Anesthetic Management of Cardiac Tumor Resections on Cardiopulmonary Bypass (CPB)

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INTRODUCTION

Cardiac tumors present a rare learning opportunity themselves, but a primary (1º) malignant myocardial tumor represents **less than 25%** of those cases. [2] Multimodality imaging (e.g. Echo, MRI, CT) has become a major component in identifying the etiology of cardiac masses, allowing tumor classification without the need for tissue biopsy in many cases. The classification of the tumor itself, however, is often not the cause of patients' symptoms; the clinical manifestations are primarily dependent on the **size** and **location** of the mass, as well as **invasion** into adjacent tissues. [1,2] A tumor located in the pericardium is often more accessible for surgical resection, leading to low risk of regrowth and high long-term survival rates. A 1º malignant tumor invading the myocardium, on the other hand, is less accessible and will likely require cardiopulmonary bypass (CPB) for resection, as well as having increased risk of recurrence and low survival rates, even after complete resection. [1,7] The best option for long-term survival in the case of an invasive myocardial tumor is **complete resection** of the mass with **clean margins**. Obtaining clean margins requires tedious resecting after removal of the bulk of the mass, and pathology consultation to confirm the margins are circumferentially tumor-free. While the dissection and removal of *any* myocardial tissue has the potential to disrupt the cardiac conduction pathway, resection of invasive and sizeable myocardial tumors will likely impact both the structure and function of the heart significantly. [8]

CPB, while an incredible advancement and adjunct in cardiothoracic surgery, has many adverse effects on the human body. One of the most notable effects from an anesthetic perspective is profound **multifactorial coagulopathy**; coagulopathic bleeding is one of the biggest risks with cardiac surgery, specifically when CPB is used. [4] Of the many factors contributing to post-CPB coagulopathy—including inhibition and/or destruction of fibrinogen and coagulation factors—**platelet dysfunction** and **destruction** are the primary causes of CPB-associated hemorrhage. [3] Platelet **count** is decreased significantly by hemodilution and high shear stress in the CPB circuit. Platelet **dysfunction** is due to several factors including the artificial surface of the circuit, heparin and protamine administration, and controlled hypothermia. [3, 6] Because platelet dysfunction is such a significant factor in post-CPB coagulopathy, platelet count alone is not a sufficient measurement to guide platelet replacement therapy and could lead to unrecognized **thrombocytopenia coagulopathy** in the event of post-CPB blood loss. In addition to coagulopathy, patients undergoing CPB are prone to metabolic abnormalities such as electrolyte imbalances and **dysoxia**. Dysoxia leads to aerobic metabolism dysfunction, and compensatory anaerobic metabolism causes **hyperlactatemia** in 10-20% of CPB cases. Subsequent **hypoxic lactic acidosis** is associated with severe metabolic acidosis and increased mortality. [6]

LEARNING OBJECTIVES

1. Clinical considerations and outcomes of myocardial tumors compared to pericardial tumors.
2. Significant coagulopathic effects of CPB and the major contributing factors.
3. Importance of coagulopathic monitoring and replacement throughout procedures involving CPB.

PATIENT DESCRIPTION

60-year-old female, ASA 4, presenting for open sternotomy and pericardial tumor resection, with possible tricuspid valve replacement, on cardiopulmonary bypass (CPB). Past medical history includes newly diagnosed rare-type non-small cell lung cancer, heart failure with preserved ejection fraction (HFpEF), moderate mitral (MR) and tricuspid regurgitation (TR), COPD requiring home O2 at 3 L/min via nasal cannula, iron-deficiency anemia requiring bimonthly iron infusions.

Patient presented to outside hospital ~ 2 weeks prior to previously stated procedure with complaints of chest pain, shortness of breath (SOB), and abdominal pain. She was admitted and found to have newly-onset atrial fibrillation (AF) with rapid ventricular response (RVR) and volume overload. Additional testing showed no acute abnormalities. After unsuccessful treatment with IV Furosemide and Diltiazem, patient was stabilized with a loading dose of Digoxin, and Metoprolol was used for rate control. Further review of chest CT scans from December 2024 and January 2025 showed mild **growth in known pericardial mass**. Patient was transferred to cardiothoracic care unit for consultation and evaluation for surgical intervention.

PRE-SURGICAL EVALUATION & TIMELINE

Transthoracic Echocardiogram (TTE): estimated EF 50–55%

Carotid Ultrasound: minimal atherosclerosis and/or stenosis

Left Heart Catheterization (LHC): no evidence of coronary artery disease (CAD)

Cardiac Magnetic Resonance Imaging (C-MRI): **9.1cm mass** centered in inferior pericardium, causing delayed relaxation of bilateral ventricles; suspected primary cardiac tumor rather than metastatic disease from lungs; appeared inseparable from right ventricle (RV) and inferior interventricular septum – characteristics suggesting **possible invasion** of RV and inferior septum; inferiorly bordered by suprahepatic region of inferior vena cava (IVC) and the diaphragm, no invasion suspected.

Patient scheduled for surgical intervention. **Initial estimated intraoperative mortality risk of 20% based on evaluation, imaging, and consultations.**

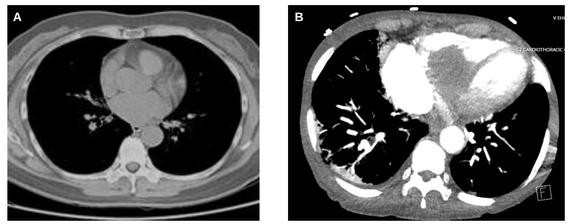

Figure 1. Normal thoracic CT scan (A) and CT scan of invasive myocardial tumor discussed in presented case (B).

