procedure is exceedingly rare [1–6]. To date, seven cases have been reported in the English literature where TC occurred as a result of performing an esophagogastroduodenoscopy (EGD) or colonoscopy [4–6]. We describe a case of TC with complete heart block (CHB) that developed shortly after intubation during an EGD.

Case report

An 81-year-old female with gastroesophageal junction thickening on a recent computed tomography and a decreased appetite presented for an elective EGD. Her medical history included hyperlipidemia, stage IIA adenocarcinoma of the ascending colon status post (s/p) curative right hemicolectomy 4 years ago, and common bile duct (CBD) stone with a papillary stricture s/p sphincterotomy followed by plastic stent placement via endoscopic retrograde cholangiopancreatography (ERCP) 1 week ago. A colonoscopy completed 6 months ago revealed only diverticulosis. Upon

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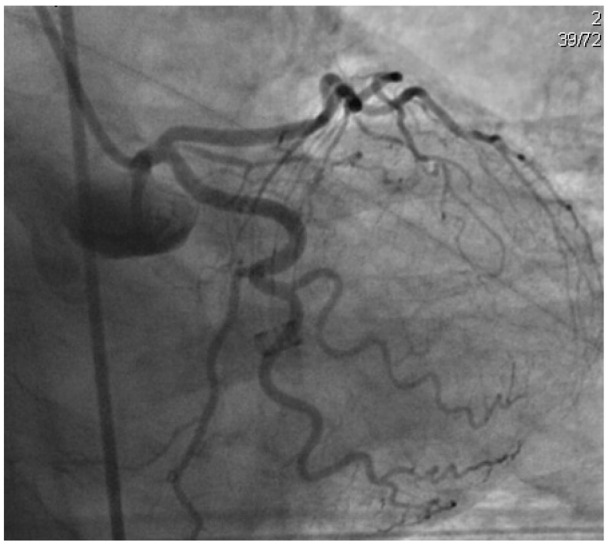
preoperative assessment, the patient expressed anxiety, but this had dissipated at the inception of the procedure. During the endoscopy, 50 µg of intravenous (IV) Fentanyl and 100 mg of IV Propofol were given for sedation, after which, the gastroscope was inserted and advanced through the proximal esophagus. At this point, bradycardia with a heart rate in the 30 s was noted which persisted despite the administration of Glycopyrrolate, necessitating the withdrawal of the gastroscope. Atropine and Epinephrine were subsequently administered; however, the patient further deteriorated, requiring cardiopulmonary resuscitation for 1 min before she regained consciousness. Although her hemodynamics briefly improved, hypotension and bradycardia soon resumed. A 12-lead electrocardiogram (ECG) was obtained which demonstrated CHB. She was resuscitated with normal saline, Ephedrine, and Dopamine, but a repeat ECG revealed new left bundle branch block. The patient then immediately underwent cardiac catheterization and was found to have normal coronary arteries but an ejection fraction (EF) of 30% on ventriculogram, with mid-apical left ventricular ballooning suggestive of stress cardiomyopathy (TC, Fig. 1 a–c). Given the patient's CHB, a transvenous pacemaker (TVP) was placed (Fig. 2). An echocardiogram revealed severe hypokinesis of the mid-apical walls of the LV with an EF of 25–30%, a dilated left atrium, moderate calcification of the mitral valve, and a normal right ventricle (Fig. 1d). An echocardiogram from 1 year ago reported an EF of 75% and moderate mitral valve calcification. Due to the persistent TVP-dependent CHB, the patient received a biventricular implantable cardioverter defibrillator on day 3 post-EGD. Lisinopril and Carvedilol were subsequently initiated, and the patient was discharged on day 5 post-EGD. She underwent an ERCP 2 months later for the removal of the previously placed CBD stent and the extraction of CBD stones. She further returned for an EGD 5 months later showing gastritis and a hiatal hernia. The patient tolerated both procedures without complications.

