

# LV Failure during Thoracoabdominal Aneurysm Repair

Mikiel Ewida SAA, Christopher Lawton DO, Brian Shin MD, Alex Steed CAA  
Indiana University School of Medicine and Indiana University Health - Indianapolis, Indiana



Indiana University Health

## Introduction

The patient is a 66-year-old male who presented for an extensive IV thoracoabdominal and iliac aneurysm repair. The patient has a significant medical history, including Marfan's syndrome, residual thoracoabdominal aortic dissection, extensive IV thoracoabdominal aortic aneurysm, and bilateral common iliac artery aneurysms. The patient's prior surgical history includes replacement of the ascending aorta with aortic valve replacement, and thoracic endovascular repair for a type B aortic dissection of zones 2-6. The patient still has residual chronic dissection from zones 6-11 with a left common iliac artery aneurysm of 4.2cm and a right common iliac artery aneurysm of 3.2cm. A cardiac workup, including an echocardiogram, revealed normal Left Ventricular Ejection Fraction (LVEF) and severe concentric left ventricular hypertrophy. A left-sided cardiac catheterization showed no significant coronary artery disease.

## Materials & Methods

The anesthetic plan for this case included the administration of general anesthesia with a left-sided double lumen endotracheal tube (ETT). Monitoring and lines for this case included the use of a transesophageal echocardiogram (TEE), cerebral oximetry, neuromonitoring such as Motor Evoked Potentials (MEP) and Bispectral Index (BIS), an awake radial arterial line placed in the right radial artery, a Quad-Lumen and MAC introducer Central Venous Catheter (CVC) and a 20G IV for access. A lumbar drain was placed prior to the procedure to help prevent spinal cord ischemia. The induction plan included the use of propofol, fentanyl, and rocuronium. Maintenance of anesthesia was achieved through the use of a Total Intravenous Anesthesia (TIVA) with propofol and remifentanyl, as well as nasal and bladder temperature probes. The hemodynamic goals during the procedure were to prevent hypertension while maintaining adequate perfusion.

During the surgical procedure, techniques used included cardiopulmonary bypass (CPB) and deep hypothermic cardiac/circulatory arrest. The patient was positioned in a right lateral decubitus position. Type and Cross was obtained as well.

## Echocardiogram

### TEE Measurements – Preintervention

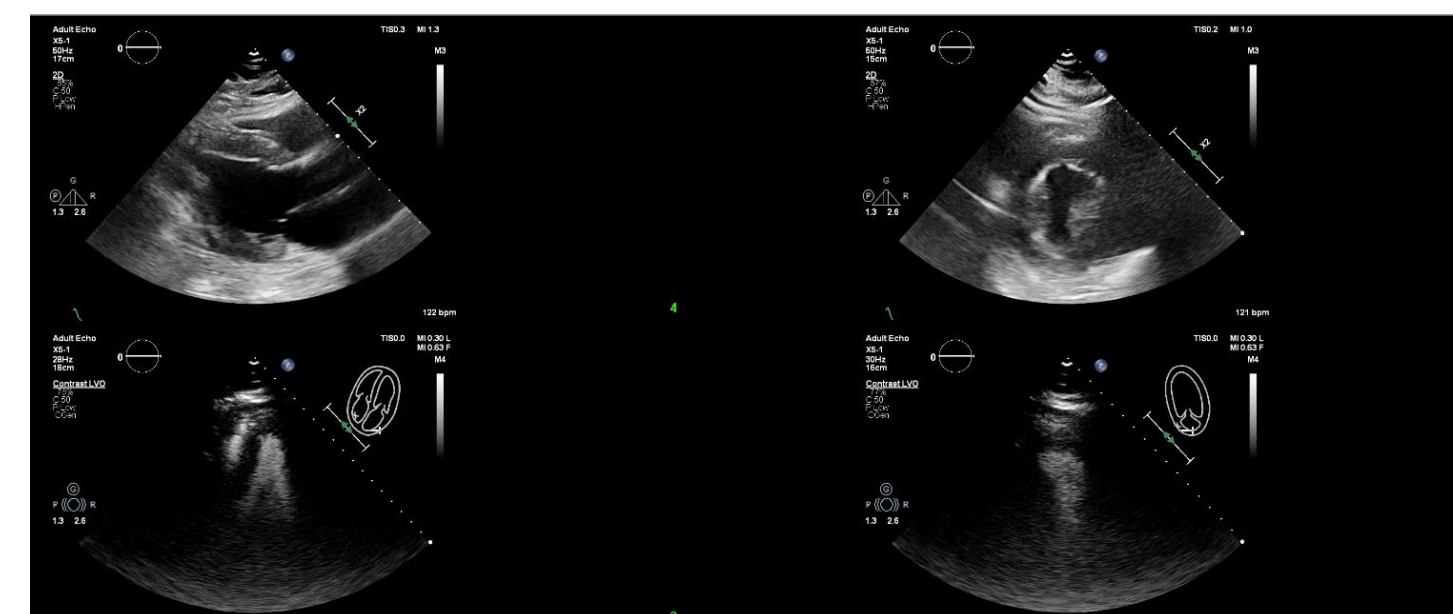
Left Atrium: Dilated.  
Interatrial Septum: Normal.  
Left Ventricle: Chamber Size Normal, Wall Thickness Moderate-Severe Concentric LVH, LVEF 55% Visually Estimated.  
Tricuspid Valve: Moderate Regurgitation  
Pulmonary SL Valve: Moderate Regurgitation.  
Mitral Valve: Mild Regurgitation  
Aortic SL Valve: Bioprosthetic AV Valve Visualized, Mild Central Jet and no Prosthetic leak. Mean PG Under Anesthesia 9mmHg and calculated AV1 1.92cm<sup>2</sup>.

