From Takotsubo Syndrome to Spontaneous Coronary Artery Dissection: A Case Report of Recurrent Chest Pain and Late Acute Myocardial Infarction

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Abstract

Background: Takotsubo syndrome can mimic acute coronary syndrome, occasionally coexisting with coronary artery disease.

Case Summary: A 58-year-old woman presented with acute chest pain, apical ballooning, and non-obstructive left circumflex coronary artery stenosis. On day 13, she developed recurrent severe chest pain with marked cardiac biomarker elevation. Emergency angiography showed total left circumflex coronary artery occlusion with a tapered appearance, and the intravascular ultrasound revealed intramural hematoma and extensive coronary dissection.

Conclusion: This case highlights that recurrent chest pain after Takotsubo syndrome should prompt urgent reassessment for acute coronary syndrome, including myocardial infarction related to spontaneous coronary artery dissection.

Keywords: microvascular dysfunction, coronary artery dissection, coronary spasm, intramural hemorrhage

Introduction

Takotsubo syndrome (TTS), also known as Takotsubo cardiomyopathy or broken heart syndrome, is characterized by acute dysfunction of the left ventricle without an identifiable coronary obstruction. The clinical features and prognosis of TTS often mimic those of acute coronary syndrome (ACS). Notably, the in-hospital mortality rate of TTS ranges from 4% to 10%, which is comparable to that of ACS.[1]

Acute myocardial infarction (AMI) can lead to TTS as a result of myocardial and coronary artery damage or stress. Recent new hypotheses suggest that TTS may arise from tiny coronary problems, and could potentially lead to ACS. Very early stages of AMI may possibly manifest subsequent to TTS.[2,3] A few limited case reports have highlighted the occurrence of acute coronary events triggered by the rapid progression of TTS.[3] Here, we describe a unique case of persistent TTS accompanied by a striking blockage of the coronary artery.

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Case Presentation

A 58-year-old woman with a history of smoking and hyperlipidemia presented with acute chest pain radiating to the back. During diagnosis, coronary angiography (CAG) revealed 50% stenosis in the left circumflex (LCX) coronary artery, with TIMI (thrombolysis in myocardial infarction) III flow. No intravascular imaging was conducted due to the absence of thrombus. The ventriculogram revealed systolic apical ballooning (Figure1). The following day, the patient's electrocardiogram displayed new T-wave inversions in multiple leads (Figure 2), and the cardiac biomarkers (CKMB) decreased rapidly (from 76.3 to 12.2 ng/mL). The patient was maintained on dual antiplatelet therapy (aspirin and ticagrelor), atorvastatin and nebivolol for the coronary lesion.

One week later, the patient experienced recurrent chest pain, whereupon we performed a chest computed tomography (CT) scan to exclude acute aortic syndrome. The scan showed patent great arteries but partial pericardial thickening (Figure 3), while the cardiac biomarkers were normal. The echocardiogram showed mild apical dyskinesia and ejection fraction improved from 55% to 66%.

On the 13th day, the patient experienced another episode of severe chest pain, accompanied by sustained T wave inversion in multiple leads on the electrocardiogram. Within six hours, the cardiac biomarker (CKMB) rapidly increased from 2.3 to 156.8 ng/mL. Emergency CAG revealed complete occlusion of the proximal LCX artery segment with a tapered appearance (Figure 4). We advanced a wire to the obtuse marginal (OM) branch of the LCX artery, and then smoothly crossed the LCX artery. After thrombus suction, a subsequent angiography revealed spiral dissection in the LCX artery, accompanied by TIMI I flow. The intravascular ultrasound (IVUS) revealed compression of the true vascular lumen by an intramural hematoma and dissected plaque. It also revealed wide-ranging coronary dissection from the opening of the OM branch to the distal part of the LCX artery. The stable hemodynamic condition and the absence of abrupt chest pain during the procedure raised the suspicion of spontaneous coronary artery dissection (SCAD). Despite some difficulty in identifying the true lumen of the native vessel, an acceptable coronary

