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SPOTLIGHT

Takotsubo cardiomyopathy: A case report with severe electrolyte abnormality

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65 years old female admitted via primary percutaneous coronary intervention pathway for ST elevation myocardial infarction (Figure 1). Coronary angiograms showed normal coronary arteries (Figure 4) and left ventriculogram revealed apical and mid ventricular hypokinesia and hyperkinetic base, Takotsubo pattern (Figure 3). Troponin T was 2021 ng/mL. Simultaneously, she was found to have severe hypocalcemia (adjusted calcium 1.85 mg/dL) and hypomagnesaemia (<0.20 mmol/L) likely secondary to omeprazole, proton pump inhibitor (PPI). Other causes of electrolyte imbalance in her case were presumably due to nausea, vomiting, and stop taking her regular calcium supplements.

She was treated with intravenous magnesium, calcium replacement, and omeprazole was stopped. After intravenous calcium and magnesium replacement therapy, electrolytes have risen up to adjusted calcium 2.10 mg/dL and magnesium 0.46 mmol/L when electrolytes replacement was continued by oral calcium and magnesium supplements. Following this, the symptom of tetany was resolved.

Left ventricle (LV) was severely impaired on initial echocardiogram (Figure 2), then improved on follow-up cardiac MRI (Video S1) which also showed absence of scar on gadolinium delay enhancement images. She was treated with angiotensin-converting enzyme inhibitor (ACE-I) and ß-blocker then transferred to local district general hospital and subsequently discharged home from there.

Takotsubo cardiomyopathy (TC) was first described in Japan in 1990.[1] It is an increasingly recognized cardiac syndrome with a prevalence of 1.7–2.2% among suspected acute coronary syndrome (ACS) patients.[2] TC mostly affects postmenopausal women with presentation similar to ACS. TC is a reversible cardiomyopathy with a morphological feature of transient LV apical ballooning. Other variants are mid ventricular, basal and focal variants however the apical variant is the most reported one.

The exact pathogenesis remains unclear but catecholamine plays an essential role. Most hypotheses suggest multifactorial etiologies including vascular abnormalities, estrogen deficiency, both autonomic and central nervous system abnormalities and genetic factors.

The association between electrolyte abnormalities and TC has still not been well established. It is worth noting that electrolyte imbalances, such as hypomagnesemia, may trigger microvascular dysfunction, which is believed to underlie TC. In addition, electrolyte disorders may contribute to cardiac impairment, similar to what happens with hypocalcemia. Furthermore, a neurohormonal stress response may be triggered by an electrical abnormality, while heart failure could be caused by a neurovegetative mechanism.[3]

The hypothesis of PPI-related hypomagnesaemia involves inhibition of intestinal magnesium absorption. Hypomagnesemia leads to development of mitochondrial alterations leading to myocardiocyte death and hypercoagulability. Moreover, it directly provokes myocardial vasodilation, indirectly affecting cardiac contractility.[3]

TC often has triggering factors such as physical and emotional triggers but not universally. The absence of a trigger should not exclude the diagnosis.

