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Recurrence of tako-tsubo syndrome, idiopathic dilated cardiomyopathy, and iterative ventricular tachycardia: just a fortuitous coincidence or a pathophysiological link?

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Abstract. – We report the case of a 65-year-old woman with late recurrence of tako-tsubo syndrome, idiopathic dilated cardiomyopathy and prior iterative ventricular tachycardia. We hypothesize that the pathophysiological link among these clinical conditions could be the hyperactivity of the sympathetic nervous system.

Key Words:
Dilated cardiomyopathy; Sympathetic hyperactivity; Tako-tsubo syndrome; Ventricular tachycardia.

Case Report

We report the case of a 65-year-old woman referred to our Hospital for urgent coronary angiography. In January 2013, she experienced a syncopal episode after emotional stress, preceded by intense sweating and precordial pain. On admission to the Emergency Department, her blood pressure was 80/60 mmHg, heart rate 75 bpm, oxygen saturation on air 93%. Chest auscultation revealed bilateral basal crackles. She presented with obesity, dyslipidemia, and a family history of coronary heart disease. She reported to have idiopathic dilated cardiomyopathy and, seven years before, to have experienced ventricular tachycardia, and six months after she was diagnosed with tako-tsubo syndrome (TTS). She was on treatment with ramipril, aspirin, furosemide and pravastatin.

The ECG showed sinus rhythm at 75 bpm, left anterior hemiblock, T-wave inversion in the II, III, aVF and V1-V6 leads, marked QT prolongation (600 ms) with a QTc interval using Bazett's formula of 671 ms (Figure 1). Echocardiography demonstrated mild left ventricular dilatation, left ventricular systolic dysfunction (ejection fraction 33%) with mid-apical akinesis.

The patient had already been treated with morphine, aspirin, i.v. heparin, clopidogrel, pantoprazole, furosemide, and atorvastatin before Emergency Department referral.

Coronary angiography revealed no significant stenosis in the epicardial coronary arteries, and ventricular angiography confirmed mild left ventricular dilatation with mid-apical akinesis. After the procedure, the patient was transferred to the referring hospital with the diagnosis of TTS recurrence.

Seven years previously (October 2006), the patient was admitted to another hospital and discharged with a diagnosis of TTS in the presence of idiopathic dilated cardiomyopathy with mild left ventricular systolic dysfunction. She was put on treatment with ramipril, furosemide, bisoprolol, aspirin, and lansoprazole. On admission, the ECG showed sinus rhythm at 69 bpm, left anterior hemiblock, T-wave inversion in the II, III, aVF and V1-V6 leads, QT prolongation (450 ms) with a QTc interval using Bazett's formula of 483 ms (Figure 2). Echocardiography demonstrated mild left ventricular dilatation with mid-apical akinesis (ejection fraction 40%). Coronary angiography was unremarkable.

Notably, six months previously (April 2006) the patient was urgently admitted to a private clinic for iterative ventricular tachycardia at 160 bpm with right bundle branch block morphology and right axis deviation (Figure 3). On that occasion, echocardiography showed mild left ventricular dilatation with mild global hypokinesis (ejection fraction 48%), coronary angiography did not reveal any significant coronary stenosis, and ventricular angiography confirmed mild left

