The Journal of Tehran University Heart Center

Case Report

An Unusual Presentation of Takotsubo Cardiomyopathy: A Case Report

Gajendra Dubey, MD, DM, Rujuta Parikh, MD, DM*, Karthik Natrajan MD, DM, Kewal Kanabar, MD, DM

Department of Cardiology, U. N. Mehta Institute of Cardiology and Research Centre, Civil Hospital Campus, Gujarat, India.

Received 08 December 2022; Accepted 07 April 2023

Abstract

Tachyarrhythmias have been well-defined in patients with Takotsubo cardiomyopathy (TTCM) and are estimated to occur in almost 13.5% of patients. However, limited data are available on bradyarrhythmias in patients with TTCM. The pathophysiology, clinical implications, and manage-ment are not well defined in this subgroup. We describe a 53-year-old woman presenting with complete heart block with TTCM and a 73-year-old woman presenting with syncope with complete heart block with TTCM. Both had persistent conduction delays despite recovery of ventricular function and eventually required permanent pacemaker implantation. The dependency on pacing was up to 90% in both patients at a 6-month follow-up.

J Teh Univ Heart Ctr 2023;18(4):298-302

This paper should be cited as: Dubey G, Parikh R, Natrajan K, Kanabar K. An Unusual Presentation of Takotsubo Cardiomyopathy: A Case Report. J Teh Univ Heart Ctr 2023;18(4):298-302.