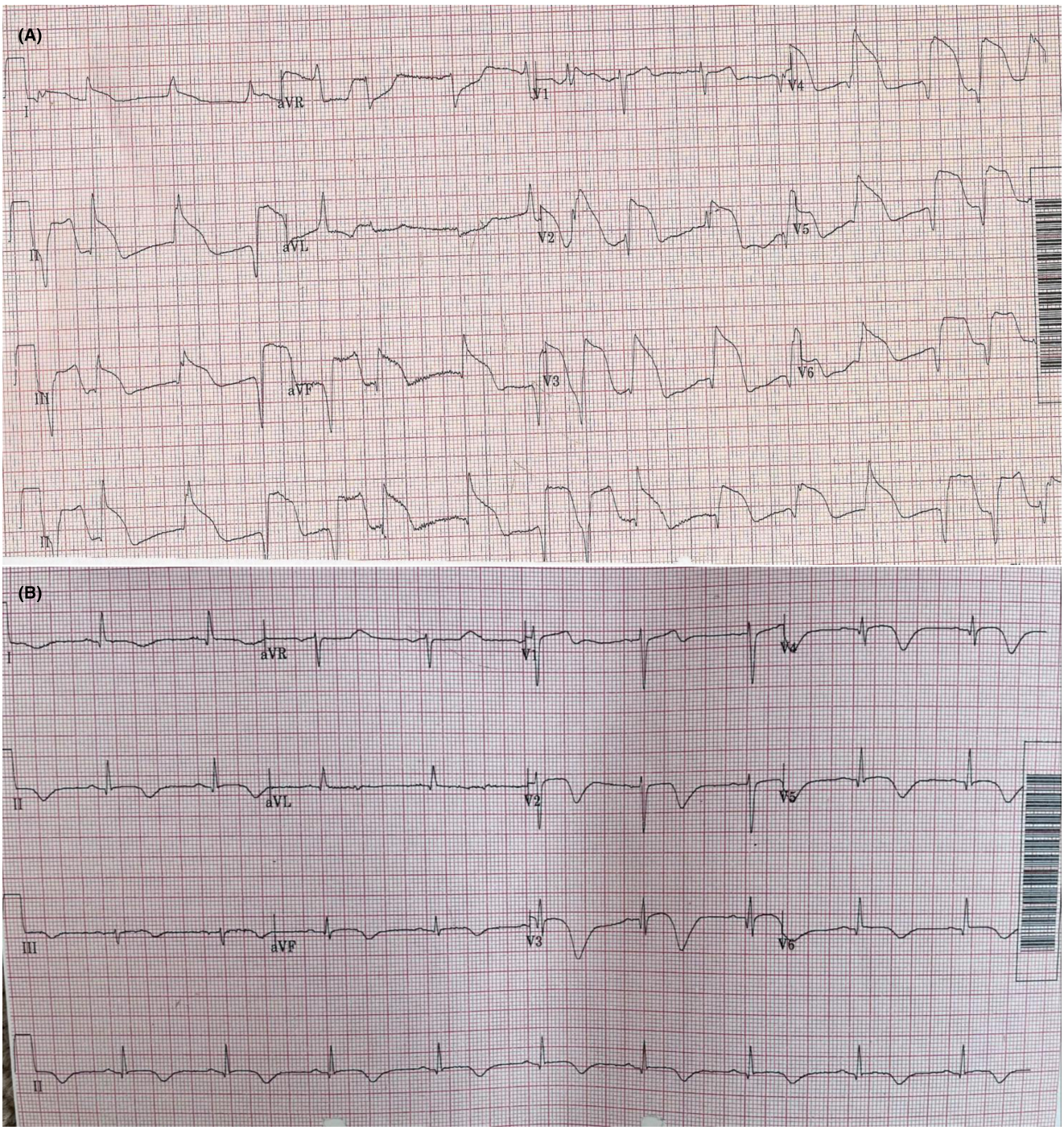

FIGURE 1 (A) ECG on admission showing an escape rhythm with widespread ST elevation and frequent ectopics. (B) ECG before discharge showing prolong QT (560 s) with widespread deep T wave inversion.

2008 modified Mayo Clinic diagnostic criteria consists (1) temporary wall motion abnormalities in LV segments with or without apical involvement; (2) the lack of significant coronary artery disease; (3) recent changes detected in the electrocardiogram (ECG) (ST-segment elevation and/or T-wave inversion) or significant elevation of serum cardiac troponins; and (4) nonexistence of pheochromocytoma or myocarditis.[4]

Imaging usually shows transient LV systolic impairment. Coronary angiography is normal or nonsignificant atherosclerosis. Cardiac magnetic resonance imaging (CMRI) helps to exclude other

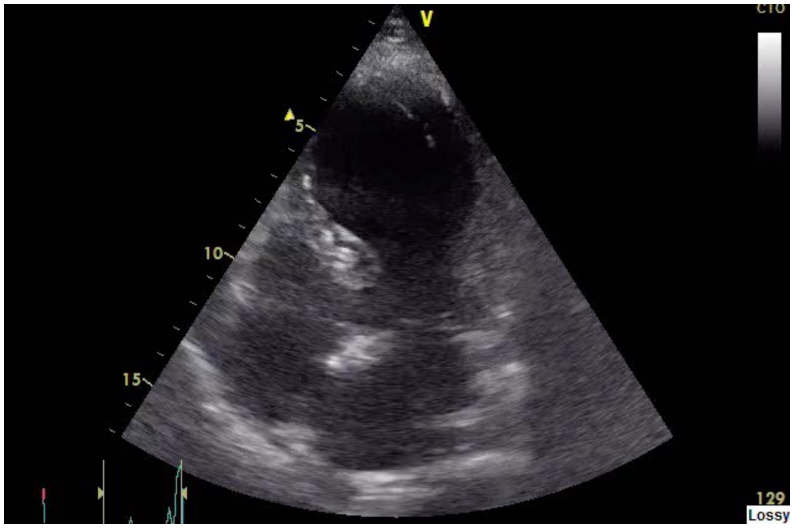

FIGURE 2 Transthoracic echocardiogram (Apical four chambers view) showing apical ballooning of left ventricle.

