

## **PBLD # 6**

### **Cardiac arrest in a child with aortic stenosis undergoing orthotopic liver transplantation.**

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#### **Objectives:**

- Understanding the role of aortic stenosis in cardiac ischemia and treatment options during a hemodynamically challenging case
- Identifying the role of the anesthesiologist during cardiac arrests and how to run a code during surgery
- Discuss the physiologic changes during the reperfusion phase of a liver transplant

An 8 y/o, 18 kg boy with a past medical history of familial hyperlipidemia and mild-to-moderate aortic stenosis (AS), is scheduled for orthotopic liver transplantation.

Past medical history:

The patient was diagnosed with familial hypercholesterolemia at age six and was referred for the liver transplantation after failing statin therapy. As part of his pre-transplant workup he underwent a cardiac catheterization which demonstrated a 70% narrowing of the left main and left anterior descending artery coronary arteries, severe stenosis of the right coronary artery and mild supravalvular aortic stenosis. Coronary artery bypass grafting was performed.

Six months after the CABG surgery, he presented for orthotopic liver transplantation .

Intravenous induction was uneventful. At the end of the preanhepatic stage of surgery (before clamping the IVC), the systolic blood pressure was in the low 70s and ST segment changes (depression) were noticed on EKG. Symptoms

improved after administration of a fluid bolus and phenylephrine. TEE was performed by a cardiologist, which showed moderate-to-severe AS (peak velocity 4.0-4.5 m/s, peak gradient 64-80 mm /Hg) with no regional wall motion abnormalities.

Phenylephrine and nitroglycerine drips were started, resulting in an elevation of BP and an improvement in EKG changes. A PRBC transfusion was also initiated to increase oxygen carrying capacity.

During the beginning of the anhepatic stage of surgery (after clamping the IVC and removing the liver) the patient developed ventricular tachycardia (VT) and blood pressure was not detected by the arterial line.

The emergency staff assist was called, the surgeon was asked to start the chest compressions and the anesthesiologist initiated the PALS algorithm.

Spontaneous circulation resumed after resuscitation involving chest compressions, two electrical shocks and two rounds of epinephrine. The team including the anesthesiologist, surgeon and cardiologist discussed the possible hemodynamic changes during the organ reperfusion and the decision was made to initiate ECMO by the in-house cardiac surgeon. Reperfusion of the transplanted liver was uneventful and child was weaned off ECMO at the conclusion of surgery

- **What is familial hyperlipidemia?**

Familial hypercholesterolemia is a genetic disorder. It is caused by a defect on chromosome 19.

The defect makes the body unable to remove low density lipoprotein (LDL) cholesterol from the blood. This results in high levels of LDL in the blood.

Atherosclerotic changes can be seen at an early age. The condition is typically

passed down through families in an autosomal dominant manner. In rare cases, a child may inherit the gene from both parents. When this occurs, the increase in cholesterol levels is much more severe. The risk for heart attacks and heart disease are high even in childhood.

Symptoms that may occur include:

- Fatty skin deposits called xanthomas over parts of the hands, elbows, knees, ankles and around the cornea of the eye
- Cholesterol deposits in the eyelids (xanthelasmas)
- Chest pain or other signs of coronary artery disease; these may be present at a young age
- Cramping of one or both calves when walking
- Sudden stroke-like symptoms such as trouble speaking, drooping on one side of the face, weakness of an arm or leg, and loss of balance

- **What tests would you prefer to have prior to liver transplantation? How do the results affect your preparation for the case?**

Since the child is in high risk for the atherosclerosis, coronary artery disease and aortic valve stenosis, baseline ECG, ECHO and cardiac catheterization should be obtained.

The echocardiogram showed mild supra-ventricular aortic stenosis with a peak velocity of 3.1 m/sec, normal systolic function, mild septal dyskinesia, with paradoxical diastolic motion and LV EF-52%.

- **What monitors would you use and what lines would you place? What medications would you have ready? Room set up.**
  - Infusion pumps, drips : epinephrine, dopamine, rocuronium, fentanyl
  - Fluid warmers#2, level one or rapid infusing system

- TEE
- Transducers#2, for the arterial line and CVP
- Central line kit, arterial line set
- Blood products
- Albumin
- Forced air warmers#2
- Drugs: calcium chloride, sodium bicarbonate, insulin, epinephrine, atropine
- Defibrillator pads

- **What is the difference between conventional versus Piggy-back techniques? Would you discuss it with the surgeon?**

With the conventional technique the two IVC and portal vein anastomoses is performed before the unclamping.

