

CARDIOVASCULAR FLASHLIGHT

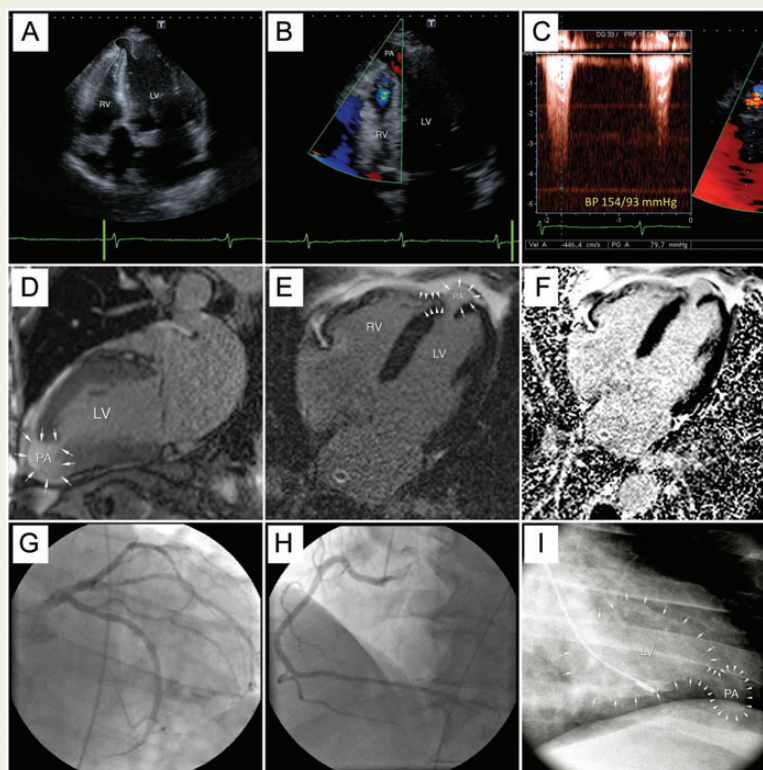
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Ventricular septal defect originating from an apical left ventricular pseudoaneurysmJan Vontobel¹, Urs Hufschmid¹, Michael J. Zellweger², and Bernhard C. Friedli^{1*}¹Cardiology, Department of Internal Medicine, Cantonal Hospital Baden, CH-5404 Baden, Switzerland and ²Department of Cardiology, University Hospital, Basel, Switzerland*Corresponding author. Tel: +41 56 486 26 36, Fax: +41 56 486 26 37, Email: bernhard.friedli@hin.ch

We report the case of a 63-year-old man with an apical trabecular ventricular septal defect (VSD) with a restrictive unidirectional left-to-right shunt (peak gradient 80 mmHg), originating from a perfused, dyskinetic apical pseudoaneurysm (PA; 38 × 31 mm) primarily diagnosed by transthoracic echocardiography (Panels A–C). The communication between the left ventricular (LV) cavity and the PA was narrow (8 × 10 mm). The pulmonary artery pressure was normal. Cardiac magnetic resonance imaging confirmed the LV apical lesions (the PA containing no muscular tissue; arrows in Panels D and E, arrow heads in Panel E) with a small shunt volume (Q_p/Q_s 1.15), a slightly hypertrophied right ventricle with good function and no signs of volume overload of the LV (LVEF 59%). A 12-lead ECG was normal and ergometry showed excellent exercise capacity and no ischaemia.

Pseudoaneurysms normally are the result of healed myocardial free wall rupture after myocardial infarction. Additional causes include local or systemic inflammation, trauma, or cardiomyopathies. Despite extensive diagnostic work-up, the patient surprisingly did not show any of them [normal late gadolinium enhancement images (Panel F), no significant coronary artery disease (CAD) at coronary angiography (Panels G–I)]. The patient's history without trauma, prolonged infective illness, and a cardiac murmur diagnosed at the age of 20 suggested congenital aetiology of both the PA and the VSD. The risk of myocardial rupture remains up to speculation. Transcatheter closure and cardiac surgery were therapeutic options, but the patient refused both. In an asymptomatic patient without CAD, a small restrictive VSD and a long history of cardiac murmur, a close clinical follow-up seemed to be the most reasonable approach.

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