### TEE Measurements – Postintervention

A Final TEE exam was performed while on VA ECMO and vasopressor support. Inferior and lateral wall akinesis was seen initially, which gradually improved. LV function moderately decreased by visual estimation. Mildly Dilated RV Noted with Mild Dysfunction. Venous ECMO cannula terminates in SVC.

### Figure 1.

Pre-Operative & Final Intraoperative TEE Report.



### Figure 2.

Post Operative Echocardiogram with Cardiac Doppler. Findings included a LVEF of 53% and mild concentric LVH.

## Results

Upon the patient's arrival to the operating room, standard monitors including cerebral oximetry were placed and baseline values were recorded. An R radial arterial line was inserted while the patient was awake, and the patient was induced with fentanyl and propofol. A muscle relaxant was administered, and a left double lumen tube was placed for ventilation. A propofol and remifentanyl infusion was initiated, as well as nasal and bladder temperature probes and a transesophageal echo probe. Additionally, a quad lumen and MAC introducer CVC was inserted. The patient was positioned, and a baseline ACT and TEG were within normal levels. Amicar was given, and CSF was regularly drained during the procedure.

During the surgical procedure, entry to the surgical field was achieved without significant incident. Cardiopulmonary bypass was initiated without issue and deep hypothermic circulatory arrest was also performed, with the patient being cooled to 19.2 degrees Celsius for 22 minutes before being gradually rewarmed. Cerebral oximetry values remained within normal limits throughout the procedure. After the repair was completed, the patient was gradually rewarmed and an attempt to take the patient off cardiopulmonary bypass was made. However, the patient experienced unstable blood pressure despite aggressive vasopressor support. A transesophageal echocardiogram revealed an enlarged right ventricle and decreased biventricular activity, and it was decided to revert to cardiopulmonary bypass. During the second attempt at weaning off bypass, milrinone was also added. The TEE showed a moderately decreased ejection fraction while on CPB with lateral and inferior wall akinesis. The decision was made to convert to extracorporeal membrane oxygenation. At the end of the procedure, decreased MEP signals of the lower extremities were observed.

