

Coronary Artery Occlusion; the Night Mare Post AVR

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Introduction

Iatrogenic intra operative acute coronary artery obstruction is a rare and potentially fatal complication of valve surgery [1]. Sudden coronary spasm, embolization of calcium plaque, and surgical factors can induce acute coronary obstruction after AVR [2]. Initially described in 1967, following aortic valve surgery, it is now recognized that patients who develop myocardial ischemia postoperatively following valve surgery, should be investigated deeply to exclude this rare phenomenon as the cause of myocardial dysfunction [3].

Although most reports highlight the potential for coronary artery stenosis in the months following valve surgery there are few documented cases of intraoperative coronary embolism causing circulatory collapse and requiring prompt treatment [5].

After Ethical Committee approval and after written consent taken from the patient family for publication, we present a case of acute intra operative left main coronary artery obstruction after Aortic Valve Replacement (AVR) and its immediate management.

Case Presentation

41-year-old male patient known to be diabetic, hypertensive and ex-smoker. The patient was presented by shortness of breath on moderate exertion. Patient was subjected for routine work up and investigations which revealed Lymphadenopathy in different anatomical areas including mediastinal LN and Para aortic LN and final diagnosis was pending based on thoracic LN biopsy to be taken on time of cardiac surgery. Patient also diagnosed to have moderate restrictive lung disease and hepatitis B positive with liver impairment.

Trans thoracic echocardiography TTE revealed severe mitral regurgitation, severe aortic regurgitation, and mild tricuspid regurgitation. Coronary Angiography showed normal coronaries.

Operative Events

On 10 December 2017 after taking the consensus in the conjoint cardiac team patient was taken to the Operating Room where the aortic valve was replaced by Edwards Perimount Tissue Valve size 23mm and mitral valve was repaired using physio semi rigid ring annuloplasty size 28mm. The patient weaned off smoothly from cardiopulmonary bypass with minimal cardiac supports, while after 50 minutes of weaning off and during hemostasis stage and giving of protamine, the patient had episode of ventricular tachyarrhythmia which dissociated to ventricular fibrillation. Direct-current DC shock was given, and amiodarone was given according to ACLS guide lines, and the patient returned to sinus rhythm, but shortly he went again to ventricular fibrillation that was resistant to treatment (medical and electrical). Establishment of cardiopulmonary bypass regained. TEE was done that showed well-functioning aortic valve and mitral valve with severe impairment of both left and right ventricular function. After stabilization by vasopressors and inotropic support, ventricular function improved and we got able to wean off from cardiopulmonary bypass and maintained on moderate doses of inotropic and vasopressor support. Fifteen minutes later again new ventricular fibrillation storm happened with severe hemodynamic decompensation refractory to medical supports, so we elected to put the patient on central ECMO to provide better hemodynamic support and sending the patient for coronary catheterization safely.

Cath Lab Events

Safe transfer to the Cath lab was established while Coronary Angiography revealed Distal Left main thrombus, partially extended and occluding the left anterior descending and the left circumflex coronary arteries (figure 1). Thrombus aspiration was done, the left circumflex was freed, but the thrombus fragments shifted to the proximal left anterior descending and diagonal with TIMI flow 3 (figure 2).



Figure 1: Coronary angiography showing LM occluded with thrombus

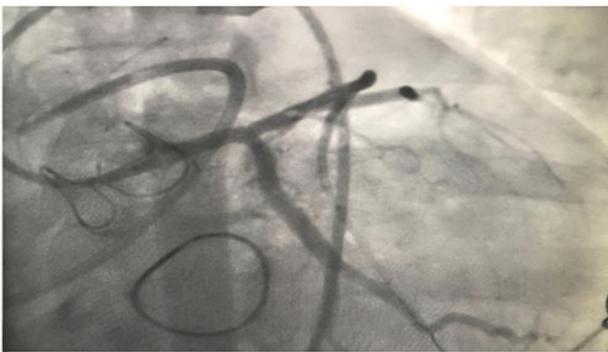


Figure 2: Showing TIMI3 flow in the LM and LAD after suction thrombectomy

Cardiac Intensive Care Events

The patient was transferred to the ICU on full flow central ECMO, open chest on big dose of vasopressors and inotropic supports in order to be able to maintain satisfactory perfusion pressure to maintain patient vitality and organs function. The main challenge was massive bleeding and severe vasoplegia in spite of massive transfusion of blood and blood products (12 units of PRBC, 12 units FFPs and 24 units platelets were given). ECMO flow was not sufficient due to loss of volume secondary to massive bleeding and failure of medical control of coagulopathy. Full resuscitation by fluids plus vasopressors and blood product failed to stabilize the patient so, he was shifted to the OR for surgical control of bleeding which done while inspection of the heart showed moderate to severe biventricular ventricular impairment.

