

Case Report

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Anesthesia Management of Emergent Sternotomy for Acute Cardiac Tamponade in a Pediatric Hematopoietic Stem Cells Recipient

Sengottaian Sivakumar^{1*}, Stacey Watt² and Justin Eckler³¹Clinical Fellow, Department of Pediatric Anesthesiology, SUNY at Buffalo²Program Director, Anesthesia Residency & Pediatric Anesthesia Fellowship, SUNY at Buffalo³Clinical Instructor of Anesthesiology, SUNY at Buffalo**ABSTRACT**

Hematopoietic stem cell transplantation (HSCT) is the infusion of hematopoietic stem and progenitor cells to reconstitute the bone marrow. Due to the long-term consequences of HSCT, anesthetic management of these patients could be complicated. Here the authors describe the clinical features and management of a hematopoietic stem cell recipient with a systemic form of GVHD, who presented with cardiac tamponade due to right ventricular injury, after pericardiocentesis for a severe pericardial effusion, which led to bedside pericardial window and sternotomy with multiple cardiac arrests in the operating room.

***Corresponding author**

Sengottaian Sivakumar MD, Clinical Fellow, Department of Pediatric Anesthesiology, SUNY at Buffalo, Newyork, E-mail:drsenkottaian@live.com

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Case Description

This is a case report of a 3-year-old male with a history of acute lymphoblastic leukemia currently in remission and diagnosed with BCR-ABL positive chronic myeloid leukemia (CML). He had a history of lymphoblastic crisis, and post haploidentical allogeneic stem cell transplantation (SCT), and there was a subsequent graft rejection and relapse of CML with involvement of ocular and central nervous system. This necessitated another SCT from an unrelated matched donor. Tacrolimus, Mycophenolate, Fludarabine, Melphalan and total body irradiation (400Gy) were given for Graft Versus Host Disease (GVHD) prophylaxis starting from day 1 post hematopoietic stem cells infusion. Ursodiol was also started for prophylaxis of veno occlusive disease (VOD) of the liver.

After seven days of second HSCT, the patient presented with maculopapular skin rash over his trunk and lower extremities, diarrhea, and fever due to hyperacute graft versus host disease. The patient was made nil per oral and total parenteral nutrition was started for gut GVHD. Skin findings continued to worsen, and his abdomen became more distended. As the patient's clinical status worsened, intravenous Solumedrol and Rituximab were started for hyperacute GVHD. He continued to receive Tacrolimus continuous infusion daily. Oral Beclomethasone was added to decrease gut inflammation and to prevent GI protein-losing enteropathy. He also received mesenchymal stem cells for his gut GVHD. He developed steroid-induced cushingoid facial features, hypertension and hyperglycemia, necessitating glargine insulin administration and intravenous enalaprilat to which labetalol and

nicardipine infusions were later added. Oral antihypertensives were deferred due to active gut GVHD. Due to hypoalbuminemia, he developed anasarca with moderate ascites, and this necessitated albumin infusions.

After a month post-HSCT, the patient developed painful hepatomegaly, jaundice, ascites, fluid retention, and weight gain. The ultrasound revealed dampened triphasic waveforms without a reversal in hepatic veins confirming VOD. Hydromorphone 0.5mg/mL patient-controlled analgesia was started for his abdominal pain. Due to transplant associated-thrombotic microangiopathy (TA-TMA), eculizumab was started. Due to pancytopenia secondary to marrow suppression by cytotoxic drugs, the patient continued to receive packed red blood cells and platelet transfusions with a transfusion trigger of 7gms for hemoglobin and 30,000 for platelets. Pain medicines were slowly transitioned to methadone.

On his 71st-day post HSCT, the patient developed pulmonary edema due to acute kidney injury and fluid overload manifested as declining saturations despite high flow nasal cannula oxygen and increasing renal parameters. He was intubated, albumin was discontinued, and he was started on fentanyl and dexmedetomidine infusions. Hemodialysis was initiated after securing a femoral venous catheter. He initially tolerated hemodialysis but later developed hypotension and was slowly transitioned to continuous renal replacement therapy (CRRT). He was extubated on the third day with good clinical improvement after fluid removal.

On his 87th-day post SCT, an echocardiogram was performed to rule out cardiac vegetations due to positive blood culture for *Streptococcus mitis*. The findings were negative for pericardial effusion and vegetations. CT of the abdomen was done due to abdominal distension, and it showed colonic pneumatosis.

Stool culture was positive for stenotrophomonas and distension decreased after starting Levaquin.