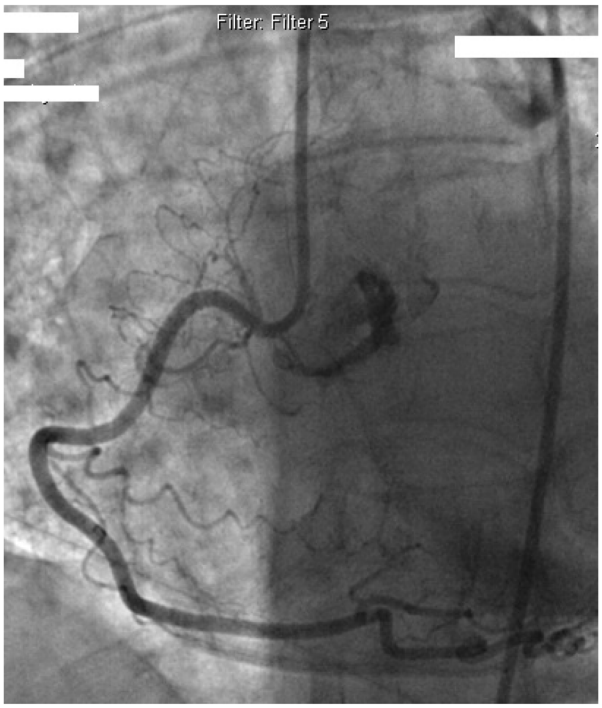
Discussion

TC is an extremely rare complication of endoscopy [4–6]. TC with concurrent CHB occurring during an EGD or colonoscopy has not been previously reported. Common triggers for TC include physical stress such as stroke, infection, cancer, or surgery, and emotional stress, engendered by both tragic and joyful events, with the former conferring a worse outcome than the latter [1–3]. TC has an incidence of 0.7%, but the underlying etiology is largely unknown [2]. A few mechanisms have been proposed, including the catecholamine surge, inhibitory G protein signaling at the LV apical adrenergic receptors, microvascular dysfunction, and loss of the estrogen-mediated adrenergic suppression after menopause [2, 3]. Diagnosis is made based on the modified Mayo Clinic diagnostic criteria (Table 1), the hallmark of which is regional myocardial dysfunction in the absence of coronary artery stenosis [7]. Brain natriuretic peptide usually rises out of proportion to troponin, and cardiac magnetic resonance typically reveals intense myocardial edema [2, 3]. Four subtypes of TC have been described depending on the location of dyskinesia: apical, mid-cavity, basal, and focal [3]. Treatment in the acute setting is supportive care, including mechanical cardiac support if necessary [2, 3]. Angiotensin-converting enzyme inhibitors may reduce mortality and recurrence at 1 year, but beta blockers have no short- or long-term benefits [3]. Contrary to traditional beliefs, the long term mortality of TC approximates that of myocardial infarction, and in the acute setting, mortality can reach 3–5% [1, 3].

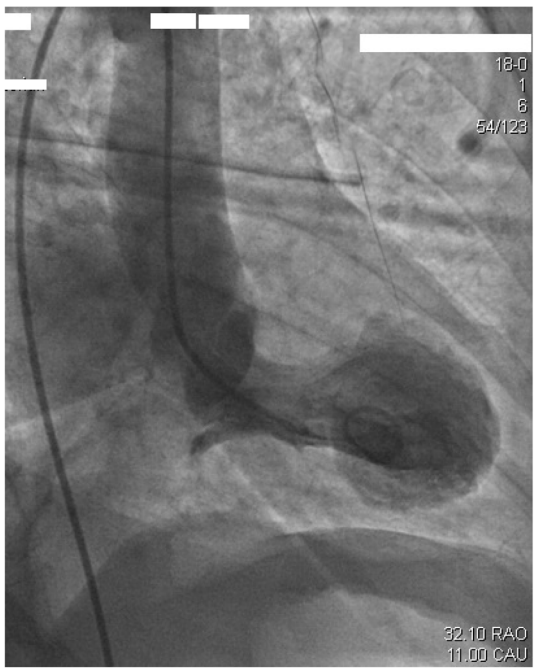
Arrhythmias associated with TC are well recognized, and can originate from both the atrium and the ventricle [8–10]. Life threatening arrhythmias (LTAs) in particular, account for 12.2% of all TC-related arrhythmias and include ventricular fibrillation (2.4%), ventricular tachycardia (5.6%), asystole (1.7%), pulseless electrical activity (0.3%), complete atrioventricular block (CAVB, 3.0%), and sinoatrial block (SAB, 1.0%) [11]. Atrial arrhythmias (AAs) occur at an incidence of 24.8% and mainly comprise atrial fibrillation (20.1%) and atrial flutter (8.4%) [10]. Both LTAs and AAs confer a worse outcome in TC. A large German study revealed that the 1-year mortality was 44% in TC patients experiencing LTAs compared to 10% in TC patients without arrhythmias [8]. Similarly, Jesel et al. showed an increase in the length of stay, in-hospital mortality, and 1-year mortality in TC patients with LTAs [9]. Low EF and long QRS (> 105 ms) were independent predictors of LTAs in TC [9]. The same pattern was seen in AAs, with worse in-hospital, 30-day, and long-term (up to 7 years) mortality in TC with AAs compared to without [10]. Interestingly, different types of arrhythmias behave differently. Polymorphic ventricular arrhythmias tend to resolve prior to discharge, whereas severe conduction abnormalities such as CAVB can persist for years even after the EF has recovered to normal [9, 11]. Jesel et al. showed that 6 out of 6 TC patients requiring a pacemaker placement had persistent heart block (5 CAVB and 1 SAB) at 5-year follow up [9]. These data suggest that TC-associated conduction defects are prolonged and their clinical course does not parallel that of the cardiac dysfunction. A permanent pacemaker (PPM) should be considered in these patients prior to discharge; however, it is worth noting that the management of bradyarrhythmias such as CHB in TC remains highly controversial and the decision to place a PPM or an implantable cardioverter defibrillator is highly case-dependent [9, 11, 12]. A few mechanisms potentially explaining the risk of arrhythmia in TC are myocardio edema along with micronecrosis and subtle fibrosis



