Cardiac arrest during anesthesia induction in a patient with LV pseudoaneurysm.

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Abstract

Free wall rupture of the Left Ventricle (LV) is a rare life-threatening complication of acute myocardial infarction. Very rarely such a rupture may be contained by the adhering pericardium thus creating a pseudoaneurysm. This condition warrants for emergency surgery. Hemodynamic management is very important in such patients during induction of anesthesia. We report a case of 58 years old male patient who collapsed during induction of anesthesia in the operation theatre.

Keywords: Coronary angiography, LVSD, Electrocardiogram, Pulmonary artery pressure, LVDD, Myocardial infarction, LVEF.

Abbreviations:

MI: Myocardial Infarction; LVEF: Left Ventricular Ejection Fraction; LVSD: Left Ventricular Systolic Dysfunction; LVDD: Left Ventricular Diastolic Dysfunction; SBP: Systolic Blood Pressure; CPR: Cardio-Pulmonary Resuscitation; IABP: Intra Aortic Balloon Pump; DVT: Deep Vein Thrombosis, SVCAD: Single Vessel Coronary Artery Disease; NTG: Nitroglycerine

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Introduction

Out of all mechanical complications of acute MI, rupture of the free wall of the LV is catastrophic. The precise incidence of aneurysm and pseudoaneurysm of the heart is unknown. LV pseudoaneurysm is formed if cardiac rupture is contained by the pericardium, organizing thrombus, and hematoma. This condition calls for urgent surgical repair [1]. Hemodynamic stability/proper monitoring is very important in these patients during induction of anesthesia. Hypertension can lead to rupture, and cardiac tamponade and hypotension can lead to LV pump failure or reduce coronary perfusion and cardiovascular collapse.

Case Report

A 58 years old hypertensive and diabetic male with c/o chest pain and breathlessness admitted in a local hospital where he was diagnosed as infero-lateral wall myocardial infarction. He was managed conservatively with antiplatelets, statin, and Beta-blockers before planned CAG. On the day of CAG, he developed hypotension and shock, 2D-Echo showed a pericardial collection of fluid with the presence of a clot over the right ventricular surface with a postero-lateral aneurysm suggestive of sealed cardiac rupture. After 7 days he came to our institute for further management.

All blood investigations such as complete blood count, renal and liver function tests, Coagulation profiles, and electrolytes were within normal limits. Chest X-ray showed enlarged cardiac shadow. ECG showed anterolateral infarct, LVH and left the anterior fascicular block. 2D-Echo showed LVEF 20% to 25%; severe LVSD, Grade I LVDD, inferoposterior LV free wall rupture with defect measuring 4.5 cm- resulting in pseudoaneurysm of 5 × 9 cm and minimal pericardial effusion. Surgery was planned for aneurysm repair (Direct Closure) +CAG on Table

The pre-op patient was on Tab. Ecosprin 75 mg, Ivabrad 5 mg, Angispan TR 2.5 mg, Dytor 5 mg and Aldactone 25 mg once a day. On arrival in OT after CAG (right femoral artery sheath *In situ*) which had revealed SVCAD-100% occlusion in mid-segment of Left Circumflex artery, Baseline ABP-130/80, Heart Rate-70/min, SpO_2-98% on room air. Intra-op Monitoring used were 5 lead ECG, ABP, PAP, SpO_2, $ETCO_2$, and Cerebral oximetry Patient was induced with Inj. Midazolam (0.05 mg/kg), Inj. Buprenorphine 2 mcg/kg, Inj. Etomidate (0.3 mg/kg) Inj. Vecuronium (0.1 mg/kg) and Sevoflurane through the mask. It was unanticipated difficult intubation. During the attempt, SBP went up to 190 mm Hg. NTG infusion was (25 mg/250 ml) started before the next attempt.

Soon, ABP fell down to 64/32 mm Hg. NTG was stopped, Domamine (200 mg/50 ml) and Noradrenaline (2 mg/50 ml) infusions were started at 20 ml/hr but the patient was not responding and soon arrested, CPR started. 100% O_2 given through a mask. Inj. Adrenaline 1 mg bolus was given. The patient was intubated with bougie on the 3rd attempt.

The patient developed ventricular fibrillation, DC shock with 200 J was given twice. CPR continued, Noradrenaline and dopamine infusions were further increased, and adrenaline boluses were given intermittently. With CPR, Mean BP was maintained around 50. Soda bicarb 100 ml and Inj. calcium gluconate 1 gm were given.

