

# Dynamic Left Ventricular Outflow Tract Obstruction with Cardiogenic Shock in Apical Ballooning Syndrome

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Apical ballooning syndrome, also called Takotsubo cardiomyopathy, is characterized by transient systolic dysfunction of mid to apical segments and hyperkinesis of basal segments of the left ventricle that mimic acute myocardial infarction without significant coronary artery stenosis. We reported a 51-year-old man with chest distress, hypotension and abnormal electrocardiogram. Echocardiography revealed extensive akinesia of the mid to apical portions of the left ventricle, hyperkinesis of basal segments of the left ventricle, increasing left ventricle outflow tract velocity and severe mitral regurgitation. Cardiac catheterization showed a normal coronary angiography and an obvious pressure gradient between the left ventricle and aorta. After we discontinued administration of nitrates, provided mild hydration and initiated intravenous dopamine infusion, the patient's hypotension, left ventricular to aortic pressure gradient, and severe mitral regurgitation resolved the next day. Thereafter, apical ballooning syndrome with dynamic left ventricular outflow tract obstruction, severe mitral regurgitation and cardiogenic shock was diagnosed.

**Key Words:** Apical ballooning syndrome • Cardiogenic shock • Left ventricular outflow tract obstruction • Mitral regurgitation

## INTRODUCTION

Apical ballooning syndrome (ABS), a disease mimicking acute myocardial infarction (AMI), is not rare in Taiwan.<sup>1,2</sup> Although ABS' etiology is not completely understood, patients with the disease usually spontaneously recover well within one month. However, dynamic left ventricular outflow tract (LVOT) obstruction may occur in ABS due to compensated hypercontractility of basal segments of the left ventricle (LV) after dysfunction

of apex and systolic anterior motion (SAM) of anterior mitral leaflet via the Venturi effect.<sup>3-5</sup> The conventional medical therapy for AMI may not be suitable for this condition, and may even worsen the LVOT obstruction.<sup>1,3,6,7</sup> Here we present the first case report of ABS with dynamic LVOT obstruction, severe mitral regurgitation (MR) and cardiogenic shock in Taiwan.

## CASE REPORT

Our patient is a 51-year-old man who suffers from hypertension, but who benefits from regular medical control. Prior to admission to our facility, he suffered from sudden onset chest pain and cold sweating for 1 hour after drinking alcohol and quarrelling with his friends. He visited a local hospital for preliminary medical assistance, where acute anterior wall ST-elevation myocardial infarction was impressed. After receiving

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aspirin, clopidogrel and intravenous nitrate infusion, he was then transferred to our emergency department. The patient's initial blood pressure was 114/71 mmHg and the heart rate was 98 beats per minute. There was a grade 3/6 systolic murmur at the left sternal border and apical regions upon physical examination. The initial cardiac biomarkers were normal, and applied electrocardiogram revealed normal sinus rhythm, 2mm ST elevation in V2-V3, and inverted T waves in V4-V6 (Figure 1). Chest radiography showed no sign of interstitial congestion. Echocardiography revealed extensive akinesia of the mid to apical portions of the LV, and hyperkinesis of basal segments of LV (Figure 2A). Also, velocity of the LVOT increased, along with an eccentric severe MR due to SAM of the anterior leaflet of the mitral valve (Figure 2C). We performed emergency cardiac catheterization which revealed a normal coronary angiogram with an approximate 40 mmHg pressure gradient between the LV and the aorta (Figure 2E). We did not perform LV angiogram at that time due to high left ventricular end-diastolic pressure (near 40 mmHg). Because the noted persistent hypotension (79/45 mmHg) and the impression of cardiogenic shock, we discontinued the nitrate infusion and prescribed 0.9% normal saline of 500 ml for hydration, and dopamine via intravenous infusion as an inotropic agent. Thereafter, the patient's hypotension gradually improved. About 20 hours later, we repeated a detailed echocardiography for the patient. It revealed

normal LV size and wall thickness, no aortic stenosis, slight hypokinesia on apex, only mild MR and no significant pressure gradient between the LV and the aorta (Figure 2D). To confirm the diagnosis, an LV angiogram was performed 36 hours after the patient arrived, which showed no significant LV-aortic pressure gradient, minimal MR, and almost recovered LV systolic function (Figures 2B and F). ABS with transient dynamic LVOT obstruction and severe MR was diagnosed. During the entire hospitalization, we sequentially checked cardiac biomarkers three times, including creatine phosphokinase and troponin-I. Only one mild elevation of troponin-I (0.64 ng/ml) was noted. The patient recovered well with beta-blocker therapy and was discharged 3 days later.

