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Case Report

Post Coronary Artery Bypass Grafting – Coronary Artery Spasm: A Case Report

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Abstract

Coronary artery spasm after coronary artery bypass grafting (CABG) is a rarephenomenon often resulting in heart failure. We report a case of 56-yearr-old male, who had coronary artery spasm after CABG, which was successfully treated with intracoronary injection of nitroglycerine and heparin.

Keywords: Coronary Artery Bypass Grafting Vasospasm, Coronary Artery

1.Introduction

Coronary artery spasm after coronary artery bypass grafting (CABG) is a rare cause of post-operative ischemia [1], but it frequently causes grave complications. Though basic mechanism for this remains unclear, endothelial dysfunction seems to plays a major role. Here we report a case of coronary artery spasm following CABG, which was successfully treated with intracoronary injection of nitroglycerin and heparin.

2. Case report

A 56-year-old man had been taking medication for chronic stable angina pectoris for 7 months, and for hypertension for 8 years. He was referred to the cardiovascular and thoracic surgery department as coronary angiography (CAG) reports were suggestive of coronary artery triple vessel disease. The patient was a chronic smoker, and was put on beta blocking agent, calcium channel blocker (CCB), and acetyl salicylic acid (ASA) on admission. CAG showed a 90% stenosis of mid left anterior descending artery, 80% ostial lesion of first diagonal branch, proximal 50% plaque in left circumflex branch, proximal 80% lesion in second obtuse marginal artery and right coronary artery (RCA) dominant with 90% long diffuse lesion. Operation performed was off pump CABG, and the left internal mammary artery (LIMA) to the left anterior descending artery and separate reverse saphenous vein grafts to the obtuse marginal branch and distal RCA were done with RCA local endarterectomy. The patient was transferred to intensive care unit with stable hemodynamics, blood pressure (180/90 mmHg), pulse rate (90 beats/min) and minimal inotropic support of noradrenaline 0.02 µgm/kg/min for initial 2 hours. Then electrocardiogram (EKG) showed ST-segment elevation in all the leads, the blood pressure fell to 100/60 mmHg and the pulse rate reached to 100beats/min.

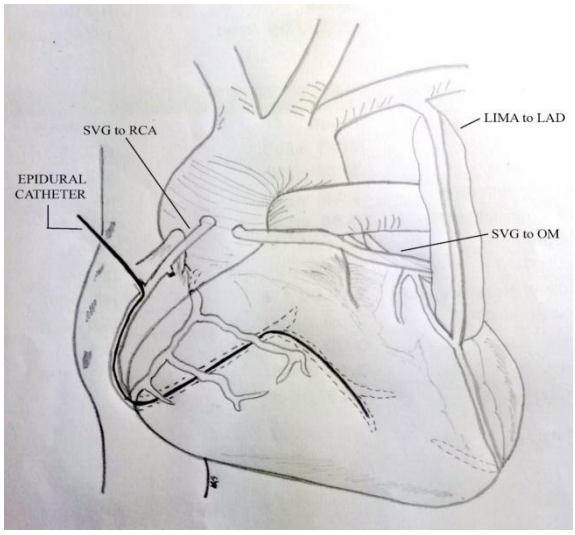
The patient had persistent signs of ischemia including ST-segment elevation (more pronounced in inferior leads than anterior and lateral leads), hypotension, LVEF of 30% and severe global hypokinesia on echocardiogram. Arterial blood gas and electrolytes were corrected and inotropes initiated were dobutamine at 4 µgm/kg/min, noradrenaline at

0.2 µgm/kg/min and nikorandil at 0.45 µgm/kg/min, and IABP was inserted. With this ST came down from 2.3 mm to 0.9 mm in lateral leads and 3.2 mm to 1.2 mm in inferior leads.

Hemodynamics were stable for an hour but there after patient had ventricular tachycardia (VT)/ventricular fibrillation (VF) which reverted back to DC shock and intravenous lignocaine and amiodarone. Hemodynamics were stable for an hour. After an hour of stabilisation patient had another incident of VT/VF which made us to do an emergency exploration to check for tamponade.