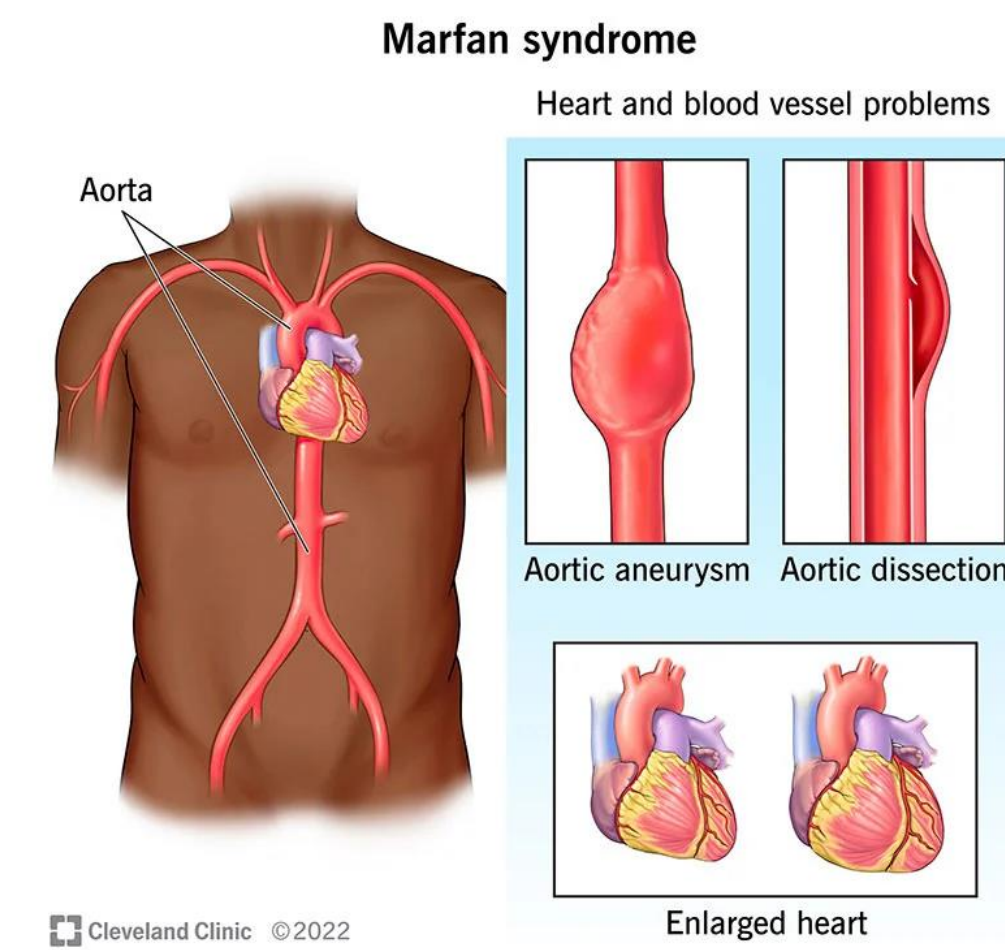
## Extent IV Thoracoabdominal Aneurysm



### Figure 4.

CTA. Extent IV thoracoabdominal aneurysm seen. Bilateral common iliac aneurysm seen.

## Marfan Syndrome



### Figure 3.

Marfan's syndrome is a connective tissue disorder that manifests by changes in the skeleton, eyes, and cardiovascular system. The prevalence is 1 in 5000 and is transmitted by autosomal dominant inheritance. It results from a defect in the extracellular glycoprotein called fibrillin-1. The loss of function of this glycoprotein results in loss of structural support in microfibril-rich connective tissue and excessive activation of (TGF)- $\beta$  signaling.

## Discussion

The nature of this surgery was high-risk due to the extensive IV thoracoabdominal aneurysm repair, which can carry a high mortality rate. The patient's prior echocardiogram revealed moderate to severe Left Ventricular Hypertrophy (LVH) with a normal ejection fraction as shown in figure 1. Due to the nature of the patient's diastolic heart failure exacerbated by the patient's Marfan's syndrome, the heart failed to function when provided with volume during the attempt at weaning off cardiopulmonary bypass (CPB). Despite vasopressor and inotropic support, the first attempt at coming off CPB resulted in unstable hemodynamics. The patient was given multiple doses of calcium bolus, vasopressin, and norepinephrine infusion with no improvement. During the second attempt, milrinone was added via bolus and infusion to provide lusitropy and inotropy. In figure 3, eccentric hypertrophy is commonly associated with Marfan syndrome, but the patient had the opposite effect, with concentric disease leading to diastolic dysfunction.