Keywords: *Atrioventricular block; Bradycardia; Cardiomyopathy; Takotsubo*

Introduction

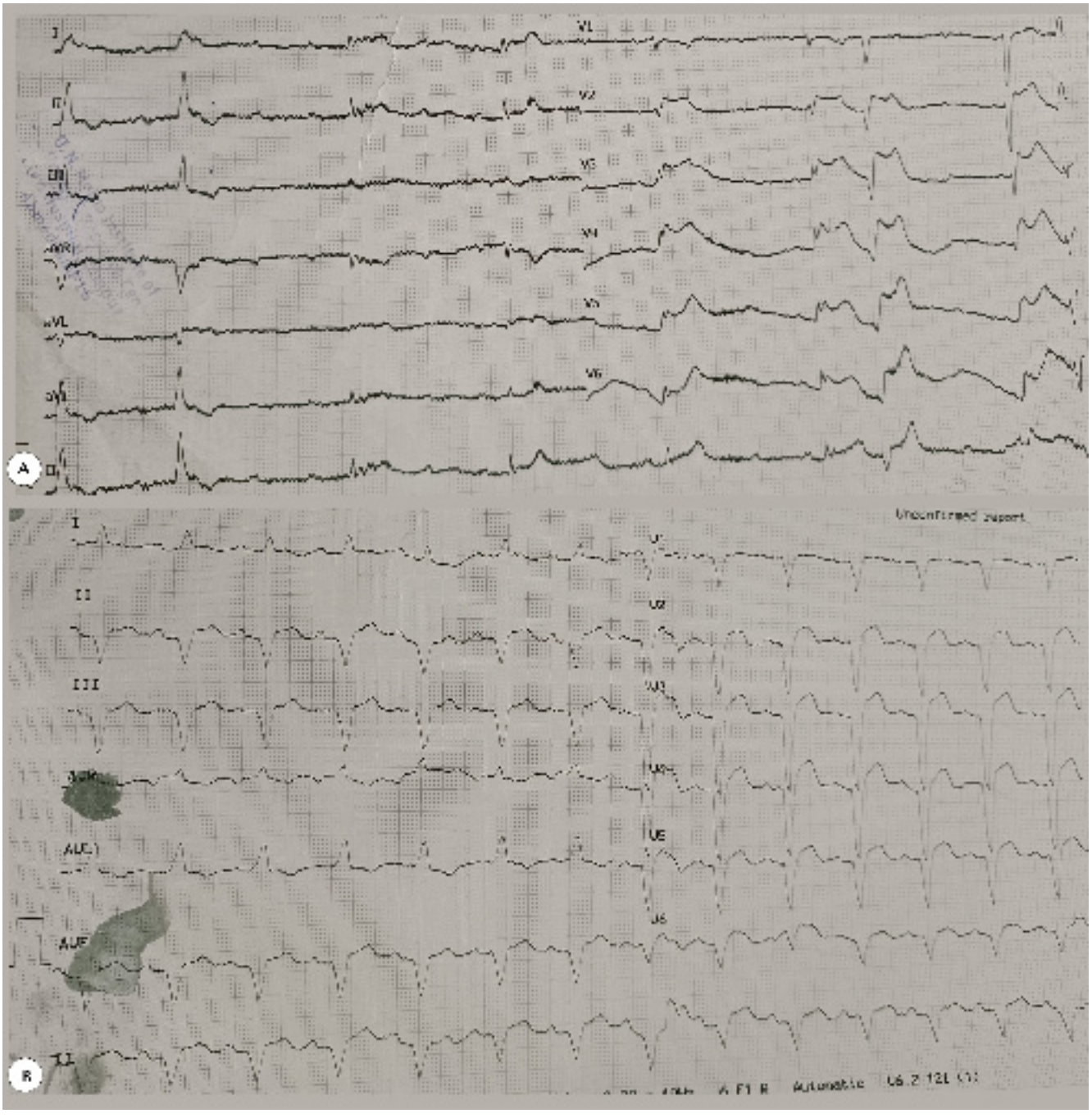
Takotsubo cardiomyopathy (TTCM) is characterized by transient reversible left ventricular (LV) dysfunction, new electrocardiographic changes (ST-segment elevation/ T-wave inversion), elevated troponin levels, and angiographically determined nonobstructive coronary artery disease. The most common electrocardiographic abnormalities on presentation include ST-segment elevation (35.6%), T-wave inversion (17.2%), and ST-segment depression (1.2%) involving the anterior chest leads.[1] Nonetheless, while a variety of arrhythmias, such as paroxysmal atrial fibrillation and ventricular tachycardia, are associated with TTCM (782 and 314 per 10,000 patients, respectively), bradyarrhythmias have rarely been described.[2] Herein, we report 2 cases of TTCM presenting with complete heart block (CHB) with persistent conduction system disease despite the recovery of LV function, eventually necessitating permanent pacemaker implantation.

Case #1

A 53-year-old woman with no history of diabetes or hypertension presented to the emergency department with acute-onset chest pain and dyspnea. An electrocardiogram (ECG) showed ST-segment elevation in leads V2–V6

*Corresponding Author: ***Rujuta Parikh,*** *Assistant Professor of Cardiology, Department of Cardiology, U. N. Mehta Institute of Cardiology and Research Centre (UNMICRC), Civil Hospital Campus, Asarwa, Ahmedabad-380016, Gujarat, India. Tel: +91 9427475822. Fax: +79 22682092. E-mail: rujutaparikh1992@gmail.com.*

with complete atrioventricular (AV) dissociation and a heart rate of 46 beats per minute (Fig. 1A: Panel 1). A 2D echocardiogram showed severe LV dysfunction with an LV ejection fraction of 28% by the Simpson method (Fig. 1B: Panel 1), akinesia in the mid and basal portions of the LV wall, and moderate mitral regurgitation (Fig. 1C: Panel 1). The troponin-I level was 3587.1 ng/L (positive), and the B-natriuretic peptide level was elevated. A working diagnosis of anterior wall ST-elevation myocardial infarction (STEMI) with CHB and severe LV dysfunction was made. A coronary angiogram showed unobstructed coronaries. The patient was managed conservatively with a temporary pacemaker, afterload-reducing agents, and other supportive treatments (Fig. 1D: Panel 1). Serial echocardiograms showed partial recovery of LV function (up to 45%), but CHB persisted. Hence, a dual-chamber permanent pacemaker was implanted. She was subsequently discharged in hemodynamically stable condition. At a 6-month follow-up, she remained pacing-dependent (up to 90%) with an LV ejection fraction of 50%.



