

## Catastrophic Intracardiac Thrombosis: A Case Report

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### Abstract

Intracardiac thrombosis is a rare and often fatal event. We present an unusual case report of a young male developing multiple intracardiac thrombi intraoperatively and surviving.

**Keywords:** Intracardiac Thrombosis; Thoracoabdominal Aortic Aneurysm (TAAA); Transoesophageal Echocardiogram (TOE)

### Introduction

Intracardiac thrombosis is a potentially catastrophic life-threatening rare event carrying a mortality rate of more than 85% [1]. We present a case of a 31-year-old male who suffered multiple intracardiac thrombosis and survived.

### Case Report

A 31-year-old male with a past medical history of Marfan Syndrome presented with a Type A aortic dissection in June 2020. He required an emergency ascending aorta replacement and mechanical aortic valve replacement. The patient re-presented in September 2020 with worsening back pain and was found to have a rapidly expanding residual extent II thoracoabdominal aortic aneurysm (TAAA), with a maximum diameter of 7.7 cm. He was transferred to a tertiary cardiothoracic specialist hospital with a plan of urgent surgery.

Pre-operatively, the patient had a transoesophageal echocardiogram (TOE) demonstrating mildly impaired right ventricular (RV) function and good left ventricular (LV) function. Intraoperatively the patient was found to have a large extensive TAAA aneurysm and a very frail aortic wall.

Half dose heparin was given. Left heart bypass was commenced between the right femoral artery and the left inferior pulmonary vein. Upon clamping the mid-descending thoracic aorta (DTA), the patient suffered an acute intraoperative aortic dissection from the clamp site down to the infra-renal aorta clamping of the aorta and had excessive bleeding throughout the procedure. An extent II TAAA repair was completed from the proximal DTA to just above the aortic bifurcation. Upon completion of the TAAA repair, left heart bypass was discontinued and the venous cannula removed from the left inferior pulmonary vein. Protamine was given together with cryoprecipitate, protamine prothrombin complex and platelets based upon heparin thromboelastography.

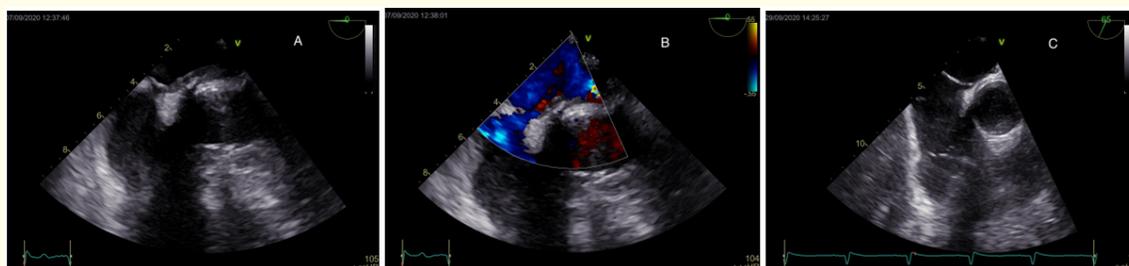
During haemostasis the patient experienced two episodes of severe desaturation and hypotension. TOE demonstrated a large clot in the right atrium (RA), RV, main pulmonary artery (PA), and through a patent foramen ovale into the left atrium (LA). The patient was unresponsive to inotropic support and hence cardiopulmonary bypass recommenced under full re-heparinisation via the femoral vein and artery.

An incision was made in the main pulmonary trunk and clot was suctioned from the PA and RV by crossing the pulmonary valve. The LA was entered via the left inferior pulmonary vein and clot evacuated. Further incision was made in the RA and clot suctioned out. Though these manoeuvres improved oxygen saturations, there remained significant clot burden on TOE and an akinetic RV. The decision was made to convert bypass to veno-veno-arterial extracorporeal membrane oxygenation (V-V-A-ECMO) and the patient was transferred to the intensive care unit. No further protamine was administered.

