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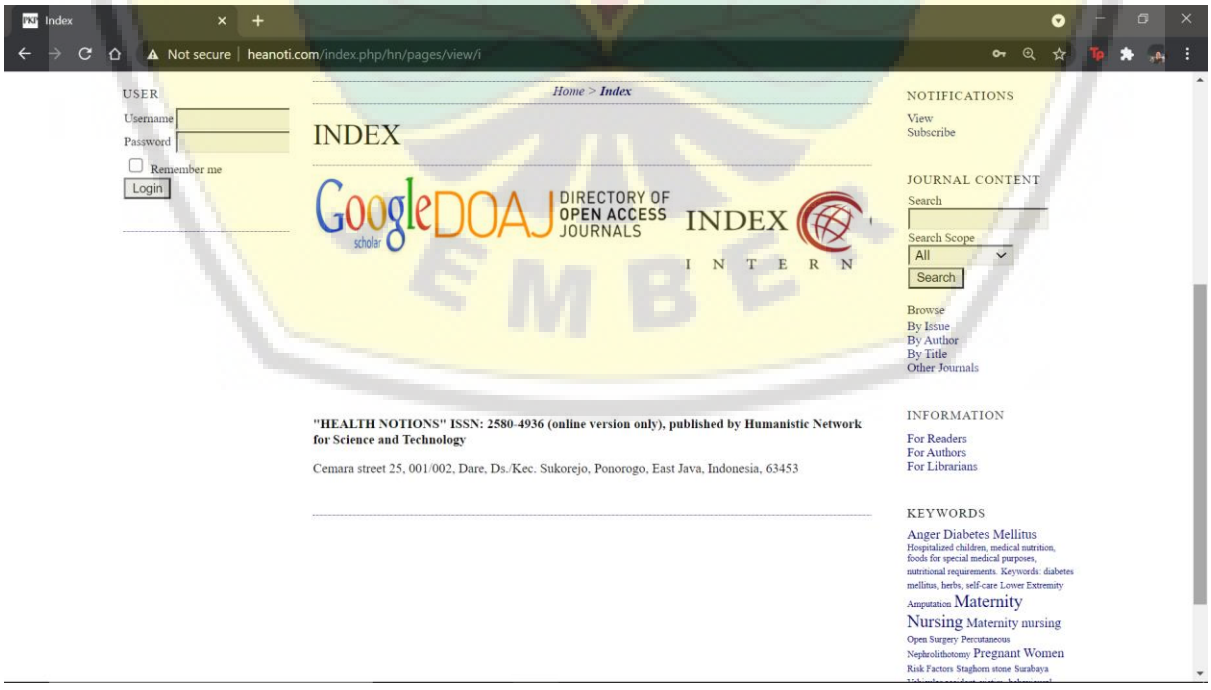