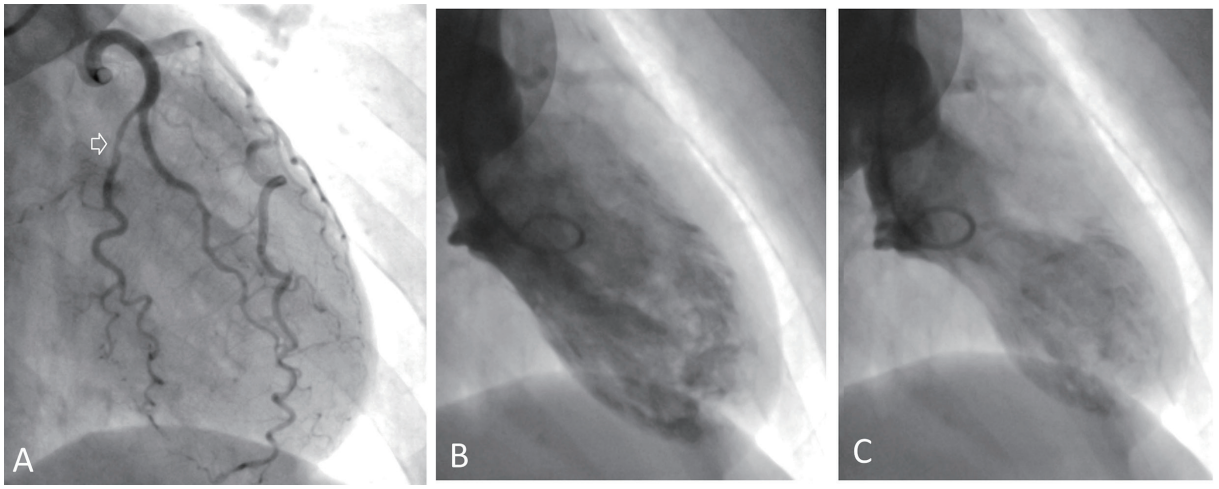

Figure 1. The first angiography.
The coronary angiography revealed a 50% stenosis of the left circumflex coronary artery with TIMI III flow (A). The left ventriculogram revealed an image typical of apical ballooning, which is indicative of Takotsubo syndrome. The images were taken during the end-diastolic phase (B) and end-systolic phase (C).

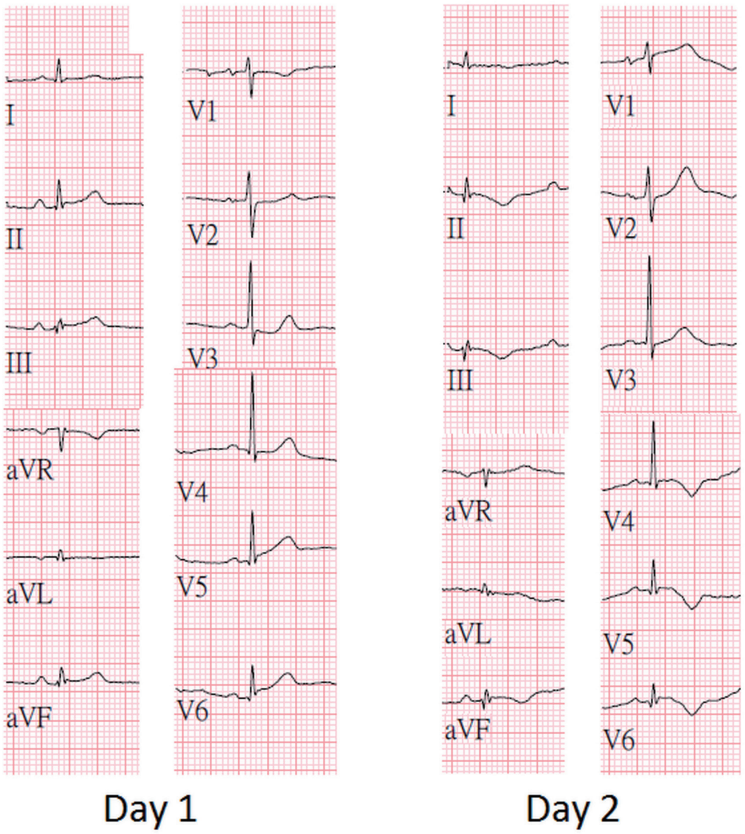

Figure 2. The electrocardiogram.
The electrocardiogram shows rapid changes with new T wave inversions in multiple leads within one day.

