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Case Report

A Case of Takotsubo Cardiomyopathy with Parkinson's and Possible Heroin Abuse

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ABSTRACT

Takotsubo cardiomyopathy, in essence, is a reversible abnormality of the heart muscle resembling myocardial infarction in the absence of any disease of the coronary artery. A middle-aged man with medical history of Parkinson's disease (PD) and substance abuse with heroin presents with chest pain. Apart from high blood pressure, he was haemodynamically stable. His troponins were elevated with a positive delta but had no ischemic changes on his electrocardiograms. He was treated as a non ST-segment elevated myocardial infarction (NSTEMI) and started on appropriate medications. His ejection fraction was found to be 35% and he had a large territory of the left ventricle apical akinesis. He was subsequently taken for a coronary angiogram which did not reveal any obstruction in his coronary arteries. This was deemed to be takotsubo cardiomyopathy and he was managed on Guideline Directed Medical Therapy. He was eventually discharged without any symptoms. Years of data have helped us gain insight into the etio-pathogenesis of this novel and rare condition, and although literature is limited, this case paves a path toward broadening our understanding about PD, coronary microvascular disease and takotsubo cardiomyopathy and their inter-connection.

Keywords: Takotsubo cardiomyopathy, Parkinson's disease, Microvascular dysfunction, Heroin, Heart failure

INTRODUCTION

Takotsubo cardiomyopathy (TC) is defined by a temporary and reversible systolic abnormality of the left ventricle's apical area resembling myocardial infarction (MI) in the nonexistence of coronary artery disease.

The pathophysiology is still not completely known, but a sudden catecholamine surge either from extreme stress or a tumour secreting the chemicals is thought to be a major factor. About 2% of all acute coronary syndrome presentations are attributable to this, especially in postmenopausal women.[1]

Here, we describe a case of a middle-aged man with Parkinson's disease (PD) and illicit drug abuse, without prior cardiac conditions, with acute onset chest pain consistent with acute coronary syndrome, which was later attributed to TC.

CASE REPORT

A 56-year-old man with medical history notable for PD, polysubstance abuse, with relapse into heroin since the past 2 years came in with chest pain which started after he used heroin a few hours prior. Apart from elevated blood pressure of around 170/100 mmHg, he was haemodynamically stable on room air. His troponins were elevated initially at 364 pg/mL followed by 632 pg/mL at 1 h with a positive delta of 268, but had no ischemic changes on his serial electrocardiograms. He was treated as non ST-segment elevated myocardial infarction and given aspirin followed by a heparin drip. His transthoracic echocardiogram revealed ejection fraction of 35% and large territory of the left anterior descending territory akinesis as seen in Figure 1. He was subsequently taken for a coronary angiogram which did not reveal any obstruction in his coronary arteries. Interestingly, his urine toxicology turned out to be negative for any drug although he admitted to using heroin just prior. This was deemed to be TC due to typical shape of heart on imaging and no blockages in his coronaries. He was started on carvedilol, dapagliflozin, sacubitril-valsartan and spironolactone for his reduced ejection fraction and elevated blood pressure. He was discharged in 12 days without any chest pain or elevated blood pressure.

DISCUSSION

This unique heart disease was originally described in 1990 in the Japanese population and was called 'Tako-Tsubo

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cardiomyopathy', named after the octopus trapping pot with a round bottom and narrow neck, which resembles the left ventriculogram during systole in these patients.[2]

According to Harmonizing Outcomes with Revascularization and Stents in Acute Myocardial Infarction (HORIZONS-AMI) trial, there is an average of 1–3% of acute coronary syndrome cases that can be labelled as being due to TC. This gives a rough idea of the population under consideration.

