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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