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A case study of post-myocardial infarction ventricular septal defect. Is there always a solution to this deadlock?

Przypadek kliniczny pacjenta z niedokrwiennym ubytkiem w przegrodzie międzykomorowej. Czy istnieje możliwość wyjścia z tego impasu?

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Abstract

This case report describes a patient with an ischaemic inferior wall aneurysm coincidental with a large post-myocardial infarction ventricular septal defect (VSD) treated with surgical closure of VSD combined with coronary artery bypass grafting. It is emphasized that there is a pivotal need for an individualized approach considering the timing of treatments, the need to stabilize haemodynamic status, and the choice between surgical, percutaneous closure, or palliative therapy.

Key words: cardio surgery, myocardial infarction, ventricular septal defect

Folia Cardiologica 2022; 17, 3: 183–186

Introduction

Post-infarct ventricular septal defects (VSDs) are severe but rare acute myocardial infarction (MI) complications. The post-infarct VSD prevalence has declined due to the early reperfusion therapy [1]. Nevertheless, patients with delayed ST-segment elevation myocardial infarction (STEMI) presentations are at risk for this often-lethal complication. Cardiosurgical repair is the decisive treatment, but it complements high morbidity and mortality. This case report describes a patient with an ischaemic inferior wall aneurysm coincidental with a large post-MI VSD treated with surgical closure of VSD combined with coronary artery bypass grafting.

Case report

A 76-year-old man presented to a hospital following a 2-week history of dyspnoea on exertion and typical chest pain. A 12-lead electrocardiogram revealed left bundle branch block; T wave inversion in inferior leads. The clinical presentation and electrocardiographic changes were suggestive of prior inferior wall myocardial infarction.

He was on acetylsalicylic acid, bisoprolol, and atorvastatin. On physical examination, a pansystolic murmur on auscultation had been audible, with bilateral, fine basal crepitations.

Differential diagnoses included viral and bacterial respiratory tract infection and pneumonia, including

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COVID-19 infection; acute myocardial infarction (AMI) with possible mechanical complications, decompensated heart failure; and pulmonary embolism.

Respiratory viral tests were negative for influenza A and B. Meanwhile, reverse transcription-polymerase chain reaction (RT-PCR) testing was negative for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2).

Due to the elevated D-dimer level patient underwent a computed tomography angiogram of pulmonary arteries that revealed lobar pulmonary embolism. Doppler ultrasound displayed lower extremities bilateral deep vein thrombosis.

The patient remained haemodynamically stable with a systolic blood pressure hovering between 90–100 mm Hg thanks to cautious fluid resuscitation. His renal function remained stable at the KDIGO G3b class.

Transthoracic echocardiogram (TTE) demonstrated an ejection fraction of 45% with concomitant dyskinetic inferior wall and inferior septum. Ruptured and dissected inferior septum coexisted with an oblique, tortuous left-to-right shunt with a neck of 30 mm (Figure 1A, 2A and 3A). Moreover, moderate tricuspid regurgitation with an estimated systolic pulmonary artery pressure of 50 mm Hg was noted. Right ventricular function was mildly reduced (tricuspid annular plane systolic excursion of 16 mm; S'10 cm/s).

The patient's coronary angiography displayed left main and triple vessel disease: tight proximal lesion in the left anterior descending artery and its first diagonal branch; tight proximal circumflex artery lesion (Figure 4); and right coronary artery proximal amputation (Figure 5) corresponding to previous inferior wall myocardial infarction. Multivessel coronary disease indicated surgical repair over transcatheter closure.

After extensive discussion with the two independent cardio surgery centres, the patient was disqualified from percutaneous closure of VSD. The Heart Team believed that cardiosurgical intervention was required and would aid this patient due to the need for concomitant coronary artery bypass grafting and detrimental myocardial rupture location.

He did receive a low-molecular-weight heparin injection, a beta-blocker, and a loop diuretic intravenous infusion. According to European Society of Cardiology guidelines [2], intra-aortic balloon pump counterpulsation was considered due to cardiac output and coronary blood flow improvement; however, the patient remained haemodynamically stable.