The patient was shifted again to the ICU with open chest; with adequate ECMO flow 4.2 liters per minute on high doses of vasopressors and inotropic supports with borderline hemodynamic profile and serial blood gases showed metabolic lactic acidemia. Urine output started to come down, so CVVHD started, immediately and follow up showed gradual improvement in hemodynamic and metabolic profile during the second day postoperative and the lactate started to decrease.

ECMO flow gradually reduced to 3.5 L/min with satisfactory perfusion parameters and good hemodynamic profile. On follow up patient experienced ST segment elevation in the anterior lead without troponin leak and without hemodynamic decompensation and consensus was to manage conservatively.

Follow up TTE revealed moderate to severely impaired left and right ventricular function.

On 5th POD while the patient was hemodynamically stable with good perfusion parameters on moderate doses of inotropes and vasopressors, we planned to shift the ECMO from central ECMO to peripheral ECMO and we closed the chest and to stop sedation for better neurological assessment.

Serial follow up showed pulmonary infiltrates and arterial blood gases and oxygenation is totally dependent on ECMO while neurologically patient still being comatose and did not show any sign of recovery.

On 7th POD, patient was hemodynamically stable, on moderate inotropes, ECMO 4.6 liters per minute. Inotropes and vasopressors gradually weaned to moderate doses with acceptable hemodynamic and metabolic parameters.

From 8th to 9th POD, there was significant lung, also no metabolic acidosis. TTE showed left ventricular EF=25-30% and moderate right ventricular dysfunction. Neurological assessment did not show any signs of recovery while the neurology consultation cannot rule out metabolic from structural brain disease unless CT scan brain to be done while the patient critical condition cannot tolerate safe transfer to radiological department.

In 10th POD the ECMO flow gradually tapered to 1.5 liter / Minutes for 24 hours with acceptable hemodynamic, metabolic and arterial blood gases profiles and consensus was to wean ECMO off in OR.

Hours after ECMO weaned off patient developed severe pancreatitis and showed severe hemodynamic, metabolic and respiratory decompensation with escalation of inotropic and vasopressor supports and increasing ventilator setting to maintain acceptable arterial blood gases.

In 11th POD the patient continued deterioration and required more vasopressors and inotropes with severe lactic acidosis while on CCVH and maximum support.

Cardiopulmonary arrest and CPR done but he did not revive and declared death at 1045H.

Discussion

Coronary artery stenosis post valve surgery is a rare entity in cardiac surgery, with incidence anywhere between 0.3 and 5% [1, 6]. Initially described by Roberts and Morrow in 1967, during the postmortem histological analysis of coronary arteries in patients who had undergone AVR, it is now a recognized phenomenon as a cause of postoperative angina and ventricular impairment developing in individuals post valve-surgery with preoperative normal coronary arteries [2,7,8].

A potential risk factor for acute coronary ostial obstruction in AVR is occlusion of the left main coronary ostium due to the surgical procedure itself, which can occur with improper positioning of the prosthesis [9]. Indeed, Pillai et al. reported that direct post-traumatic thrombosis due to excessive aortic traction or over-tightening of the new aortic valve suture ring can lead to sudden intraoperative hemodynamic collapse and fatal outcomes [10]. Farid et al., conducted a postmortem autopsy analysis and identified a number of hospital deaths attributed to coronary ostial obstruction after AVR [10]. In their study, the authors suggested that patients' underlying conditions and aortic root size could influence the risk of coronary artery compromise; however, the cause of death in these patients was attributable, at least in part, to encroachment of the coronary ostia due to surgical factors such as a high level AV suture line [10, 11].

Examples of theories that were mentioned as a possible cause of coronary obstruction:

1. The insertion of coronary cannula for cardioplegia, causing immediate traumatic insults on the coronary vessels; intimal thickening, and fibrous proliferation.
2. Secondary to turbulent blood flow around the prosthetic valves.
3. Immunological reaction to the graft utilized in valve surgery; and genetic predisposition, has all been postulated as potential causes [5, 11, 12, 13, 14].