On patient's 158th-day post SCT, a large PE (Fig. 1) was noted and was closely monitored with serial echocardiograms. After 27 days of diagnosis, the cardiologist decided to tap the effusion due to the patient's clinical symptoms including cough, dyspnea, orthopnea, jugularvenous distention, and pulsus paradoxus. By subcostal approach, 170 ml of fluid was tapped, and a pericardial drain was placed. Immediately post-procedure, the patient received a crystalloid bolus dose of 20mL/kg total to augment preload following decompression of right atrium and ventricle. Approximately three hours following the pericardial drain placement, the surgeon was called to the bedside for hypotension in the setting of escalating vasopressor requirement.

Epinephrine and norepinephrine infusions were started and up titrated, and a stat transthoracic echocardiogram revealed reaccumulation of circumferential pericardial effusion with tamponade physiology. The volume elicited manually from the pericardial drain was 70 ml of frank blood (without resolution of tamponade physiology). The patient lost pulses and cardiopulmonary resuscitation was initiated for pulseless electrical activity. General surgery and cardiology were paged STAT to the bedside to perform a pericardial window. The pericardial space and myocardium explored, and a small laceration or punctate lesion of apex at the right ventricle near posterior descending coronary

artery was noted. Eleven hundred milliliters of blood were lost during the procedure in the PICU, and the patient was transferred to the operating room for further management.

In the operating room, the patient was mechanically ventilated in pressure control mode, and rocuronium was given for neuromuscular paralysis. Suture placement in the right ventricle resulted in ventricular fibrillation and internal paddle defibrillation was performed after removing the sutures. Multiple doses of adrenaline and calcium chloride, as well as a single dose of 20 mEq of sodium bicarbonate, were given during initial resuscitation in the operating room. With ongoing resuscitation, the surgeon placed pledgeted sutures on the right ventricle. 700 ml of packed red blood cells and 300 ml of fresh frozen plasma were given during the surgery, but after transfusion, the patient gradually became bradycardic and hypotensive. At this time i-STAT measurement of arterial blood showed a pH of 6.9 and potassium of 7.1 meq/l (Table 1- column under 11.14 A.M). Two units of plain insulin bolus were given, and 10 % dextrose infusion was started. Another dose of 20 mEq of sodium bicarbonate was repeated with several doses of adrenaline. Return of spontaneous circulation was obtained and the surgeon attached atrial and ventricular pacing leads for a temporary external pacemaker. The sternum was closed, the patient was transferred back to the PICU with epinephrine and norepinephrine infusions, and milrinone infusion was added later to increase the cardiac output.

Table 1: Perioperative blood gas values

	Preoperative values (VBG)	Intra-op (i-STAT values) (ABG)				Postoperative Values (VBG)	Post op Day 1 (VBG)
Time	6.22	10.39	10.53	11.14	11.42	12.46	8.29
PH	7.15	6.92	7.17	7.03	7.10	7.05	7.41
PCo2 _{mmHg}	32	58	38	51	52	56	36
Po2 _{mmHg}	28	58	83	87	73	59	53
BE _{mEq/L}	16.2	-21	-14	-17	-14	-15.6	-1.4
Hco3 _{mmol/L}	22.4	12.2	14.4	14	16.5	12	24
Tco2 _{mmol/L}		14	16	16	18		
Saturation %	33.4	74	95	93	90	75	87.4
Sodium _{mmol/L}	135	143	148	143	144	148	143
Potassium _{mmol/L}	4.8	6.8	5.5	7.6	4.7	3.5	4.1
Calcium _{mmol/L}	6.4	6.1	2.6	7.7	8.4	7.6	8.5
Glucose _{mmol/L}	141	140	152	115	240	160	188
Hemoglobin _{g/dl}	<6.5	11.6	7.8	12.2	17.2	16.6	13.7
Hematocrit	16	34	23	36	36	50	41
Lactate _{mmol/L}	3.8					11.5	2.3

(VBG- Venous blood gas, ABG- Arterial blood gas)

