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Editorial

The Ongoing — and Resurgent — Challenge of Post-Infarct Ventricular Septal Defect Management

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A curious, but important, collateral impact of the shelter-in-place requirements occurring during the COVID-19 pandemic has been the reduction in patients presenting with acute vascular events such as myocardial infarctions and cerebrovascular accidents. This has likely led to increased out-of-hospital mortality [1] but also likely late-term complications in survivors. One of these late-term post-myocardial infarction complications is the development of a post-infarct ventricular septal defect (VSD), and there have been whisperings that these have been seen more commonly during the pandemic [2]. Therefore, it is timely to have an article re-assessing the management of patients with a post-infarct VSD, particularly with respect to the appropriate timing of closure.

Heckle and colleagues [3] review the published retrospective case studies of percutaneous closures of VSDs, and compare outcomes when the repair was done within 14 days of diagnosis versus later. As the authors acknowledge, without randomization, it is impossible to know whether such findings are the result of the specific treatment strategy or are due to patient confounders, including survival and selection biases. Nevertheless, this study is informative in providing a summary of current outcomes of this uncommon but potentially lifesaving procedure.

The current report reiterates the findings of prior percutaneous and surgical studies, that early closure of post-infarct VSDs is consistently shown to be associated with higher mortality than later attempts [4,5]. Certainly, waiting at least 14 days will lead to a lower procedural mortality due to survival biases, but that does not mean it is safe to wait those 14 days, as some patients may require something to be done early in order to survive. Although the incidence of initial cardiogenic shock was similar between the two groups, the amount of left to right shunting was dramatically different between the two groups, pointing to the fact that the patients treated earlier were sicker. Furthermore, patients who presented later actually had no evidence for left to right shunting, raising the question as to what the clinical indications for closure in those individuals were who proved themselves as survivors.

Although not formally studied, it is hoped that intervening early may attenuate the increased risk of the left to right shunt exacerbating a myocardial infarction with subsequent ventricular dysfunction. It also may theoretically reduce the stress on the friable tissue, limiting further tearing. It has also been suggested that percutaneous VSD closure may be preferable to surgery for stabilizing patients by avoiding the risks of cardiopulmonary bypass and the challenges of surgical repair in the early stages of the VSD presentation. In some patients, the optimal management of a post-infarct VSD may best be considered as a staged procedure — device closure or surgery (possibly with concomitant CABG), followed some time later with the alternative to complete the closure. It is tempting to consider using a percutaneous ventricular assist device as a means to unload the ventricle while providing time for tissue healing and delay percutaneous or surgical VSD repair [6,7], but this requires independent testing.

It is well-known that surgical VSD repair is associated with much greater success when it is done after some time has passed to allow for healing (and to "weed out" the patients who are most likely to die early) [4]. Not surprisingly, with percutaneous closure as well, the outcomes are better when the procedure can be delayed. In fact, the mortality rates in the study by Heckle and colleagues are not terribly different than what has been reported for surgery [4]. Much of this is for the reason described above — the worse cases will die early – the stronger survive. If a patient can survive 14 days, they are less likely to die from the defect, or following the VSD closure, and the closure can be done to reduce the hemodynamic impact of the shunt —, which can include left ventricular dilation and volume overload with congestive heart failure, as well as increasing pulmonary artery pressures.

Early intervention carries the disadvantage that the defect may extend spontaneously or as a result of the procedure, as has been observed with surgical repair. Surgeons typically describe the tissue as being

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extremely friable early after the VSD develops but less so with late repairs [8]. The previously published pooled analysis by Schlotter and colleagues showed a strong association between early percutaneous VSD closure ≤14 days and higher mortality [5], which is consistent with the finding in this current update. What these studies do *not* provide are guidance for clinicians as to the optimal timing for intervention.

In considering the likelihood of benefit of early percutaneous closure, it is important to be aware of the serious potential complications. Aside from the usual risks of vascular access and contrast administration, it may be possible to extend the defect, or otherwise cause tearing of the fragile myocardial tissues, due to tension during the procedure or from the device. Many operators will adopt a strategy of placing the largest possible device, either an off-label atrial septal occluder device or the HDE approved post-infarct VSD device (which is made in larger sizes than the FDA approved congenital VSD occluder devices) but which may increase tension on the defect. The defect may also extend independently, such that a seemingly reasonable closure (which in and of itself may be challenging to judge) may become worse over time. Or the device initially may only provide partial closure (seen in 75–80% of the patients reported on by Heckle) [3], given the eccentric nature of these types of post-infarct defects, and the challenges of device sizing and characteristics of available devices. In addition, there is a possibility of interfering with the mitral or tricuspid valves, leading to significant regurgitation.

A curious and unexplained observation is that patients who presented later had no evidence for right to left shunting, raising the question as to what the clinical indications for closure were in those individuals who proved themselves as survivors. Perhaps future reports can elaborate further on the clinical and/or hemodynamic indications for late closure of a post-infarct VSD.

The question of whether a patient presenting with a post-infarct VSD should be treated as soon as possible or stabilized, giving time for the myocardial infarct to mature and scar, cannot be answered by any observational study. Given the infrequency of patients presenting with post-infarct VSDs, even in these challenging times, this is unlikely to be studied in a randomized trial. But this may not be justification for clinical nihilism. Successful device placement and reduction of the left to right shunt can be achieved in upwards of 80% of patients [5,9]. Despite high mortality, for a severely decompensated patient with a large ventricular shunt, even partially reducing the shunt may be critical to changing the patient's clinical course. Therefore, hemodynamically compromised patients with significant shunting should be considered for urgent closure, even "early" post-infarct. Ultimately, the decision to fix a VSD urgently or after a waiting period for clinical stabilization, whether surgically or percutaneously, depends on multiple patient factors and local expertise, as well as VSD defect size and complexity, with the decisions best determined by the heart team.

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