Post-Myocardial Infarction Ventricular Septal Defect: Still a Deadly Complication 30 Years after the First Transcatheter Closure

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KEY WORDS: intracardiac shunt, percutaneous coronary intervention (PCI), surgical closure, transcatheter closure, ventricular septal defect (VSD)

IMAJ 2018; 20: 720–721

Post-myocardial infarction (MI) ventricular septal defect (VSD) is a rare complication in the era of primary percutaneous coronary intervention (PCI) [1,2]. Still, it is associated with a high mortality rate [3]. Our clinical case emphasizes the complexity of the management and the transcatheter closure of this type of VSD, especially 1 week after a myocardial infarction. Successful closure could decrease the 30 day mortality rate to 30–40%.

A diagnosis of VSD should be considered in cases of pathologic cardiac auscultation and confirmed by emergent transthoracic echocardiography (TTE). Hemodynamic stabilization, mainly with the insertion of an intra-aortic balloon pump (IABP), is the first step in its clinical management [4]. With an IABP, there may be a decrease in the left-to-right shunt, afterload, and oxygen consumption. It also increases the coronary perfusion. In cases of refractory cardiogenic shock, a veno-arterial extracorporeal membrane oxygenation (ECMO) should be considered.

We describe a clinical case to emphasize the complexity of the management of a VSD complication. It is one of the rare cases cited in the literature in which the transcatheter closure of VSD was performed after 7 days.

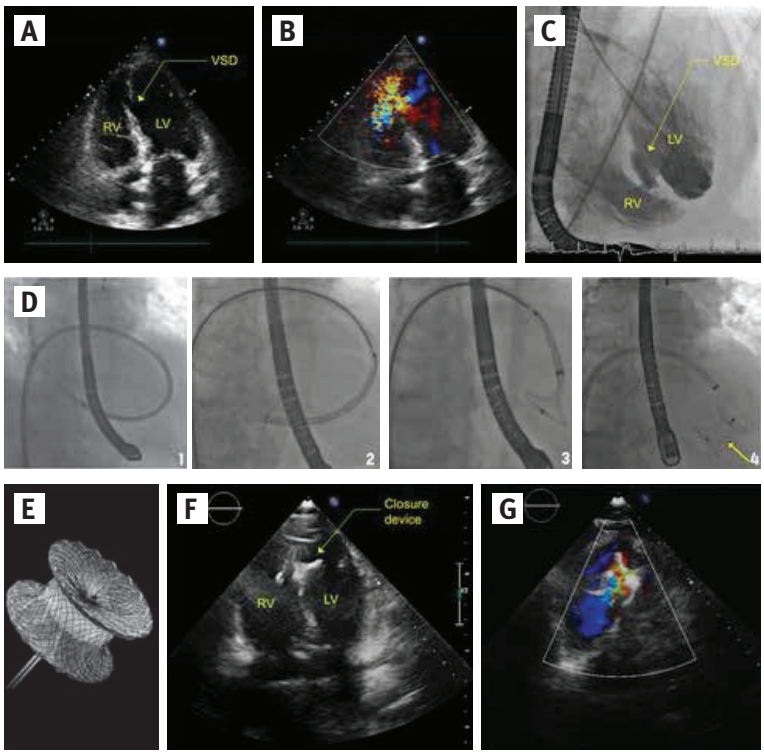

Figure 1. Post-myocardial infarction ventricular septal defect: a deadly complication

[A] Transthoracic echocardiogram (TTE) apical view showed the presence of a large ventricular septal defect (VSD) **[B]** TTE apical view highlighted with color Doppler showed left-to-right shunt through the VSD **[C]** The left anterior oblique at a 45 degree and cranial 25 degree ventriculogram showed a VSD with sufficient distance from the apex and a 14 mm defect size **[D]** The steps of the percutaneous VSD closure with the insertion of the closure device using a transeptal access via the transfemoral venous route. The defect was crossed using a balloon tip catheter, and an Amplatz Super Stiff™ ST-1 guidewire (Boston Scientific, USA) was subsequently positioned in the right pulmonary artery to advance the 12F delivery system **[E]** A 20 mm Amplatzer™ Post-MI VSD Occluder (St. Jude Medical, USA) **[F]** TTE apical view showed the device at the level of the interventricular septum **[G]** TTE apical view highlighted by color Doppler showed a mild residual leak