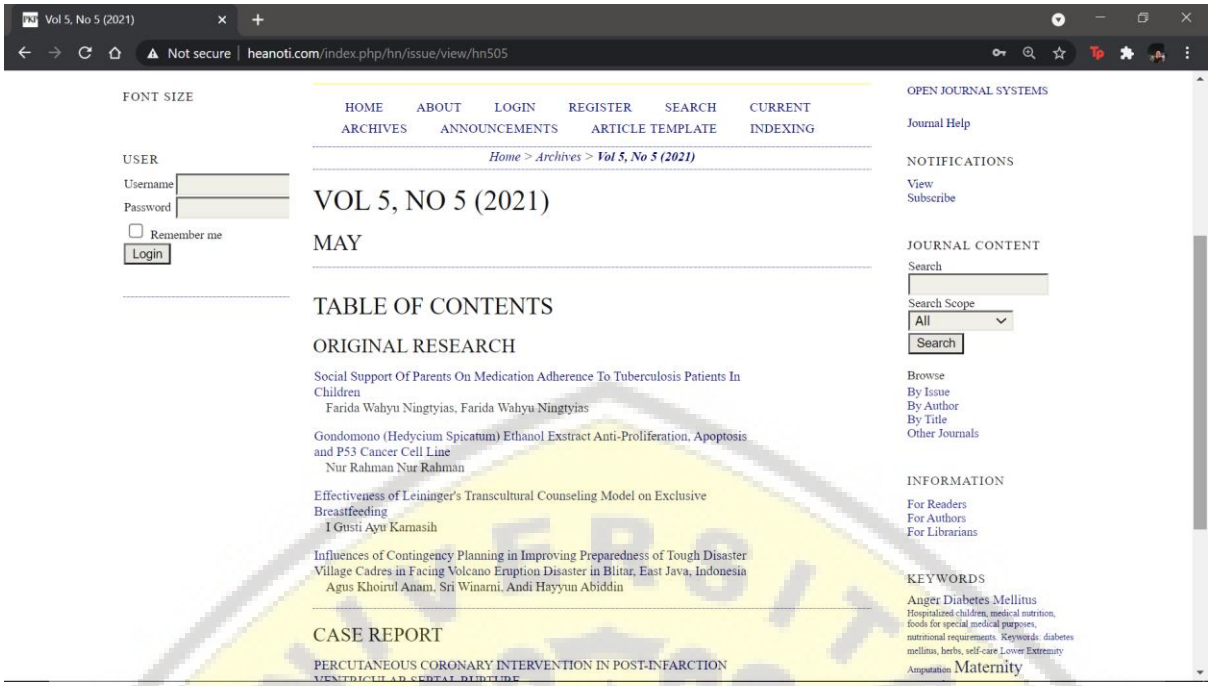

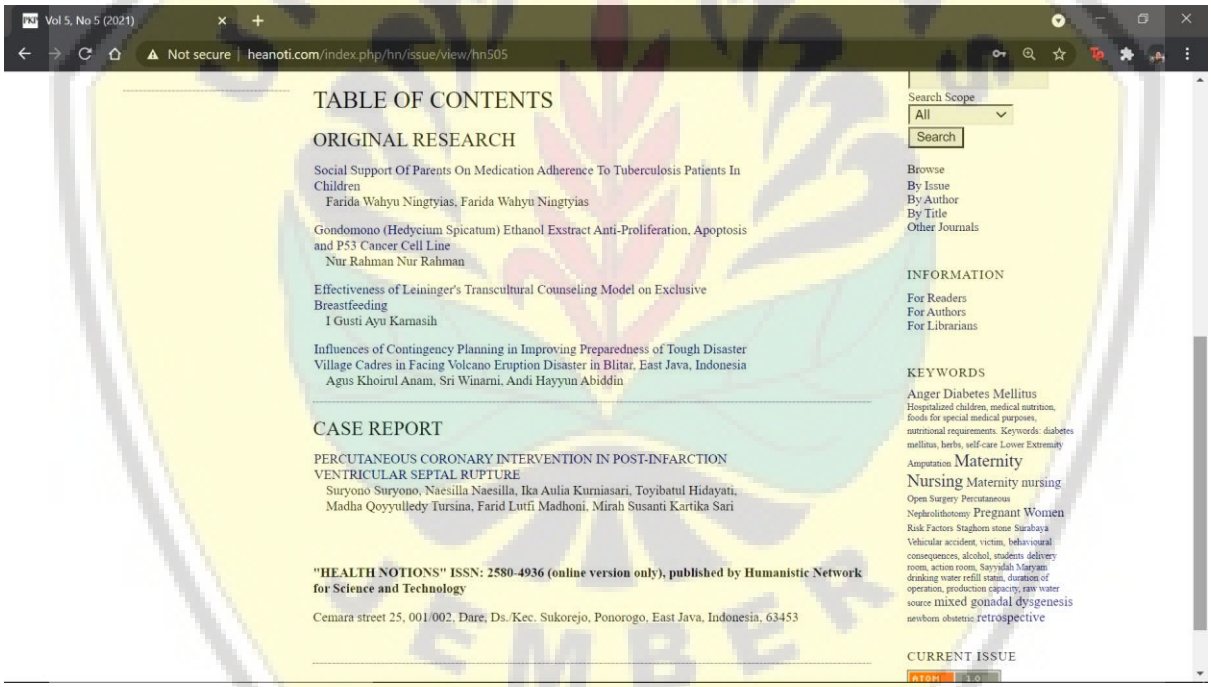

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PERCUTANEOUS CORONARY INTERVENTION IN POST-INFARCTION VENTRICULAR SEPTAL RUPTURE

