Clinical Case Report

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Takotsubo cardiomyopathy and giant r wave syndrome mimicking acute myocardial infarction

A case report

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Abstract

Rationale: The clinical features of Takotsubo cardiomyopathy largely overlap with those of acute myocardial infarction, especially in the presence of ST-segment elevation on the initial electrocardiogram. Giant R wave syndrome has mainly been observed in the hyperacute phase of acute myocardial infarction.

Patient concerns: In this study, we report a unique case of Takotsubo cardiomyopathy that caused giant R wave syndrome.

Diagnosis: A 71-year-old woman was transferred to hospital with new onset chest pain. An initial electrocardiogram showed ST-segment elevation in the inferior wall and anterior wall leads. Her initial cardiac troponin I levels were elevated. Acute myocardial infarction was suspected and the patient underwent emergent cardiac catheterization. A coronary angiography showed no overt stenosis in the coronary artery. After 2 hours, her chest pain disappeared and an electrocardiogram revealed that the ST segment had decreased markedly. However, on day 3, an electrocardiogram of the V1–V6 leads revealed the formation of giant R wave syndrome: giant R waves merging with the markedly elevated ST segments and the obliteration of S waves. Cardiac echocardiography showed hypokinetic apical mid-segments and hyperkinetic basal segments of the left ventricle, low left ventricular ejection (42%), and enlargement of the left ventricle. On the basis of these findings, the patient was diagnosed with early recurrent Takotsubo cardiomyopathy.

Interventions: The patient has been treated by levosimendan and furosemide to improve cardiac function before leaving the hospital. After discharge, she was treated with a beta blocker.

Outcomes: The patient was discharged 2 weeks later in stable condition without chest pain. One year later, during her follow-up, a repeat echocardiogram and ECG showed normal findings.

Lessons: To the best of our knowledge, this is the first report of giant R wave syndrome on electrocardiogram following Takotsubo cardiomyopathy. Takotsubo cardiomyopathy, especially presenting with giant R wave syndrome on electrocardiogram, remains a challenging condition given its similarity to acute myocardial infarction in its early phase.

Abbreviations: AMI = acute myocardial infarction, BNP = brain natriuretic peptide, ECG = electrocardiogram, GRWs = giant R wave syndrome, TTC = Takotsubo cardiomyopathy.

Keywords: acute myocardial infarction, case report, electrocardiogram, giant R wave syndrome, Takotsubo cardiomyopathy

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The publication of this case report was in accordance with the Declaration of Helsinki and was approved by the ethics committee of Affiliated Hospital of Shandong University of Traditional Chinese Medicine (ethical approval number: 2013-259-28-01).

Written informed consent for the publication of the manuscript and any accompanying images was obtained from the patient in this case report.

Availability of data and material: All data are available in the manuscript.

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1. Introduction

Takotsubo cardiomyopathy (TTC), also known as stress cardiomyopathy, is characterized by transient regional systolic and diastolic dysfunction on the apical and mid-segments of the left ventricle and hyperkinesia of the basal left ventricular segments without obstructive coronary artery lesions.[1] The clinical features of TTC largely overlap with those of acute myocardial infarction (AMI) including chest pain, modestly elevated cardiac troponin, and electrocardiogram (ECG) changes, especially in the presence of ST-segment elevation on the initial ECG .[2]

Giant R wave syndrome (GRWS) is characterized by the appearance of a giant R wave, loss of the S wave, and merging of the QRS complex with the ST segment, causing a monophasic QRS-ST complex. This ECG pattern mimics a bundle branch block or ventricular tachycardia, especially when the rapid ventricular rate obscures the P wave. Such transient GRWS has mainly been observed following the hyperacute phase of myocardial infarction, occasionally following variant angina, and after percutaneous transluminal coronary angioplasty or experimental coronary artery ligation .[3]

Herein we present an unusual case of TTC that caused GRWS and had a very early recurrence.