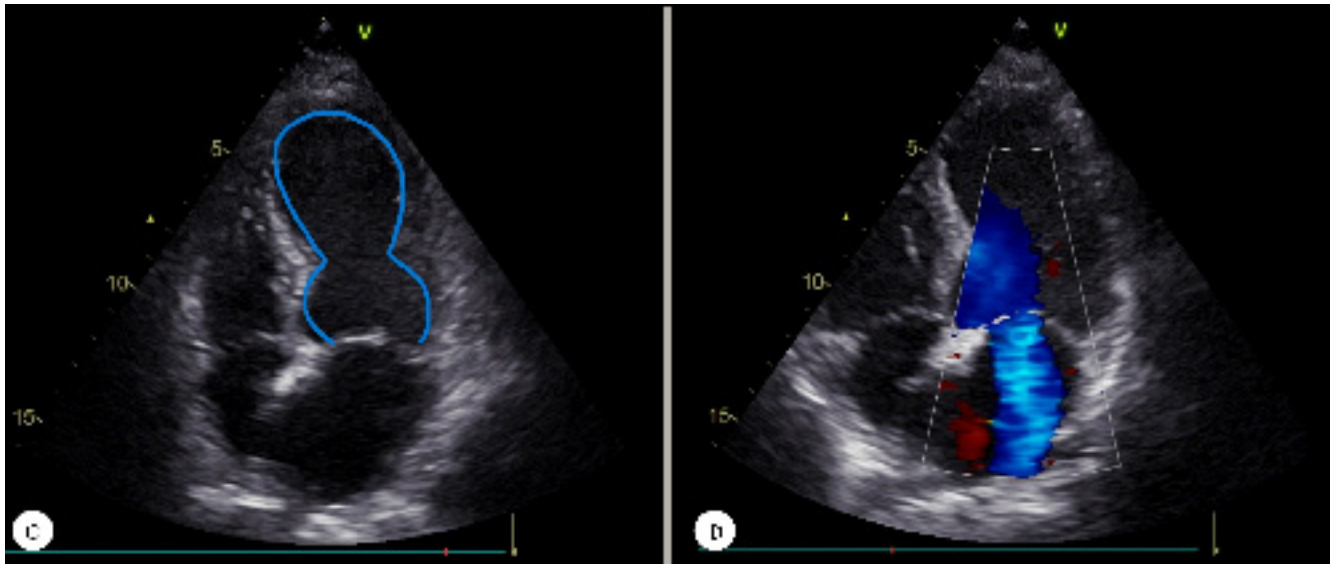


Figure 1. The images show the patient's

A) ECG on presentation showing ST elevation in anterior leads with complete AV dissociation