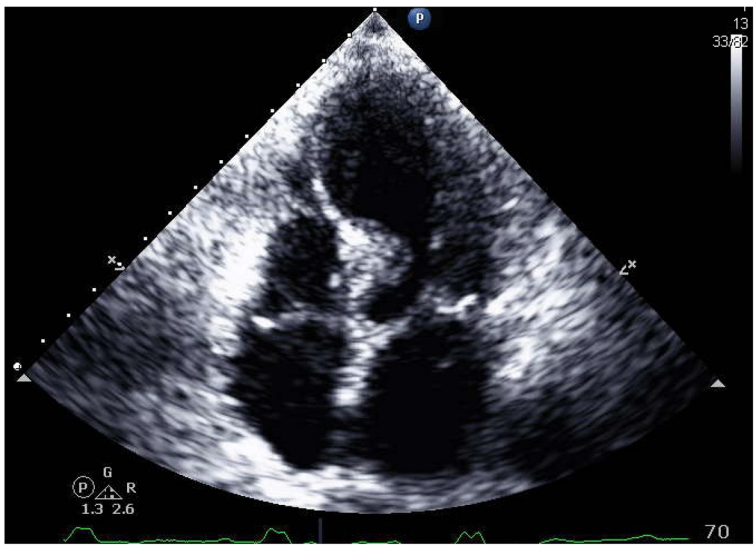
a Cardiac catheterization: right anterior oblique view with caudal angulation, showing normal left coronary circulation.



b Cardiac catheterization: normal right coronary circulation.



c Left ventriculogram showing mid-to-apical wall ballooning and basal hyperkinesis during systole



d Echocardiogram: four-chamber view, showing left ventricular ballooning during systole.

Fig. 1 **a** Cardiac catheterization: right anterior oblique view with caudal angulation, showing normal left coronary circulation. **b** Cardiac catheterization: normal right coronary circulation. **c** Left ventriculogram showing mid-to-apical wall ballooning and basal hyperkinesis during systole. **d** Echocardiogram: four-chamber view, showing left ventricular ballooning during systole

of the myocardium, QT prolongation, catecholamine excess, and a secondary increase in the vagal tone in the case of bradyarrhythmias [8]. The recurrence rate of TC-associated arrhythmias is thought be 22%, according to one study [9].

In our case, it was likely that TC occurred secondarily to the CHB as a result of a compensatory catecholamine surge. However, one cannot exclude the possibility that the two phenomena occurred simultaneously [13]. Other plausible triggers for TC in this case included a gastroscope-induced vagal response, excessive adrenergic stimulation during the resuscitation, anesthesia, and preoperative anxiety [3, 14, 15]. Specifically, local anesthesia accounts for