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ABSTRACT

Background: Ventricular septal rupture (VSR) is a fatal mechanical complication following acute myocardial infarction (AMI). The definitive treatment remains surgical repair, however several aspects are still debatable, including the timing of surgery. Patient's refusal of treatment and lack of medical facilities put other challenges in management of VSR.
Case presentation: A-48-year-old male with a history of diabetes mellitus and hypertension came with a late-presentation anterior AMI, and refused the reperfusion therapy. By the third day, he developed VSR and cardiogenic shock. The patient was also against any referral plan for further therapy. Hemodynamic status was optimally controlled with vasopressor and inotropic agents. The patient was discharged with a grade II-III New York Heart Association (NYHA) on the tenth day. In follow up evaluation a week later, the patient presented limb swelling and functional NYHA class III-IV. The patient agreed to received percutaneous coronary intervention (PCI). Drug eluting stent (DES) in left anterior descending (LAD) coronary artery was implanted successfully without defect closure. Follow-up after 12-months revealed no signs or symptoms of decompensated heart failure.

Keywords: ventricular septal rupture, percutaneous coronary intervention, acute myocardial infarction

INTRODUCTION

Major complications of acute myocardial infarction (AMI) include heart failure, arrhythmias, embolism, inflammation, and mechanical complications.[1] One of the mechanical complications involving ventricular septal rupture (VSR), a rare fatal complication. The incidence of post-infarction VSR is 1-2%. This number has dropped significantly (0.17-0.31%) since the development of effective reperfusion therapy.[1,2] Its mortality rate is 24-87% and 90% in patients with and without definitive surgery, respectively.[3,4,5] Defect size, hemodynamic status, and comorbidities may predispose the mortality on VSR cases. [3,4]

Surgical closure should be performed immediately in large VSRs (≥ 15 mm) to prevent embolizations. Meanwhile, amplatzer closure is recommended for small-medium VSRs ≤15 mm to stabilize the patients' hemodynamic and may also be used as a temporary correction.[6] The American College of Cardiology-American Heart Association recommends immediate closure surgery regardless of the hemodynamic status. Combination of definitive VSR therapy with other therapeutic options such as reperfusion therapy with the primary percutaneous coronary intervention (pPCI) is frequently used to overcome the high mortality rate postoperatively. Inadequate stenting will require coronary artery bypass grafting (CABG) to prevent the risk of surgical complications and continued with the defect closure.[3,7,8,9] Therefore, management of VSR involves a multidisciplinary approach. Meanwhile, patient delay (refusal of treatment) and lack of medical facilities put other challenges in VSR management. This article reported a post-infarction VSR patient who received the percutaneous coronary intervention (PCI) without defect closure.

CASE REPORT

A-48 year-old hypertensive and diabetic male sought medical care to the Emergency Department (ED) due to increasing-intensity chest pain lasting for 13 hours. He had the blood pressure of 160/93 mmHg, heart rate (HR) of 100 bpm, the temperature of 36.50C, and respiration rate (RR) of 20x/min and peripheral oxygen saturation (SpO2) of 96% (room air). He had no history of smoking or heart disease

Initial electrocardiogram showed normal sinus rhythm, HR 105 bpm, normal axis, ST-segment elevation in leads II, III, aVF, V1-V6, concluded as anteroinferior STEMI and the laboratory tests showed positive cardiac troponin. The chest X-ray showed cardiomegaly without any signs of pulmonary oedema (Figure 1).

The patient was diagnosed with anteroinferior ST-segment elevation myocardial infarct (STEMI). Despite extensive education, the patient refused reperfusion therapy. Thus, the patient was treated with the standard medication, including loading dose 300 mg, loading dose clopidogrel 300 mg, atorvastatin 40 mg, glimepiride 1mg, and IV nitroglycerin 10 mcg/min and loading dose enoxaparin 0.3 cc IV followed by 2 x 0.6 cc subcutaneously.

On the 3rd day, the patient experienced cardiogenic shock with BP of 80/60 mmHg, HR 120 bpm, RR 30 x/min, with cold clammy extremities (Killip score IV). A new 3/6 pansystolic murmur was found in the parasternal linea ICS IV 3/6. Echocardiography showed a VSR sized 0.98 cm in diameter and a left ventricular ejection fraction of 48% (Figure 3). The final diagnosis was anteroinferior STEMI complicated with VSR and cardiogenic shock. Nitroglycerin IV was discontinued, and the patient received dobutamine started at 10 mcg/min and norepinephrine started at 0.5 mcg/min.