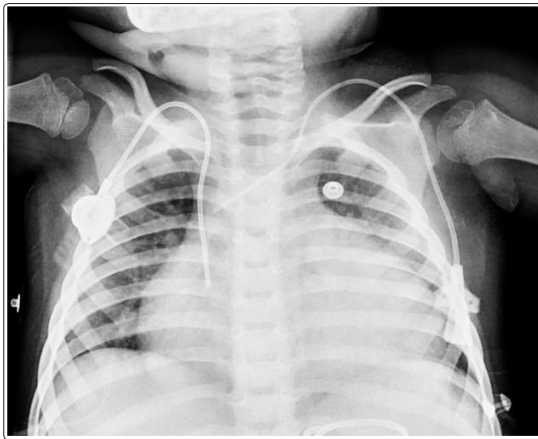


Figure 1: Pre-operative chest X-ray demonstrating severe pericardial effusion

Bedside EEG monitoring with time-locked bedside video monitoring for the first three postoperative days showed no seizure activities. Postoperative echocardiography showed moderate systolic dysfunction and later improved. His immediate postoperative chest X-ray showed a reduced size of the cardiac silhouette (Fig 3). Ventricular and atrial leads were removed on the 16th POD. CRRT was continued till 10th POD and the femoral venous catheter was removed after improvement in renal function. Extubation was delayed till 18th POD due to significant lung infection due to his profound immunosuppressed state. He initially had slow responses to verbal commands but improved after several days.

Discussion

SCT patients have a complicated medical history, with a possible variety of medications (steroids, antibiotics and sedatives), and may have multiple organ system dysfunctions. Thus, a methodical review of the history and a carefully directed physical examination will allow for the planning and delivery of safe anesthesia.

Tyrosine kinase inhibitors like Dasatinib and Imatinib are increasingly used in CML and can cause serosal inflammation, including pleural and pericardial effusions due to its antifibrotic activities. The pathogenesis may involve inhibition of platelet derived growth factor or expansion of cytotoxic T and natural killer cells [1]. Our patient also suffered from inflammation of other serosal surfaces like paranasal sinuses and ascites. Furthermore, fluid overload related to AKI secondary to nephrotoxic anticancer medications and imbalanced Starling forces related to hypoalbuminemia secondary to gut GVHD, contributed to severe pericardial effusion.

Preoperative period of this patient is complicated by the following:

- Polymicrobial infections due to profound immunosuppressive state,
- GVHD of skin and gut,
- Microvascular hemolysis due to transplant-associated thrombotic microangiopathy (TA-TMA),
- Liver dysfunction due to venoocclusive disease also known as sinusoidal obstruction syndrome,
- Refractory hypertension secondary to fluid overload due to acute kidney injury probably due to nephrotoxic medications and
- TA-TMA induced acute kidney damages and gross abdominal distension due to hepatomegaly, colonic pneumatosis and ascites.

In most cases, draining the pericardial fluid results in complete, asymptomatic recovery. The pericardial space normally contains 10-50 mL of fluid. A pericardial effusion (PE) is said to exist when the intrapericardial volume exceeds these limits and can be classified according to size as small (< 10 mm), moderate (10-20 mm), or severe (> 20 mm), according to onset as acute (<1 week), subacute (1 week to 3 months) and chronic (>3 months) and according to location as circumferential and loculated. Management is mainly guided by the hemodynamics of the patient [2]. So, in this case report, the effusion can be classified as severe (28mm), subacute and circumferential (Fig. 1). The pressure-volume curve of the normal pericardium is a J-shaped curve (Fig.2). The compliance of the parietal pericardium increases slowly, although it can eventually accommodate large volumes without any hemodynamic compromise only when accumulation occurs over weeks to months. However, in the setting of ventricular perforation due to percutaneous intervention, fluid accumulation is abrupt and as little as 100 cc may result in hemodynamic decompensation [3,4]. In this case bleeding from right ventricular injury caused rapid decline in hemodynamic status as evidenced by preprocedural echocardiogram with diastolic collapse of right atrium and right ventricle.

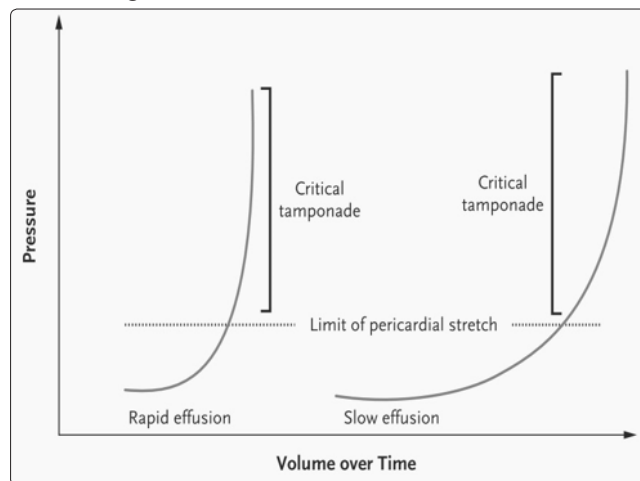


Figure 2: Compliance curve of the pericardium



Figure 3: Post-operative chest x-ray

The Subxiphoid pericardial window with pericardiostomy tube serves to drain the pericardium and relieve any potential tamponade. If bleeding ceases and the patient remains stable, sternotomy may be avoided. The whole procedure can be performed under local anesthesia with adequate sedation.

