



Takotsubo triggered by acute myocardial infarction: a common but overlooked syndrome?

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Abstract

Takotsubo cardiomyopathy (TCM) is an acute cardiac syndrome characterized by extensive, but potentially reversible, left ventricular dysfunction in the absence of an explanatory coronary obstruction. Thus, TCM is distinct from coronary artery disease (CAD) and acute myocardial infarction (AMI). However, substantial evidence for co-existing CAD in some TCM patients exist. Herein, we take this association one step further and present a case in which the patient simultaneously suffered from AMI and TCM, and in which we believe that a primary coronary event triggered TCM. An 88-year-old female presented with chest pain. Echocardiography revealed apical akinesia with hypercontractile bases. An occluded diagonal branch with suspected acute plaque rupture was identified on the angiogram, but could not explain the extent of akinesia. Cardiac function recovered completely. Thus, this patient adhered to current diagnostic criteria for TCM. TCM is a well-known complication for other conditions associated with somatic stress. It is therefore intuitive to assume that AMI, which also associates with somatic stress and elevated catecholamine, can cause TCM. Our case illustrates that TCM and AMI may occur simultaneously. Although causality cannot be conclusively inferred from this association, the somatic stress associated with AMI may have caused TCM in this patient.

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1 Introduction

Takotsubo cardiomyopathy (TCM) has emerged as an important differential diagnosis in patients presenting with acute chest pain. TCM is associated with severe emotional or somatic stress and is characterized by extensive left ventricular dysfunction in the absence of coronary artery obstruction of sufficient magnitude to explain the extent of left ventricular involvement. Thus, TCM is distinct from coronary artery disease (CAD) and acute myocardial infarction (AMI). Although patients with concomitant significant CAD traditionally were excluded from the TCM cohorts,^[1] several groups, including our own, have provided substantial evidence for co-existing CAD in some TCM patients.^[2–4]

Here, we take this association one step further and present a case in which the patient simultaneously suffered from AMI and TCM, and in which we believe that a primary coronary event triggered TCM.

2 Case report

An 88-year old woman presented to the emergency department with acute onset of chest pain. Her only concomitant condition was chronic obstructive pulmonary disease (COPD). Her COPD was considered mild and had remained stable for many years. Over the last year she had single-handedly been caring for her demented husband, a situation she described as increasingly stressful and anxious.

Electrocardiography (ECG) revealed atypical ST elevation in precordial leads (Figure 1). Echocardiography was performed immediately on arrival and revealed typical left ventricular apical akinesia. Acute coronary angiography revealed a plaque rupture in the first diagonal branch (Figure 2A and 2B) and percutaneous coronary intervention (PCI) was performed. Ventriculography revealed typical apical ballooning (Figure 2C and 2D). Three days after the

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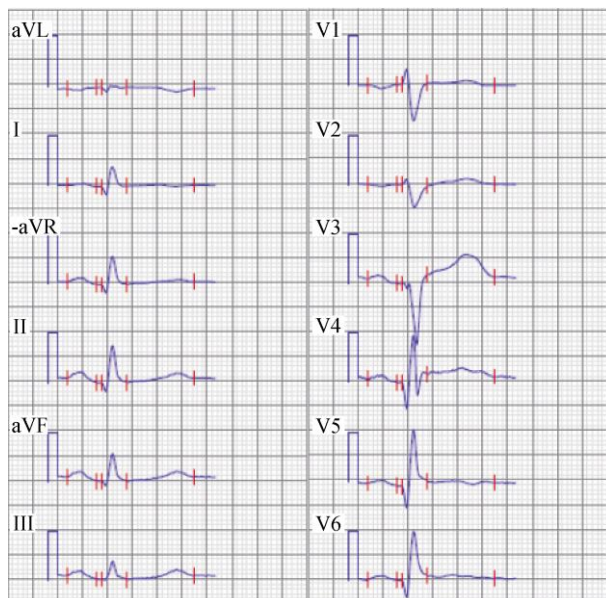


Figure 1. The patient presented with anterolateral ST-elevation on the electrocardiogram.

