**SUMMARY**

Ventricular septal rupture is a life threatening complication of myocardial infarction leading to cardiogenic shock. Echocardiography, cardiac catheterization and ventriculography confirm the diagnosis. Timely management of cardiogenic shock with inotropes and intra aortic balloon pump and surgical repair of defect can save a patient. Here, we present the anaesthetic management strategy of such case.

**Keywords**: Myocardial infarction, Ventricular septal rupture, Cardiogenic shock, Intra aortic balloon pump.

**Introduction**

Post infarction ventricular rupture is an infrequent but serious complication of myocardial infarction. Ventricular septal rupture complicating myocardial infarction was first described at autopsy by Latham. One of the first antemortem diagnosis of postinfarction ventricular septal rupture (VSR) was made by Brunn in 1923. Sagar in 1934 established specific clinical criteria for diagnosis and stressed the association of postinfarction VSR with coronary artery disease. In 1957, Cooley and colleagues reported the successful surgical repair of postinfarction VSR in a patient nine weeks after the diagnosis of septal rupture. Incidence of VSR is 1 to 3%. It usually occurs between 2 to 6 days after onset of acute myocardial infarction, although it may occur anytime within about first 2 weeks after an acute infarction. Ventricular septal rupture is associated with high mortality with an incidence of 1 to 5% of all infarction related death. This complication is usually associated with total occlusion of a single coronary artery, occurs with both anterior and inferior or posterior infarction, and is secondary to infarction of the interventricular septum. Surgical repair is the treatment of choice. Surgery is done as an emergency procedure. Hereby we are discussing the anaesthetic management of post myocardial infarction ventricular septal rupture who presented in cardiogenic shock.

**Case report**

A 69 year old male presented to casualty with complains of chest pain and breathlessness. History revealed that he had myocardial infarction two days back for which he was admitted to a hospital. He was stabilized there and streptokinase was given. However, two days after myocardial infarction patient suddenly developed breathlessness. They suspected VSR and patient was referred to our hospital. On examination patient was conscious but restless and was complaining of chest pain. Pulse rate was 124 min⁻¹ thready and of low volume. Blood pressure was not recordable. Extremities were cold and pallor was present. Tongue was moist. Elasticity of skin was normal. On auscultation there was pansystolic murmur heard along the lower left sternal border. Blood investigations revealed haemoglobin 13.5 gm%, blood urea 135 mg%, serum creatinine 2 mg%, blood sugar 170 mg%, serum sodium 128 meqL⁻¹ and, serum potassium 5.1 meqL⁻¹. Chest X-ray (PA view) showed evidence of increase pulmonary blood flow. Echocardiography was done which revealed apical muscular VSR with left to right shunt with a gradient of 38 mmHg. Left ventricular ejection fraction (LVEF) was 25% to 30%. Cardiac catheterization was done which showed step up of blood oxygen saturation from right atrium (70.6%) to right ventricle (89.6%) and confirmed the left to right shunt. Angiography revealed that left anterior descending artery was significantly blocked and there was apical muscular VSR.

As the patient was in cardiogenic shock dopamine 10 gkg⁻¹min⁻¹ and dobutamine 5 gkg⁻¹min⁻¹ were started. Intra aortic ballon pump (IABP) was inserted to improve coronary circulation and to reduce afterload. After taking informed consent patient was shifted to O.T. with no premedication for VSR repair. In the operation theatre monitors were placed, ECG leads for heart rate and rhythm, pulse oximeter for SPO₂, arterial pressure by femoral sheath (which was used for cardiac catheterization). Arterial blood sample was collected for blood gas analysis. He was induced...
with inj. midazolam 3 mg and inj. fentanyl 100 g and trachea was intubated with 9.0 mm PVC cuffed oral endotracheal tube (portex) after relaxation with inj. rocuronium 100 mg in divided doses. Patient was ventilated with O₂:Air:50:50 (50%) to maintain normocarbia (PaCO₂ between 35-40 mmHg) and avoid hyperoxia (PaO₂ between 150-200 mmHg). Before going on to cardiopulmonary bypass (CPB) augmented systolic pressure was maintained between 110-130 mmHg and pulse rate was between 90-120 min⁻¹. After midsternotomy and before cannulation of ascending aorta and right atrium, patient was heparinized with heparin 3 mg·kg⁻¹·h⁻¹ and before cannulation of ascending aorta and right atrium, heparin level was monitored by ACT. After repair, patient was heparinized with heparin 3 mg·kg⁻¹·h⁻¹ to keep the activated clotting time (ACT) above 480 sec. Patient was put on CPB in a routine manner. Patient was cooled gradually to nasopharyngeal temperature of 28°C. After aortic cross clamping (Aoxcl) antegrade cold blood cardioplegia (St. Thomas) was given and on arrested heart VSR was approached through right ventriculotomy and defect was repaired with Goretx patch. During CPB, IABP was put off and perfusion pressure was maintained between 70-90 mmHg. Heparin level was monitored by ACT. After repair, patient was weaned off CPB gradually and heparin was reversed with protamine. The total Aoxcl time was 61 minutes and CPB time was 108 minutes. On completion of surgery patient was shifted to ICU on infusion dopamine (10 g·kg⁻¹·min⁻¹), NTG (0.5 g·kg⁻¹·min⁻¹), and supported by IABP. Total intraoperative urine output was 700 ml. In ICU, the patient was gradually weaned off from IABP (20 hr). During that period patient was ventilated electively with intermittent dose of pancuronium, midazolam and morphine. The patient was extubated after 36 hr postoperatively when patient was fully awake and maintaining adequate hemodynamics. He had no neurologic deficits. The dopamine and NTG were gradually tapered and stopped. Total chest tube drainage was 310 ml in first 24 hour and 80 ml in next 24 hour. The patient was shifted to ward on 4th postoperative day and then discharged from hospital after 7 days.