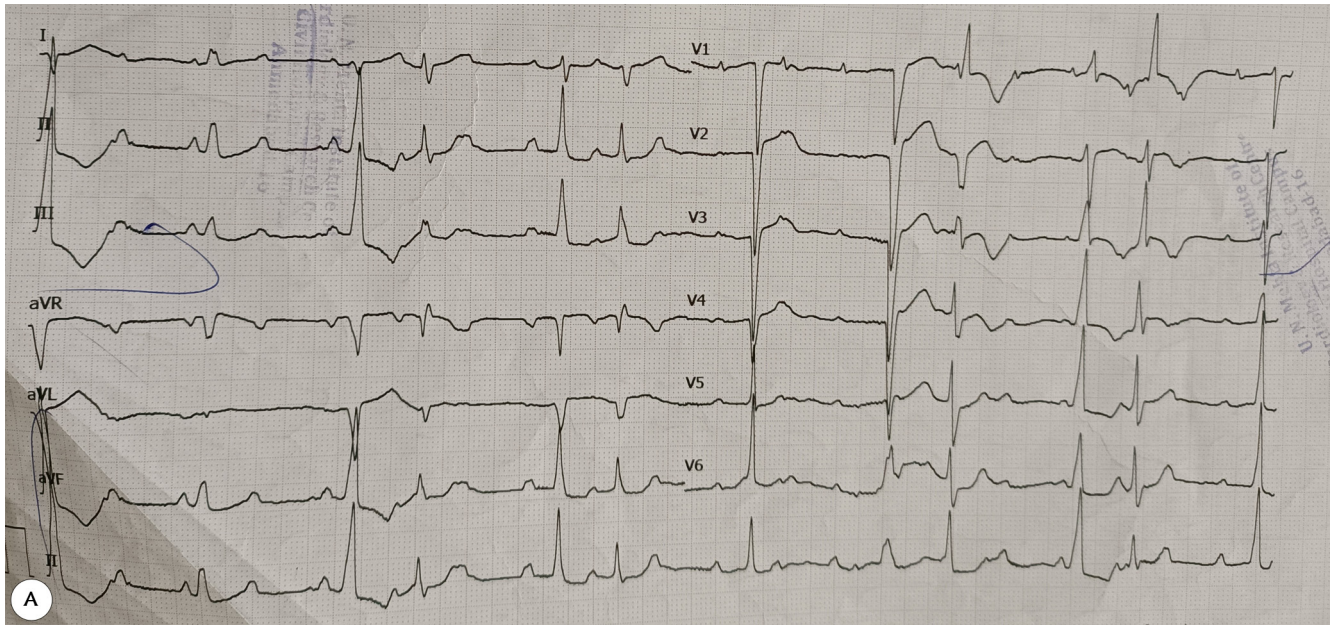
B) ECG post PPI showing A sensed V paced rhythm

C) Apical 4 chamber showing apical ballooning with akinesia of mid and apical LV

D) Apical four chamber view showing moderate mitral regurgitation

Case #2

A 73-year-old hypertensive woman presented with exertional dyspnea of 2 weeks' duration and syncope on the admission day (Fig. 2A: Panel 2). ECG was suggestive of CHB. The patient had an elevated troponin-1 level (219 ng/L). A 2D echocardiogram showed hypokinesis in the anterior, apical (Fig. 2B: Panel 2), anterolateral, and lateral walls with hyperkinesia in the inferior and basal septa with an LV ejection fraction of 30% and moderate mitral regurgitation (Fig. 2C: Panel 2). A working diagnosis of non-STEMI with CHB was made. A coronary angiogram showed unobstructed coronaries. She was managed conservatively with a temporary pacemaker and supportive treatment. Subsequently, her LV function improved to 52%. Nevertheless, since her CHB persisted, the patient underwent dual-chamber pacemaker implantation. She was discharged in hemodynamically stable condition (Fig. 2D: Panel 2). At a 6-month follow-up, she had a pacing requirement of 98% with an LV ejection fraction of 55%.



