

Case Report

A Case Report: Iatrogenic Aortic Dissection During Minimally Invasive Mitral Valve Surgery: A Successful Management

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Abstract

Minimally Invasive Valve Surgery (MIVS) are on the rise in the field of heart valves surgery, and considered more favorable than traditional open approach. Nevertheless, the complication of Iatrogenic Aortic Dissection (IAD) remains a concern. We present a case, highlighting the clinical management of Acute IAD of ascending aorta during Minimally Invasive Mitral Valve Surgery (MIMVS) in a 61-year-old lady. Diagnosis of primary Mitral Regurgitation (MR) secondary to mitral valve prolapse was made and surgery was conducted. Exploration showed a hematoma in ascending aorta, which was confirmed by Trans-Esophageal Echocardiography (TEE) leading to Aortic Dissection diagnosis. Thoracotomy was done. Elephant stent graft and mitral valve repair were conducted successfully.

Keywords: Iatrogenic aortic dissection; Mitral valve replacement; Thoracic endovascular aortic aneurysm repair; Case report.

Introduction

MIVS gained popularity due to its rapid progress and nowadays preferred over full thoracotomy because of its more favorable outcomes in terms of post-surgery complications in native and redo cases [1]. However, MIVS might come with an increased risk for phrenic nerve palsy, stroke, or IAD. IAD is a very rare and severe complication, with an incidence of only 0.15% during or after non-aortic surgery and high mortality between 30-50% [2]. So far, only cases of IAD after Minimally Invasive Aortic Valve Surgery and Right Atrial Cryoablation during port access have been discussed [3]. And presenting our experience of an IAD during MIMVS, will bring more light to surgeons to

better manage these cases as this procedure is innovative and little is known about how to manage IAD.

Patient's information

A 61-year-old woman, with no pre-existing medical conditions, was admitted to our hospital with effort intolerance; shortness of breath during physical activity for about 2 weeks.

Examination

Auscultation showed normal vesicular breath sounds and a pan-systolic murmur (3/6) at the apex, radiating to the left axilla, left lung base, with a mid-systolic click. Normal jugular vein pressure and no pedal edema were noted.

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Table 1: Examination findings on admission.

Blood pressure	117/64 mmHg
Arterial pO ₂	76mmHg
ECG	Mild left ventricular hypertrophy (Sokolow-Lyon-Index 4,6mV) Sinus rhythm (82 bpm)
TTE	Mild displacement of the mitral leaflets (P1) into the left atrium during systole, slight leaflet thickness and stiffness. EF=72% LA=30mm, LV =42mm, IVS = 10mm
Doppler color flow imaging	Mitral valve orifice area = 7.9 cm Tricuspid valve pressure gradient = 21 mmHg mild pulmonary regurgitation
Sonography of vessels	Carotid artery (7mm), jugular vein (8 mm) and femoral artery (7.2 mm) and vein (7.9 mm) were without pathological findings.
Thoracic Contrast Tomography (CT) scan	Mild calcification of aortic wall Pleura slightly thickened
Coronary CT scan	Mild stenosis (about 10%) of the proximal RCA
Brain CT scan	Small focal infarction in the basal ganglia region Mild cerebral atrophy
Lung function	FEV1 = 2.1L MVV= 81 L/min