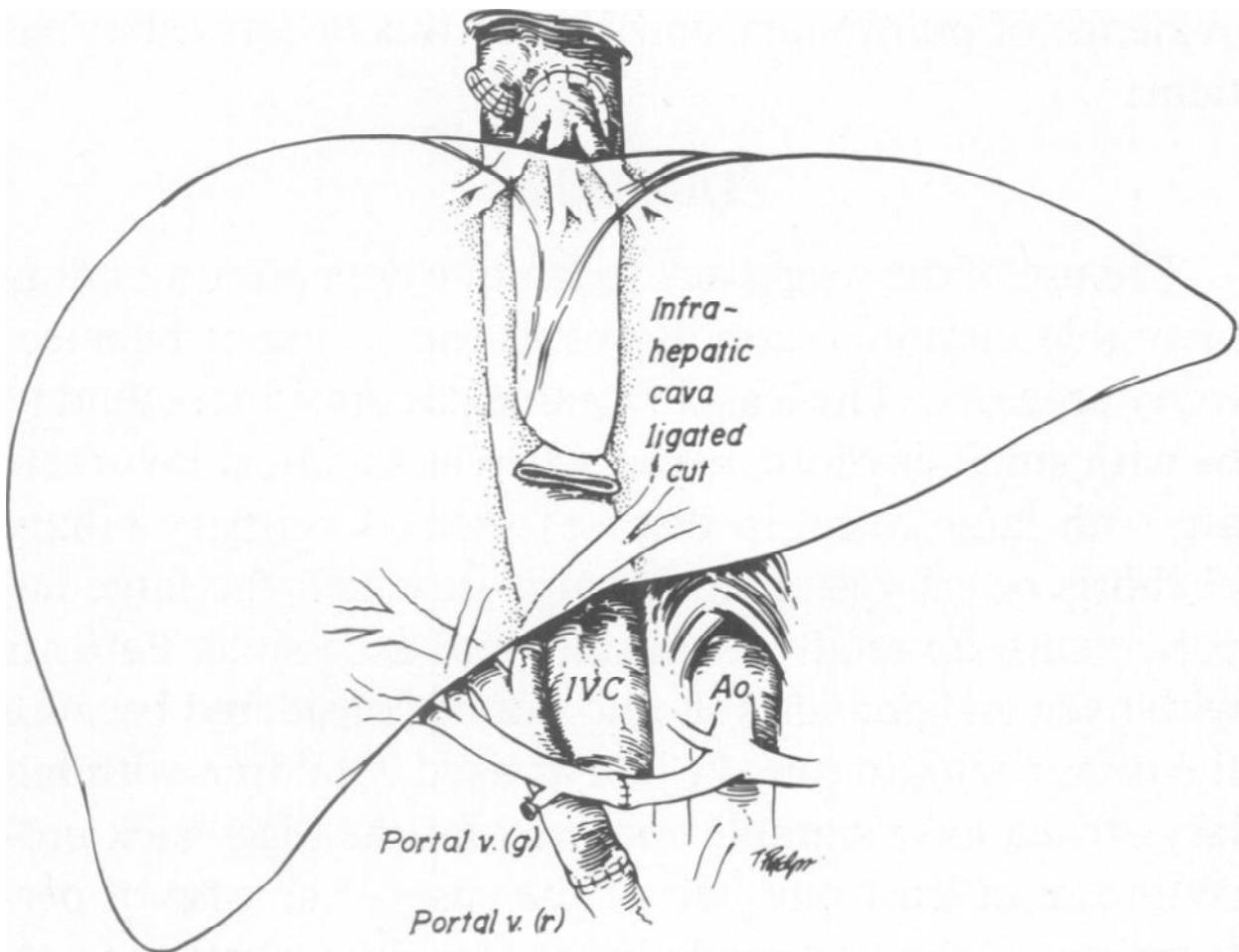
Piggy-back technique:

The cava anastomosis is performed between the suprahepatic part of donor IVC and common orifice of all three hepatic veins

1. Continuous venous decompression during the anhepatic phase is provided without venovenous bypass.
2. Venous return to the heart and renal venous outflow is maintained.
  - Mean arterial pressure, CO, SVR, CI show minimal changes
  - Acute renal failure is less frequent
3. Warm ischemia time can be shortened because there is no need for the infrahepatic vena cava anastomosis.
4. Reduction of red blood cell transfusion
5. Reduction of vasoactive drug use
6. Improvement in graft survival

Disadvantages:

1. Venous outflow obstruction
- 2 Thrombosis (venous)



**Intravenous induction was uneventful. At the end of the preanhepatic stage of surgery (before clamping the IVC), the systolic blood pressure was in the low 70s mmHg and ST segment depression was noticed on EKG. Symptoms improved after administration of a fluid bolus and phenylephrine boluses. TEE was performed by a cardiologist, which showed moderate-to-severe AS (peak velocity 4.0-4.5 m/s, peak gradient 64-80 mm /Hg) with no regional wall motion abnormalities.**