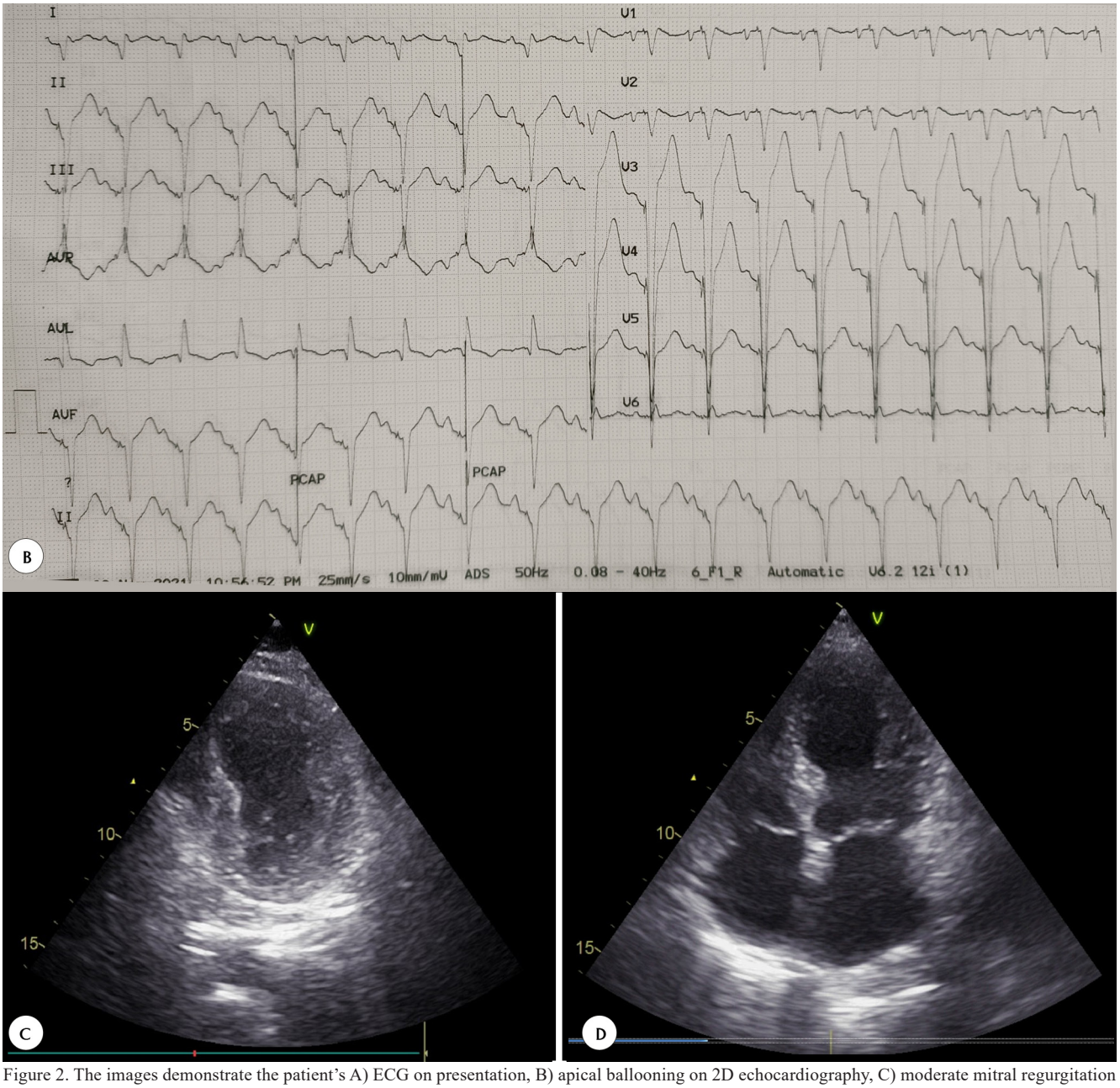


Figure 2. The images demonstrate the patient's A) ECG on presentation, B) apical ballooning on 2D echocardiography, C) moderate mitral regurgitation on echocardiography, and D) ECG after permanent pacemaker implantation.

Discussion

TTCM was first described in Japan by Sato et al[3] in 1991 and is most commonly seen in post-menopausal women. The prevalence of life-threatening arrhythmias in patients with TTCM is estimated at 13.5%. TTCM is most commonly associated with a prolonged QTc and includes monomorphic ventricular tachycardia (4.5%), polymorphic ventricular tachycardia (2.8%), asystole (2.8%), ventricular fibrillation (2.2%), and complete AV block (2.2%).[4] Mortality 1 year after the index event is 44% in patients with arrhythmias compared with those without arrhythmias (10%).[3]