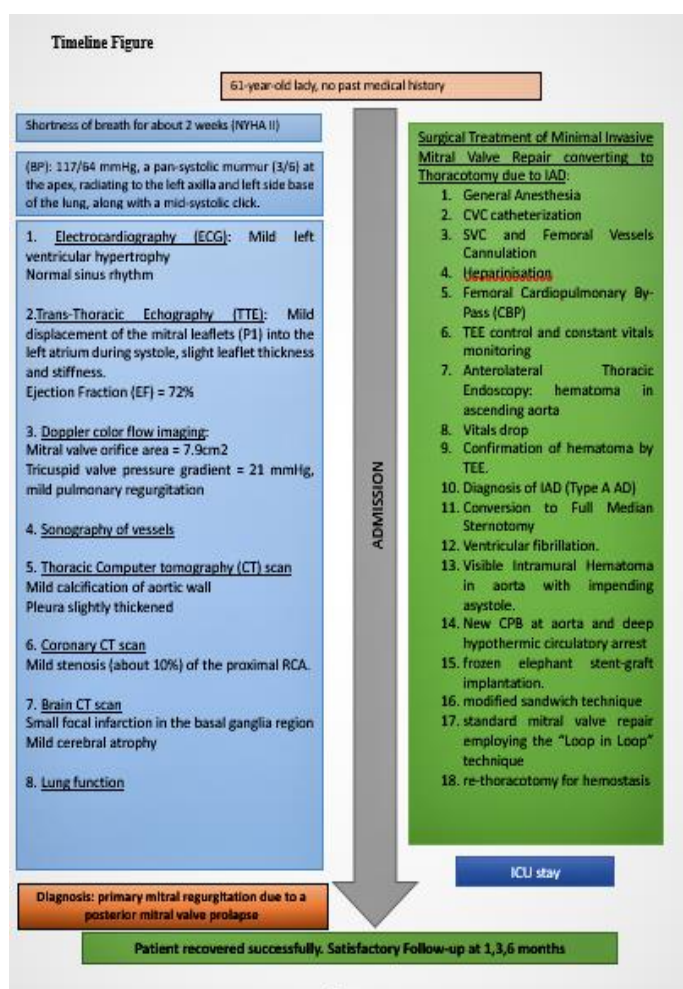


Figure 1: Timeline figure of events.

Diagnosis

Investigations were done, (as shown in Table 1). It was determined to be primary MR due to a posterior mitral valve prolapse. Mitral valve repair is mostly selected over mitral valve replacement in primary MR. As symptomatic patients have poor long-term prognosis, early surgical intervention in high-throughput centers with good expectable outcomes should be targeted.

Surgical management

Post general anesthesia, a Central Venous Catheter (CVC, Arrow International Inc. Pennsylvania, 19605, USA) and a guide-wire for later Superior Vena Cava (SVC) cannulation into the inner jugular vein were placed. Afterwards, we made a 2-cm incision at the right groin to prepare the femoral vessels. After heparinisation and sufficient Activated Clotting Time (ACT), Cardiopulmonary Bypass (CPB) was installed by cannulating the femoral vessels and SVC via jugular vein and connected them to the heart-lung machine. The operation was carried out under constant TEE control. Invasive blood pressure monitoring was installed in the right radial artery and rectal temperature was followed.

Whilst still creating the endoscopic access, we asked the Perfusionist to run simultaneously the CPB and decrease the body temperature in order to continue the surgery without disturbance.

The surgical field was exposed using the anterolateral port access approach in the proximal axillary line of the 4th right intercostal space [4]. Subsequently, the thoracic endoscope was inserted; a slight adhesion of the pleural leaflets was detached, exposing the surgical field. A hematoma was observed in ascending aorta (Figure 2A). The Anaesthesiologist noted a drop in BP, pulse, a spike in CVP. TEE confirmed the hematoma in ascending aorta and diagnosis of a type a acute Aortic Dissection was made (Figure 2B).

Immediately, a shift to full median sternotomy was performed. Suspecting the dissection might originate from false-lumen cannulation of the femoral artery, we immediately stopped the CPB. The heart rate decreased considerably but not fully stopped.

Post-thoracotomy, a thinned aortic wall with intermural hematoma was conspicuously seen, confirming IAD. Ventricular fibrillation was noted.

