

- 16 **Xiao RP**, Hohl C, Altschuld R, *et al.* β2-adrenergic receptor-stimulated increase in cAMP in rat heart cells is not coupled to changes in Ca²⁺ dynamics, contractility, or phospholamban phosphorylation. *J Biol Chem* 1994;**269**:19151–6.
- 17 **Altschuld RA**, Starling RC, Hamlin RL, *et al.* Response of failing canine and human heart cells to β2-adrenergic stimulation. *Circulation* 1995;**92**:1612–18.
- 18 **Kuschel M**, Zhou YY, Spurgeon HA, *et al.* β2-adrenergic cAMP signaling is uncoupled from phosphorylation of cytoplasmic proteins in canine heart. *Circulation* 1999;**99**:2458–65.
- 19 **Brodde OE**. β1- and β2-adrenoceptors in the human heart: properties, function, and alterations in chronic heart failure. *Pharmacol Rev* 1991;**43**:203–42.
- 20 **Bristow MR**, Ginsburg R, Minobe W, *et al.* Decreased catecholamine sensitivity and β-adrenergic receptor density in failing human hearts. *N Engl J Med* 1982;**307**:205–11.
- 21 **Kiuchi K**, Shannon RP, Komamura K, *et al.* Myocardial β-adrenergic receptor function during the development of pacing-induced heart failure. *J Clin Invest* 1993;**91**:907–14.
- 22 **Gu XH**, Kompa AR, Summers RJ. Regulation of β-adrenoceptors in a rat model of heart failure: effects of perindopril. *J Cardiovasc Pharmacol* 1998;**32**:66–74.
- 23 **Dorn GW II**, Tepe NM, Lorenz JN, *et al.* Low- and high-level transgenic overexpression of β2-adrenergic receptors differentially affect cardiac hypertrophy and function in Gaq-overexpressing mice. *Proc Natl Acad Sci USA* 1999;**96**:6400–5.
- 24 **Maurice JP**, Hata JA, Shah AS, *et al.* Enhancement of cardiac function after adenoviral-mediated in vivo intracoronary β2-adrenergic receptor gene delivery. *J Clin Invest* 1999;**104**:21–9.
- 25 **Shah AS**, Lilly RE, Kypson AP, *et al.* Intracoronary adenovirus-mediated delivery and overexpression of the β2-adrenergic receptor in the heart: prospects for molecular ventricular assistance. *Circulation* 2000;**101**:408–14.
- 26 **Turki J**, Lorenz JN, Green SA, *et al.* Myocardial signaling defects and impaired cardiac function of a human β2-adrenergic receptor polymorphism expressed in transgenic mice. *Proc Natl Acad Sci USA* 1996;**93**:10483–8.
- 27 **Dishy V**, Landau R, Sofowora GG, *et al.* Beta2-adrenoceptor Thr164Ile polymorphism is associated with markedly decreased vasodilator and increased vasoconstrictor sensitivity in vivo. *Pharmacogenetics* 2004;**14**:517–22.
- 28 **Bruck H**, Leineweber K, Park J, *et al.* Human beta2-adrenergic receptor gene haplotypes and venodilation in vivo. *Clin Pharmacol Ther* 2005;**78**:232–8.
- 29 **Bruck H**, Leineweber K, Ulrich A, *et al.* Thr164Ile polymorphism of the human beta2-adrenoceptor exhibits blunted desensitization of cardiac functional responses in vivo. *Am J Physiol Heart Circ Physiol* 2003;**285**:H2034–8.

IMAGES IN CARDIOLOGY

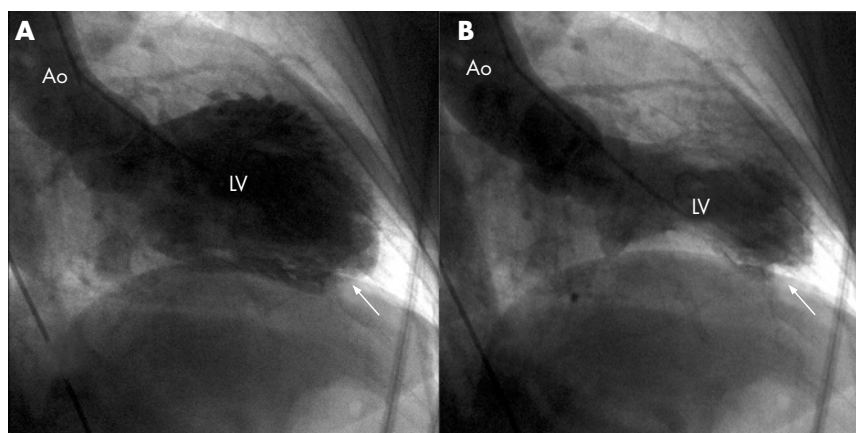
doi: 10.1136/hrt.2006.095372

Left ventricular thrombus associated with left ventricular apical ballooning

A 74-year-old woman who had been experiencing mental stress was admitted to our institution with a history of continuous atypical chest pain. The evolutive ECG showed T wave inversion in leads V2–V6, I AVL and III, with a prolonged QT interval. Mild enzymatic changes were found in blood chemistry examinations. Coronary angiography showed no significant stenosis, but left ventriculography demonstrated apical asynergy with basal hyperkinesia (apical ballooning). A striking filling defect highly suggestive of a thrombus was also viewed at the apex (panel A, diastole; panel B, systole; and video 1, white arrows; to see video footage visit the *Heart* website—<http://heart.bmj.com/supplemental>). Left ventricular ejection fraction was 40%. The apical ballooning and intraventricular thrombus were confirmed by transthoracic echocardiography. The patient was discharged under anticoagulant treatment.

After 2 months the ECG showed normal findings, and a transthoracic echocardiogram showed absolutely normal left ventricular (LV) wall motion and complete resolution of the apical thrombus.

Direct evidence of a LV thrombus associated with takotsubo-like ventricular dysfunction has not been demonstrated, although there have been reports regarding the embolic complications of this disorder.



End diastolic (panel A) and end systolic (panel B) ventriculograms of the patient, showing akinesia of apical segments of the left ventricle (LV) and hypercontraction of basal and mid segments. A striking filling defect highly suggestive of a thrombus was also viewed at the apex (white arrow). Ao, aorta.

Some articles reported that the akinetic LV wall in the setting of myocardial infarction is an important cause of LV thrombus. Given that the LV thrombus in the present case was caused by a wall motion abnormality, its clinical appearance seems rather late. We report a case of transient LV apical ballooning with LV thrombus demonstrated in the early angiographic procedure. With restoration of LV apical wall motion and warfarin treatment, the LV thrombus disappeared. LV thrombus

should be considered an early and delayed complication of transient LV apical ballooning.

Pablo Robles, Jose Julio Jimenez, Manuel Alonso

probles@fhcalcorcon.es; problesve@yahoo.es



To view video footage visit the *Heart* website—<http://heart.bmj.com/supplemental>