Subsequently, the patient underwent coronary artery bypass grafting with first grafting to a left anterior descending artery and consecutive open surgical repair of VSD with a Dacron patch and a buttressing pledget of Teflon. After the procedure, he remained in a serious but haemodynamically stable condition. The renal function deteriorated with a GFR of 16 mL/min/1,73 m^2 on the 14th postprocedural day. A repeat transesophageal echocardiography demonstrated no residual shunt.

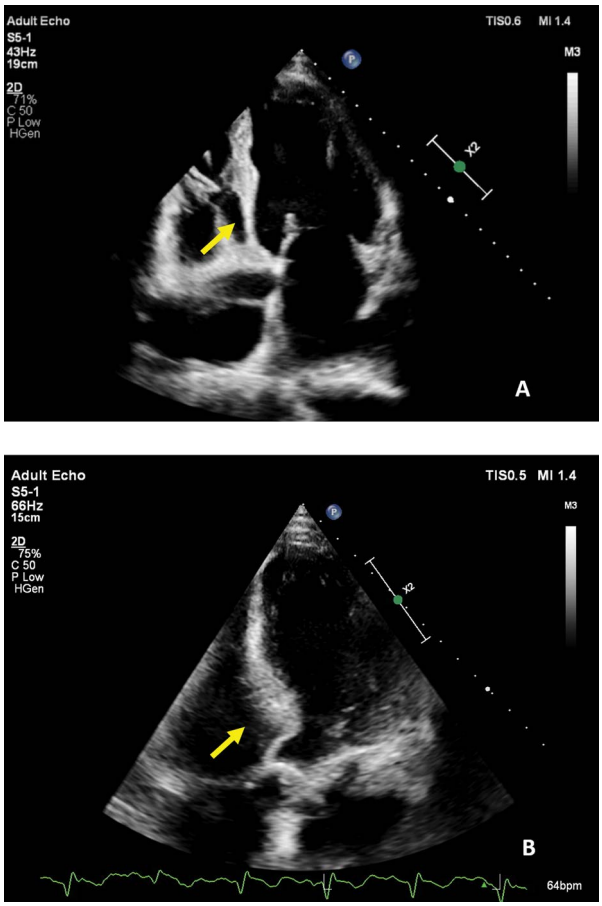

Figure 1. Rupture and dissection of the inferior septum (indicated by the arrows) before (**A**) and post-surgical repair (**B**) of ventricular septal defect with a Dacron patch (apical 4-chamber view)

A 3-month outpatient follow-up revealed the patient's clinical state improvement without dyspnoea and no residual shunt in TTE (Figure 1B, 2B and 3B).

Discussion

Ventricular septal defects complicating STEMIs are associated with high mortality, prevailing at 50% even after surgical repair [3]. Fatality is significantly higher in patients of advanced age [4].

Owing to bidirectional heart damage affected by AMI and sudden-onset cardiac shunt [5], patients with post-infarct VSD are likely to be haemodynamically compromised.

Nevertheless, there are no unequivocal scientific studies analysing surgical and percutaneous closure of post-infarct VSD. Surgical closure offers a negligible improvement in mortality compared to pharmacotherapy; however, benefits are not apparent due to myocardial necrosis in the first two weeks after AMI with subsequent fibrosis of VSD's edges and friable scar formation. Moreover, surgical intervention has self-constraints, including the restriction