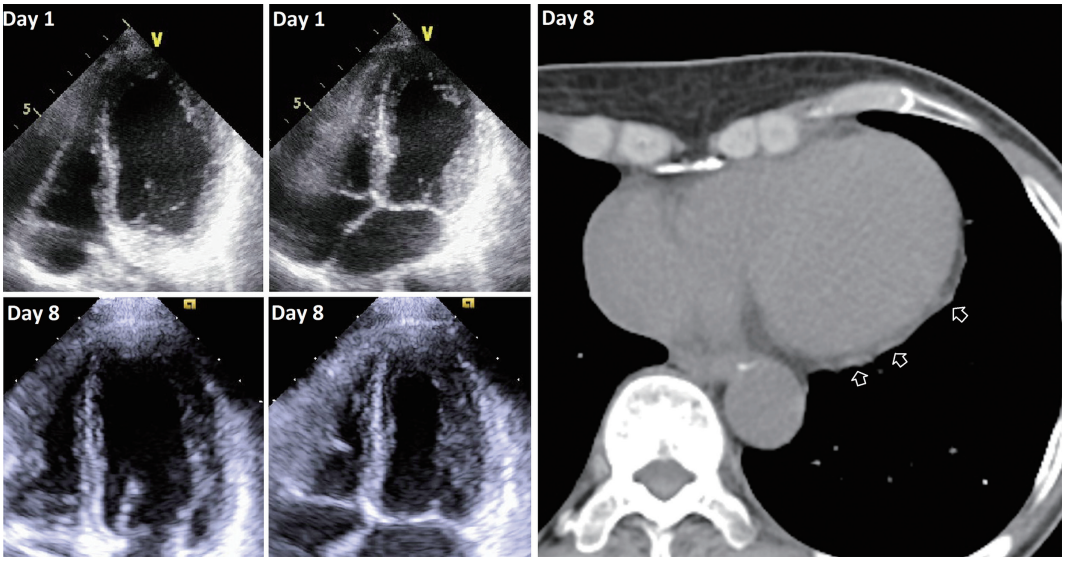

Figure 3. Echocardiography and the first computed tomography.
Echocardiography one week later revealed improved left ventricular apical wall motion, indicating an improvement in cardiac function compared to the initial echocardiogram. (Left: end-diastolic phase; Middle: end-systolic phase) However, the CT scan revealed increased thickness of the pericardium (Right).

