

PLUG THE VSD-CHANNEL DESPITE OF SEPTAL PATCHES AND NON-TRIVIAL ANATOMY

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HISTORY AND PHYSICAL

On 12th of December last year, the 60-years old male patient developed symptoms of cardiogenic shock. ECG showed ST-segment elevations in the inferior leads. Echocardiography unveiled a 24-mm measuring defect of the ventricular wall. Catecholamines could not control hypotonic circulation, so a VA-ECMO was installed. Three L/min and 10 mg/h dobutamine were sufficient to stabilize the patient. Coronary angiography showed an occlusion of the proximal LAD (receiving collaterals from RCA) as well as the distal CX, significant stenosis of the RCA. On 15th of December, the VSD could be fixed via a patch plastic, LAD and CX were bypassed via LIMA and a venous graft, respectively. No rest shunt was visible in echocardiography afterwards. ECMO could be weaned on 18th of December and the patient could leave the hospital on 31st of December. Well, already on 4th of January he was sent back from rehabilitation. Transesophageal echocardiography showed an VSD of 13 mm, just below the valves. A shunt volume of 8.2 L/min was quantified invasively, so re-VSD patch operation was indicated and performed on 17th of January with good result. However, on 23rd of January cardio-respiratory deterioration took place. Diagnostics displayed pericardial effusion as well as right-sided hemothorax, which was treated with his 3rd operation. On 25th of January he could be moved to the intermediate care station. Echocardiography showed a recurrent VSD, right heart catheterization displayed a significant shunt again, hence – another open surgery was technically not doable – an invasive attempt to close the shunt was scheduled for 7th of February, which remained unsuccessful. After thorough reviewing the images (CT, MRI, echocardiography), showing an intraseptal channel, another interventional approach was scheduled. In the case of failure, heart transplantation would be his treatment option.

IMAGING

Coronary angiography: 13th of December: proximal occlusion of LAD (collaterals via RCA), distal occlusion of CX, high-grade stenosis of the proximal RCA.

Echocardiography: 12th of December: initial echo in our hospital showing a VSD of about 20 mm. Several echocardiographies followed. TOE on 10th of March monitored the interventional therapy.

CT scan 13th of December and 3rd of February: ventricular septal defect.

Cardiac MRI: 2nd of February: inferioseptal VSD, channel in baso-caudal direction.

INDICATION FOR INTERVENTION

The patient suffered from dyspnea NYHA III-IV, open surgery was not doable after two surgical patch insertions.

INTERVENTION

On 10th of March the channel could be probed via a left sided approach. A wire could be advanced into the channel under TOE control. Using an 8-F guide catheter, the 8 mm Amplatzer™ muscular VSD occluder could be placed into the proximal part of the channel. Assessed via echocardiography as well as angiography, the shunt appeared to be significantly reduced, with a weak jet just through the meshes of the occluder. Monotherapy with ASS should allow thrombose.

LEARNING POINT OF THE PROCEDURE

Interventional opportunities should be maxed out before sending patients to heart transplantation. Especially in patients with symptomatic heart failure due to left ventricular shunting innovative and individual plug technics should be considered. Skillful interventional occlusion of the VSD was possible after thorough examination of the anatomy and pathology via 3D echocardiographic guidance, which paved the way to successful intervention in the catheter lab. The patient is still in clinic and recovering.