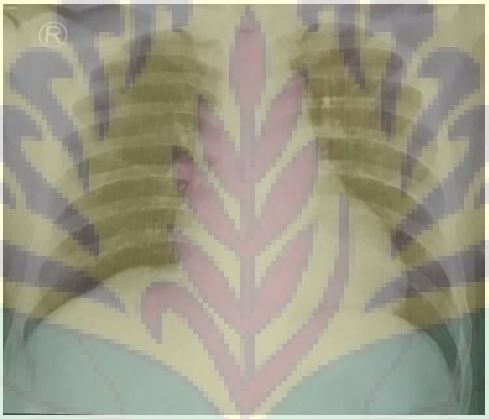

Figure 1. Chest X-ray showed cardiomegaly

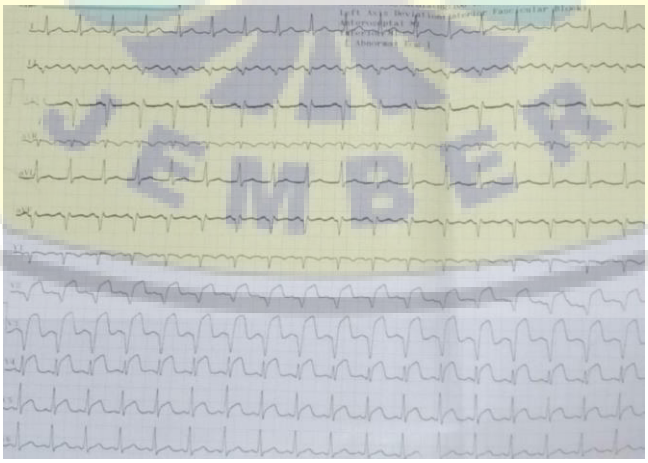

Figure 2. Electrocardiogram in third day of admission.

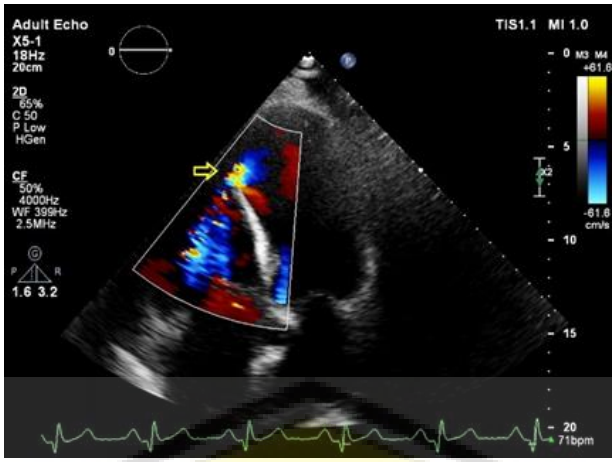

Figure 3. Four chamber echocardiography showed the presence of a VSR type with a diameter of 0.98 cm and the left ventricular ejection fraction of 48%.

The patient's hemodynamic status gradually improved with vasopressors, subsequently, acute heart failure had resolved. On day-5, his blood pressure was 110/70 mmHg, the patient was treated with furosemide 1 x 20 mg and ramipril started 1 x 1,25 mg. Dobutamine and norepinephrine were gradually tapered and discontinued the next day. The following day, he received bisoprolol 1 x 1.25 mg, spironolactone 1 x 25 mg and increased dose of ramipril (1 x 2.5mg). On day 10 of hospitalization, the patient was no longer having complaints and discharged.

Seven days after hospital discharge, the patient developed symptoms of heart failure: shortness of walking and had lower limbs swelling. The patient was classified as NYHA Functional Class III-IV. The patient and his family were re-informed about the condition and willing to be having PCI. Once the hemodynamic status stabilized, the coronary angiography was performed via the right femoral artery using 6 Fr sheath. It revealed insignificant stenosis in the proximal right coronary artery and left circumflex artery. Critical stenosis was shown in the proximal LAD (Figure 4). DES was placed in the proximal left anterior descending artery (LAD) using a guiding catheter launcher JL 4.0/6Fr, wire run-through NS, pre dilatation using a 2.0 x 20 mm Across Up balloon inflated up to 10 atm. After recanalization and pre-dilatation with a balloon in the left anterior descending artery, a 3.0 x 20 mm CRE8 stent was placed to expand it to 14 atm and evaluated by angiogram (Figure 5). No complication emerged during and after PCI.

