Patient with Hypertrophic Cardiomyopathy with Apical Aneurysm and Thrombus Presenting with Progressive Congestive Heart Failure

Christina Doesch1,2, Rainer Schimpf1,2, Stefan Haneder1,3, Martin Borggreve1,2, Theano Papavassiliou1,2

1First Department of Medicine, Cardiology affiliated at the DZHK (German Centre for Cardiovascular Research), 2DZHK partner site, Mannheim, Germany, 3Institute of Clinical Radiology and Nuclear Medicine, University Medical Center Mannheim, Medical Faculty Mannheim, University of Heidelberg, Germany

A 55-year-old woman presented with a progressive deterioration in physical work capacity and dizziness. Cardiac enzymes were within the normal range. The ECG showed sinus rhythm with left axis deviation, Q waves inferior, T-wave inversion anteroseptal, anterior, anterolateral and lateral, as well as a loss of R-waves in the precordial leads (Figure 1A, B). A cardiovascular magnetic resonance scan revealed hypertrophic cardiomyopathy (HCM) with mid-ventricular hypertrophy (maximum septal wall thickness 18 mm), an increased left ventricular mass index 110 g/m^2, slightly impaired left ventricular (LV) function (ejection fraction, EF 46%), an apical aneurysm resulting in an hourglass-shaped LV cavity, and a large apical mass (3.3 x 4.1 x 4.3 cm) (Figure 2A). Early enhancement, one minute after administration of contrast agent with a high inversion time of 400 ms, and late gadolinium enhancement (LGE) confirmed the diagnosis of an apical thrombus (Figure 2B). LGE images in the four- and two-chamber views show the large thrombus attached to the completely fibrotic LV apical aneurysm (Figure 2D, E). The basal short-axis view revealed the patchy LGE typical of HCM at the anterior insertion of the right ventricle (Figure 2C). The mid-ventricular short-axis view (Figure 2F) illustrated the beginning of the thrombus and showed extensive LGE from anteroseptal to inferoseptal and inferolateral. This large amount of LGE explains the ECG changes described above. Coronary angiography ruled out concomitant coronary artery disease.

Although the underlying pathological mechanisms are unknown, approximately 2% of patients with hypertrophic cardiomyopathy present with apical LV aneurysms.1 Despite anticoagulation with warfarin, the apical thrombus persisted during follow up. This case demonstrates the phenotypical diversity of patients with HCM.

References

Figure 1. See text for explanation.

Figure 2. See text for explanation.