The revised Mayo Clinic criteria are widely used diagnostic criteria for TC. They are as follows:

- Transient dyskinesis of the left ventricular midsegments, with or without apical involvement; the regional wall-motion abnormalities extend beyond a single epicardial vascular distribution, and a stressful trigger is often, but not always, present
- Absence of obstructive coronary disease or absence of angiographic evidence of acute plaque rupture
- New electrocardiogram abnormalities (ST-segment elevation and/or T-wave inversion) or modest elevation in the cardiac troponin level
- Absence of pheochromocytoma and myocarditis.

The precise cause, pathogenesis and pathophysiology of TC are still uncertain. Recently, the most accepted theories are catecholamine-induced cardiotoxicity and microvascular dysfunction, alongside involvement of the cognitive centres of the brain and hypothalamic-pituitary-adrenal axis.

Literature has pointed to potential underdiagnosed microvascular disease in TC, but the cause or effect analogy with respect to the acute TC episode is yet to be determined. Microcirculatory dysfunction is transient, and its resolution correlates with improved cardiac function. This suggests that the progression of TC is probably linked with changes in coronary microcirculation.

Dysfunctional left ventricular segments in patients with TC have a lower blood flow compared with those segments showing normal wall motion in the acute phase. Other non-invasive modalities have also shown similar results, prompting some investigators to relate coronary microvascular dysfunction to the pathogenesis of TC.[3] Myocardial oedema secondary to intramyocardial catecholamine inflammatory process may occur in some patients with TC, leading to compression of the microvascular system causing dysfunction.[4]

Naturally, there are some studies that talk against the microvascular theory. Despite ST-elevation MI (STEMI)-like EKG changes in more than 30% of patients with TC who usually have extensive left ventricle wall motion abnormalities, they usually have only modest troponin elevation.[5] This would go against ischemia, causing STEMI-like EKG changes, induced by microvascular dysfunction as a cause of TC.

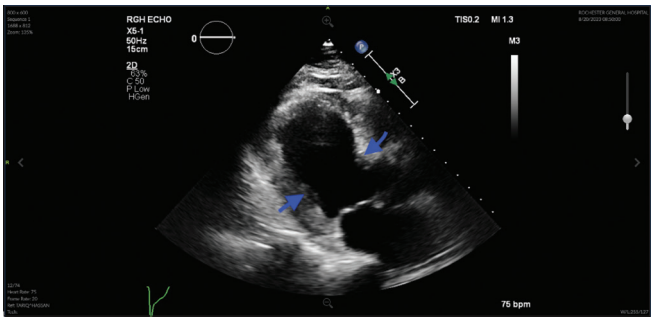

Figure 1: During systole, left ventricle showing apical ballooning/akinesis in left anterior descending (LAD) territory. The blue arrows signify normally constricting myocardium and the apex seems to be ballooning above it. This is an image of the echocardiogram of the patient.

Systematic reviews show that about two-third of drug-induced TCare due to direct or indirect catecholamine stimulation.[6] There are case reports talking about opioid withdrawal complicating hospital stays with TC.[7] However, there are no reports of heroin abuse leading to TC. Heroin is an opioid, which is a Central Nervous System depressant and should theoretically not cause TC as there would be no catecholamine surge.

The case that we present is unique in the following facts.

- The patient is a male and only around 10% of all TC cases are seen in males.[8]
- The patient admitted to using heroin since the last couple of years and was abusing it almost every night to help him sleep. He was found to be a case of NSTEMI but the coronary arteries were normal and the transthoracic echocardiogram demonstrated apical ballooning and akinesis. His urine toxicology turned out to be negative for any drug although he admitted to using heroin just prior. He was not in withdrawal as well when he started having chest pain or when he was diagnosed with TC a few hours later. There is some evidence of cardio-protective effect of opioid through opioid receptors in the myocardium. Differential activation may be a hypothesis where in heroin could cause damage.
- The confounding factors that may have contributed to his TC may be his insomnia and underlying PD and anxiety/depression. In a prospective study, the prevalence of depression/anxiety was 78%, much higher than in patients with acute coronary syndrome (ACS).[9]
- In PD, there is depletion of dopamine in the CNS, which is the substrate to make catecholamines. Moreover, there is a loss of noradrenergic neurons at the cardiac level, this may account for the rarity of association.

