A 67-year-old man, with known coronary artery disease, diabetes mellitus, renal insufficiency and hypertension, had undergone quadruple bypass surgery 7 years earlier for three-vessel disease, with internal mammary artery graft to the left anterior descending artery and saphenous vein grafts to the first diagonal branch, distal circumflex and right coronary arteries. A recent exercise electrocardiogram had to be interrupted because of a pathological blood pressure fall (drop in systolic pressure from 220 to 195 mmHg). Because no clinical or electrocardiogram signs of ischaemia were observed, an exercise echocardiogram with continuous echocardiography monitoring during exercise was performed.

Blood pressure at the beginning of the test was elevated at 150/100 mmHg. Rest images showed slightly increased left ventricular (LV) wall thickness (11 mm) and mass (117 g/m²). LV volume and ejection fraction (LVEF), calculated by biplane Simpson’s rule, were normal (LV end-diastolic volume [LVEDV], 41 mL/m²; LVEF 68%). Furthermore, Doppler indices suggested elevated LV filling pressures (septal E/e’, 23) (Fig. 1). Pathological blood pressure fall occurred again at low-level exercise (50 watts; 68% of age-predicted maximal heart rate) from 220 to 195–190 mmHg, accompanied by severe dyspnoea. Echocardiography did not disclose regional wall motion abnormalities, dynamic obstruction or new mitral regurgitation. Global LV volumes and LVEF remained stable (LVEDV, 42.5 mL/m²; LVEF, 71%). However, the study revealed severe pulmonary hypertension during
Dyspnoea and blood pressure fall at exercise: Usefulness of exercise echocardiography

Figure 1. Normal pulmonary pressure at rest and severe pulmonary hypertension at low-level exercise (tricuspid regurgitation pressure gradients 24 and 64 mmHg, respectively). Doppler indices suggest increase in left ventricular filling pressure during exercise (inversion of E/A ratio, shortening of mitral deceleration time [DT] and augmentation of septal E/e’ from 23 at rest to 42.5 at exercise).

Exercise with an increase in tricuspid regurgitation velocity from 2.45 m/s at rest to 4 m/s at exercise (Fig. 1). Furthermore, there was significant worsening of indices of LV filling pressure (inversion of E/A ratio; septal E/e’, 42.5) (Fig. 1). Subsequent pulmonary investigations were normal, including high-resolution computed tomography, pulmonary ventilation — perfusion scan, pulmonary function tests, 6-minute walk test and blood gas analysis at rest [PaO₂, 10.8]). Pulmonary embolism, chronic thromboembolic disease and underlying significant airway or parenchymal lung disease could be ruled out by these investigations. Left heart catheterization did not show pulmonary hypertension at rest or signs of restrictive or constrictive haemodynamic pattern but elevation of LV end-diastolic pressure to 22 mmHg.

Pharmacological 201-thallium myocardial perfusion scintigraphy with dipyridamole was normal, with no significant changes in LV volumes and LVEF between rest and peak pharmacological stress (Fig. 2). At 30-month follow-up, there were no significant changes in wall thickness and calculated LV mass. A repeat exercise echocardiogram after optimization of antihypertensive medication showed similar pulmonary pressure and LV filling variables at rest and exercise, however, at a slightly higher workload and without pathological blood pressure response.

Pulmonary hypertension due to LV diastolic dysfunction revealed by exercise echocardiography is most likely responsible for the pathological exercise test. The cause of the LV diastolic dysfunction is not completely clear, but is probably related to hypertensive heart disease. This case underlines that pathological blood pressure fall at exercise may occur without evidence of myocardial ischaemia or critical aortic stenosis.

Conflict of interest statement

None.