

APICAL BALLOONING SYNDROME

A 65-year-old female presented to the emergency room with severe retrosternal chest pain and dyspnea. Her symptoms began approximately one and one-half hours after she was informed of the death of her mother. This emotional stress was also exacerbated by other personal and social issues on the evening of her admission. The patient had no prior history of any cardiac disease. Her risk for vascular disease, however, included hypertension, hyperlipidemia, and insulin dependent diabetes. After her hospital arrival, she was immediately brought to the cardiac catheterization laboratory for urgent angiography. The patient was found to have diffuse coronary artery disease but no evidence of a “culprit lesion” characterized by either a vessel occlusion or acute plaque rupture. Left ventriculography demonstrated an extensive area of anteroapical and inferoapical wall akinesis and an apical ballooning configuration with an ejection fraction in the range of 25%. Although the troponin I was modestly elevated, serial CPK levels remained normal. She was discharged on aspirin, Lipitor, insulin, beta blocker therapy, and an ACE inhibitor. She was discharged 48 hours from her admission in stable condition with plans for a follow-up 4-6 week echocardiogram.

This case illustrates an example of “tako-tsubo” syndrome first reported by Japanese authors in 1990 characterized by chest pain and hyperacute electrocardiographic changes associated with transient, but often severe, apical wall akinesis. The name tako-tsubo was used because of the unusual shape of the left ventriculogram which resembled a Japanese tako-tsubo pot - a pot with a round bottom and narrow neck - used for octopus trapping in Japan. Because left ventricular function usually returns to normal, the term transient left ventricular apical ballooning syndrome has more recently been used when discussing this entity. This event often occurs in the absence of any significant coronary disease or evidence of acute plaque rupture, although the patient discussed above certainly did have coronary artery disease related to her underlying risk factors. This syndrome is of interest in that it seems to be prevalent in post menopausal women and often is associated with very mild or, as in this case, essentially normal enzymes despite severe left ventricular dysfunction. This syndrome also seems to be precipitated by a severe episode of emotional stress as was seen in the patient above. This fact seems to suggest an adrenergic stimulus to the event. Although the precise mechanisms are unknown, the apex may be particularly vulnerable to this condition because the left ventricular apex lacks the usual three layered myocardial structure present in other areas of the left ventricle and the apical area may be more prone to ischemic insult and more easily damaged.¹ In addition, there is increased adrenergic receptor density in the cardiac apex which may further make this area susceptible to acute injury.² In support of this association is a recent report of transient left ventricular apical ballooning syndrome associated with cocaine use.⁶ An elevated catecholamine state may precipitate this acute event by a) epicardial coronary artery spasm, b) coronary microvascular compromise, or c) a direct cardiotoxic effect. It is important to recognize, however, that despite what initially may seem to be profound left ventricular dysfunction, most individuals recover full left ventricular contractility often within days of the initial event. Left ventricular dysfunction should be treated with the usual medical armamentarium used for the treatment of most patients with left ventricular dysfunction and heart failure.

However, if vasospasm can be shown to be a factor in the acute event, calcium channel blockers may be more appropriate in some situations instead of beta blocker therapy. Although once felt to be an unusual syndrome, 22 patients were prospectively identified over a 32 month period at one institution³ and it is certainly possible that this syndrome may have masked what traditionally has been felt to be a myocardial infarction with a “plaque rupture” when nonobstructive disease may have been found in the setting of an acute cardiac event. This author has seen three such patients in an 18 month period. Although several recent reports in the literature have discussed this unique acute myocardial event,¹⁻⁶ this syndrome is important not only to clinically recognize as an entity (Table 1) but to understand that the resolution of the often profound left ventricular dysfunction may lead to complete recovery of underlying left ventricular function; Yet there have been reports of late recurrences of this condition¹ and for the primary care physician, it is therefore important to note that until further information is known, lifetime treatment with antiadrenergic medications (beta blockers) and neuro-hormonal antagonists (ACE inhibitors, angiotensin receptor blockers, and spironolactone) may be necessary. It would therefore be premature to discontinue therapy if an echocardiogram reveals normal recovery of left ventricular function. In this respect, initial recognition of this syndrome by emergency room personnel and cardiologists and the choice of appropriate medical therapy may be equally complimented by long term choices in medical therapy as well.

References

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4. Elian D, et al. Left ventricular apical ballooning: not an uncommon variant of acute myocardial infarction in women. *Clin. Cardiol*. 2006;29:9-12.
5. Guttormsen B, et al. Transient left ventricular apical ballooning: a review of the literature. *Wis Med J*. 2006;105:49-54.
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TABLE 1: Proposed Mayo Criteria for the Clinical Diagnosis of the Transient Left Ventricular Apical Ballooning Syndrome*

1. Transient akinesis or dyskinesis of the left ventricular apical and mid-ventricular segments with regional wall-motion abnormalities extending beyond a single epicardial vascular distribution
2. Absence of obstructive coronary disease or angiographic evidence of acute plaque rupture
3. New electrocardiographic abnormalities (either ST-segment elevation or T-wave inversion)
4. Absence of
 - Recent significant head trauma
 - Intracranial bleeding
 - Pheochromocytoma
 - Obstructive epicardial coronary artery disease
 - Myocarditis
 - Hypertrophic cardiomyopathy

* All 4 criteria must be met.

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