Of note, there may be a correlation between microvascular dysfunction and PD. There is a case report of a patient with PD having TC without any known emotional trigger.[10]

CONCLUSION

TC as a condition may be self-limiting and reversible in a way but can still pose substantial health risks. Yet, we do not have all the answers to the cause and prevention of this disease. Substantial literature over the past decade has hinted toward microvascular dysfunction as one of the main of pathogenesis of TC. The triggers for this dysfunction are numerous and although the data are limited, there may be a relationship between PD and microvasculature in the coronary arteries as well, which can be explored further. Moreover, the suspected heroin use could have been another factor adding to the microvascular maladaptation. Hence, this case can add to the deficiency in our understanding of microvascular disease and TC.

Ethical approval

Institutional Review Board approval is not required.

Declaration of patient consent

Patient's consent is not required as patients identity is not disclosed or compromised.

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Conflicts of interest

There are no conflicts of interest.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

REFERENCES

1. Akashi YJ, Nef HM, Lyon AR. Epidemiology and Pathophysiology of Takotsubo Syndrome. Nat Rev Cardiol 2015;12:387-97.
2. Kurisu S, Sato H, Kawagoe T, Ishihara M, Shimatani Y, Nishioka K, *et al*. Tako-tsubo-like Left Ventricular Dysfunction with ST-segment Elevation: A Novel Cardiac Syndrome Mimicking Acute Myocardial Infarction. Am Heart J 2002;143:448-55.
3. Abdelmoneim SS, Mankad SV, Bernier M, Dhoble A, Hagen ME, Ness SA, *et al*. Microvascular Function in Takotsubo Cardiomyopathy with Contrast Echocardiography: Prospective Evaluation and Review of Literature. J Am Soc Echocardiogr 2009;22:1249-55.
4. Eitel I, von Knobelsdorff-Brenkenhoff F, Bernhardt P, Carbone I, Muellerleile K, Aldrovandi A, *et al*. Clinical Characteristics and Cardiovascular Magnetic Resonance Findings in Stress (Takotsubo) Cardiomyopathy. JAMA 2011;306:277-86.
5. Montone RA, Galiuto L, Meucci MC, Del Buono MG, Vergni F, Camilli M, *et al*. Coronary Slow Flow is Associated with a Worse Clinical Outcome in Patients with Takotsubo Syndrome. Heart 2020;106:923-30.
6. Kido K, Guglin M. Drug-induced Takotsubo Cardiomyopathy. J Cardiovasc Pharmacol Ther 2017;22:552-63.
7. Launer H, Nelson D, Dietzen A, Singla A. Can Breaking Heroin Addiction Lead to a Broken Heart? A Case of Reverse Takotsubo Cardiomyopathy in a Patient with Heroin Withdrawal. Tex Heart Inst J 2023;50:e217604.
8. Arcari L, Núñez Gil IJ, Stiermaier T, El-Battrawy I, Guerra F, Novo G, *et al*. Gender Differences in Takotsubo Syndrome. J Am Coll Cardiol 2022;79:2085-93.
9. Ghadri JR, Wittstein IS, Prasad A, Sharkey S, Dote K, Akashi YJ, *et al*. International Expert Consensus Document on Takotsubo Syndrome (Part I): Clinical Characteristics, Diagnostic Criteria, and Pathophysiology. Eur Heart J 2018;39:2032-46.
10. Lazaros G, Dimitriadis K, Xanthopoulou M, Latsios G, Antoniou C, Lazarou E, *et al*. Takotsubo Cardiomyopathy and Parkinson's Disease: An Exceptionally Uncommon Clinical Duet. Hellenic J Cardiol 2019;60:334-5.

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