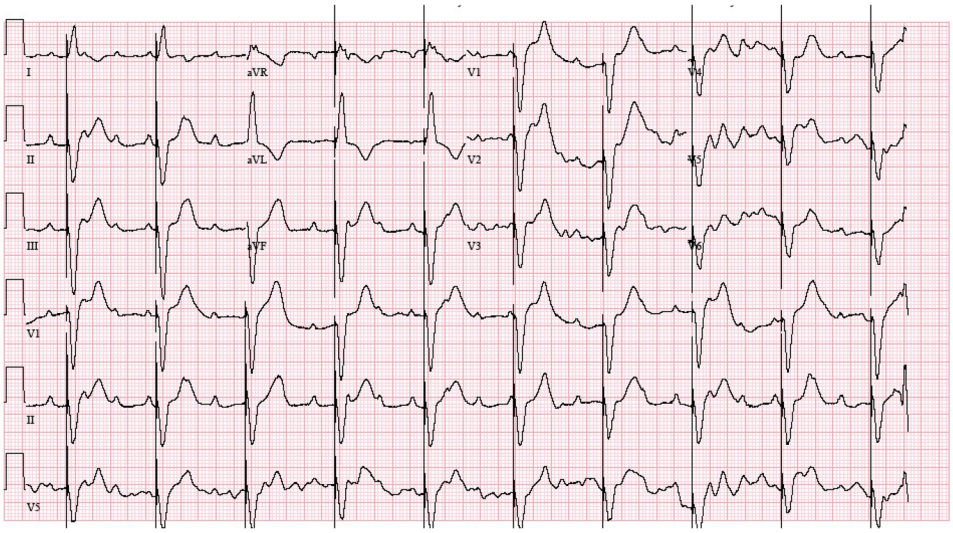


Fig. 2 Electrocardiogram: sinus tachycardia with complete heart block and a ventricular paced rhythm

Electrocardiogram: sinus tachycardia with complete heart block and a ventricular paced rhythm

Table 1 Mayo Clinic diagnostic criteria for Takotsubo cardiomyopathy (apical ballooning syndrome) [7]

1. Transient hypokinesis, akinesis, or dyskinesis of the left ventricular mid segments with or without apical involvement; the regional wall motion abnormalities extend beyond a single epicardial vascular distribution; a stressful trigger is often, but not always, present[a]
2. Absence of obstructive coronary disease or angiographic evidence of acute plaque rupture[b]
3. New electrocardiographic abnormalities (either ST-segment elevation and/or T-wave inversion) or modest elevation in cardiac troponin
4. Absence of: pheochromocytoma, myocarditis

[a]There are rare exceptions to these criteria such as those patients in whom the regional wall motion abnormality is limited to a single coronary territory

[b]It is possible that a patient with obstructive coronary atherosclerosis may also develop Takotsubo cardiomyopathy. However, this is very rare in our experience as well as in the published literature, perhaps because such cases are misdiagnosed as an acute coronary syndrome

3% of all perioperative TC [16]. However, the exact role of any particular anesthetic agent in the pathogenesis of TC is unclear. There is also no clear strategy to prevent anesthesia related TC, although adequate preoperative stress relief using anxiolytics has been emphasized [16]. In general, TC recurs at the rate of 1.8% per patient-year with a cumulative recurrence of 1.2% at 6 months and 5% at 6 years [16]. Recurrence particularly related to endoscopy is not known. Independent of TC, CHB may also result from anesthesia and the procedure itself. Propofol, as compared to midazolam or fentanyl, is associated with a higher incidence of cardiac arrhythmias, including bradycardia, heart block, ventricular fibrillation, and asystole [17, 18]. The onset of arrhythmias, however, would be difficult to predict. Our patient had previously received both general and local anesthesia during a hemicolectomy and ERCP, respectively, but had no perioperative complications. EGD alone can additionally be associated with a 38.5% risk of arrhythmias [19]. However, rarely does this manifest as CHB requiring PPM placement, especially in a patient with no underlying cardiac impairment. It should be stressed that our patient was subjected to the same operative conditions during a prior ERCP and colonoscopy and endured no cardiac events, highlighting the unpredictability of the onset of cardiac complications during an endoscopy.

Overall, our case illustrates that serious cardiovascular complications, such as TC and CHB, can occur during an EGD in the most unpredictable manner. TC patients experiencing arrhythmias have worse clinical outcomes including increased all-cause mortality compared to those without arrhythmias. The endoscopist should be aware of the potential cardiac complications involving an endoscopy as well as their prognosis, and the endoscopic facility should be adequately equipped to respond to these critical events as they arise.

Funding This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Compliance with ethical standards

Conflict of interest The authors declare that there is no conflict of interest regarding the publication of this article.

Human and animal rights All procedures followed have been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments.

Informed consent An informed consent from the patient was obtained.

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