Worsening of the situation; an impending asystole, hypothermia and lack of time, we urgently needed an alternative circulation. As a retrograde dissection caused by the tip of the cannula was suspected, peripheral femoral cannulation was aborted in favor of direct ascending aortic cannulation for a new CPB (Fig-

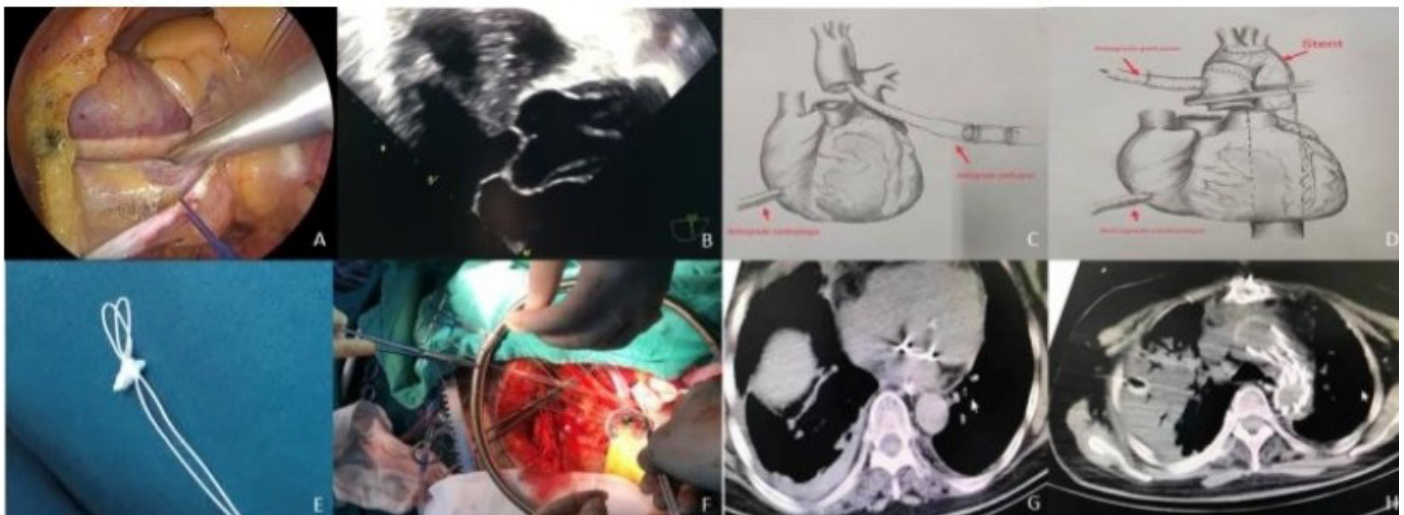


Figure 2: Steps in Surgery.

ure 2C). Axillary cannulation was not also preferred due to the emergency. RCA involvement was ruled out.

Histidine-Tryptophan-Ketoglutarate (HTK) solution (Custodiol; Dr. Franz Kohler, Chemie GmbH, Bensheim, Germany) was administered into both coronary sinuses for myocardial protection. Once reconstitution of body perfusion attained, Deep Hypothermic Circulatory Arrest (DHCA) was induced. No intimal damage or entry site was visible in the ascending aorta and aortic arch. We implanted an elephant stent-graft (MicroPort Medical Co, Ltd, Shanghai, China) into the true lumen of the aortic arch and the proximal part of the descending aorta. After inflation of the stent-graft, we fenestrated the part covering the three arch branches by an island anastomosis with interrupted 2-0 prolene suture, fixing the branches to the window of the stent. To elongate the proximal end of the stent-graft, we sewed an artificial vessel to the attached vascular graft, gaining space for the cannula insertion. In the next step, we clamped the artificial vessel and cannulated the descending aorta via the artificial vessel. We reinforced the proximal end through vascular patches and wrapped the artificial vessel with a slice of the autologous pericardium. After 19 minutes of DHCA, we reconstituted the body perfusion and started to increase the temperature slowly; this gave us time to reinforce the aortic root using a modified sandwich technique, as described by Tang et al [5].