- **What are anesthesia considerations in a patient with aortic stenosis?**

Goals during management of anesthesia in patients with aortic stenosis are maintenance of normal sinus rhythm and avoidance of extreme and prolonged alterations in heart rate, systemic vascular resistance, and intravascular fluid volume. Preservation of normal sinus rhythm is critical because the left ventricle is dependent on properly timed atrial kick to ensure optimal left ventricular filling and stroke volume. Marked increases in heart rate (higher than 100 beats/min) decrease the time for left ventricular filling and ejection, and decrease coronary blood flow while increasing myocardial oxygen consumption. Coronary blood flow to the left ventricle occurs during diastole, and changes in heart rate primarily affect diastolic time. Bradycardia (lower than 50 beats/min) can lead to acute overdistention of the left ventricle. Tachycardia may lead to myocardial ischemia and ventricular dysfunction. Although increased SVR may cause obstruction to left ventricular ejection, decreases in systemic vascular resistance may be associated with large

decreases in systemic blood pressure and coronary blood flow and myocardial ischemia. Intra-arterial pressure monitoring is essential and can speed identification and treatment of hemodynamic changes. Prophylactic infusions of vasoconstrictors, such as phenylephrine, may reduce hemodynamic changes. A prophylactic intravenous infusion of phenylephrine can be started prior to induction to reduce hemodynamic changes. A cardiac defibrillator should be promptly available when anesthesia is administered to patients with aortic stenosis since external cardiac compressions are unlikely to generate an adequate stroke volume across a stenosed aortic valve.

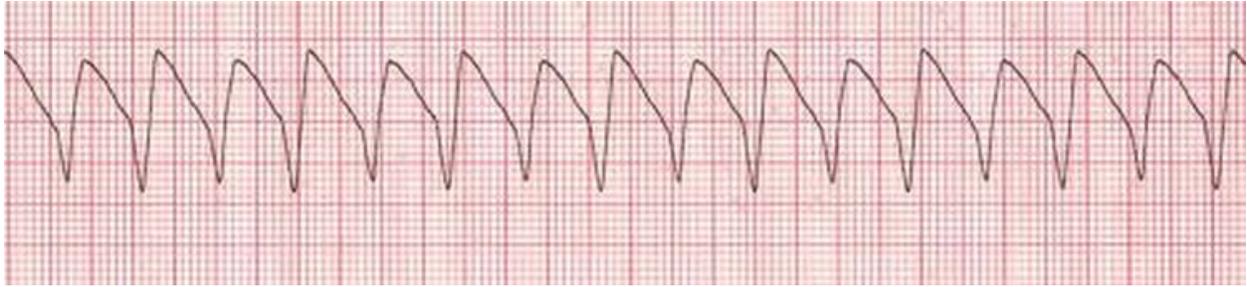
- **What is your treatment plan?**

**Phenylephrine and nitroglycerine drips were started, resulting in an elevation of BP and an improvement in EKG changes. A PRBC transfusion was also initiated to increase oxygen carrying capacity.**

**During the beginning of the anhepatic stage of surgery (after clamping the IVC and removing the liver) the patient developed ventricular tachycardia (VT) and the blood pressure was undetectable by the arterial line.**

PALS protocol for VT

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- - Initiate CPR (Push hard, push fast)
  - Defibrillation - 2J/KG
  - Re-initiate CPR (Push hard, push fast)
  - Increase joule setting to 4J/Kg, then repeat defibrillation if still unsuccessful
  - Re-initiate CPR (Push hard, push fast)
  - Check rhythm. If still in V-Fib, administer medication.
  - **Epinephrine** (1:10,000) 0.01 mg/kg IV, may repeat every 3-5 minutes
  - Repeat defibrillation if still unsuccessful
  - Re-initiate CPR (Push hard, push fast)
  - Check rhythm. If still in V-Fib, administer medication
  - **Amiodarone** 5 mg/kg IV bolus
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- **Running a code during surgery**
  - **The role of the anesthesia provider**
  - **Assigning roles to the OR team**

## **ACRM (Anesthesia Crisis Resource Management)**

The primary goal of crisis management is to detect and correct an evolving problem to prevent an adverse outcome. Management of dynamic situations depends on the ability of the anesthetist to respond to many sources of rapidly changing information.

### **Seven Key Points**

- Call for Help early enough to make a difference.
- Take an appropriate leadership role.
- Communicate effectively.
- Distribute the workload.
- Anticipate and plan
- Know your work environment.
- Utilize all available resources.

### **Take command as team leader**

- Make sure the team understands who is in charge.
- The team leader decides what needs to be done, prioritizes the necessary tasks, and assigns them to specific individuals.

### **Communicate effectively**

- Communications between team members is crucial in a crisis.
- State your commands or requests clearly (closed loop communication).