On the third post-operative, computed tomography (CT) Head demonstrated longstanding infarcts with possible hypoxic-ischaemic injury. CT pulmonary angiogram showed no central pulmonary embolism of intra-cardiac filling defects. TOE on day 3 showed severely impaired RV function with residual clot in the RV and mild-moderate LV impairment. A repeat scan one-week post-operatively demonstrated slight improvement in RV function but residual thrombus at RV apex and LA appendage thrombus. Both clots resolved but there continued to be mild-moderate impairment in the LV and RV. The arterial limb was successfully decannulated from ECMO around one week post-operatively, but the patient required a further week of veno-venous ECMO support, being successfully decannulated from this two weeks post-op. He required a percutaneous tracheostomy be inserted for a prolonged weaning period. Despite suffering from delirium, the patient underwent tracheostomy decannulation 4 weeks after his surgery and was discharged to the ward 7 weeks after his operation. He was then repatriated to his local hospital for ongoing rehabilitation needs and psychological support. At his latest follow up, the patient remains neurologically intact and is fully functional and independent.



**Figure 1:** Transoesophageal echo imaging taken during surgery. (A) Thrombus present in the right ventricle. (B) Thrombus present in the right ventricle inflow/outflow track. (C) Thrombus cleared from right ventricle.



**Figure 2:** Transoesophageal echo imaging taken during surgery. (A) Thrombus present in intra-atrial septum. (B) Colour doppler over intra-atrial septum. (C) Thrombus cleared from intra-atrial septum.

### Discussion

Catastrophic intracardiac thrombosis is a rare and life-threatening complication of cardiac surgery. There is little literature available on such events, consisting of case reports and small case series. To date there exists no reports of survival from multiple intracardiac thrombosis after TAAA surgery.

Williams., *et al.* [1] performed a systematic review identifying the common features of intra-cardiac thrombosis (ICT) and pulmonary embolism after cardiopulmonary bypass (CPB). In 48 patients they demonstrated a high mortality of 85.4% with common features including congestive heart failure (50%), platelet transfusion (37.5%), cardiopulmonary bypass duration greater than 3 hours (37.5%) and aortic injury (27.1%). In the current case the last three of these common features are applicable.

There was significant endothelial injury due to the patient having an intraoperative dissection which promotes an increase in plasma fragments as well as increase production in tissue factor and collagen [1,2]. Prolonged CPB times seen in aortic surgery usually predispose to hypercoagulability but the increased thrombin generation that occurs may also favour hypercoagulability [1-4]. Furthermore, haemodilution as well as an acquired antithrombin deficiency may have occurred, thus diminishing the anticoagulation properties of heparin [1].

As in this case, the majority of post bypass ICT is precipitated by heparin reversal with protamine or the administration of procoagulant products, such as cryoprecipitate, protamine prothrombin complex, fresh frozen plasma, platelets, and tranexamic acid. However, the authors feel that this case was not unusual in terms of the post-bypass management. Following extent II Thoraco-abdominal aneurysm repair, it is standard protocol to administer haemostatic blood products to counter the expected coagulopathy associated with prolonged bypass time and heparinisation. The choice of haemostatics was also guided by thromboelastography. On this occasion, following such a clinical pathway resulted in a pro-coagulable state and massive ICT. However, there is little in the patient's history or intra-operative events that would predict this. As such it is difficult to implement changes to prevent future events. The authors recommend vigilance of all members of the operative team as well as developing strategies to deal with this rare but life-threatening complication.

We attribute three contributing factors to the ultimately successful outcome achieved in this case. Firstly, the expertise of the cardiac anaesthetist in TOE, allowing early diagnosis and the sites of thrombosis to be identified and targeted. Secondly, the approach and diligence of the operative surgeon in ensuring a rigorous thrombectomy, and finally, the capability of the unit to support the patient on VA-ECMO and allow sufficient time for the patient to be anticoagulated and any remaining ICT to dissipate. The role of all members of the multi-disciplinary team working together to achieve a successful outcome cannot be over-emphasised in this case [5].

### Conclusion

Catastrophic intracardiac thrombosis is a rare and life-threatening complication of cardiac surgery. Despite being difficult to predict prior to the event, strategies can be implemented to optimise treatment including routine use of TOE, rigorous surgical thrombectomy and the use of extra-corporeal membrane oxygenation as a bridge to recovery.

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