A hypertensive 89 year old woman was admitted to our institution 72 hours after complaining of chest pain. The electrocardiogram (ECG) showed an inferior ST-elevation myocardial infarct (STEMI). Clinical examination revealed sinus tachycardia, normal blood pressure, and holosystolic murmur on the precordium graded 4/6 with jugular venous distension. Urgent TTE showed a left ventricle ejection fraction of 45% with inferoseptal and inferior akinesia as well as the presence of a large VSD with left-to-right shunt highlighted on the color Doppler [Figure 1A, 1B].

Coronary angiogram revealed a sub-occlusion of the posterior descending, and retroventricular arteries were treated by two drug eluting stents due to ongoing ischemia. Right heart catheterization confirmed an important left-to-right shunt and low cardiac output. The left ventriculogram showed a VSD with sufficient distance from the apex and a 14 mm defect size [Figure 1C]. An IABP was inserted.

The subsequent modality of closure, either surgical or transcatheter, as well as the ideal timing, was discussed in the heart team. Past research has suggested a post-MI VSD closure at least 7–10 days or later after the STEMI because of the risk of a crumbly septum muscular wall in the acute setting. Successful closure decreases the 30 day mortality rate to 30–40% [3,4].

After 9 days, the transcathteter closure of this VSD was performed with a 20 mm Amplatzer™ Post-MI VSD Occluder (St. Jude Medical, USA) [Figure 1E]. Figure 1D shows the steps of the transcatheter VSD closure with the insertion of the closure device using a transeptal access via the transfemoral venous route. The defect was crossed using a balloon tip catheter, and an Amplatz Super Stiff™ ST-1 guidewire (Boston Scientific, USA) was subsequently positioned in the right pulmonary artery to advance the 12F delivery system. A mild residual leak was seen on post-procedural TTE [Figure 1F, 1G]. The patient was treated with aspirin and clopidogrel.

It is important to note that in the acute setting, the closure of the VSD decreased the shunt but it did not eliminate it completely. Maintenance of the IABP at least 48 hours or more, depending on the hemodynamic repercussion, should be considered. Unfortunately, in our case, 1 day after the insertion of the IABP, the patient still could not be weaned and the patient requested a therapeutic withdrawal.

Although 30 years have passed since the first transcatheter closure, the best timing of post-MI VSD closure and the regimen of antiplatelet therapy after the closure remains uncertain [5]. Surgical or transcatheter closure after 7 to 10 days could ideally be the best timing due to the weakness of the septum; however, in case of hemodynamic instability, this timing may not be observed. Treatment with aspirin alone for 6 months seems to be proper treatment. In cases of persistent significant shunt, antiplatelet therapy would not increase success. However, progress in the field of percutaneous interventions and mechanical support could improve the outcomes of this kind of rare and severe complication.

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Capsule

The genetic basis and cell of origin of mixed phenotype acute leukaemia

Mixed phenotype acute leukaemia (MPAL) is a high-risk subtype of leukaemia with myeloid and lymphoid features, limited genetic characterization, and a lack of consensus regarding appropriate therapy. **Alexander** and colleagues showed that the two principal subtypes of MPAL, T/myeloid (T/M) and B/myeloid (B/M), are genetically distinct. Rearrangement of *ZNF*384 is common in B/M MPAL, and biallelic *WT*1 alterations are common in T/M MPAL, which shares genomic features with early T-cell precursor acute lymphoblastic leukaemia. The authors showed that the intratumoral immunophenotypic heterogeneity characteristic of MPAL is independent of somatic genetic variation, that founding lesions arise in primitive haematopoietic progenitors, and that individual phenotypic subpopulations can reconstitute the immunophenotypic diversity in vivo. These findings indicate that the cell of origin and founding lesions, rather than an accumulation of distinct genomic alterations, are prime tumor cells for lineage promiscuity. Moreover, these findings position MPAL in the spectrum of immature leukaemias and provide a genetically informed framework for future clinical trials of potential treatments for MPAL.

Nature 2018; 562: 373

Eitan Israeli

"If life had a second edition, how I would correct the proofs"

John Clare, (1793–1864), English poet