Lastly, we connected the aortic root to the artificial vessel to complete the aortic repair. (Figure 2D). A shunt was established between the artificial chamber and the right atrium via an end-to-side anastomosis with an artificial blood vessel. After stabilizing the vitals, we opened the left atrium, exposing the mitral valve apparatus. The mitral valve was slightly thickened, with ruptured P1, P2 papillary muscles and mitral prolapse. Standard mitral valve repair employing the "Loop in Loop" technique was performed (Figure 2E). When the water probe indicated a satisfying closure and no significant regurgitation, we implanted a #28 mitral annulus (Medtronic® semi-rigid band) (Figure 2F). After the air exclusion with the head-down position, the atrial septum incision was closed. The heart started to beat again after an external 200 Joules defibrillation. Blood was returned to the patient and she was finally weaned off the CPB. Total CPB time was 280 minutes, aortic cross-clamp time was 115 minutes. Three drainage tubes were inserted followed by chest closure. The patient was transferred to the ICU.

Post-surgical management

The same night; a re-thoracotomy was performed for hemostasis, requiring massive blood transfusion.

Patient's ICU stay was 17 days. Total intubation time was 3 days, with Bi-Level Positive Airway Pressure (BIPAP) assistance for another 6 days and double channel oxygen for 8 days. Furthermore, CT scan, X-ray, transthoracic echocardiography and blood gas analysis were routinely performed.

Follow-up and Outcomes

The patient's recovery was good. TEE showed normal EF of LV (55%). Day 32 post-operative, the patient was discharged in stable condition. One, three, six months of postoperative examinations were normal.

Discussion

Preoperatively, the typical risk factors for AD were not found. Sonographic evaluation of femoral and jugular vessels did not show pathological findings. However, pre-operative assessment of aortic wall by angiography was not done, as no history of peripheral vascular disease was reported and examination results were normal. We assumed a retrograde dissection from femoral cannulation. The cannula tip might have hurt the intima layer of the fragile aortic arch, inducing a Stanford type A aortic dissection. The most common entry site for IAD in non-aortic cardiac surgery is the abdominal aorta [6,7].

Dissection can be identified as follows: Blue discoloration and expansion of the ascending aorta, intractable bleeding from aortic sites, and high perfusion line pressures with associated systemic hypotension. In our case, we started the CPB at a point under TEE surveillance; however, we had not yet obtained visual control of the surgical field. IAD was confirmed at the moment both by TEE and the naked eye. Although coming with an increased risk for unobserved AD allows the induction of hypothermia while still establishing the surgical field, this practice is timesaving. A delayed induction of hypothermia after complete set up would require the entire team to wait for an additional 10-15 minutes. By starting CPB before pericardiotomy and soon after thoracotomy, minimally invasive cardiac surgery becomes feasible in patients unable to bear unilateral lung ventilation [8]. We suggested that CPB is started only when the surgical field and TEE monitoring are already seen at the same time to minimize the risk of IAD.

Once IAD is confirmed, the surgeon discontinued the CPB immediately and secure an alternative peripheral site of cannulation. This ensures that the blood flow is through the true aortic lumen. We described direct aortic cannulation as an emergency method to reconstitute bridging perfusion under a very urgent situation of low blood pressure.

To maintain an adequate perfusion pressure, we directly cannulated the ascending aorta after probing for the true lumen and assessing the intimal condition, similar to the "Samurai" cannulation method proposed by Kitamura et al. However under different circumstances [9].

Conclusion

To summarize; close attention, discovery on time, effective treatment is required in a modern minimally invasive surgery team. Regarding the effective management of IAD, the surgeon's experience, degree of specialization, and ability are decisive.

Abbreviations: MIVS: Minimally Invasive Valve Surgery; IAD: Iatrogenic Aortic Dissection; MIMVS: Minimally Invasive Mitral Valve Surgery; TEE: Trans-Esophageal Echocardiography; MR: Mitral Regurgitation; ACT: Activated clotting time; CPB: Cardiopulmonary bypass; RCA: Right Coronary Artery.

Declarations

Ethics Statement and Informed Consent: The written consent of the patient was taken for the publication of any potential images or data published in this article.

Authors Contribution: All the authors have contributed equally in writing this article.

Conflict of interest: No conflicts of interest relevant.

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