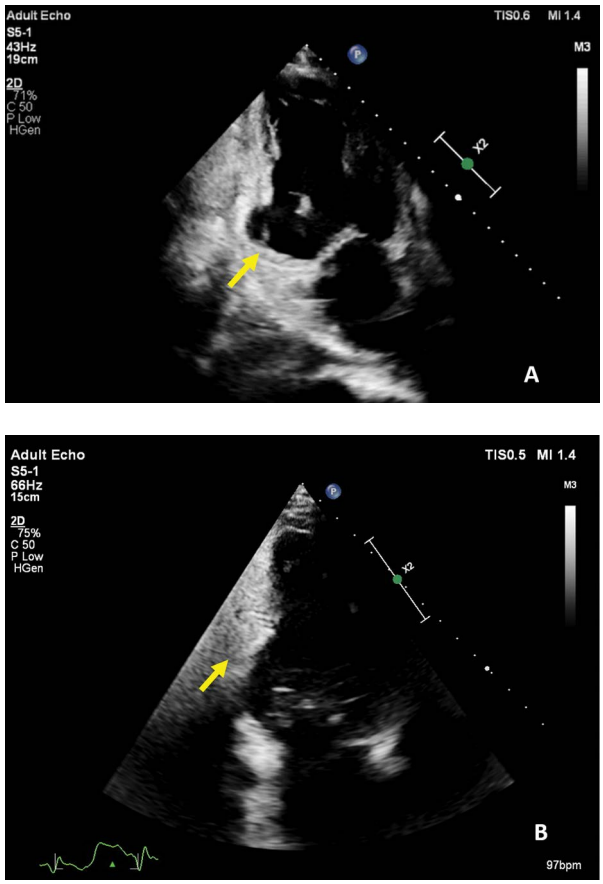

Figure 2. Rupture and dissection of the inferior septum (indicated by the arrow) before (**A**) and post-surgical repair (**B**) of ventricular septal defect (apical 2-chamber view)

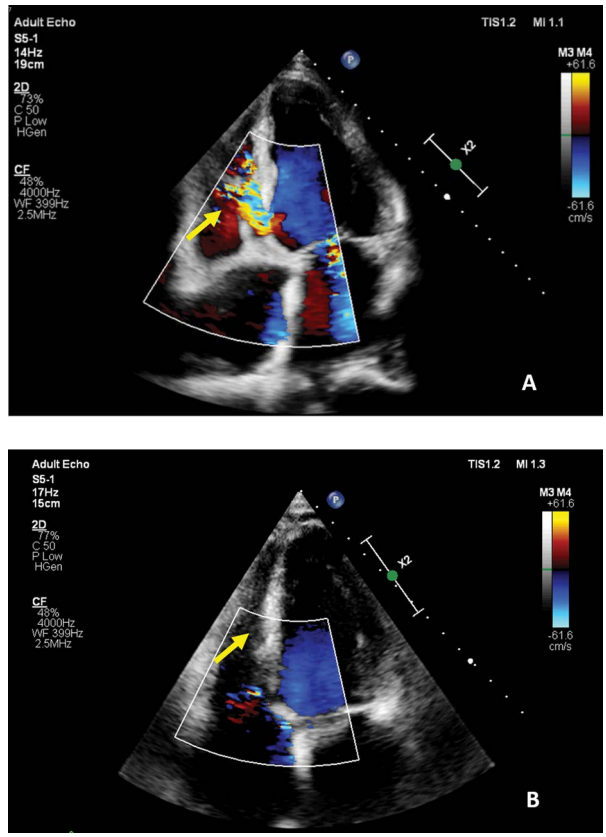

Figure 3. A tortuous left-to-right shunt with a neck of 30 mm before (**A**) and post-surgical repair (**B**) of ventricular septal defect with a Dacron patch (indicated by the arrows)

of advanced age and surgical expertise that is not easily accessible [6].

Anatomical factors favouring surgical repair are large defect size (> 24 mm), the need for complimentary coronary artery bypass grafting, accompanying severe valvular disease, multiple serious defects, as well as a previous failure of transcatheter repair, institution expertise, and patient's decision [5].

Transcatheter closure of post-infarct VSD could be an encouraging alternative treatment method that is less invasive and advocated by national guidelines [5]. Although an absence of firm tissue to seat the device for significant defects may be problematic [6]. Post-infarct VSD is usually diverse in shape, which may change throughout the cardiac cycle and could be accompanied by septal rupture [7], which led in the present case to the decision of surgical intervention.

According to Sabiniewicz et al. [8], reporting over a decade-long experience with transcatheter closure of the post-infarction ventricular septal defect, only technical success of the procedure and white blood count before the procedure were significantly associated with 30-days mortality.

