



## Case Report

# Takotsubo Cardiomyopathy Complicated By Arrhythmia: A Rare Presentation after Renal Transplant

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### Abstract

Takotsubo cardiomyopathy (TTC) is a rare clinical condition after kidney transplantation of unexplained origin mimicking an acute myocardial infarction (AMI). While there have been three renal transplant recipients reported to have developed TTC worldwide, we report the first patient to experience TTC and arrhythmia, requiring a double chamber pacemaker.

**Case Report:** The patient is a 60-year-old female with a history of end-stage renal disease secondary to hypertension and diabetes. She received a kidney transplant from a deceased donor with no intraoperative complications. The renal graft demonstrated good function initially until the subsequent events. On post-operative day one, the patient developed acute respiratory failure and hypotension requiring ventilatory support and vasopressor agents were required. Left ventricular function was severely depressed in a pattern consistent with stress cardiomyopathy in cardiogenic shock. An IMPELLA device was placed for 7 days and improved her hemodynamics. As a complication of this event, the patient continued to have symptomatic and frequent significant conversion pauses that has been managed by permanent dual chamber pacemaker. The patient eventually was discharged home with good kidney function after a short period of continuous renal replacement therapy (CRRT). **Conclusion:** Given the nonspecific symptoms and signs of TTC after kidney transplant cases, a high clinical index of suspicion is necessary to detect the disease in different clinical settings and scenario.

**Keywords:** Arrhythmia; Kidney transplantation; Takotsubo cardiomyopathy

### Introduction

Takotsubo cardiomyopathy (TTC) is an acute reversible clinical condition of unexplained origin mimicking an acute myocardial infarction (AMI). It is further recognized as broken heart syndrome, stress cardiomyopathy, or transient left ventricular apical ballooning syndrome.

Clinical components of this syndrome include abrupt onset

of chest pain, ST-segment changes, and reversible left ventricular dysfunction on echocardiography, normal coronary angiogram, and minimal elevation of cardiac enzymes.

TTC linked with systemic diseases and emotional or physical stress. TTC usually develop in the perioperative course and among extremely ill patients. The diagnosis of TTC should be based on clinical symptoms and differentiated from a range of differential diagnoses by using cardiac echocardiography, serum biomarkers, and cardiac catheterization.

TTC has been reported rarely after kidney and liver

transplants [1-4]. So far, there have been only three case reports of TTC in the renal transplant recipient population. Still, none of them reported an arrhythmia and a need for a double chamber pacemaker as a complication of TTC. We present a case herein of a 60-year-old woman who had a renal transplant complicated by TTC, which has been followed by arrhythmia. She underwent IMPELLA device insertion and a double chamber pacemaker.

## Case Report

The patient is a 60-year-old female with a history of end-stage renal disease secondary to hypertension and diabetes; she has been on dialysis for 2 years before transplant. The patient has other medical history notable for morbid obesity, atrial fibrillation on warfarin, HTN, and hypothyroidism. Her surgical history is significant for Roux-en-Y Gastric Bypass.

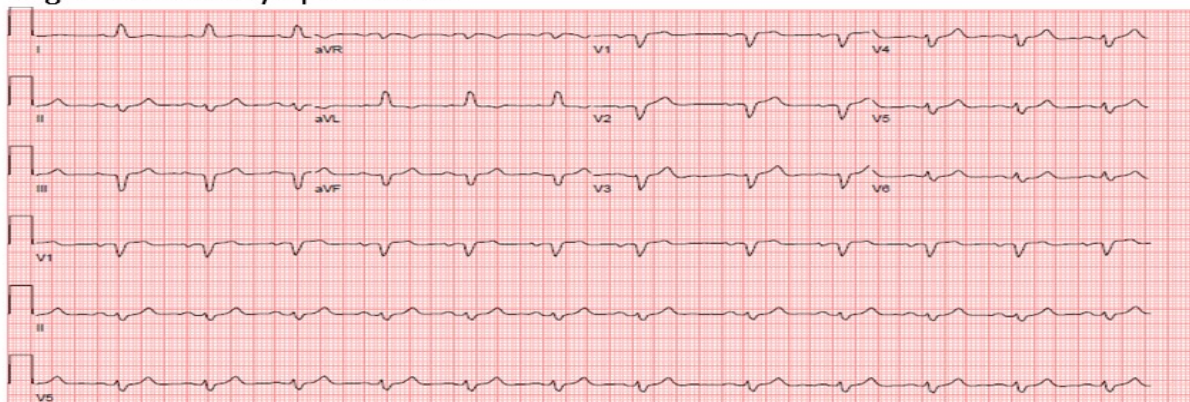
She received a kidney transplant from a deceased donor, with KDPI 57% and zero cross-match. There were no intraoperative complications; she received 1 unit of PRBCs and 2 units of FFP. The estimated blood loss was 150 ml, and she started to make urine

immediately. She received thymoglobulin for induction therapy at the time of transplant. The patient was extubated immediately after the surgery and moved to the PACU in a stable condition. She was admitted to the Transplant Intensive Care Unit due to concern of bleeding as her procedure was performed with a therapeutic INR, and she had an atrial fibrillation episode during the surgery.