The decision to use extracorporeal membrane oxygenation (ECMO) was made after much discussion with the surgeon, and after multiple attempts at weaning off CPB failed. Cerebral oximetry levels were within normal ranges throughout the procedure, and the total time of hypothermic circulatory arrest was 22 minutes, well within the range of ischemic tolerance time. Bleeding also occurred, in which the patient was given Profil-9, desmopressin, and heparin reversal while on ECMO. The patient was given 13 units of Fresh Frozen Plasma (FFP), 12 units of Packed Red Blood Cells (PRBC), 5 units of Cryoprecipitate, 1750mL of albumin, and 2350mL of Cell Saver Blood. These changes in clotting factors, volume, and metabolites could have also played a role in the failing heart.

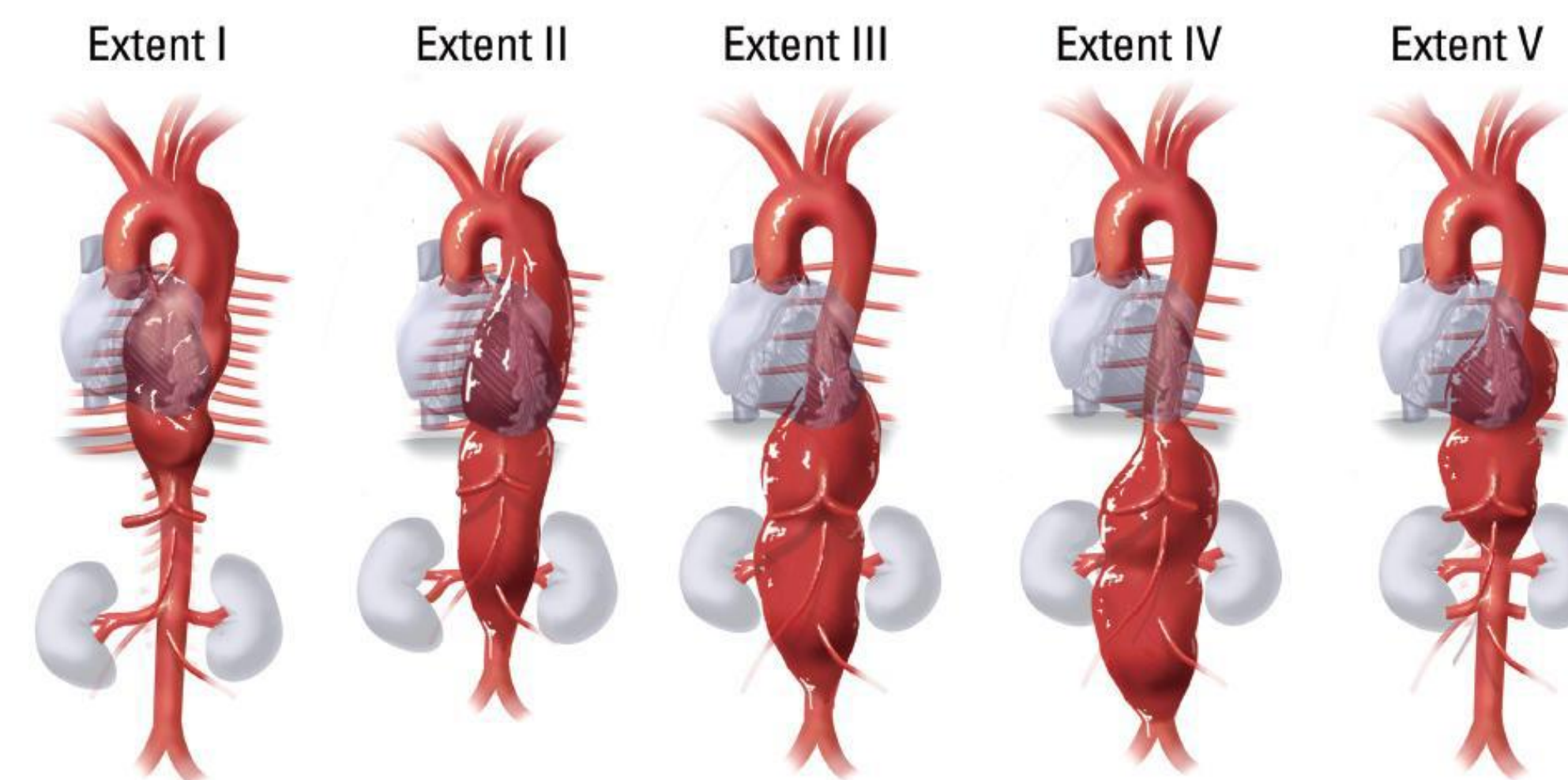
## Conclusions

On the first attempt of weaning off cardiopulmonary bypass (CPB), epinephrine boluses of up to 0.7mg as well as an infusion were used, along with a vasopressin infusion, and a norepinephrine infusion at a rate of 10mcg/min. During the second attempt at coming off CPB, milrinone was added at a rate of 1.8 mg/hour. Due to the patient's prior knowledge of the transesophageal echocardiogram (TEE) showing severe ventricular hypertrophy, the decision could have been made to maintain maximum hemodynamic support from the very first attempt at coming off CPB. Even with a normal ejection fraction, the heart can still be functioning at a suboptimal level.

In this case, the patient had prior aorta repairs that included bypass, and there was evidence to support that the patient had a weak heart to begin with, considering his pre-procedure TEE exam. The nature and extent of the surgery, which included massive fluid shifts and metabolic changes, majorly contributed to this result. The patient's prior comorbidities, such as Marfan's syndrome, were also major contributors to the failure of the Left Ventricle.