2. Case presentation

A 71-year-old woman was transferred to the emergency department with new onset chest pain for 9 hours. Her past medical history included hypertension, diabetes, and gall stones. Her regular medications included levamlodipine besylate 5 mg and metformin hydrochloride 1.0 g. She was a nonsmoker with no alcohol consumption. An ECG before admission was normal. Upon the patient's arrival, her blood pressure was 90/65 mm Hg and pulse rate and respiratory rate were 120 and 30 per minute, respectively. The initial ECG conducted at the emergency department showed ST-segment elevation in the II, III, aVF, and V1–V5 leads; slight ST-segment depression in the aVR lead; a prolonged QT interval with QTc of 470 ms; and premature ventricular beats (Fig. 1A). Her initial cardiac troponin I(c-TnI) and brain natriuretic peptide (BNP) levels were elevated: c-TnI was 2.1 ng/mL (normal level <0.03 ng/mL) and BNP was 273 pg/mL (normal range 0–100 pg/mL). In view of her history of hypertension and diabetes, AMI was suspected. After receiving a load dosage of aspirin (300 mg) and ticagrelor (180 mg), the patient underwent emergent cardiac catheterization. A coronary angiography showed no evidence of coronary artery disease (Fig. 2A). After 2 hours, her chest pain disappeared and an ECG revealed ST-segment elevation had fallen slightly to 50% in the

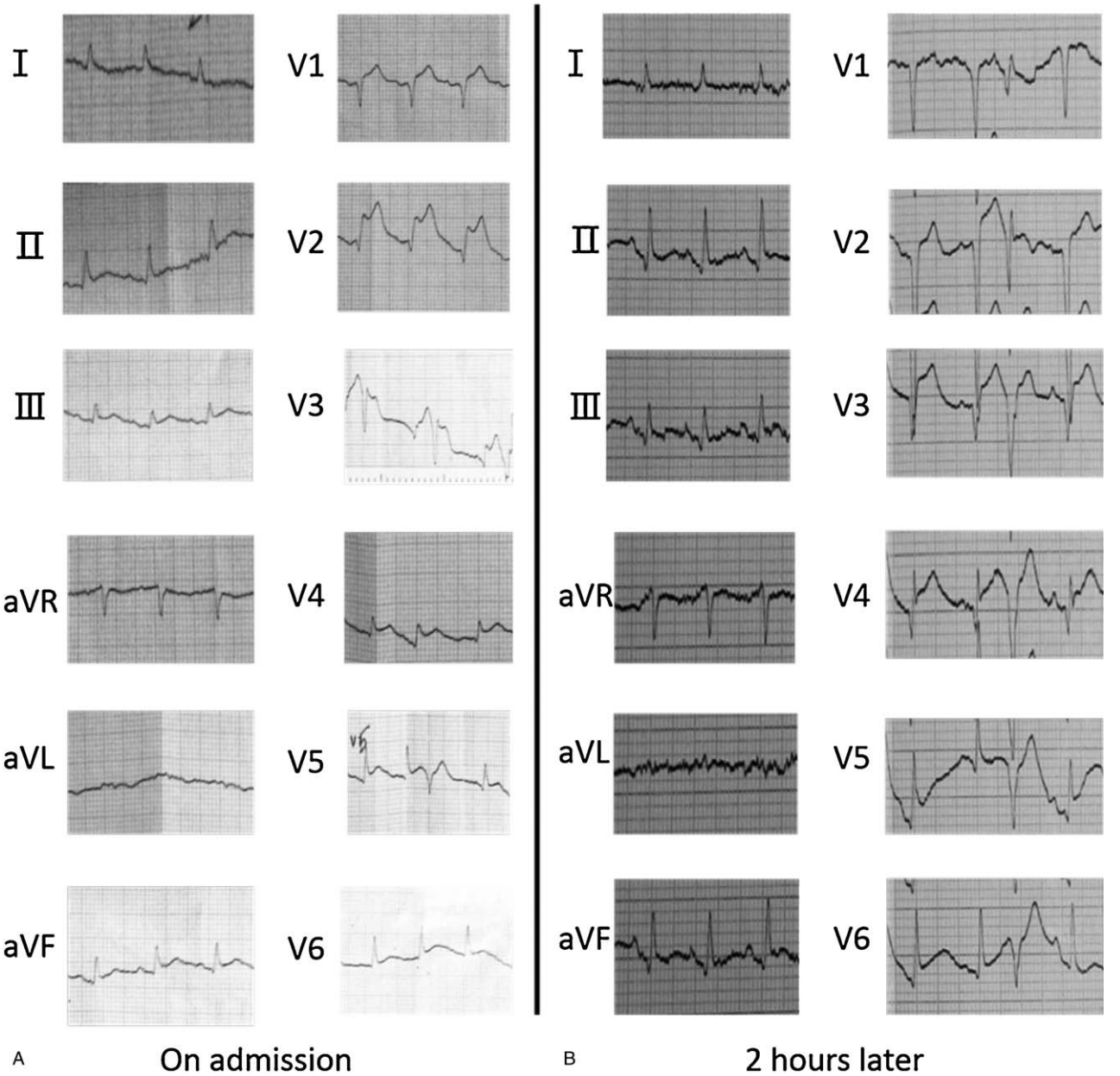

Figure 1. (A) Initial electrocardiogram of the patient at admission time ST-segment elevation in the II, III, aVF, and V1–V5 leads; slight ST-segment depression in the aVR lead; and a prolonged QT interval. (B) ECG recorded approximately 2 hours after first ECG showing that ST-segment elevation had fallen slightly, to >50% in the V1–V2 lead.

