Reversal of Acute Pulmonary Oedema with Beta-blockers in Hypertrophic Cardiomyopathy

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A 41-year old male was admitted with dyspnoea in 1990. His ECG demonstrated sinus rhythm, voltage-criteria for left ventricular hypertrophy and Q waves in the lateral leads. Transthoracic echocardiography demonstrated asymmetrical septal hypertrophy (17 mm), partial systolic anterior movement of the anterior mitral valve leaflet, a dynamic sub-aortic gradient of 30 mmHg and the presence of diastolic dysfunction as demonstrated by a reversal of the E:A ratio. The diastolic dysfunction was thought to be responsible for the patient’s presenting symptoms. Hypertrophic cardiomyopathy (HCM) was diagnosed but the patient declined any medical treatment or follow-up.

He presented in June 2000 with a right hemiparesis. The ECG on admission showed atrial fibrillation with a ventricular rate of 110 beats per minute (bpm). Computerized tomography of the brain did not reveal evidence of a haemorrhagic bleed and treatment was commenced with intravenous heparin and an oral anticoagulant. An echocardiogram on day 5 demonstrated no changes from his previous study. In the absence of a severe outflow tract gradient, he was commenced on verapamil 120 mg twice daily, for ventricular rate control and treatment of diastolic dysfunction.

On day 18, the patient became acutely dyspnoeic. Examination revealed a heart rate of 65 bpm, blood pressure of 120/70 mmHg, a late systolic murmur loudest at the left sternal edge and crackles throughout the lung fields. A chest radiograph confirmed pulmonary oedema and treatment with intravenous diuretics and oxygen therapy was immediately initiated. The patient was unresponsive to therapy and so an emergency echocardiogram was performed. This revealed normal systolic function of the left ventricle, a maximum dynamic gradient of 150 mmHg in the left ventricular outflow tract and mild mitral regurgitation (Fig. 1). An intravenous bolus of esmolol was administered and within 10 min there was rapid improvement in the patient’s symptoms. Real-time echocardiography demonstrated a dramatic reduction in the outflow tract gradient to 16 mmHg (Fig. 2). An esmolol infusion was continued for a further 8 h and then replaced with oral beta-blocker therapy. The remainder of the patient’s stay was uneventful and he was discharged on oral...
anticoagulation and bisoprolol 5 mg twice daily. He remained well at outpatient review and was commenced on amiodarone with successful electrical cardioversion 2 months later.

This case illustrates the importance of a provokable increase in gradient and the use of negative inotropes in HCM. Verapamil is routinely prescribed for its beneficial effect on diastolic dysfunction and reducing outflow tract gradients in selected HCM patients[1–3]. However, it must be used with caution as side effects including cardiac rhythm disturbances and heart failure, have been reported with this calcium-antagonist[4–7]. Initiation of verapamil in this patient precipitated pulmonary oedema secondary to a significantly increased outflow tract gradient, most likely due to peripheral vasodilatation. The pulmonary oedema was resistant to treatment with standard medical therapy and the use of esmolol, a short-acting beta-blocker, reversed the gradient leading to resolution of the patient’s symptoms. Esmolol has been reported to be of beneficial use in cardiac emergencies related to hypertrophic cardiomyopathy[8,9] but not in cases of pulmonary oedema.

**References**


