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Apical hypertrophic cardiomyopathy masked as vasovagal syncope: a clinical pathway to implantable cardioverter defibrillator implantation

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Introduction: Apical hypertrophic cardiomyopathy (ApHCM) is a rare form of hypertrophic cardiomyopathy (HCM) in Western populations constituting around 1-10% of all HCM cases. It has traditionally been considered a more benign HCM phenotype with a lower risk of ventricular arrhythmias. However, recent studies show that patients with ApHCM remain at a substantial risk for serious cardiac events, especially when high risk factors are present.[1,2]

Case report: 60-year-old man with history of arterial hypertension presented with brief loss of consciousness, which corresponded to vasovagal syncope. He also reported mild non-anginal chest pain. However, 12-lead ECG showed signs of left ventricular hypertrophy, laboratory testing revealed mildly elevated hs-troponin I and NT-proBNP, all leading to hospital admission for further evaluation. Echocardiography showed preserved left ventricular systolic function (LVEF 60%) with predominant midventricular and apical hypertrophy, reaching 22 mm. There was no significant LVOT or midventricular obstruction. Coronary angiography was performed and showed a 90% stenosis of the posterior descending artery, which was successfully treated with drug-coated balloon angioplasty. Cardiac magnetic resonance confirmed predominantly midventricular and apical hypertrophy, with almost complete systolic obliteration of the apical cavity, without signs of apical aneurysm. Patchy mid-myocardial late gadolinium enhancement (LGE) was present in several apical segments and the apex, involving approximately 25% of the left ventricular myocardial mass. Genetic testing showed variants of uncertain significance. During continuous monitoring there were no significant ventricular arrhythmias recorded. The calculated HCM Risk-SCD was below 4%, indicating low sudden cardiac death (SCD) risk. However, taking into account high LGE burden, an implantable cardioverter defibrillator (ICD) implantation was recommended as the next therapeutic step, but patient initially opted for continued surveillance. Several months later, the patient remained asymptomatic, but 24-h Holter ECG monitoring documented one nine-beat episode of non-sustained ventricular tachycardia lasting 5.2 seconds. Recalculated HCM Risk-SCD remained below 4%. However, considering the newly documented NSVT, extensive LGE, primary-prevention ICD implantation was reconsidered and successfully performed.

Conclusion: ApHCM is a rare form of HCM which may present with a variety of symptoms. There is growing evidence that the risk of SCD is similar to risk in other HCM phenotypes, however traditional SCD risk scores may underestimate the need for ICD implantation. Extensive LGE and repeated assessment of arrhythmias can significantly influence the decision regarding ICD implantation and improve risk stratification, especially when calculated SCD risk remains low.

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