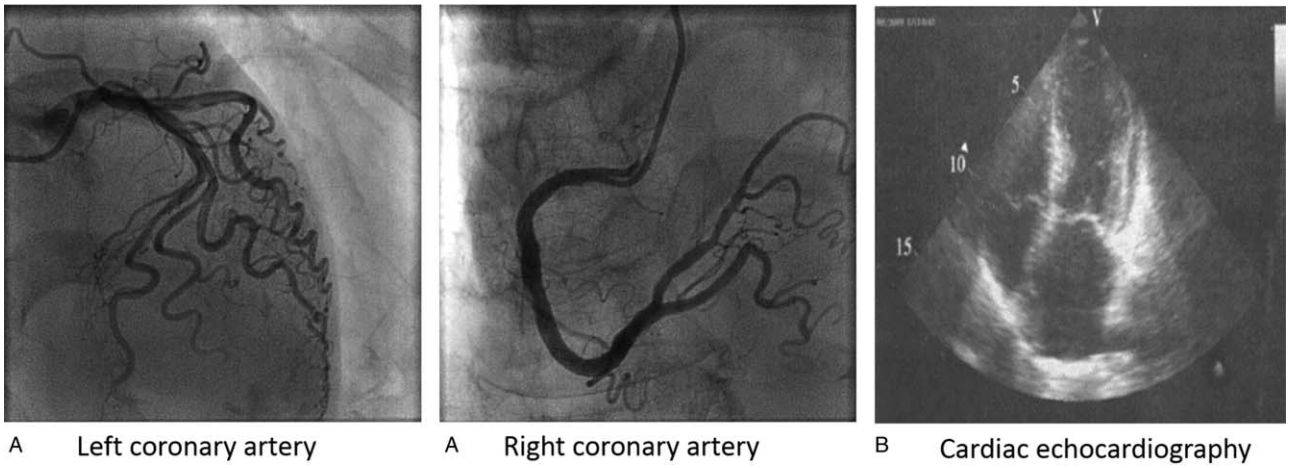

Figure 2. (A) Coronary angiography showing no obvious coronary artery stenosis. (B) Cardiac echocardiography showing hypokinetic apical mid-segments and hyperkinetic basal segments of the left ventricle.

V1–V2 leads (Fig. 1B). Thrombus autolysis was suspected, and conventional AMI therapy was initiated.

However, on day 3, an ECG (Fig. 3A) revealed ST-segment elevations in leads II, III, aVF, and V1-V6 and ST-segment depressions in lead aVR without obvious chest pain. A Q wave was noted in leads V1–V2, with Q waves inscribed in leads II, III, aVF, and V3–V6. The ECG pattern in the V1–V6 leads also revealed giant R waves merging with markedly elevated ST segments, the obliteration of S waves, and the formation of monophasic QRS-ST complexes. Physical examination revealed that her blood pressure was 105/65 mm Hg, her pulse rate was 95 beats/min,and respiratory rate was 22/min. As the first cardiac catheterization showed no coronary artery occlusion, her family refused to repeat the coronary angiography. A bedside cardiac echocardiography showed hypokinetic apical mid-segments and hyperkinetic basal segments of the left ventricle with left ventricular ejection of 42% (Fig. 2B). Laboratory examinations revealed increased levels of c-TnI (4.11 ng/mL) and creatine phosphokinase (333.30 U/L). Her BNP level rose markedly to 1630 pg/mL. Physical examination indicated: oedema of both legs, lung moist rale, and cold extremities. Cardiac insufficiency and mild organ hypoperfusion were considered. On the basis of these findings, the patient was diagnosed with very early recurrent Takotsubo cardiomyopathy. Levosimendan (0.1 μg/kg min) and furosemide were administered to improve cardiac function.