Intraoperative TEE is a useful tool for identifying the mechanisms of hemodynamically urgent situations in cardiac surgery [13]. TEE can provide detailed information during cardiac valve replacement surgery, including prosthetic valve motion, pathological leaks, and findings related to other valvular diseases. On the other hand, it is easy to overlook adjacent structural problems with TEE [14]. Cardiologists or skilled cardiothoracic anesthesiologists typically focus on prosthetic valvular leaflet motion and paravalvular leakage, which can indicate that the AV is well sutured or well positioned [13,14]. However, it is much more important with TEE to closely evaluate the new relationship between the bilateral coronary ostium and new prosthetic valve, as well as to monitor for new RWMA [15]. We know that the coronary arteries run near the valves, and the coronary ostium is located near the aortic valve.

In the present case, detection of the RWMA by TEE was critical for deciding to perform Coronary Angiography, and doing so to prevent a poor outcome due to abrupt coronary arterial obstruction.

Ono et al., reported a case of left coronary ostial obstruction that occurred approximately 20 minutes after withdrawing CPB [16]. In their case, the suggested mechanism of obstruction was a floating calcified plaque that obstructed the coronary ostium during decalcification of AV lesions.

Presentation consisting of angina and ventricular arrhythmias, usually occurring some months after initial valve surgery is common, although cases of electromechanical dissociation as

early as 12 days post the operation have been documented . The diagnosis is often made by angiography with revascularization, either by percutaneous intervention or a coronary artery bypass graft [17, 18].

Our patient developed acute obstruction of the proximal LM intra operatively could not be attributed to specific factors while a lot of pathological conditions been accused for example protamine reversal versus embolization from cannula site or other sites or related to undiagnosed hypercoagulable state related to paraneoplastic disorder which cannot be proven or disprove all are being in assumption form. The acuteness would imply that the mentioned theories for coronary stenosis are not applicable in this instance. The clinical sequelae in this patient may strongly points to calcium emboli mobilized during the initial aortotomy, causing occlusion of the proximal LM. Pifarre et al., document a similar occurrence, in their patient undergoing AVR [19]. Sanchez-Recalde et al., describe a patient who developed total ostial occlusion of the RCA and died twelve days post AVR for aortic stenosis. The post mortem results showed accumulation of calcium-like material occluding the RCA ostium [20].

Kinoshita et al. described a case of coronary artery spasm occurring in cardiac procedures for both revascularization and valve surgery [21]. In their series of five patients, all exhibited features of hemodynamic collapse and ventricular tachyarrhythmia during the period of reperfusion following discontinuation of CPB. In addition, the free wall of the RV exhibited poor contractility and dilatation. As in our patient, an intra-aortic balloon pump was initially utilized to improve the cardiac index but no improvement so AV-central ECMO was inserted. However, they rule out mechanical obstruction as the cause of the circulatory failure although, in one patient undergoing re-fixation of an aortic valve, a probe was inserted into the RCA ostium. Treatment consisted of nitroglycerine and diltiazem to unmask the effects of the vasospasm, with success in four of the five patients strongly pointing to spasm as the cause.

Although CABG has been the choice of treatment for left main disease for many years, PCI and stenting might be equally effective in certain cases due to the continuous improvement and refinement of these techniques over the past 20 years. Thus, coronary obstruction induced by calcium debris is not a contraindication for PCI, despite the associated risk of certain complications [22, 23].

Conclusion

We have demonstrated a rare phenomenon of acute occlusion of the LM coronary ostium post AVR and thereby leading to hemodynamic instability. Rapid intervention and ECMO implantation and percutaneous revascularization was successful in returning the patients hemodynamic status even for a period of time.

Sudden hemodynamic collapse with left heart failure occurs during any point of the intra operative period following AVR, acute coronary ostial obstruction should be considered. Surgeons should be highly aware of this serious complication after AVR, and

anesthesiologists skilled at TEE should work with surgeons to facilitate rapid identification of the etiology of potential coronary complications. Through vigilant awareness and appropriate intervention, it is possible to avoid fatal compromise from acute coronary ostial obstruction, which can ultimately reduce patient mortality and morbidity.

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