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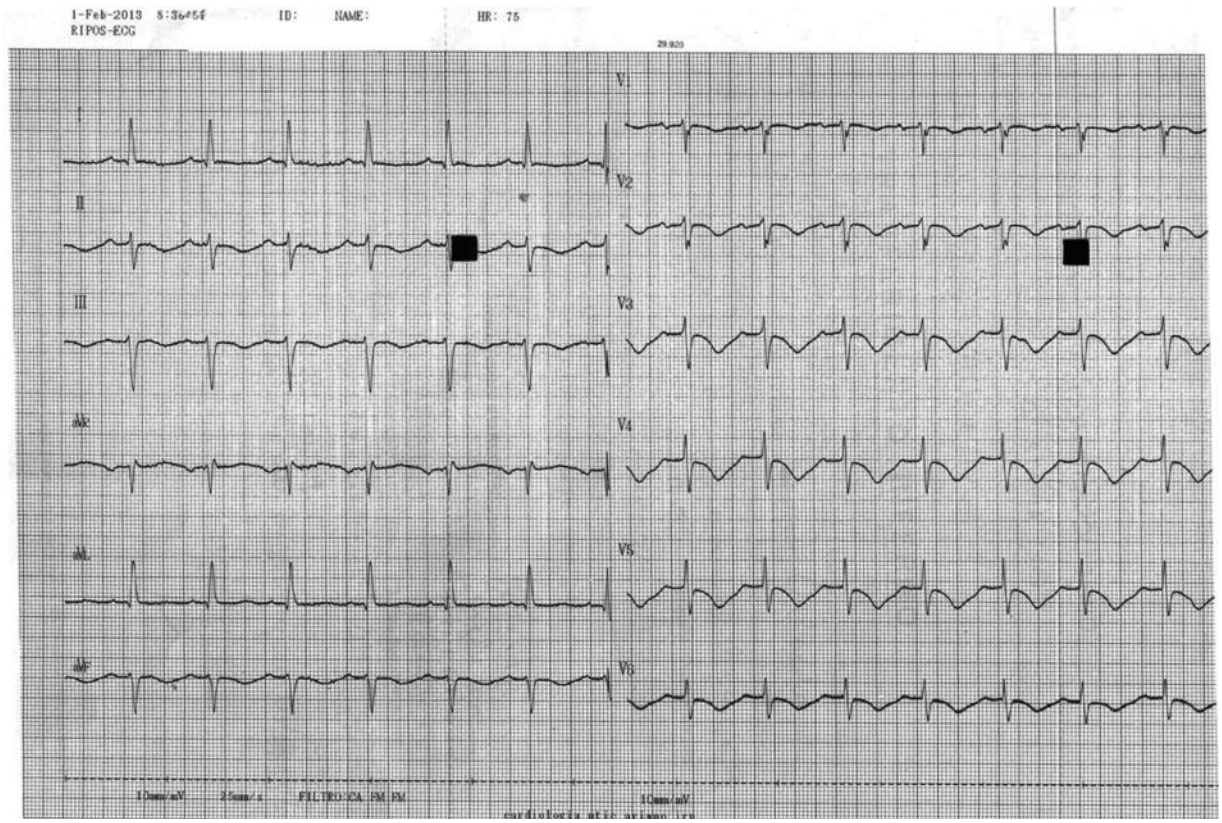

Figure 1. ECG performed in January 2013 showing sinus rhythm at 75 bpm, left anterior hemiblock, T-wave inversion in the II, III, aVF and V1-V6 leads, marked QT prolongation (600 ms) and QTc interval of 671 ms.

ventricular dilatation with mild and diffuse contractile dysfunction (ejection fraction 45%). No significant arrhythmias were inducible at electrophysiological testing, and radiofrequency catheter ablation was unsuccessful. The patient was discharged on therapy with amiodarone, ramipril, and aspirin.

Discussion

TTS, also called *"apical ballooning syndrome",* is a clinical entity, much more common in postmenopausal women, usually triggered by intense emotional and/or physical stress, characterized by reversible left ventricular wall dyskinesia, transient changes of ST segment, myocardial enzymatic release, without significant epicardial coronary artery stenoses, that can mimic acute myocardial infarction[1]. This syndrome, first described in 1990 by Sato et al[2], is called "*takotsubo"* because the typical apical dyskinesia of the left ventricle resemble a Japanese octopus trap.

This case report highlights several points of interest.

First, the occurrence of typical TTS in a patient with idiopathic dilated cardiomyopathy. Although this association is unusual, it may not be fortuitous but both diseases may share a common pathophysiological cause. The literature data have consistently demonstrated complete recovery of myocardial contractility in nearly all patients surviving the acute phase of TTS, which makes the hypothesis of a previously misdiagnosed TTS evolved into dilated cardiomyopathy very unlikely. An enhancement of the sympathetic activity as well as abnormalities in cardiovascular autonomic regulation may indeed represent the link between dilated cardiomyopathy and TTS. In the early stages of dilated cardiomyopathy, the progressive activation of neurohormonal mechanisms and the vasoconstrictor responses to increased activity of both the adrenergic and renin-angiotensin systems[3] may predispose to the development of TTS, given that a catecholamine surge has been suggested as a main pathogenetic mechanism of this syndrome[4].