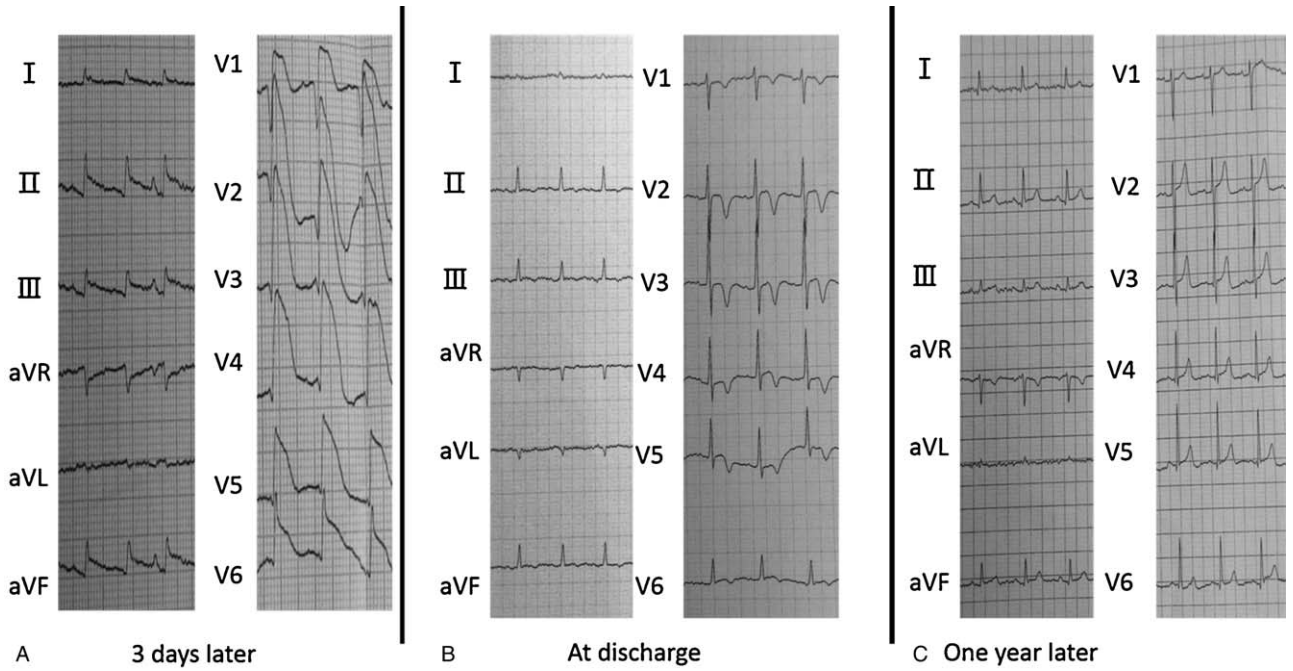

Figure 3. (A) Electrocardiogram on the third day of admission revealing giant R waves merging with the markedly elevated ST segments. (B) Electrocardiogram at discharge revealing T wave inversion over leads V1–V6 and no Q waves over leads II, III, aVF, and V1-V6.C. ECG recorded one year after discharge showing no obvious abnormalities.

Subsequent progress was satisfactory: the levosimendan and furosemide were withdrawn. The patient was discharged 2 weeks later in stable condition without chest pain. An ECG at discharge showed T wave inversion over leads V1-V6 and no Q waves over leads II, III, aVF, and V1–V6 (Fig. 3B). Her BNP level had fallen to 450 pg/mL, and her c-TnI level was in the normal range. The patient was subsequently treated with a beta blocker. One year later, during her follow-up, a repeat echocardiogram and ECG showed normal findings (Fig. 3C).

3. Discussion and conclusion

TTC mainly occurs in elderly women and is often triggered by emotional and/or physical stress, but 28.5% of TTC patients have no evident trigger .[1] The clinical scenario of TTC mimics that of AMI, and approximately 2% of all patients presenting with apparent symptoms of AMI might in fact be diagnosed with TTC .[4] Diagnostic criteria were proposed at the Mayo Clinic 2004 and were modified in 2008 .[5] Several mechanisms, including multivessel coronary artery spasm, coronary microvascular dysfunction, and catecholamine-induced cardiac toxicity, have been proposed to explain the pathophysiology, but the precise mechanism remains unclear .[5] Although patient in this report had no evident emotional and/or physical stress triggers, this case fulfilled the criteria. The entire course was well documented by ECG abnormalities, absence of obstructive coronary disease, modest elevation of cardiac troponin I, marked elevation of BNP, hypokinetic apical mid-segments, and hyperkinetic basal segments of the left ventricle with left ventricular ejection of 42%.