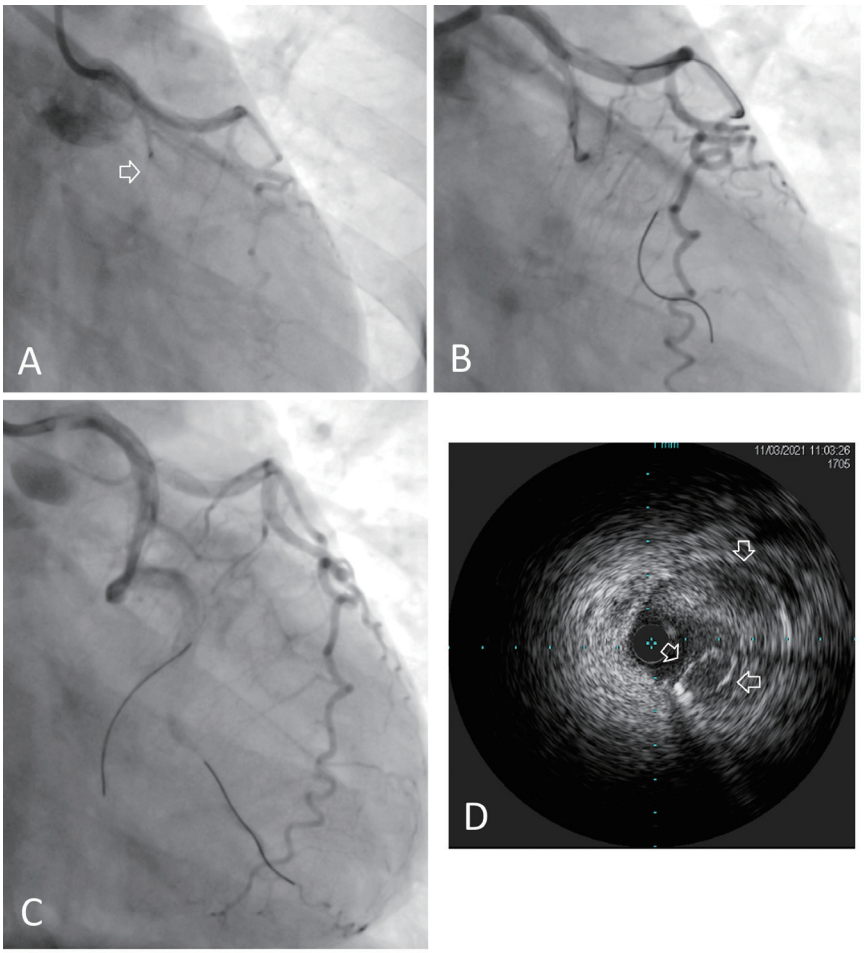

Figure 4. The coronary angioplasty.
Emergency coronary angiography and intravascular ultrasound on day 13.
Emergency coronary angiography revealed total occlusion of the proximal left circumflex artery with a tapered appearance (A). After wire crossing (B) and thrombus aspiration, angiography revealed extensive spiral dissection with impaired distal flow (C). Intravascular ultrasound showed compression of the true lumen by intramural hematoma and dissected plaque (D).

flow was eventually established. The peak value of the cardiac biomarker (CKMB) was 280.3 ng/mL, and echocardiography revealed global hypokinesis of the left ventricle with a low ejection fraction of 38%. One month later, a CT coronary angiography showed good healing of the dissected LCX artery, but residual thickening of the pericardium remained (Figure 5). A follow-up echocardiogram six months later revealed an LVEF of 54% with no wall motion defects, and the patient remained asymptomatic at her three-year follow-up.

Discussion

This case illustrates three important issues: 1. the diagnostic overlap between TTS and ACS, 2. the potential mechanisms linking TTS to coronary artery events, and 3. the need for urgent reassessment when chest pain recurs after an initial diagnosis of TTS.

The differential diagnosis of TTS, ACS and SCAD remains challenging. We diagnosed TTS based on the Revised Mayo Clinic Diagnostic Criteria. Although the presence of a non-

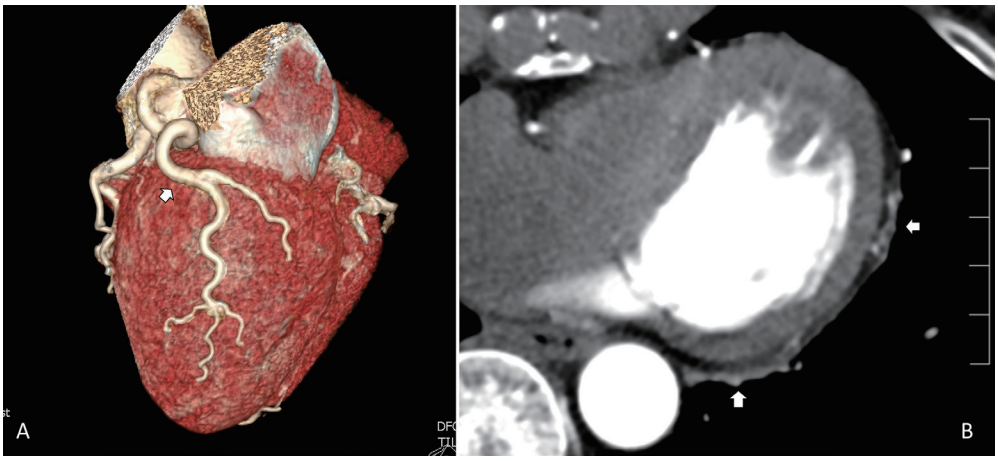

Figure 5. The computed tomography coronary angiography.
On day 43, CT coronary angiography revealed a significant improvement in the healing of the dissected LCX artery (A), while the pericardium remained thickened (B).