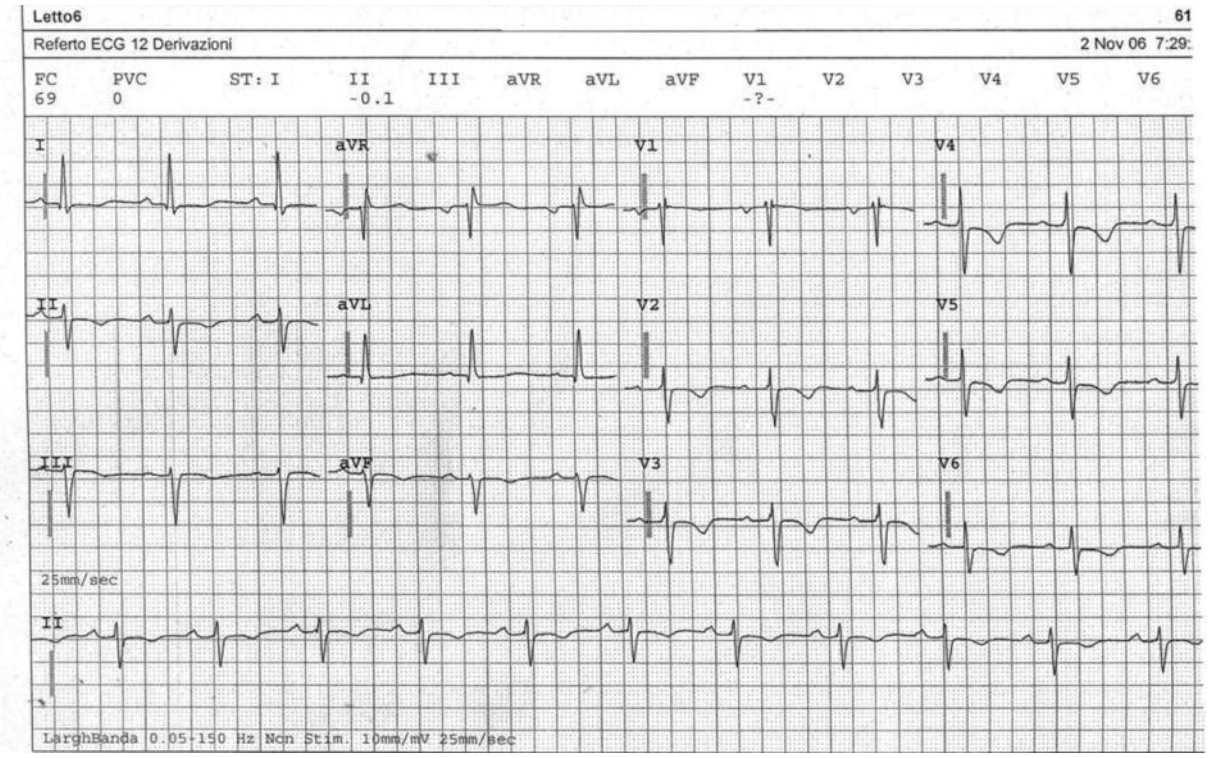

Figure 2. ECG performed in October 2006 showing sinus rhythm at 69 bpm, left anterior hemiblock, T-wave inversion in the II, III, aVF and V1-V6 leads, QT prolongation (450 ms) and QTc interval of 483 ms.