While reexploration; patient had no evidence of myocardial infarction or tamponade. He had persistent ST elevation in all leads but more in inferior leads. Trans esophageal echocardiography (TEE) was done which showed gross akinesia of inferior wall with reasonable movement of anterolateral wall and septum. Hence we decided to put the patient on normothermic CPB to give rest to heart. ST changes persisted in all leads so aorta was cross clamped and hot shot warm cardioplegia was given with LA vented. After 15 minutes cross clamp removed and grafts deaired but there were persistent ST changes on EKG with inferior wall akinetic (even confirmed on TEE). As EKG and echocardiography findings were suggestive of inferior wall akinesia, venous tributary of graft to PDA was opened and epidural catheter was introduced to check patency (Figure 1).

Figure 1: Depicting various Grafts and Epidural Catheter course.



It was found patent. Then we injected intracoronary nitroglycerin and heparin through epidural catheter. Deairing was done and reperfusion was started. ST changes came down and contractility improved on TEE. Patient gradually weaned off CPB smoothly with inotropic support of adrenaline of 0.08 µgm/kg/min, noradrenaline 0.17 µgm/kg/min and levosimendan at 0.07 µgm/kg/min with IABP support, and was shifted to recovery room with stable hemodynamics.ST changes came down in all leads and EKG become normal in six hour. TEE showed improved contractility with ejection fraction of 40%. Thereafter, he was on mechanical ventilation till 4th postoperative day (POD), inotropes were gradually tapered off and IABP was removed on 7th POD. He was discharged on the 11th POD with normal EKG. Post-operative echocardiography suggested absence of regional wall motion abnormalities with ejection fraction of 55%.On first follow up patient's EKG was normal with no regional wall motion abnormalities on echocardiography.

3. Discussion

Perioperative myocardial infarction is the first cause of early and long term mortality after cardiac surgery [2]. Coronary artery spasm before CPB during cardiac surgery was shown by MacAlpin *et al*. in 1973, and the incident case of coronary vasospasm in the postoperative period after CABG was reported in 1980 by Pichard *et al*. [3,4].

Since numerous factors are involved [3-6] the exact incidence of postoperative coronary artery spasm is not known. Buxton *et al*. reported incidence 0.8% of whom undergone CABG [3,7], Skarvan *et al*. 2.5%, and Lockerman *et al*. about 8% [3,6].

The etiology of coronary artery spasm after CABG is unclear, but several factors namely brief high-release of noradrenaline and adrenaline in the immediate post CPB period, administration of catecholamine such as dopamine, preoperative use of beta blockers or calcium channel blockers (CCB), high dosage of nitroglycerin before surgery, vascular injury by manipulation during surgery, platelet activation at vascular endothelial injury site, high level of potassium in the local area, respiratory alkalosis as a result of hyperventilation, hypomagnesaemia, hypothermia and stimulation by achest tube have been reported to potentiates vasospasm[3-7].

On the contrary Lockerman *et al* [6] showed that there is no relation between withdrawal of CCB, nitrate, nitroglycerin and the occurrence of postoperative coronary artery spasm. Postoperative coronary vasospasm is most commonly manifested by ST-segment elevation and shock without specific cause [3-7].

Coronary artery spasm most commonly occurs within two hours in post-operative period. We aimed to treat coronary vasospasm which resulted in shock in immediate post-operative period. Most data

supports use of nitroglycerine, heparin or CCB alone or in combination, and all these are more effective via intracoronary route [3-8]. As shown by Tarhan *et al*, to prevent spasm of grafting artery, donor vessel should be carefully handled to minimize surgical trauma during harvest, endothelial injury must be avoided by maintaining the blood flow until just before distal anastomosis, body temperature and temperature of harvested vessel have to be strictly controlled as normothermia, and medication such as nitroglycerin and/or CCB also should be applied systemically ortopically [9].

Although the incidence of coronary artery spasm after CABG is not high, we should get close observation of the patient who is in high risk group, because it can cause disastrous result without prompt adequate management. When unexplainable, sudden circulatory collapse after CABG accompanying ST-segment change comes up, we must keep in mind the spasm of untouched native coronary artery, grafted coronary artery, and grafting artery with high suspicion.

In this scenario, we need to observe regional hypokinesia of myocardium on echocardiogram and to confirm vasospasm by CAG. When coronary artery spasm and/or grafting artery spasm are certified by the means mentioned above, intracoronary infusion of nitroglycerin or CCB only or in combination should be performed, even in the hypotension status. Also, in case of highly suspicious vasospasm with difficulty of CAG, immediate intravenous administration of nitroglycerin or CCB provides fair results. Moreover the application of IABP also might be helpful.

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