Myocardial Infarction-induced Ventricular Septal Defect
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Clinical History
A 63-year-old man presented to an outside hospital with a two-week history of recurring chest pain. His presenting ECG was consistent with an evolving inferior wall myocardial infarction (MI). On physical exam he was tachycardic with a holosystolic 3/6 murmur on his left sternal border. He was transferred for emergent percutaneous coronary intervention (PCI) and further management.

Findings
Coronary angiography showed one vessel disease, with subacute thrombotic occlusion of the distal right coronary artery (Fig. 1). An echocardiogram (not shown) revealed a large basal ventricular septal defect (VSD) with left-to-right shunt, a small inferior basal pseudoaneurysm, normal biventricular function and valve morphology and function. The VSD was surgically repaired with a pericardial patch, and the pseudoaneurysm was excluded (David technique). The patient did initially well, but represented three months later with right-sided heart failure. An echocardiogram showed a residual VSD (Fig. 2), a large inferior basal pseudoaneurysm, reduced left ventricular ejection fraction (LVEF 38%) and right ventricular global hypokinesis. A cardiac CT (Fig. 3 and 4) was requested for surgical planning prior to repeat cardiac surgery and confirmed a residual VSD and a recurrent large inferior basal pseudoaneurysm. Unfortunately, surgical repair of the residual VSD, pseudoaneurysm or the implantation of a ventricular assist device were considered to be technically unfeasible. The patient underwent cardiac transplantation, but eventually died of the complications of an invasive pulmonary aspergillosis.

Discussion
VSD is a mechanical complication of myocardial infarction and typically occurs three to five days after an acute MI. Nevertheless it has been observed also within the first 24 hours or as late as two weeks [1]. In a study of 6678 consecutive MI patients during the last 30 years, it accounted for about 2% of the total population [2]. The incidence has been diminishing over the last decades thanks to the introduction of timely reperfusion therapy [2]. VSD is observed with equal frequency in anterior and non-anterior infarctions [3]. In case of an anterior MI, the defect is most commonly found in the apical septum; in inferior MI, it usually occurs in the basal segments.

Rupture develops at the limit between the necrotic and non-necrotic myocardium. The defect can be either a direct through-and-through hole, or have a more irregular and serpiginous path [4]. The size of the VSD determines the magnitude of left-to-right shunt, which in turn correlates with survival. Cardiac CT has excellent spatial resolution, and is well suited to delineate the morphology and extent of pseudoaneurysm and septal defects.

REFERENCES
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