The principal pathophysiologic mechanism postulated to be responsible for TTCM is increased catecholamine levels. The elevation stimulates β-adrenergic receptors from Gs to Gi, which causes negative inotropy and LV dysfunction by "stimulus trafficking." This process explains the apical form of this syndrome since β receptors are maximally concentrated at the apex.[4]

Multivessel coronary artery spasms causing ischemia, particularly due to the involvement of the right coronary artery, may induce high-grade AV block. Ischemia and fibrosis of the conduction system may be responsible

for persistent AV block despite the resolution of LV dysfunction.[5]

Presently, there is no confirmed cause-effect relationship between high-grade AV block and TTCM. There have been reports of 24 patients in whom AV node conduction abnormality was present on admission and persisted even after the resolution of LV dysfunction. Twenty-one of them required permanent pacemaker implantation. Bradycardia causing excess adrenergic responses and consequent pathophysiologic alterations of TTCM seem likely.[6]

The coexistence of CHB with TTCM poses a therapeutic dilemma between dual-chamber pacemaker implantation and cardiac resynchronization therapy with defibrillators. However, LV dysfunction is transient and reversible in TTCM. The conduction disturbance is not known to improve despite the restoration of LV function, with follow-up studies showing a pacing requirement of up to 99%.[7] A study on the role of defibrillators in TTCM documented no tachyarrhythmias on long-term follow-up. In that study, defibrillators were not found necessary, and the long-term prognosis concerning tachyarrhythmias was similar between patients with TTCM and those with acute coronary syndromes.[8]

Thus, in view of the likelihood of pacing requirements for bradyarrhythmias, dual-chamber pacemaker implantation is appropriate in the setting of TTCM with CHB.

Conclusion

Reports of associations between arrhythmias and TTCM are increasing. Arrhythmias may constitute a substrate for the development of cardiomyopathy, or they may occur as a result of LV dysfunction and QTc prolongation seen primarily. CHB is a rare presentation and usually requires pacemaker implantation.

References

1. Sanchez-Jimenez EF. Initial clinical presentation of Takotsubo cardiomyopathy with-a focus on electrocardiographic changes: A literature review of cases. World J Cardiol 2013;5:228-241.
2. Ramphul K, Mejias SG, Sombans S, Sonaye R, Joynauth J. Cardiac arrhythmias associated with Takotsubo cardiomyopathy and ST- segment Elevation Myocardial Infarction (STEMI). Am J Cardiol 2020;127:195.
3. Dote K, Sato H, Tateishi H, Uchida T, Ishihara M. Myocardial stunning due to simultaneous multivessel coronary spasms: a review of 5 cases. J Cardiol 1991;21:203-214.
4. Stiermaier T, Eitel C, Denef S, Desch S, Schuler G, Thiele H, Eitel I. Prevalence and Clinical Significance of Life-Threatening Arrhythmias in Takotsubo Cardiomyopathy. J Am Coll Cardiol 2015;65:2148-2150.
5. Limm BN, Hoo AC, Azuma SS. Variable conduction system disorders in takotsubo cardiomyopathy: a case series. Hawaii J Med Public Health 2014;73:148-151.
6. Korantzopoulos P, Nikas DN, Letsas K, Gkirdis I, Kolios MD, Ntalas IV, Goudevenos JA. High-Grade Atrioventricular Block and Takotsubo Cardiomyopathy: Case Report and Review of the Literature. Int Cardio Res J 2015;9:e11407.
7. Afzal A, Watson J, Choi JW, Schussler JM, Assar MD. Takotsubo cardiomyopathy in the setting of complete heart block. Proc (Bayl Univ Med Cent) 2018;31:502-505.
8. El-Battrawy I, Erath JW, Lang S, Ansari U, Behnes M, Gietzen T, Zhou X, Borggrefe M, Akin I. Takotsubo syndrome and cardiac implantable electronic device therapy. Sci Rep 2019;9:16559.