## DISCUSSION

Apical ballooning syndrome (ABS), also called "Takotsubo cardiomyopathy", was originally described about 20 years ago in Japan, and is characterized by an acute onset of chest distress associated with electrocardiographic changes (ST elevation, T-wave inversion or transient left bundle branch block) with or without elevation of cardiac enzymes, hypokinesia, akinesia or dyskinesia of the LV mid to apical segments and basal hyperkinesia in echocardiography or ventriculogram.

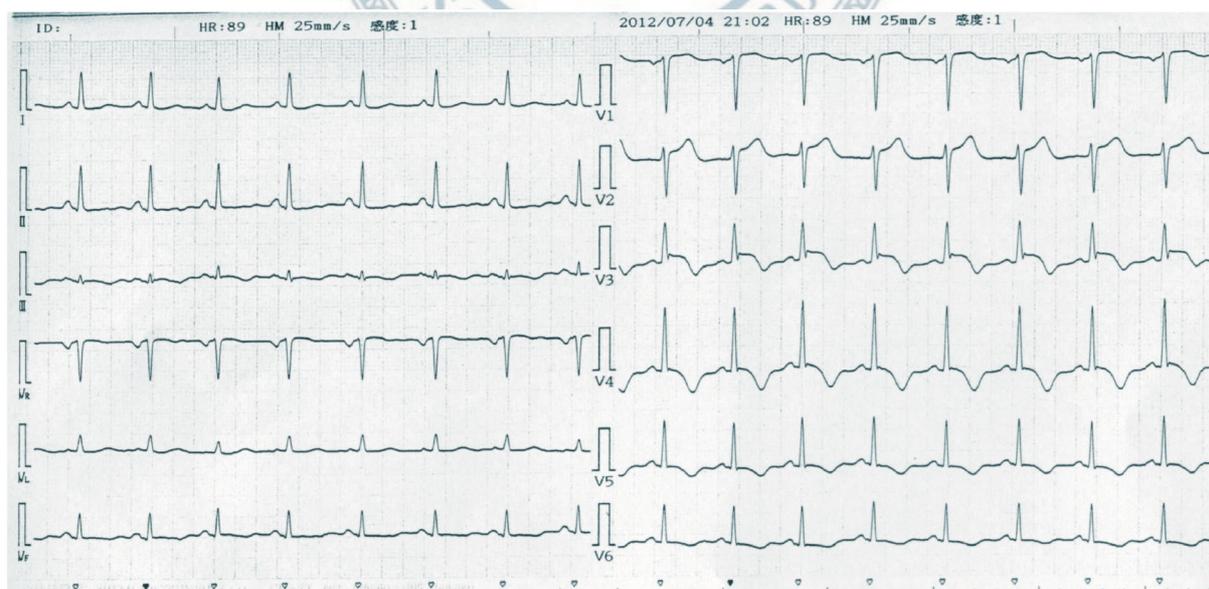
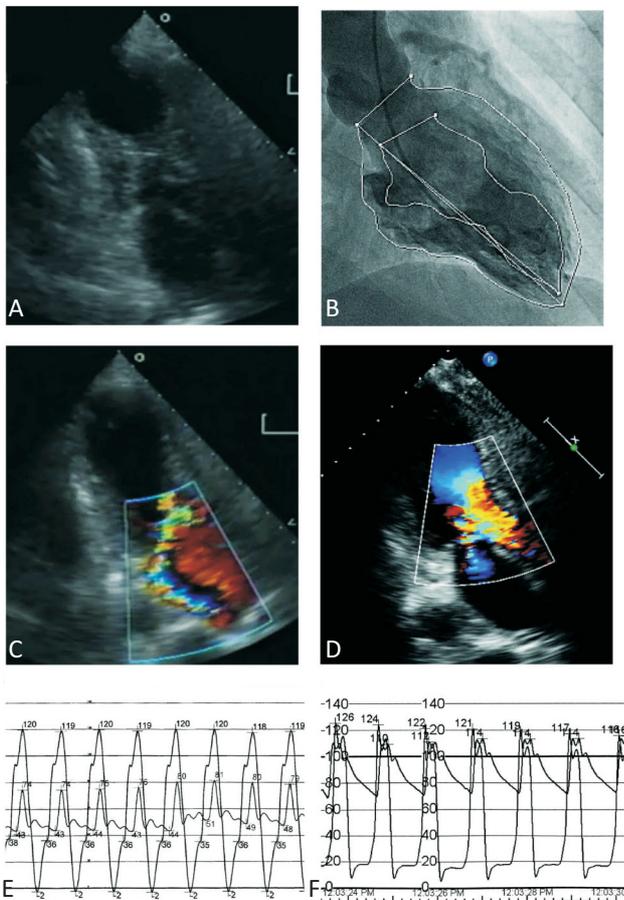


Figure 1. The initial electrocardiogram showed normal sinus rhythm, 2 mm ST elevation in V2-V3, and inverted T waves in V4-V6.