## References

1. Frederick, J., & Woo, Y. (2012). Thoracoabdominal aortic aneurysm. *Annals Of Cardiothoracic Surgery*, 1(3), 277-285. doi:10.3978/j.issn.2225-319X.2012.09.01
2. Judge, D. P., & Dietz, H. C. (2005). Marfan's syndrome. *Lancet (London, England)*, 366(9501), 1965-1976. https://doi.org/10.1016/S0140-6736(05)67789-6
3. Robbins, S. L., Cotran, R. S., Abbas, A. K., Aster, J. C., Kumar, V., Turner, J. R., & Perkins, J. A. (2021). *Robbins & Cotran pathologic basis of disease*. Elsevier.
4. Robinson, P. N., Arteaga-Solis, E., Baldock, C., Collod-Bérédou, G., Booms, P., De Paeppe, A., Dietz, H. C., Guo, G., Handford, P. A., Judge, D. P., Kieley, C. M., Loeys, B., Milewicz, D. M., Ney, A., Ramirez, F., Reinhardt, D. P., Tiedemann, K., Whiteman, P., & Godfrey, M. (2006). The molecular genetics of Marfan syndrome and related disorders. *Journal of medical genetics*, 43(10), 769-787. https://doi.org/10.1136/jmg.2005.039669
5. Tomislav, M. (2022). Marfan syndrome: Causes, symptoms, diagnosis & treatments. Cleveland Clinic. Retrieved January 21, 2023, from https://my.clevelandclinic.org/health/diseases/17209-marfan-syndrome



### Figure 5.

Comparison of thoracoabdominal aneurysm extent based on the Crawford Scheme. In 1986, Crawford represented the first Thoracic-Abdominal Aortic Aneurysm schematic based on the anatomical locations of the aneurysm. Extent I involves most of the thoracic descending aorta, from the origin of the Left innominate to the suprarenal abdominal aorta. Extent II covers the innominate to the aortoiliac bifurcation and is the most extensive. Extent III covers the distal thoracic aorta to the aortoiliac bifurcation. Type IV is limited to the abdominal aorta exclusively, below the diaphragm. This scheme was modified by Safi to include Extent V, which covers the distal thoracic aorta and including the celiac and superior mesenteric origins.