Meanwhile, the chest was opened with continuous CPR, Rhythm came back. Simultaneously Left femoral artery was

cannulated. The patient was taken on CPB after heparinization. On opening Pericardium, there was no hemopericardium but the size of pseudoaneurysm was enlarged. Defect closure was done. CPB time was 2 hours 20 minutes, Aortic cross-clamp time was 40 minutes. IABP inserted while coming off CPB Intra-op patient was transfused 5 whole blood, 6 packed cells, 2 single donor platelets, and 8 fresh frozen plasma units. The patient was shifted to ICU with Inj. Dopamine (200 mg/50 ml) at 5 ml/hr, Inj. Noradrenaline (2 mg/50 ml) at 5 ml/hr and Inj. Adrenaline (1 mg/50 ml) at 5 ml/hr.

Course in the ICU-On day 1, he was conscious, oriented, following verbal commands. There was no neurological deficit. He was extubated uneventfully after 24 hours. He had retained secretions for which mini-tracheostomy was done. Tapering of Inotropes was done with stable hemodynamics; all invasive lines were removed and the patient was mobilized in next 7 days.

On 7thday, he complained of mild pain and swelling of left lower limbs. Venous Doppler of left Lower Limb revealed DVT. Anticoagulant was continued. 2D-Echo was done to see PAP, which was normal. DVT stockings were used. Rest of the course was uneventful. Mini-tracheostomy was removed on the 10^{th} post-op day. The patient was shifted to the ward on the 11^{th} post-op day and was discharged from the hospital on 17^{th} post op day. Post-op Echo showed LVEF-25%, moderate LVSD, Grade I LVDD, Trivial to mild localized pericardial effusion-lateral to LV 6 mm.

Discussion

Left ventricular pseudoaneurysm is a type of rare, lethal condition developed from rupture of the ventricular free wall, but enclosed by the adherent pericardium or scar tissue [2]. The incidence of LV pseudoaneurysm was 0.23%, as reported by Csapo et al. [3] Though mortality rates in patients who underwent surgery was approximately 23%, but untreated pseudo aneurysms had an approximately 30% to 45% risk of rupture, so surgical resection was considered the most appropriate way of management of LV pseudoaneurysm.

Though nonspecific, LV pseudoaneurysm usually presents with symptoms such as congestive heart failure and chest discomfort (chest pain or dyspnea), syncope or cough [2] Because of nonspecific symptoms, diagnosis of LV pseudoaneurysm is always made tardily, and may even be reported 4 months after infarction [4].

Frances et al. reviewed data from 290 patients and reported that the most common etiology of LV pseudoaneurysm is myocardial infarction followed by cardiac surgery [5]. If the etiology was myocardial infarction these patients usually present with LV pseudoaneurysm within 2 months of the event [6]. The risk factors for the LV pseudoaneurysms are old age, hypertension and inferior and lateral wall involvement in the myocardial infarction. The common location for LV pseudoaneurysm is postero-inferior followed by the postero-lateral and anterior wall, in contrast to a true LV aneurysm which is more commonly located in the anterior and apical walls [5].

Once the diagnosis is confirmed, urgent surgical intervention is necessary for acute LV pseudoaneurysm (>7 days old) as the risk of rupture outweighs the risk of surgery [7]. However, Small retrospective studies have shown that patients with incidental finding of chronic (>3 months old) small LV pseudoaneurysm less than 3 cm in size and patients with increased surgical risk can be managed conservatively [8].

In our case, pseudoaneurysm was semi-acute (15 days old) initially we thought the patient collapsed because of rupture of pseudoaneurysm and cardiac tamponade due to a rise in ABP following failed first intubation attempt. But on the opening pericardium, we found though pseudoaneurysm had enlarged in size, there was no hemopericardium or cardiac tamponade. The cardiovascular collapse was due to hypotension, not hypertension [9].

In our case, while trying to avoid hypertension and possible rupture we started NTG infusion (25 mg/250 ml) in small doses. Even with transient NTG, preload had been reduced and hypotension occurred reducing forward flow from LV to Aorta and LV cavity distended. It facilitated the flow of more blood from the LV cavity to a low resistance pseudoaneurysm cavity. However, due to the adherent pericardium (7 days), it did not rupture. Hypotension led to a reduction in coronary perfusion and LV pump failure. As the heart was already bad (2D-Echo) the patient arrested. Pre-op insertion of IABP may have helped in improving left ventricular function and afterload reduction [10].

Conclusion

Both hypertension and hypotension are dangerous in a patient with pseudoaneurysm during induction of anesthesia. So induction of anesthesia should be accomplished with minimal or no compromise in cardiac function.

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