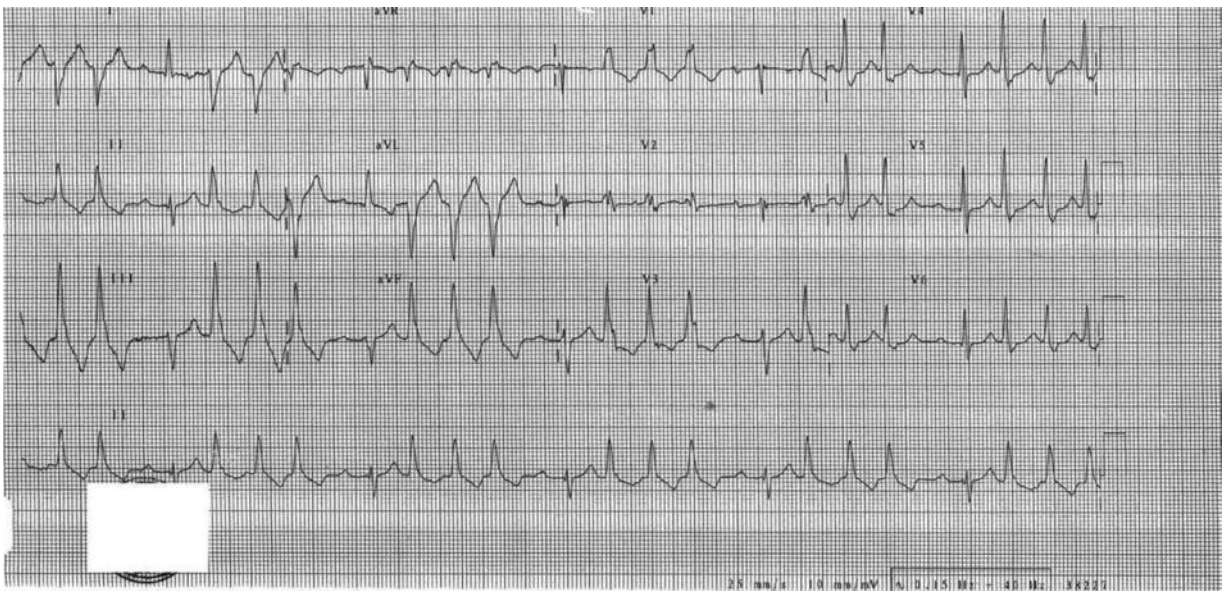

Figure 3. ECG performed in April 2006 showing iterative ventricular tachycardia at 160 bpm with right bundle branch block morphology and right axis deviation.

Second, the recurrence of TTS after seven years from the first diagnosis. Recurrent TTS is uncommon, though not rare, with a reported prevalence in case series ranging from 0%[5] to 11.4%[6]. Longer-term studies are warranted to identify high-risk clinical characteristics for TTS recurrence.

Third, the onset of iterative ventricular tachycardia a few months before the occurrence of TTS. It is reasonable to believe that we are facing a mere coincidence. However, as evidenced by Poirot's dictum that «one coincidence is just a coincidence, two coincidences are a clue, three coincidences are a proof», it may be hypothesized that, in the absence of inducible tachyarrhythmias at electrophysiological testing, abnormal automaticity could be responsible for induction of ventricular tachycardia. It is well known that in TTS increased plasma catecholamine levels and the absence of scarred myocardial tissue may contribute to initiation of ventricular arrhythmias due to enhanced automaticity or triggered activity rather than re-entry[1].

Conclusions

In patients with an intrinsic predisposition, as in our case, sympathetic hyperactivity may represent the pathophysiological link that explains the association among idiopathic dilated cardiomyopathy, TTS and ventricular tachycardia.

Conflict of Interest

The Authors declare that there are no conflicts of interest.

References

1) Rotondi F, Manganelli F. Takotsubo cardiomyopathy and arrhythmic risk: the dark side of the moon. Eur Rev Med Pharmacol 2013; 17: 105-111.
2) Sato H, Tateishi H, Uchida T, Dote K, Ishihara M. Stunned myocardium with specific (tsubo-type) left ventriculographic configuration due to multivessel spasm. In: Kodama K, Haze K, Hori M, eds. Clinical Aspects of Myocardial Injury: From Ischemia to Heart Failure. Kagakuhyouronsya Co; Tokyo 1990; pp. 56-64.
3) Waagstein F, Bristow MR, Swedberg K, Camerini F, Fowler MB, Silver MA, Gilbert EM, Johnson MR, Goss FG, Hjalmarson A. Beneficial effects of metoprolol in idiopathic dilated cardiomyopathy. Metoprolol in Dilated Cardiomyopathy (MDC) Trial Study Group. Lancet 1993; 342: 1441-1446.
4) Prasad A, Lerman A, Rihal CS. Apical ballooning syndrome (Tako-Tsubo or stress cardiomyopathy): a mimic of acute myocardial infarction. Am Heart J 2008; 155: 408-417.
5) Kurisu S, Sato H, Kawagoe T, Ishihara M, Shimatani Y, Nishioka K, Kono Y, Umemura T, Nakamura S. Tako-tsubo-like left ventricular dysfunction with ST-segment elevation: a novel cardiac syndrome mimicking acute myocardial infarction. Am Heart J 2002; 143: 448-455.
6) Elesber AA, Prasad A, Lennon RJ, Wright RS, Lerman A, Rihal CS. Four-year recurrence rate and prognosis of the apical ballooning syndrome. J Am Coll Cardiol 2007; 50: 448-452.