**Figure 2.** (A) The initial echocardiography revealed extensive akinesia of the apical and mid portions of the left ventricle (LV) and hyperkinesia of the basal segments of LV. (B) One and a half day later, LV angiogram showed an almost recovered LV systolic function with ejection fraction 81.6%. (C) An eccentric severe mitral regurgitation and increasing LV outflow tract velocity were noted in the initial echocardiography. (D) The following echocardiography revealed only a mild mitral regurgitation 20 hours later. (E) The initial emergent cardiac catheterization showed a 40 mmHg pressure gradient between the LV and aorta, hypotension, and high LV end-diastolic pressure. (F) There was no more significant LV-aortic pressure gradient in the following cardiac catheterization.

However, patient coronary angiography study is typically normal.<sup>1,2,8-10</sup> It is an important differential diagnosis of AMI and myocarditis in clinical practice. In a cohort study in Taiwan, the prevalence of ABS presenting with acute coronary syndrome was 0.9%, and markedly higher in women.<sup>2</sup> The pathophysiology of ABS is not well understood. An excess release of catecholamine due to stress or acute medical illness with myocardial stunning was the most likely mechanism. Other mechanisms such as ischemia-mediated stunning due to

multivessel epicardial or microvascular spasm and dynamic LVOT obstruction were also documented.<sup>10</sup> From these studies, we did note that the ABS always recovered completely in two to four weeks. However, there were complications observed in some studies, such as cardiogenic shock, congestive heart failure and death, with prevalences of 6.5%, 3.8% and 3.2%, respectively.<sup>11,12</sup>

Not infrequently, dynamic LVOT obstruction may occur in the acute stage of ABS, with a prevalence rate as seen in one previous report of up to 25%.<sup>5</sup> The main mechanism of this obstruction is hypercontractility of the basal segment of the LV which causes the path of the LVOT to narrow during systole with the presentation of SAM of the anterior mitral leaflet (which also causes MR), similar to the mechanism of hypertrophic obstructive cardiomyopathy. The degree and duration of mitral SAM determine the severity of the dynamic LVOT obstruction and MR.<sup>4</sup> Our case presented both dynamic LVOT obstruction and acute severe MR due to SAM of the anterior mitral leaflet, which may result in cardiogenic shock in this AMI-mimicking condition.<sup>4,6</sup> Echocardiography should be performed as soon as possible in cases of ABS with shock for the possible concurrence of LVOT obstruction.

The medication that we prescribed should be administered with great care and caution in cases of ABS with LVOT obstruction. Nitrates, broadly used in acute coronary syndrome, may exacerbate the LVOT obstruction in ABS by decreasing preload and afterload of LV and reflex tachycardia.<sup>1,6</sup> Furthermore, inotropic agents such as dopamine and dobutamine may increase mitral SAM and worsen LVOT obstruction in ABS.<sup>3,7</sup> However, therapeutic maneuvers that may be beneficial include beta-blocker, alpha-adrenergic agonist, and volume expansion.<sup>5,6,13</sup> Beta-blockers can reduce LVOT obstruction by reducing basal hypercontractility and heart rate and increasing LV filling, thereafter improving MR. Intravenous fluid supplementation can increase intravascular and LV volumes, thereby reducing the mitral SAM, and should be given for patients without significant pulmonary congestion. Alpha-adrenergic agonists, which increase arterial impedance, may reduce LV ejection velocity and increase LV volume. The combination of increased LVOT volume/area and reduced ejection velocity reduces dynamic LVOT obstruction. This treatment

may be helpful to support blood pressure while a beta-blocker is administered. If beta-blockers are contraindicated, nondihydropyridine calcium channel blockers may be used to achieve target heart rate below 60 to 70 bpm.<sup>4</sup> Intra-aortic balloon pump may be used in cases involving cardiogenic shock, but it should be noted that there is a slight risk of afterload reduction which would worsen the degree of LVOT obstruction in ABS.<sup>3,7</sup>

## CONCLUSION

Early recognition of acute dynamic LVOT obstruction is an important clinical and therapeutic problem in patients with ABS. Conventional treatment for acute coronary syndrome such as nitrates and afterload vasodilators would likely increase the degree of LVOT obstruction, resulting in a vicious cycle of hypotension. However, beta-blockers and intravenous fluid supplementation may be beneficial and even life-saving for these patients. Additionally, alpha-adrenergic agonists and nondihydropyridine calcium channel blockers can be used as alternative drugs for these patients.

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