Treatment strategies for a giant left ventricular aneurysm and developing ventricular septal defect in a patient after anterior wall myocardial infarction

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A 66-year-old woman was readmitted to the hospital due to progressive severe heart failure (New York Heart Association class III/IV) 2 months after acute ST-segment elevation myocardial infarction of the anterior wall and percutaneous coronary intervention with implantation of 3 drug-eluting stents in the left anterior descending artery (Thrombolysis in Myocardial Infarction flow grade 1; no significant changes in other coronary arteries) (FIGURE 1A–1B). On admission, the levels of N-terminal pro–B-type natriuretic peptide were 14 000 pg/ml; international normalized ratio, 1.7; and bilirubin, 4.5 mg/dl. Transthoracic echocardiography revealed a giant left ventricular (LV) aneurysm (LVA) involving a half of the distal part of the interventricular septum (IVS), the apex, as well as the inferior, anterior, and lateral wall, with LV ejection fraction of 24% and LV end-diastolic volume of 272 ml. The IVS thickness at the level of the aneurysm was 2.5 mm. In the middle part of the IVS, a small ventricular septal defect (VSD) was found (FIGURE 1C). Cardiac magnetic resonance imaging confirmed these findings: it revealed an extensive, dyskinetic LVA involving the apex and all distal segments. The VSD in the middle part of the IVS had the maximum width of 4.5 mm (FIGURE 1C–1D). The disease progressed rapidly. Over the next days, the VSD diameter increased to the maximum width of 6.5 mm (FIGURE 1E). The patient was hemodynamically unstable and required inotropic agents, diuretics, and intra-aortic balloon pumping. The Heart Team discussed the case several times. The patient was considered ineligible for percutaneous VSD closure because of the IVS thickness and spiral shape of the VSD. Due to the risk of too low LV volume, the patient was also considered ineligible for endoventricular circular patch plasty. While awaiting the heart transplant, the patient developed progressive signs of cardiogenic shock with right ventricular decompensation secondary to VSD progression. Because of unstable condition, increasing decompensation, and no possibility of heart transplant, all treatment strategies were re-evaluated and a lifesaving surgical ventricular reconstruction with VSD excursion and mitral and tricuspid valve annuloplasty was successfully performed (FIGURE 1F). The patient survived the operation but died on day 4 after the surgery due to complications unrelated to the procedure.

Our case illustrates difficult decision making in the treatment of a patient with a VSD in progress localized in the region of the LVA requiring urgent intervention. An LVA is a common complication of acute myocardial infarction and occurs in 10% to 38% of patients.[1,2] The mortality in patients with an LVA is up to 6-fold higher than in those without this condition.[3] An LVA carries a risk of rupture; therefore, it constitutes an indication for surgical treatment. However, postoperative outcomes, including survival, are affected by the size of an aneurysm and the ejection fraction of the remaining "healthy" part of the left ventricle. Thus, multimodality imaging is necessary to obtain full details of LV morphology and to differentiate between an aneurysm

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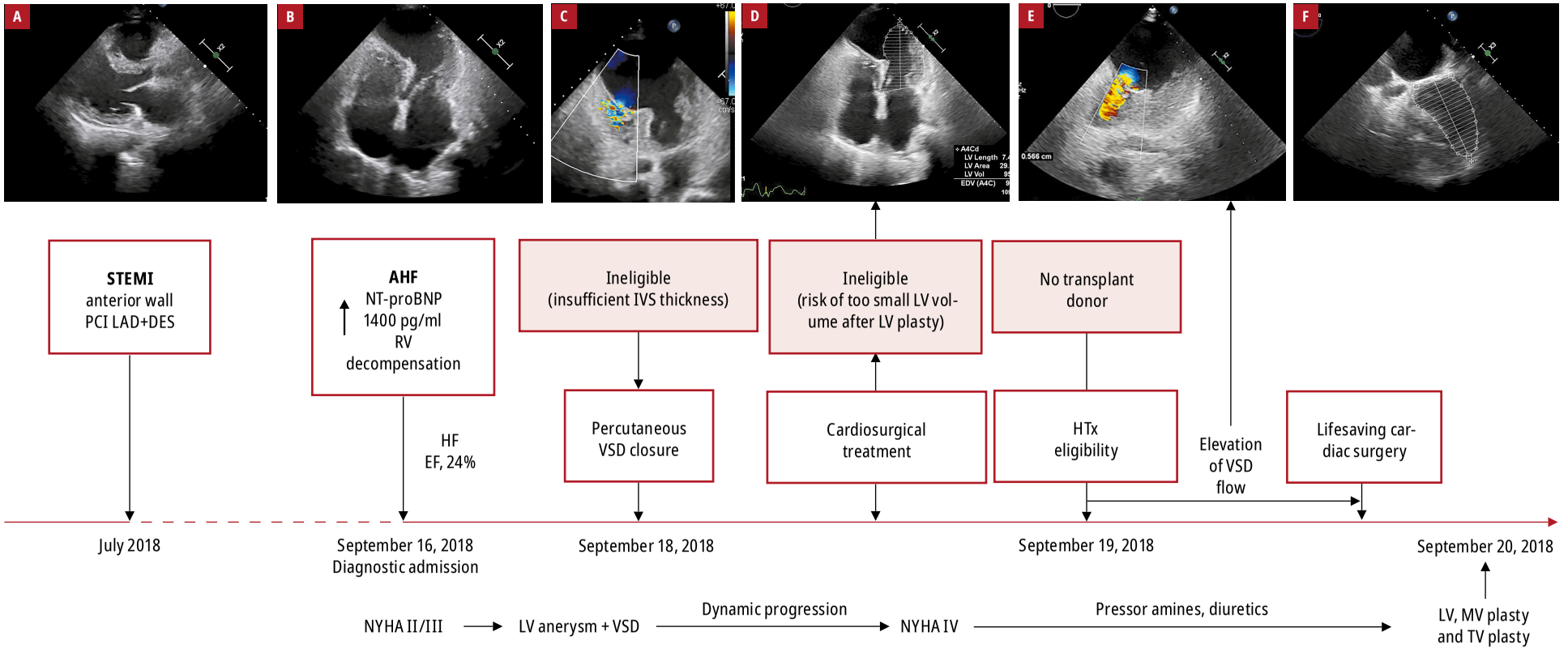

FIGURE 1 The management and imaging of a giant left ventricular aneurysm (**A–E**, transthoracic echocardiography; **F**, transesophageal echocardiography): **A** – long-axis view; **B** – left ventricular aneurysm; **C** – ventricular septal defect; **D** – left ventricular aneurysm before surgery; **E** – increased blood flow in the ventricular septal defect; **F** – the left ventricle after surgery

Abbreviations: AHF, acute heart failure; DES, drug-eluting stent; EF, ejection fraction; HF, heart failure; HTx, heart transplant; LAD, left anterior descending artery; LV, left ventricular; MV, mitral valve; NT-proBNP, N-terminal pro–B-type natriuretic peptide; NYHA, New York Heart Association; PCI, percutaneous coronary intervention; RV, right ventricular; STEMI, ST-segment elevation myocardial infarction; TV, tricuspid valve; VSD, ventricular septal defect

and a pseudoaneurysm.[4] In-hospital VSD development is rare, and its diagnosis, treatment, and monitoring require the best invasive strategy. All these data were considered in the life-saving treatment of our patient.

ARTICLE INFORMATION

CONFLICT OF INTEREST None declared.

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