The angiogram also showed the extent of LAD down to the heart base therefore stenosis in the proximal part of the LAD might cause extensive necrotizing myocardial tissue. Following the PCI procedure, the patient had no more complaints, his hemodynamic status remained stable and the patient was discharged. Follow up evaluation at 12 months showed no signs and symptoms of any complications.

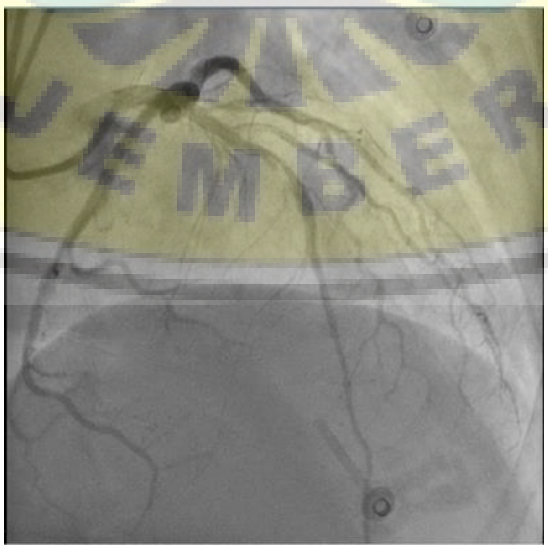

Figure 4.Coronary angiogram examination (pre stent) showed critical stenosis in the left anterior descending proximal artery

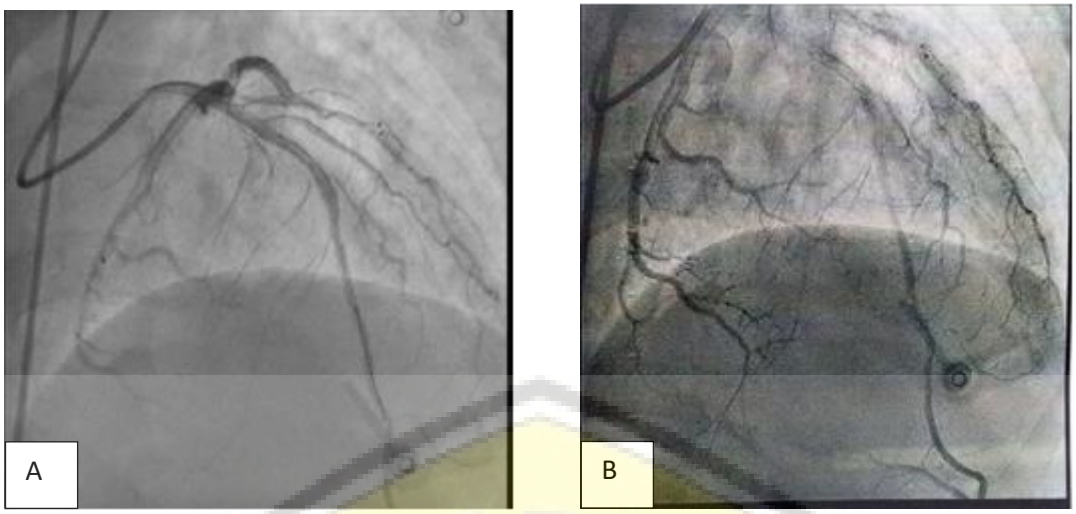

Figure 5. A. Coronary angiogram (post stent). B. left anterior descending artery (LAD) appears long to the basal

DISCUSSION

Ventricular Septal Rupture (VSR) is an interventricular septum defect due to ischemic necrosis following STEMI.[1] This complication is rarely [fo5und], nevertheless, it carries high mortality risk.[2] Only a few studies had reported VSR and the definitive management of VSR is still widely debated. As mentioned above, the American College of Cardiology-American Heart Association recommends immediate closure surgery regardless of the hemodynamic status.[10] Meanwhile, the European Society of Cardiology guidelines stated that optimal time for defect closure was yet to determined.[5]