### **Distribute the workload**

- Assign specific tasks to individuals according to their skill level.
- The team leader should only become involved in manual tasks if specific expertise is necessary to ensure correct and timely completion.

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### **Use all available resources**

Self

OR/ICU personnel

Equipment

Cognitive aids

External resources

Plans/algorithms

**After successful resuscitation involving chest compressions, two shocks and two rounds of Epinephrine, the child resumed spontaneous circulation and was put on ECMO by the in-house cardiac surgeon.**

- **Reperfusion Syndrome**

The post-reperfusion syndrome continues to be an important intraoperative risk factor for impaired graft function, morbidity and mortality

Could be defined as persistent severe hypotension (>30% decrease in MAP), arrhythmias, bradycardia, occasionally cardiac arrest, and fibrinolysis. It also can include a decrease in SVR in the face of acutely increased right ventricular filling pressures.

Hyperkalemia can be precipitated after liver reperfusion reperfusion.

Acidosis

Hypothermia

Air and microthrombi embolism

Blood loss

Risk factors for postreperfusion syndrome

- Volume status (decreased) of the recipient before reperfusion
- Myocardial depression caused by the flushing of the residual preservation solution into the circulation
- Release of vasoactive pro-inflammatory factors from the Kupffer cells of the postischemic liver

Preparation for reperfusion

- flushing the liver with blood before the reperfusion
- Optimization of volume status
- Restoration of normal pH, potassium level, ionized calcium
- Normothermia
- 100% O<sub>2</sub>

- Administration of vasoactive meds
- Blood products

- **Identify advantages of ECMO or Veno-venous bypass**
  - Reduces hemodynamic instability during anhepatic phase
  - Maintain adequate venous return and cardiac filling pressures
  - Benefit patients with portal hypertension, cardiomyopathy
  - Has been shown to maintain intraoperative renal function
  - Maintains CPP
  - Reduce blood loss

### **Discussion:**

Liver transplantation has become very successful in treating children with end-stage liver disease. The utilization of split-grafts, living related donors provides more organs for the pediatric patients. More than 500 transplantations perform each year. Pediatric patients account for about 12.5% of liver transplant recipients.

About 50% of the pediatric patients who require a liver transplant have a diagnosis biliary atresia. The next common group of patients is children with intrahepatic cholestasis: sclerosing cholangitis, Alagille's syndrome, progressive familial intrahepatic cholestasis.

Metabolic diseases are also another indication for liver transplantation.

Examples of metabolic diseases include Wilson disease, alpha 1-antitrypsin deficiency, tyrosinemia, and hemochromatosis, Crigler- Najjar syndrome, oxalaturea, familial hyperlipidemia, methylmalonyl aciduria.

Other indications include patients with acute fulminant hepatic failure, cystic fibrosis, liver tumor (hepatoblastoma).

The liver transplant operation is divided into three stages: dissection or pre-anhepatic, anhepatic and neohepatic. Every stage represents a challenge for the anesthesiologist. In this case, the patient had a severe aortic stenosis, a

condition that required tight control of preload, heart rate and cardiac output, making the case even more complicated.

During the first stage the anesthesiologist should replace the blood loss, normalize the coagulation abnormalities, maintain metabolic control, normothermia. If the patient presents with ascites, ventilation can improve after opening the abdomen. In portal hypertension or adhesions are present, the blood loss can be significant. During this stage, adequate cardiac and urine output should be maintained with administration of crystalloids, colloids or blood products. Vasoactive medications should be started if necessary. The potassium level should be checked and kept below 4 mEq/L.

During the anhepatic stage, clamping the IVC results in the loss of venous return from the lower body, and can cause decrease in CVP and CO. This can be tolerated relatively well because of the compensatory increase in SVR and HR. Patients with portal hypertension can tolerate clamping of the IVC secondary to the presence of porto-systemic collaterals. This patient did not have portal hypertension and the hemodynamic changes during this stage precipitated the decompensation and arrhythmia.

Communication with the surgical team should be maintained in order to ensure all the necessary preparations are made prior to reperfusion. Labs should be checked before hand. The anesthesiologist has to be ready for massive transfusion, electrolyte abnormalities, dysrhythmias and acidosis after reperfusion

The neo-hepatic/ reperfusion stage begins with the unclamping of the portal vein, IVC and reperfusion of the organ. Reperfusion syndrome is diagnosed by the presence of bradycardia, hypotension or heart conduction disturbances and should be treated aggressively. Small boluses of epinephrine can be necessary to improve contractility and prevent graft overload. Hyperkalemia is another concern during reperfusion: hyperventilation, sodium bicarbonate, calcium chloride , insulin/glucose, albuterol, furosemide, washed PRBC's can help in prevention or treatment. Air and microembolism can occur as well.

Once blood flow through the graft is established and bleeding is controlled, the bile duct reconstruction begins.

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