Early in the morning on day 1, the patient developed acute respiratory failure requiring emergent intubation. She also became hypotensive with a requirement of norepinephrine and became acidotic with elevation in lactic acid to 6mmol/L. During this event, she was awake and denied chest symptoms.

STAT EKG (**Figure 1**); showed Normal sinus rhythm with sinus arrhythmia, Left axis deviation, and Left bundle branch block with mild cardiac enzymes elevation (**Table 1**). The patient remained hypotensive and bradycardic; Norepinephrine was initiated with improvement. The patient was started on diuretics due to poor urine output and volume overload. However, urine output remained low. In addition, she became hyperkalemic and continuous renal replacement therapy (CRRT) was began.

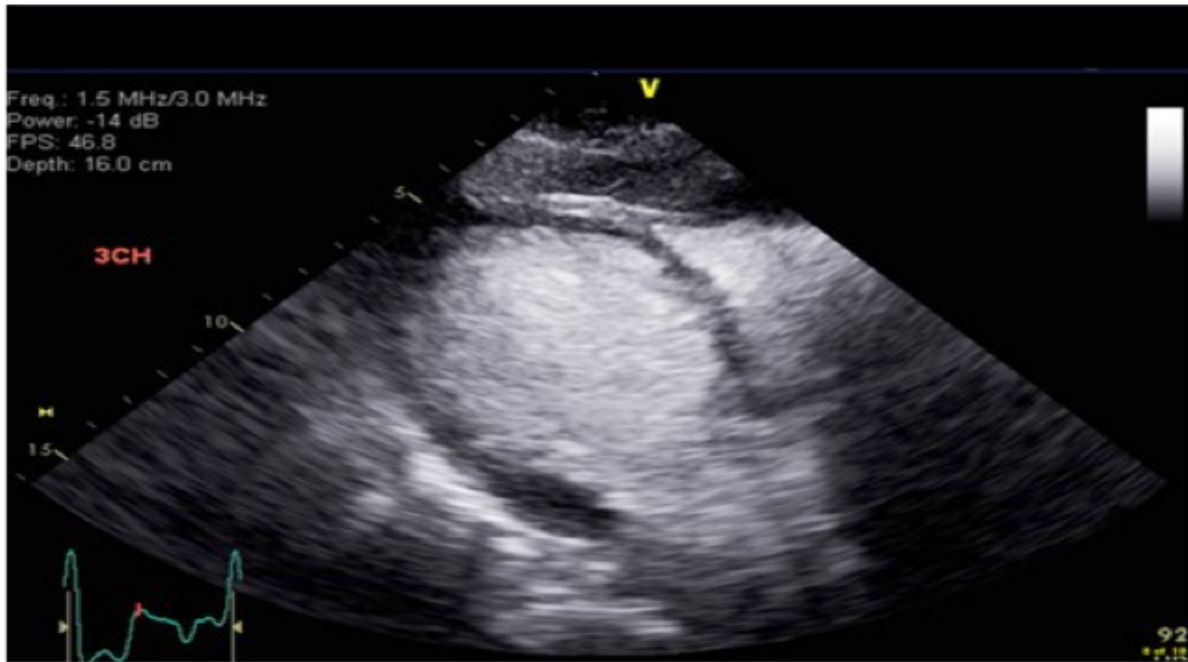
**Figure 1: EKG at symptom onset**



**EKG: normal sinus rhythm with left bundle block**

Bedside echocardiogram (**Figure 2**); Left ventricle was normal in size with severe systolic dysfunction. The quantitative LVEF based on modified Simpson's method was 15% with severe global hypokinesis. Due to hemodynamic compromise and new LBBB, cardiac catheterization was performed and showed Luminal irregularities throughout and 70% stenosis in the RCA. No intervention was done, and the patient continued to require increasing doses of NE and dobutamine was added.

**Figure 2: Bedside echocardiogram**



Echocardiogram shows left ventricle with severe systolic dysfunction that resembles stress cardiomyopathy

Left ventricular function was severely depressed in a pattern consistent with stress cardiomyopathy in cardiogenic shock. The shock code team was activated, and the decision was made to proceed with IMPELLA device insertion, which improved the hemodynamics significantly. She was off all vasopressors within hours after insertion, without impairment of hemodynamics, and removed after 7 days (**Table 1**). Three days after the IMPELLA insertion, her ejection fraction was 54%, and she was extubated.

**Table 1:** Trends in LVEF and troponin T.

Time	LV EF (%)	Troponin T (ng/ml)
POD 1	<20%	0.054
POD 2	25-29%	1.630
POD 4	55-59%	1.070
POD 7	65-69%	0.540
POD 9	>74%	X
POD 14	>74%	X

**LVEF:** Left Ventricular Ejection Fraction.

The patient continued to have symptomatic and frequent significant conversion pauses (multiple 6-8 second pauses, approximately one pause every minute for 15 minutes) and occasionally has sinus beats with PACs. The case was discussed with the Electrophysiology team, who proceeded with placement of a temporary pacing wire. After that, the patient continued to have ongoing bradycardia and need for treatment of atrial bradyarrhythmias with long conversion pauses a permanent dual chamber pacemaker was placed.