Previous studies on VSR observed that closing the defect could reverse the affected hemodynamic status (mainly due to left-to-right shunt).[11,2,12,13] Lack of expertise and medical sources are challenging for the clinicians in regional health care centres. Several predisposing factors of VSR of this patient included the presence of comorbidities such as diabetes mellitus, hypertension, no history of smoking, and anteroinferior STEMI.[11,5] VSRs following anterior STEMI are usually simple type and located in the apical septum, whereas VSRs from inferior STEMI are usually extensive complex type and located in the basal part of the septum. Inferior STEMIs are frequently complicated with cardiogenic shock.[5]

The angiography showed an anatomical anomaly of the coronary artery in this patient. Long LAD was found extending to the base of the heart (Figure 5B). Approximately, only 1.5% of angiogram showed an anomaly in the coronary artery.[14] Rupture is a result of tissue necrosis extending to the interventricular septum. This event attracted neutrophil infiltration into the necrotic tissue leading to thinning of the septum. Those neutrophils triggered the apoptosis and released the lytic enzymes that accelerate the degradation of necrotic tissue.[11] Several studies observed that VSR frequently occur 3-5 days post-infarction.[14,15,5] This finding was in concordance with our case whose VSR was found on the 3rd-day post-infarction. The longer left anterior descending artery, the more tissue it vascularizes. It means that occlusion in the proximal part of long LAD would affect a wider area. However, the association between this anomaly and VSR was not yet established, possibly related to unknown coronary artery anomalies or unreported cases.

As shown before, the patient refused the definitive therapy, only to find that he returned with unstable hemodynamic a week after discharged. Hemodynamic stabilization was the priority, followed by reperfusion therapy via PCI. PCI was aimed to restore the flow in the coronary vessels and prevent the extension of ischemic tissue.[16] Once the stent implanted, the coronary blood flow will continue to provide perfusion to the tissue and cease the apoptotic process and expansion of the necrotic tissue. The remaining non-ischemic tissue also endures the inflammatory response due to the presence of necrotic tissues. As a result, cell regenerates and scar forms as fibrous connective tissue in the area of necrosis.[17] Necrotic cardiomyocyte attracts cellular changes including cardiac hypertrophy, apoptosis, necrosis, increased collagen production, and fibroblast proliferation. Fibroblasts predominate since early proliferation phase particularly in the infarct region and may display proliferative, migratory, and secretory myofibroblast features.[18] Myofibroblasts produced most of the interstitial collagen (initially type III and later type I collagen). Collagen deposition was essential to increase tensile strength and prevent further rupture. Myofibroblasts increased the secretion of fibronectin (FN) and various cellular matrix proteins which further increase myofibroblast migration.[19] Furthermore, angiogenic signalling stimulates the proliferation and infiltration of endothelial cells leading to the formation of microvascular tissue to the infarct area. [20,21] It was essential to supply myofibroblasts with adequate oxygen and

nutrients during the remodelling process. Following the formation of a collagen-based matrix at the infarct site, the growth factors, matrix cell proteins, and myofibroblast activity would decrease.[22,18,21] Type III collagen will eventually be replaced by type I collagen. Type I collagen is modified further by cross-linking catalyzed by lysyl oxidases (LOX). Expression of the four isoforms of lysyl oxidases was increased in the infarct area and the border zone 3–7 days post-myocardial infarction.[23] The cross-linking of the collagen fibres increased the tensile strength and formed scar contraction The presence of fibrotic tissue formation prevented the extension of defect size.[24]

The extensive ischemia and lack of collateral circulation accelarated the forming of VSR, resulting in short period of time between MI and VSR. The deterioration of the patient's hemodynamic status during the formation of VSR occurred due to unexpected left-to-right shunt. Fibrosis tissue, collateralization, small defect size (<15 mm), and reperfusion therapy to restore adequate coronary blood flow might play some roles, in this case, allowing an adaptation to the new intracardiac system and maintaining the hemodynamic stability.[25,5]

CONCLUSION

Reperfusion therapy via percutaneous coronary intervention (PCI) may improves hemodynamic status in the presence of post-infarction VSR. Several factors need to be considered in choosing therapeutical approach, such as the size of the defect, the hemodynamic status of the patient, the availability of health care facilities or sources, and also timing (since onset of infarction). Regular follow up is necessary to monitor the defect size, hemodynamic status, or long-term complication in these patients.

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