Although cardiac catheterization is very important for the differential diagnosis of TTC and AMI, the initial and subsequent features of ECG in TTC are of potential clinical importance with respect to diagnosis, especially in patients in whom urgent coronary angiography may be deferred because of clinical instability. ECG changes more frequently and typically seen in TTC include the absence of abnormal Q waves or transient Q waves, ST-segment elevation/T wave inversion in multiple leads extending beyond the perfusion territory of any single coronary artery, lack of ST-segment depression in the reciprocal leads, absence of ST-segment elevation in lead V1, QTc prolongation, and deep T-wave inversions in later stages .[6] Antonio et al. demonstrated that when ST depression in aVR is accompanied by ST elevation in the anteroseptal leads (V1-V3), the diagnostic sensitivity and specificity of TTC was 12% and 100%, respectively ($P<.001$) .[2] As in our patient, ECG changes can be divided into 4 phases: initial ST-segment elevation in the II, III, aVF, and V1–V5 leads extending beyond the perfusion territory of any single coronary artery and slight ST-segment depression in the aVR lead followed by decreasing ST-segment elevation in the V1–V3 leads then giant R waves in the precordial leads and deeper ST-segment depression in the lead aVR, indicating recurrence, and finally giant T wave inversion and the absence of Q waves.

GRWS was first reported in 1960 in the context of variant angina. GRWS mainly occurs in the very early stage of AMI and disappears shortly after the inception of the ischemic insult. Therefore, its appearance may aid in identifying the very early phase of AMI. The electrophysiological mechanism of GRWS has been attributed to a focal intraventricular conduction block in the severe ischemia or myocardial infarction region caused by an ischemia-induced increase in extracellular K^+ with membrane depolarization, depletion of intracellular ATP, and action potential shortening. In this case report, we described a patient with GRWS who was ultimately diagnosed with TTC. Although ST-segment elevation occurs in approximately 44.7% of patients with TTC,[1] this was rare report of TTC displaying GRWS. Two possible mechanisms have been proposed to explain the ECG diffuse ST-segment elevation in TTC .[7] Some experts attributed these ECG changes to circumferential subepicardial ischemia caused by multivessel coronary artery spasm or microvascular dysfunction. Other experts assume that, aside from electrophysiologically mediated elevation, ST-segment elevation is mediated by the extensive mechanical stretching of the dyskinetic myocardium due to hyperkinetic basal contraction. As in this case, there was no identifiable epicardial coronary spasm, and the TIMI frame count, a method that can be used to detect coronary microvascular dysfunction, was in the normal range, so the possibility of circumferential subepicardial ischemia can be ruled out. It is more likely that GRWS in this patient was due to dyskinesia displayed by apical "ballooning." Apart from the excessive magnitude of ST-segment elevation on ECG, the patient in this study also exhibited persistent ST-segment elevation.

The recurrence of TTC is infrequent. Several studies reported an average yearly recurrence rate of TTC ranging from 1.8% to 2.9%, with a span of 25 days up to 10 years after the first event .[1] Xu et al[8] reported a patient with TTC who recurred in only 4 days, so very early recurrence is possible. As indicated by the dynamic changes on ECG, our patient had a very early recurrence. The current absence of proven medical treatment remains a significant constraint for the prevention of TTC.

In conclusion, we reported a unique case displaying GRWS on ECG following the occurrence of TTC. In the past, GRWS was always regarded as the typical ECG features of patients in the hyperacute phase of AMI. TTC that presents with GRWS on ECG remains challenging given its similarity to AMI in its early phase. Additionally, a very early recurrence was seen in this patient. The optimal diagnosis and pharmacologic management of this condition is not well established at present. Further studies are warranted.

Author contributions

Yong Wangand Jianliang Ma collected, analyzed, and interpreted the patient data and drafted the manuscript. Wei Guowere involved in direct patient care and percutaneous coronary intervention. Yong Wang, Jianliang Ma,and Wei Guo extracted the data and provided the clinical information. All of the authors read and approved the final manuscript.
Data curation: Yong Wang, Jianliang Ma.
Investigation: Yong Wang, Wei Guo, Jianliang Ma.
Methodology: Yong Wang.
Writing – original draft: Yong Wang.
Writing – review & editing: Yong Wang, Jianliang Ma.

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