Discussion

Ventricular septal rupture complicate 1 to 3% of all infarction and account for 5% of all peri infarction death.² This usually occurs between 2 to 6 days after onset of acute myocardial infarction, although it may occur anytime within about first 2 weeks after an acute infarction.³ Mortality is high if treated medically and 25% patient die from cardiogenic shock within 24 hours which reaches 90% at 2 months.⁴,⁵ Our patient presented with cardiogenic shock in the casualty. We managed the patient with vasopressors, dopamine and dobutamine. Vasodilators are ideally required along with vasopressors to decrease the afterload and left to right shunt. However they could have resulted in hypotension and further exaggerated myocardial hypoperfusion in the presence of associated coronary artery disease and may contribute to additional myocardial dysfunction. For that reason we avoided the use of vasodilators in the casualty. Echocardiography showed the presence of septal rupture and ruled out the possibility of acute mitral regurgitation, the other cause of sudden cardiogenic shock after myocardial infarction. Echocardiography also allows for quantitative assessment of the defect, size of ventricular defect and the presence of gradient between two ventricles. In an emergency situation revival and diagnostic technique was going on side by side. We shifted the patient to cath lab to confirm the diagnosis. Step up of blood oxygen saturation (more than 10%) from right atrium to right ventricle confirm the VSR. As systemic pressure was not sustained insertion of intra aortic balloon pump in cath lab improved the coronary perfusion. Patient was immediately shifted to cardiac O.T. for surgical repair.

Reduction in systemic blood flow due to left to right shunt results in a low output hypotensive syndrome that lead to cardiogenic shock⁶,⁷ and renal failure and may result in death. In this patient blood urea was 135 mg% and serum creatinine was 2 mg% and patient was not passing urine. With immediate revival and pressure support by IABP, the patient passed adequate urine. In case patient was not passing urine, diuretic inj. furosemide should be given. Operative repair also carries a high mortality rate especially when patients are associated with preoperative cardiogenic shock, low right atrial oxygen saturation (less than 60%), and anterior wall infarction.⁸,⁹,¹⁰

We induced the patient with fentanyl and midazolam to avoid any myocardial depression. Rocuronium was chosen for intubation and maintenance because the patient was in renal compromised state. Atracurium could have been ideal muscle relaxant for such patients. But atracurium was not available and instead, rocuronium could be used cautiously in such patients. After induction and intubation NTG was started to reduce afterload and left to right shunt which improved systemic blood flow and coronary perfusion. As hypocarbic alkalosis and hyperoxia reduces the pulmonary vascular resistance and increase in left to right shunt. We ventilated the patient with 50% O₂ and 50% air to maintain normocarbia (PCO₂ between 35-40 mmHg) and avoiding hyperoxia. We maintained anaesthesia with fentanyl and midazolam to avoid any myocardial depression. After
surgical repair patient was shifted to ICU and extubated on 3rd day and discharged after 7 days.

**Conclusion**

We conclude that post myocardial infarction ventricular septal rupture is a surgical emergency and patient can be saved by early referral, swift evaluation by echocardiography, cardiac catheterization and emergency surgery. Preoperative stabilization of the patient from cardiogenic shock by vasopressor and mechanical support (IABP) improve the outcome.

**References**