In this patient's case, surgical repair seemed to be applicable, with benefits outweighing the risk according to the defect size and its serpiginous morphology, as well as the presence of multivessel coronary disease requiring bypass grafting.

Conclusion

The case highlights that VSDs should remain a priority in differential diagnoses in patients with a delayed presentation of a STEMI. The authors emphasize that every postinfarct VSD is unique. There is a pivotal need for an individualized approach considering the timing of treatments, the need to stabilize haemodynamic status, and the choice between surgical, percutaneous closure, or palliative therapy.

Conflict of interest

None declared.

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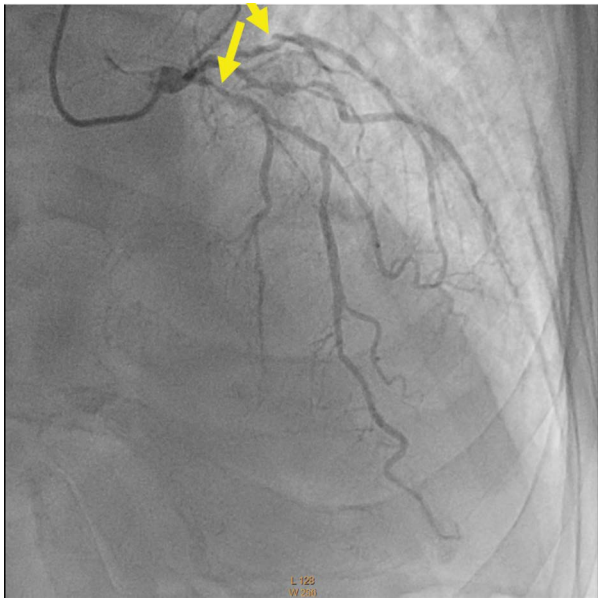

Figure 4. A tight proximal lesion in the left anterior descending artery and its first diagonal branch; tight proximal circumflex artery lesion (right anterior oblique; caudal view)

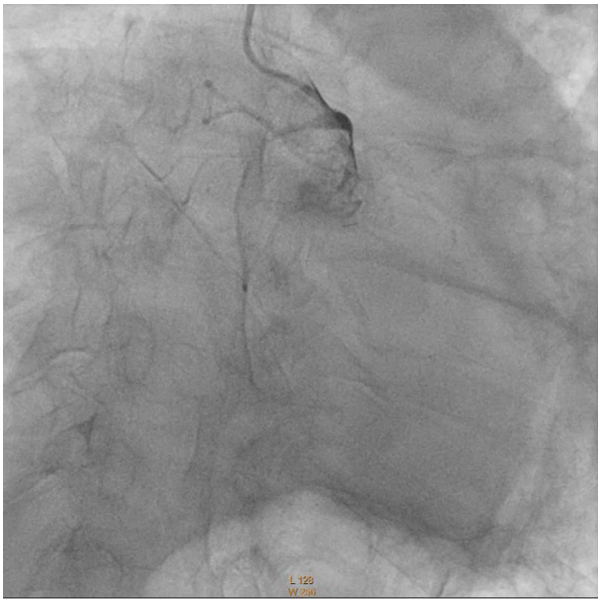

Figure 5. Right coronary artery proximal amputation (right anterior oblique projection)

Streszczenie

W niniejszej pracy przedstawiono przypadek pacjenta z niedokrwiennym tętniakiem ściany dolnej lewej komory ze współistniejącym ubytkiem w przegrodzie międzykomorowej leczonym chirurgicznie przy jednoczesnym pomostowaniu aortalno-wieńcowym. Autorzy chcieliby podkreślić wagę zindywidualizowanego podejścia do ubytku w przegrodzie międzykomorowej, w tym wyboru momentu leczenia, potrzeby wyrównania statusu hemodynamicznego pacjenta oraz wyboru metody terapii: chirurgicznej, przezskórnej czy paliatywnej.

Słowa kluczowe: leczenie kardiochirurgiczne, ostry zespół wieńcowy, ubytek przegrody międzykomorowej

Folia Cardiologica 2022; 17, 3: 183–186

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