INDIAN HEART JOURNAL 68 (2016) 99–101

Available online at www.sciencedirect.com

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Review Article

Transcatheter closure of post-myocardial infarction ventricular defect: Where are we?

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ARTICLE INFO

Article history:
Received 29 April 2015
Accepted 29 June 2015
Available online 18 January 2016

Keywords:
Post-myocardial infarction
ventricular septal defect
Transcatheter closure
Challenges

ABSTRACT

Since 1988, TCC of PMIVSD became an alternative treatment for anatomically suitable patients with high risk of surgical closure. TCC is less invasive but the optimal timing and technique have not developed much in the last four decades. The dismal prognosis at the contemporary sight should not be discouraged. The rapid innovation in TAVI is an example. The learning curve slopes down to the line of inertia in inaction. Some innovations have slept for centuries but their potential needs to be celebrated. The published experience of TCC of PMIVSD across the globe is limited as they are based on consensus. The experience related to clinical practice has heterogeneous topography around the globe because of the morbid pathology. The increasing number of onsite cardiothoracic wings, better imaging tools, LVADS, and ECMO, along with improvement in well matching hardware to the pathology of PMIVSD, build incremental confidence. The improved outcomes believes in the enthusiasm of closing the PMIVSD using either surgical or TCC approach and is recommended.

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There is phenomenal reduction in the incidence of mortality due to coronary artery disease from 1960 to 2015, which is reflected as lower incidence (0.2%) of PMIVSD. The mortality rate is more than 90% at the end of the first 2 months with current medical management. The natural history shows bimodal peak of occurrence of PMIVSD. The first and second peaks happen in 16–24 h and in 3 and 5 days after MI, respectively. The limited time interval between the detection of PMIVSD with cardiogenic shock and death is really a challenge to manage in the different hospital settings. The surgically treated patients suffer a mortality between 20% and 60%.[1] The pre-operative hemodynamic stability improves mortality rate but guideline opposes this waiting. The operative mortality rate was 54% when the surgery was done within 7 days and it reduced to 18% when the surgery was done after 7 days.[1] The mortality was quite high when the surgery was done in an emergency basis, especially within 6 h of MI. The incidence of residual defect after surgical repair of post-MI VSR was noted to be around 10–40%.[2] The TCC of PMIVSD is very useful when done after a an interval of primary PCI, after 2–3 weeks of MI, residual leak closure after surgery, and as a bail out or salvage where surgical closure weighs more risk than benefit.[3,4] The residual shunting, ventricular rupture, device embolization, and adverse events experience during follow-up in some patients are seen in 41% of patients when TCC is done in acute (<2 weeks) and subacute (2–4 weeks) conditions.[4,5] The contraindications for transcatheter therapy include defect size >35 mm, basal VSR near mitral or aortic

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http://dx.doi.org/10.1016/j.ihj.2015.06.038
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valves, and apical VSD without sufficient margins. Of course, earlier revascularisation (primary PTCA or delayed PTCA) does reduce incidence of VSR and size of VSR, and restores hemodynamic stability, good for either surgical or device closure. Because of the serpiginous border, a significant number of VSR failed to qualify for device closure. The heterogeneity in the incidence of mortality with surgical or TCC in acute or subacute cases is explained by timing of surgery for VSR, cardiogenic shock, and recurrence of VSR, posterior VSR, and inferior MI complicated with mitral regurgitation and renal failure. In the current scenario, the mortality is quite high in either surgical or TCC when performed in the first 2–3 weeks.[6] Because most of the deaths occur during in the first 2 months without intervention, American College of Cardiology and American Heart Association recommend immediate surgical repair, irrespective of shunt size. Of note is that transcatheter closure of PMIVSR is off label.

The first report of PMIVSR dates back to Latham's first description of PMIVSR at autopsy in 1847. In the year 1923, Brunn made first ante-mortem diagnosis of PMIVSR. In the year 1934, Sager established specific clinical criteria for diagnosis, stressing the association of PMIVSR with coronary artery disease. In an article in 1957, Cooley et al. performed first surgical repair of PMIVSR. In 1988, Lock et al. reported first the transcatheter closure of VSR using cardioseal device, based on their large experience of TCC of congenital VSD, and since then approximately 300 cases of TCC have been reported worldwide in the form of case reports, and small series with success rate of approximately 80–90%.[3] The periprocedural use of hemodynamic support like IABP, ECMO, and Impela device provides stability but the delay in intervention is supported by consensus. There are several innovations that have been introduced in this arena and are in pipeline. In the article in 2015, Zhou et al.[7] reported the use of parachute device in case of PMIVSD with left ventricular aneurysm. Cinq-Mars et al.[8] in an article titled ''The novel use of heart transplantation for the management of a case with multiple complications following acute myocardial infarction'', first described the role of heart transplantation to salvage a patient of PMIVSR. Ari et al.[9] reported in 2012 percutaneous Closure of PMIVSR in a patient with left ventricular apical thrombus, as a first published report. The choice of occluder is no more restricted to dedicated PMIVSD occluder because of varying anatomy and difference of time intervention from operator to operator.[10] The waist size of PMIVSD occluder should comparatively be larger by 6–8 cm when the size of VSD is compared, because of their soft and friable border. The exact three-dimensional size and shape should be well perceived before intervention. The basic imaging tools are 2D and 3D echocardiography for diagnosis and intervention. In the cases of complicated anatomy, cardiac MRI would be complimentary or can replace left ventricular angiogram in this aspect. Transjugular approach is preferred to transfemoral approach. Balloon dilatation is usually discouraged in acute or subacute setting to assess the size of VSD. Risk stratification can be supported by EURO score. The contrast-induced nephropathy and device embolization following it should not be ignored in the acute and subacute setting because the intervention in these cases is all about hemodynamically unstable patients and friable border of PMIVSD. The meta-analysis reported by De Puy et al.[3] shows the procedural success rate of 80–90% with TCC. There are two case reports and one small series has been reported from India.[10–12]Though there is drastic reduction in the incidence of PMIVSR with improvement in pharmaco-invasive therapy from 3% to 0.17% in APEX-MI registry,[13] this should neither encourage cardiologists to sit cross legged nor be completely biased by AHA/ACC decision saying that surgery is indicated in this condition irrespective of clinico-hemodynamic aspect. A scientist makes science and not the reverse. The device closure of PMIVSD is currently the second choice and is in the upslope of the learning curve. Indian researchers should take more initiative in this regard to add to the experience of transcatheter closure of PMIVSR using hemodynamic supportive measures like ECMO, Impella and IABP to improve periprocedural results. The results with surgical closure are closely similar to TCC. Therefore, TCC should be encouraged whenever hemodynamic condition and morphology are suitable to device closure. The coronary artery disease burden and certainly its related complication would arise in the coming days in India. Therefore, the cardiologist, as evidenced, should show interest in TCC as a viable alternative.

Conflicts of interest

The author has none to declare.

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