An 83-year-old woman with hypertrophic cardiomyopathy was referred with progressive exertional dyspnoea. She had marked apical and mid-ventricular hypertrophy with mid-ventricular obstruction and a small apical aneurysm (Panels A1–3, Supplementary material online, Video S1). Doppler echocardiography through the left ventricle (Panel B1) identified an early peak systolic gradient of 36 mmHg (Panel B1), mid-systolic cessation of flow (Panel B2), followed by re-emergence of forward flow in late systole and continuing into early diastole (paradoxical flow) (Panel B3). Cardiac catheterization demonstrated a 184 mmHg mid-ventricular gradient (Panel D), pulmonary artery wedge pressure of 32 mmHg, and normal coronaries. She underwent apical and mid-ventricular myectomy (Panels C1–3) which resulted in symptom improvement.

Flow through an area of obstruction is required in order to derive a Doppler pressure gradient. In our patient, complete mid-ventricular obstruction led to mid-systolic flow cessation through the left ventricular cavity and the absence of a Doppler signal to determine a pressure gradient. Therefore, the true peak mid-ventricular pressure gradient recorded on haemodynamic catheterization occurred during apical mid-systolic isovolumic contraction when cavity obliteration prevented ejection of flow from the apex (Panels B1 and D1–2). This supports the concept that significant elevations in apical intracavitary pressure due to severe mid-ventricular obstruction may contribute to the genesis of an apical aneurysm. Therefore, non-invasive Doppler echocardiography may significantly underestimate the magnitude of mid-ventricular obstruction. Mid-ventricular myectomy relieves mid-ventricular obstruction, and, in patients with significant secondary elevations in pulmonary artery wedge pressure, improves diastolic filling.

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