Review of several case reports indicate that few cases were managed with only local infiltration without any complications and some cases were managed with combination of local anesthesia and general anesthesia with endotracheal intubation [5–10]. Usually local anesthetic infiltration was provided to infiltrate the skin, subcutaneous tissues, rectus abdominis muscle and perichondrium about the xiphisternal junction to create an incision into the pericardium, thereby relieving the tamponade. Then general anesthesia with positive pressure ventilation is provided if needed, to repair ventricular injuries or bleeding from epicardial vessels.

Webster et al reported anesthesia for mini sternotomy in a pregnant patient who presented with a symptomatic anterior mediastinal mass due to Hodgkin's lymphoma and then developed acute cardiac tamponade due to chemotherapy induced pericardial effusion requiring surgical drainage [6]. This was successfully managed with awake fiberoptic intubation due to airway compression and incremental doses of ketamine and sevoflurane in minimal concentrations. In our case, local infiltration was used to drain the pericardial fluid collections. Secondary to unstable hemodynamics, our patient was intubated prior to draining the fluid for the first time, but spontaneous ventilation was maintained until the sternum was opened for further repair of the right ventricular laceration. Onal et al described cardiac tamponade after placing an umbilical venous catheter in a term infant, drainage of which was managed successfully with local anesthesia alone [7]. Jiha et al reported a case of intraoperative cardiac crystalloid tamponade after placing a subclavian central venous catheter for non-cardiac surgery, in which tamponade was diagnosed by noticing low voltage QRS complexes and electrical alternans in EKG in the middle of surgery during resuscitation for severe hypotension [8].

To summarize, patients requiring a pericardial window are susceptible to hypotension and cardiac arrest due to the induction of general anesthesia. Intravenous and inhalation anesthetics promote peripheral vasodilation and direct myocardial depression. In addition, positive-pressure ventilation will increase intrathoracic pressure thereby decreasing venous return. Both interventions lead to further impairment in cardiac output. Intubation and mechanical ventilation should be avoided unless strictly necessary, as this will tend to exacerbate cardiac failure in tamponade. Ventilated patients developing tamponade in an intensive care environment should have positive end expiratory pressure minimized to avoid limiting venous return. Invasive hemodynamic monitoring should be instigated, allowing continuous blood pressure and central venous pressure measurements to be made. Mechanical ventilation may increase pulmonary vascular resistance and decrease RV outflow, further exacerbating leftward septal shift, impairing LV filling, and potentially worsening systemic hypotension. Because of this, some clinicians suggest maintaining spontaneous ventilation. However, merely avoiding large tidal volumes and high peak airway pressures will likely minimize the impact of ventilation on systemic hemodynamics and allow for general anesthesia and adequate surgical conditions.

Ideally, unstable patients with acute cardiac tamponade are treated with percutaneous pericardiocentesis under local anesthesia. If

surgical drainage under general anesthesia is required, an intra-arterial catheter should be placed prior to induction so that acute changes in hemodynamics can be detected and corrected. For severe cardiac tamponade, the patient should undergo surgical preparation and draping with the surgeon scrubbed at the operating room table ready for incision prior to induction. The physiologic goals are to maintain tachycardia, optimize preload to maximize left ventricular filling, and maintain an elevated systemic vascular resistance. Vasodilation and hypotension should be avoided. These three parameters work together to ensure adequate cardiac output [4,11]. Medications used during general anesthesia should be consistent with these goals.

Catecholamine infusions (e.g., norepinephrine, dopamine, epinephrine) should commence prior to induction. Small boluses of epinephrine may be helpful for temporary inotropy and chronotropy. Ketamine is the preferred intravenous anesthetic due to its promotion of sympathetic output. Suitable options for maintenance include midazolam, fentanyl, nitrous oxide, and pancuronium (which also induces tachycardia). Positive pressure ventilation may be delayed until drainage of the pericardial space is imminent. Once the pericardium is drained, venous return can enter the heart and hemodynamics will rapidly normalize or swing to hypertension [4,11].

Conclusion

Meticulous surgical technique, knowledge of the possible complications, and close monitoring of the patient in the perioperative period are required in the management of acute pericardial tamponade. Our review indicates that knowledge of idiosyncrasies associated with HCT, when accompanied by careful and direct evaluation of each patient, could assist with provision of safe anesthesia.

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