obstructive LCX lesion complicated the initial diagnostic interpretation, several findings strongly favored TTS over ACS. These included the typical apical ballooning pattern, TIMI grade III coronary flow, modestly elevated yet rapidly declining cardiac biomarkers, and the prompt recovery of left ventricular function. Rapid improvement in regional wall motion seen on echocardiography, alongside residual pericardial thickening, provided further compelling evidence supporting a TTS diagnosis. Ultimately, the 50% stenosis in the LCX artery may have represented an unstable coronary lesion that initiated the cascade of events that contributed to TTS pathogenesis. Simultaneously, the effects of TTS, such as microvascular dysfunction[4,5], can influence the coronary lesion, leading to vasoconstriction and compromised blood flow in the coronary arteries, ultimately resulting in ischemia and infarction.[6] Furthermore, there is the possibility of coronary vasospasm and type 2 SCAD, which is caused by intramural hematoma and produces a smooth tubular coronary lesion without visible dissection plane. Although iatrogenic dissection during wire manipulation or thrombus aspiration cannot be completely excluded, the tapered angiographic occlusion before intervention, the absence of a clear stump, IVUS evidence of intramural hematoma with true lumen compression, and the subsequent healing on follow up CT coronary angiography all favored the understanding of a pre-existing SCAD.

There was some evidence of mechanisms linking TTS to coronary events, including coronary vasospasm, microvascular dysfunction, intramural hematoma and SCAD. Traditionally, TTS is well known to have cardiovascular complications and pericardial involvement,[7,8] as the CT examination illustrated in our case. The coexistence of AMI and takotsubo syndrome has previously been associated with a poor long-term prognosis.[9] However, recent study suggests coronary spasm might be a cause of TTS,[10] and it can be reproducible in around 20% of TTS patients.[11] In our case, intermittent spastic angina attack may have occurred with the initial narrowing of the LCX artery seen on angiography, which may have been the result of coronary spasm. Whenever the patient experienced an AMI attack, the vasospasm worsened, resulting in a short and tapered segment of the LCX artery. Given that we did not prescribe calcium channel blockers under the emergency conditions of AMI, it is not unreasonable to suggest that vasospasm might be associated with TTS initially and contribute to AMI later on. Similar to coronary spasm,

microvascular dysfunction is another pathogenic mechanism in TTS, which can be confirmed by the Angiography-derived Index of Microcirculatory Resistance (Angio-IMR), a non-invasive tool for the assessment of coronary microvascular dysfunction (CMD). Recent findings highlight the clinical utility of Angio-IMR in TTS, with studies suggesting that elevated resistance (>25 units) is common in the acute phase, particularly in the left anterior descending (LAD) artery, and often resolves during recovery.[12]

In some reported cases of TTS, including our own, SCAD has been observed.[3] It is plausible that an intramural hemorrhage occurred, resulting in type 2 SCAD,[13] and that the coronary circulation might have remained in a stunned state, as suggested by the inverted T waves on the electrocardiogram. This state could have potentially resulted in type 4 SCAD (an abrupt, total occlusion of a coronary artery caused by SCAD). The high degree of tapering of the vessels observed in the angiography of our case, without a clear stump, provides further evidence supporting the diagnosis of type 4 SCAD. Although it is possible that the dissection occurred in conjunction with coronary artery instrumentation during the restoration of coronary flow, stable hemodynamics without acute chest pain during the procedure suggested that the initial presentation of SCAD was established before the coronary intervention. Our IVUS revealed an extensive coronary dissection from the ostium of the OM branch to the distal part of the LCX artery. However, more advanced intravascular imaging techniques, such as optical coherence tomography, would have been needed to confirm whether plaque rupture was the cause of the coronary artery dissection.

Questions remain about why TTS may lead to AMI. In addition to the three possibilities mentioned earlier, one further hypothesis suggests a chicken-or-egg causality dilemma, and another considers the coexistence of TTS and AMI.[1,14] In our case, AMI occurred 13 days after the TTS diagnosis, making it appear to be a coincidence. However, based on the series of imaging findings, there is a strong suggestion that TTS and AMI could have coexisted in our case. Further investigation is needed to fully determine the pathophysiological mechanisms.

Conclusion

This case highlights a plausible pathophysiological link between TTS and subsequent SCAD. TTS may induce microvascular dysfunction, coronary spasm, or intramural hematoma, thereby triggering an AMI via SCAD mechanisms. Conversely, the intense chest pain caused by an evolving SCAD event could act as a physical and emotional stressor, ultimately precipitating TTS. Therefore, recurrent or worsening chest pain after an initial diagnosis of TTS should prompt urgent reassessment for ACS, including SCAD related myocardial infarction, particularly when accompanied by dynamic electrocardiographic changes or biomarker elevation.

Acknowledgements

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Conflict of interest

All the authors declare no conflict of interest.

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