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Abstract

This paper reports the clinic course of a patient with Post-infarction ventricular septal defect (PIVSD). At the period of surgery, the patient received levosimendan for cardioprotection. During the levosimendan infusion, the hemodynamics of the patient worsened. It might be also due to contrast induced nephropathy or to enlargement of the VSD. The mortality of the patient might be responded to the increase of the amount of left to right shunt, which had possibly occurred due to levosimendan treatment.

Keywords: Levosimendan; Postinfarction ventricular septal defect

Introduction

Ventricular Septal Defect (VSD) is a rare, but a fatal complication of acute myocardial infarction (MI) [1]. Current guidelines for the treatment of Post-infarction ventricular septal defect (PIVSD) recommend immediate surgical repair, regardless of clinical status [2]. At the period of surgery, it may be longer with some reasons, Intra-Aortic Balloon Pumping (IABP) provides temporary but effective early medical stabilization. Positive inotropic agents are used generally to patients with hypotension (<90 or 80 mmHg) or cardiogenic shock, but there is no data about the benefits or risks of this treatment [3,4]. There is growing data about perioperative use of levosimendan for cardioprotection in the patients who underwent cardiac surgery [5]. The effects of levosimendan on Acute Heart Failure (AHF) complicated by PIVSD are discussed here through a case in which levosimendan was used for cardioprotection before cardiac surgery.

Case Report

This paper reports the clinic course of a patient with PIVSD. He was 65 years old and hospitalized for subacute anterior MI. There was ST segment elevation and negative T waves on electrocardiography. Troponin levels at admission were high (Troponin I: 28 ng/ml). Echocardiography revealed an aneurysm at anteroseptal region of the Left Ventricle (LV) and Ejection Fraction (LVEF) was approximately 35%. The patient was hemodynamically stable at the first day. On the second day, the patient complained of chest pain and dyspnea, a new holosystolic harsh murmur was auscultated, and VSD was found on bed-side echocardiography. Immediately performed coronary angiography and left ventriculography confirmed the postinfarction VSD, and showed total occlusion on proximal Left Anterior Descending Artery (LAD) and severe stenosis on Right Coronary Artery (RCA). Immediate surgery for VSD and coronary bypass was planned. However, the patient could not be immediately operated because of ongoing discussions with surgeons. In the same day, blood pressure was 110/70 mmHg, but Urine Excretion (UE) felt below 30 cc/h, and the serum Creatinine levels (Cr) raised over 2 mg/dl along with increase in Blood Urea Nitrogen levels (BUN) over 60 mg/dl. Following IABP and nitroprusside infusion, UE increased over 50 cc/h and Cr felt below 2mg/dl, BUN remained at similar levels. The patient was managed via this therapeutic plan for three days. At the first day, intravenous levosimendan infusion was started and after 24 hours of infusion of levosimendan, the surgery was performed. The patient

su ered nonsustained ventricular tachycardia episodes, Cr levels raised over 2.5 mg/dl, BUN over 90 mg/dl, and UE felt below 40 cc/h during levosimendan infusion. On the operation, a saphenous vein graft between aorta and RCA was placed and left internal mammalian artery was anastomosed to LAD. The VSD was repaired via left ventriculotomy by internal patch. After these processes, the patient could not be weaned from Extracorporeal Perfusion Pump (ECP) and succumbed to death.

Discussion

Postinfarction VSD complicated 1% to 2% of acute MI before the reperfusion era [1,6]. The patient reported here did not receive thrombolytic therapy because of late admission, and had large infarct area, so an aneurysm at LV. It has been showed that levosimendan improves the outcomes of patients undergoing cardiac surgery, especially with cardiotomy and ECP become much more frequently used for this aim [5]. Despite no invasive hemodynamic measurements were performed in the patient reported here, the UE and serum urea-creatinine levels, which are important markers for cardiac output, were deteriorated after the infusion of levosimendan and ventricular arrhythmias appeared. It might be also due to Contrast Induced Nephropathy (CIN) or enlargement of the VSD or decompensation of heart failure. However, hemodynamic improvement was observed after IABP, suggesting the impairment was not responded to CIN. On the other hand, development of arrhythmias may suggest further impairment of cardiac function and not renal impairment. If the reason of hemodynamic impairment was decompensation of heart failure, levosimendan would be expected beneficial at that situation. The clinical findings suggest that levosimendan might be the reason of impairment of hemodynamics, through increasing the amount of left to right shunt, as discussed below. The favorable effects of levosimendan are known in AHF [7]. There are no data about the

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Effects of levosimendan on hemodynamic parameters in patients with post-infarction or other forms of VSD. Concluded data were reported for other positive inotropic agents when used in other forms of VSD [8,9]. Positive inotropic agents were used in PIVSD, when cardiogenic shock occurred. There are no adequate data about whether they are beneficial or harmful. Levosimendan has theoretically beneficial effects in the situation of PIVSD, in which Right Ventricular Failure (RVF) eventually occurs.

It was shown that levosimendan may improve right ventricular functions and clinical signs in patients with RVF [10]. On the other hand, the amount of left to right shunt was found higher in patients with higher EF than those with lower EF in patients with PIVSD [4,11]. This finding suggests that increasing contractility with a positive inotropic agent, e.g. levosimendan, may increase the amount of left to right shunt and compromise functions of right ventricle further and may decrease the cardiac output, as observed in this patient. The amount of left to right shunt before and after levosimendan therapy could be measured by echocardiography in this patient, although the retrospective analysis of the patient has become impossible. The preoperative LVEF was not associated with in-hospital or 30-day mortality in patients undergoing surgical or percutaneous repair [4]. Although in other reports, the patients who survived operative repair had larger shunts on average than the nonsurvivors [11]. According to this acknowledgement, the impairment of hemodynamics and mortality of the patient might be responded to the increase of the amount of left to right shunt, which had possibly occurred due to levosimendan treatment. In patients with postinfarction VSD, cardiogenic shock or AHF could be said to be secondary to mechanical problems, and, as in those with postinfarction VSD, inotropic therapy without apparent afterload reduction might bring about increased contractility and increased left to right shunt, which may result in RVF earlier. Finally, the use of levosimendan or other positive inotropic agents in patients with PIVSD with the aim of perioperative cardioprotection or treatment of AHF is a matter requiring more attention.

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