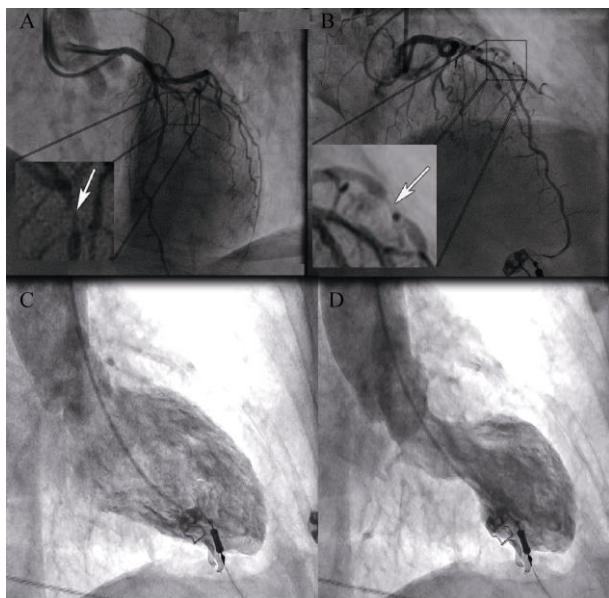


Figure 2. Invasive diagnostics. (A & B): Evidence of plaque rupture in a small diagonal branch could be detected on the acute angiogram (arrows). Ventriculography (C: diastole; D: systole) revealed typical left ventricular apical ballooning and an akinetic region that extended well beyond the vascular territory of the occluded vessel.

event, echocardiography was repeated and could not detect any akinesia (video 1). Serum levels of troponin T peaked at 764 ng/L. Six months later, cardiac functions had further improved and was estimated to be entirely normal, without any trace of regional wall motion defects. Thus, this patient adhered to the diagnostic criteria for TCM (Table 1).

3 Discussion

First and foremost, our case illustrates that TCM and AMI may occur simultaneously. Although causality cannot be conclusively inferred from this association, we believe that the somatic stress associated with AMI may have caused TCM in this patient.

Female gender and advanced age are the best known risk factors for developing TCM. Other risk factors include chronic stress and anxiety.^[5] This patient described a difficult social situation associated with considerable stress and anxiety. Thus, she can be considered to have been at increased risk of developing TCM.

TCM is typically preceded by an acute somatic or emotional stressor and is believed to be caused by elevated levels of catecholamines.^[6,7] Among the somatic stressors known to be associated with TCM are intracranial events, severe infection, surgical trauma and respiratory disease.^[8] Because a relationship has been described between TCM and many acute syndromes associated with increased somatic stress, it is intuitive to assume that AMI, another acute condition associated with considerable somatic stress, could be associated with TCM. Similar to other forms of severe somatic disease, AMI is associated with increased levels of catecholamine.^[6] However, in AMI, catecholamine is particularly high in the periinfarct zone. Thus, if anything, AMI would be expected to more frequently associate with TCM than other somatically stressful syndromes.

We propose that TCM and AMI may not be mutually exclusive conditions, and that they may be intertwined in ways we have yet to discover. Perhaps Takotsubo frequently complicates AMI and sometimes tips these patients into cardiogenic shock. If this is true, developing strategies for reversing Takotsubo may improve the outcome in patients presenting with cardiogenic shock secondary to AMI.

Recent evidence indicates that Takotsubo is a cardiocirculatory syndrome that associates with low peripheral resistance and low blood pressure despite adequate tissue perfusion.^[9] In other words, patients with TCM display a different cardiocirculatory profile compared to patients with only AMI. Because standard acute heart failure therapy with vasopressors and/or inotropes may be directly harmful in TCM, clinicians would probably do well to withhold these therapies in hypotensive patients with TCM that do not display signs of tissue hypoperfusion.^[8] Because optimal treatment of hypotensive patients may depend on whether or not AMI is complicated by TCM, swift confirmation or exclusion of TCM is desirable.

If echocardiography had not been performed in this patient immediately on admission, we believe that this patient

may have been diagnosed with only myocardial infarction and the diagnosis of TCM may have been overlooked. It may be prudent to perform echocardiography without delay in patients presenting with acute chest pain and elevated troponins.

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