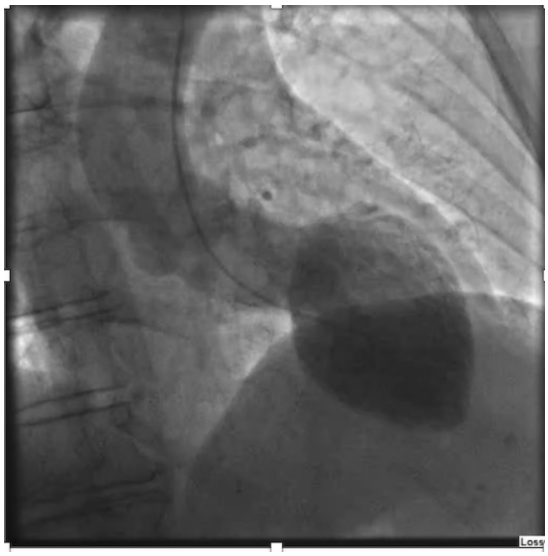

FIGURE 3 Left ventriculogram showing apical ballooning of left ventricle.

pathologies like myocarditis and MINOCA (Myocardial Infarction with no obstructive coronary arteries).

The management should focus on treatment of ACS at the first instance, ß-blocker and ACE-I or angiotensin receptor blocker in addition to supportive treatment.

The annual rate of long-term mortality was 3.5% and that of recurrence was 1%.[5] Older age, physical stressor and apical ballooning were significantly associated with an unfavorable long-term prognosis. Almost all patients with TC have favorable prognosis. LV function may recover in several days and fully recuperates in 3–4 weeks.

We reported a case of TC associated with severe electrolytes derangement. It was presented and initially managed as ACS then TC was subsequently identified. This highlights the importance of suspecting the disease to correctly identify, investigate, and manage it. This case also expresses an educational point regard to the importance of controlling unnecessary long-term prescribing of PPI and of regular testing of serum electrolyte concentrations.

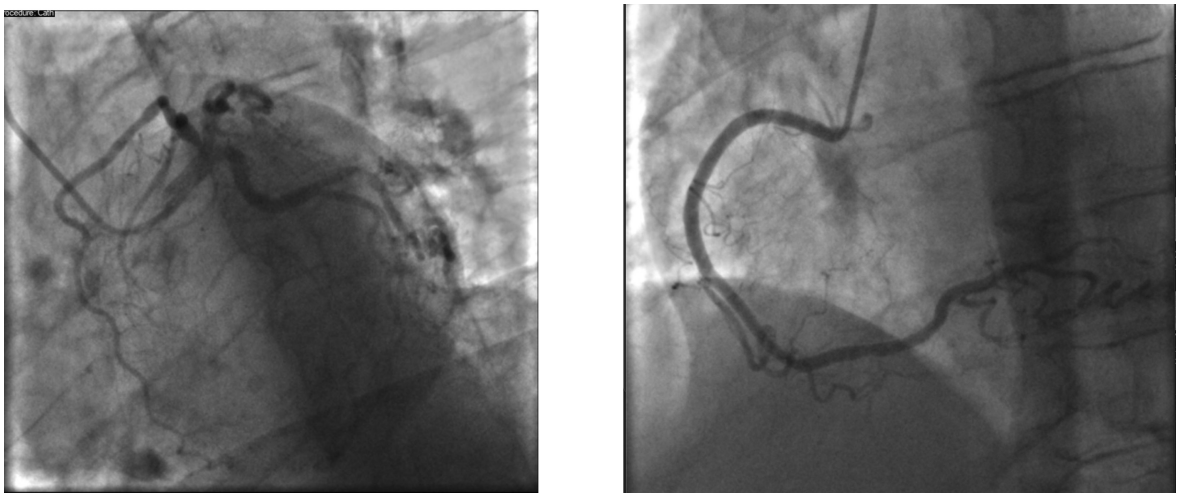

FIGURE 4 Left coronary angiogram (left) and Right coronary angiogram (right) showing normal coronary arteries.

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CONFLICT OF INTEREST STATEMENT

N/A

ETHICS APPROVAL STATEMENT

N/A

PATIENT CONSENT STATEMENT

Obtained.

CLINICAL TRIAL REGISTRATION

N/A

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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