The patient's urine output and serum creatinine improved and CRRT was discontinued. The patient eventually was discharged home after a short stay in inpatient rehabilitation unit.

## Discussion

TTC is a disease first cited by Dote and partners in 1991 at the Hiroshima City Hospital [5]. The name was obtained from the appearance on left ventriculography at the end-systolic stage, which appears as a Takotsubo (bowl for trapping octopus used by Japanese fishers)-like shape owing to extreme contraction of the base of the heart to compensate for extensive transient akinesia of the left ventricular apex.

TTC has two subtypes, primary and secondary; the typical patient with primary Takotsubo syndrome is a post-menopausal woman who has encountered serious, sudden emotional or physical stress [6]. The patients with the primary subtype present with manifestations like acute cardiac syndrome. The secondary subtype includes mainly hospitalized patients whose TTC is assumed to have been precipitated by their underlying medical or surgical conditions or operations. Most cases that surgeons encounter belong to this secondary subtype [7].

*Deshmukh A et al.* reported the prevalence of TTC based on nationwide hospitalization records; they noticed that women have greater likelihood of developing TTC (odds ratio 8.8). The overall frequency of TTC was calculated to be 5.2 per 100,000 for women and 0.6 per 100,000 for men. In an absolute number of admissions, 6,178 (90.4%) were women, and 660 (9.6%) were men. Also, they noted that women >55 years old had 4.8 times greater odds of developing TTC when matched with women <55. Whites were twice as prone to be diagnosed with TTC as Blacks, Hispanics, and Asians. TTC is assumed to be the explanation of 1-2% of patients presenting with symptoms like acute coronary syndrome, more so in females than in males. Further, they found that TTC was diagnosed in about 0.02% of all hospitalizations in the United States, particularly in old women with history of smoking, alcohol abuse, anxiety states, and hyperlipidemia [8].

A hallmark of TTC is complete reversibility of the left ventricular contraction abnormalities within days to weeks, new left bundle branch block, or QTc prolongation as electrocardiogram (EKG) evidence with slight elevations in troponin. The “*gold standard*” in ruling out a primary coronary obstructive etiology is the use of invasive coronary angiography to exclude coronary artery disorder, although cardiac computed tomography angiography, magnetic resonance imaging (MRI), and nuclear imaging may further assist in the diagnosis [9].

Reported hospital mortality ranges from 0 to 8%, males had more than two-fold greater mortality than females [6, 10]. Patients

with “*secondary*” TTC have a two-to tenfold higher mortality than those with “*primary*” TTC. Among hospital deaths, the cause in 62% of cases was related to the underlying comorbid medical condition, while in 38%, it was directly related to the cardiac complications of TTC mainly caused by refractory cardiogenic shock or ventricular fibrillation [11, 12].

TTC has widely been considered as a somewhat benign condition as many cases recover without complications. However, TTC patients are susceptible to serious complications like congestive heart failure (12-45%), pulmonary edema (8 to 20%), and cardiogenic shock (4-20%), which is associated with increased short and long-term mortality rates [13]. This group of patients may benefit from early mechanical assistance as a ‘*bridge-to-recovery*’ to reduce or avoid inotropic therapy [6, 7].

Arrhythmia, as what our patient had after the recovery of TTC, develops in up to 44% of patients and life-threatening arrhythmias develop in about 6% including atrial fibrillation (5-47%), ventricular tachycardia (1.2-4.0%), ventricular fibrillation (1-2%), and asystole (0.5-3%) [6].

Although a number of assumptions have been suggested to explain TTC, recent preclinical and clinical studies have demonstrated that the role of catecholamines is fundamental to the pathophysiology of TTC, either directly injuring the myocytes due to the excess intracellular catecholamines and/or indirect ischemic injury due to brief microvascular dysfunction [14]. This has been acceptable for many years and has led to some investigators renaming TTC as “*stress cardiomyopathy*”.

The previous three cases of TTC in renal transplant patients presented with a range of symptoms from anxiety, tachycardia, to cardiac arrest. Many had elevated troponin, left bundle branch block, and all had echocardiograms that showed evidence of Takotsubo. They could all be treated with medical management. Whereas, our patient required mechanical cardiac support. Like all the previous cases, our patient recovered her EF completely. Our patient is the only one complicated by bradyarrhythmia requiring pacemaker placement.

## Conclusion

Perioperative cases of TTC present with evidence of heart failure, arrhythmias, or cardiac arrest. Thus, one must consider TTC when any of these signs and symptoms occurs during the perioperative period. Given the nonspecific symptoms and signs after kidney transplant cases, a high clinical index of suspicion is necessary to detect the disease in different clinical settings. A key feature of TTC is recovery of normal cardiac function. The major objective of in-hospital treatment should be supportive care to sustain life and to minimize complications during recovery.

**Conflict of Interest:** There are no financial conflicts of interest to report.

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