SURGICAL & ANESTHETIC MANAGEMENT

AIRWAY: Easy bag-mask ventilation and intubation. **8.0 endotracheal tube** (ETT) placed via direct laryngoscopy (DL) with Mac 3 blade. There were **no difficulties or complications** with ventilation or maintaining oxygenation.

ACCESS: Right chest port (not used intraoperatively), **20G peripheral IV** (pIV) and **18G pIV**, **left radial** and **left femoral arterial lines**, and **central Cordis introducer** with floated **Swan-Ganz catheter** (SGC)

DECISION TREE: After sternotomy and pericardial dissection, the severity of the mass became very clear. The surgeon updated the patient's family with their options: abort the procedure and hope for a few more months or proceed with complete resection and an **updated intraoperative mortality estimate > 50%**. The family decided to proceed.

CPB START: Patient was adequately heparinized for CPB with 21,000U Heparin. Shortly after, the activated clotting time (ACT) dropped, suggesting that the patient's **low antithrombin** level was inhibiting effective anticoagulation. 500U of recombinant antithrombin (AT3) administered, correcting the ACT. Perfusion maintained heparinization throughout CPB and another 500U AT3 bolus was given about 2 hours into bypass.

RESECTION: The tumor was fully resected with minimal excess to maintain as much myocardium as possible. Several areas of abnormal surrounding tissue were **resected until confirmed microscopically clean** by pathology. All that remained of the right ventricle (RV) was the outflow tract.

RECONSTRUCTION: A new tricuspid valve was placed in the existing annulus, and the RV was repaired with endocardial *and* epicardial bovine tissue patches.

CPB STOP: With no surgical bleeding identified, but significant coagulopathic bleeding, the patient was weaned off CPB with AV pacing assistance and given 2g calcium chloride. Protamine was dosed based on blood heparin concentration (hep-con), returning the ACT to normal range.

At this point, we had given 4 units packed red blood cells (PRBCs), 6 units fresh frozen plasma (FFP), and 2 units cell-saver (CS) from perfusion.

CLOSING → ICU: Due to significant coagulopathic bleeding, 1mg activated coagulation factor VII (CoF-7a) was administered, along with additional calcium (1g), PRBCs (2), FFP (1), cryoprecipitate (2), and platelets (2).

About 1hr post-CPB, as the temporary chest closure was being sutured into place, the patient went into **severe metabolic acidosis** and **pulseless electrical activity** (PEA) with rapid drop in mean arterial pressure (MAP).

3mg epinephrine (given in 500mg boluses over < 5min) and another 1g of calcium, in tandem with **open cardiac massage**, recovered patient's pulse and MAP. Another 1mg dose of CoF-7a and 100mEq of sodium bicarbonate were administered while preparing for ICU transport, followed by additional 500mg boluses of epinephrine as needed to resuscitate and stabilize.

ICU: Patient remained unstable, requiring frequent code-dose epinephrine and immediate massive transfusion protocol (MTP) initiation. Despite aggressive resuscitation attempts and open cardiac massage, patient did not achieve return of spontaneous circulation; called within 1hr of ICU transport.

	Hb (g/dL) [11.4 – 14.4]	pH [7.35 – 7.45]	Lac (mmol/L) [0.4 – 0.8]	Plt (10E3/mcL) [150 – 400]	Fib (mg/dL) [200 – 393]	AT (%) [80 – 120]
PRE-OP	8.1	7.38	0.8	438	—	65
PRE-CPB	10.0	7.35	2.3	—	—	—
EARLY CPB	7.0	7.45	1.2	212	340	—
LATE CPB	8.4	7.39	1.9	—	—	—
POST-CPB	8.9	7.23	5.0	—	—	—
PRE-ARREST	< 6.6	7.17	6.6	—	—	—
ICU	9.0	7.17	12.75	57	184	—

Table 1. Notable lab values throughout discussed procedure. Hb: hemoglobin; Lac: lactate; Plt: platelet count; Fib: fibrinogen; AT: antithrombin.

DISCUSSION & CONCLUSIONS

While multimodal imaging is improving cardiothoracic practices in many ways, it is still lacking in its ability to comprehensively distinguish malignant invasion. Had the extent of this mass been notable in the image studies conducted, the surgical team would have perhaps advised against or cautioned the patient regarding increased intraoperative mortality.

Whether this mass was pericardial or myocardial, its size and proximity to the RV made it extremely high-risk, and this patient would have benefited greatly from preoperative hemodynamic optimization. Had her baseline levels been higher, pre-CPB autologous sequestration of both whole blood and platelet-rich plasma (PRP) could have allowed preservation of her own coagulation factors and re-administration during or after CPB weaning. [9,10]

In addition to platelet count, platelet function tests can provide valuable information on the functional status of remaining platelets via multiplate analysis. Platelet activation triggered by different pathways is quantified; three common pathways are ADP receptor-dependent (ADP test), collagen-dependent (COL test), and TRAP-6-dependent (TRAP test). [5]

According to American Red Cross, one unit of platelets should raise platelet count on average by 25,000-35,000/mcL. Theoretically, there was room to administer an additional two to four units of platelets while in the OR, regardless of platelet count; this could have significantly improved thrombocytopenic coagulopathy. Though severe metabolic acidosis, and likely hypoxic lactic acidosis due to dysoxia on CPB, was also a significant contributor to the outcome of this case.

Essentially reconstructing the bulk of the RV with nonconductive, noncontractile bovine tissue left this patient heavily dependent on external pacing and cardiac output to provide adequate passive flow. The severely reduced RV function made volume resuscitation nearly impossible and eventually overloaded the heart completely.

Cardiothoracic surgery will always present increased risk of complications and mortality, and cardiac tumors are no exception. However, there is still much to be studied and improved regarding appropriate optimization and replacement of blood products with the hope of decreasing that risk.

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