

MASSIVE UPPER GI HAEMORRHAGE ENDING IN TRANSHIATAL OESOPHAGECTOMY WITH GASTRIC PULL THROUGH FOLLOWING CONDUIT REPAIR OF PULMONARY ATRESIA: A CASE REPORT

Dr. Ranjith Karthekeyan B¹, Dr. Karthikeyan N S², Dr. Rakesh M G²,
Dr. Suresh Rao K G³, Dr. Mahesh Vakamudi³, Dr. Balakrishnan K R⁴

1. Assistant Professor, 2. Postgraduates, 3. Professor

Department of Anaesthesiology

4. Professor and Head, Department of Cardiothoracic and Vascular
Surgery

Sri Ramachandra Medical College and Research Institute, Porur,
Chennai.

Correspondence: Dr. Ranjith Karthekeyan B (ranjithb73@gmail.com)

About the Author: The author graduated from Kilpauk Medical College, Tamilnadu & did postgraduate training in anaesthesia and cardiac anaesthesia fellowship from Sri Ramachandra Medical College & Research Institute. For the past five and half years the author is a Assistant Professor in cardiac anaesthesia in Sri Ramachandra Medical College & Research Institute. Special interest include Redo cardiac surgery, TEE, Aneurysm surgery and Paediatric cardiac anaesthesia



Abstract

A five year old female with complex congenital heart disease developed gastrointestinal hemorrhage following VSD closure, RV-PA conduit, ligation of aneurysmal right MAPCA and clipping of modified pott-shunt. All conservative measures from cold saline lavage to left gastric artery embolisation were tried but none was successful. Gastroscopy was done in intensive care unit and intra-operatively during the procedure both of which were inconclusive of the origin of the bleeding. Transhiatal oesophagectomy with gastric pull through and feeding jejunostomy had to be done as a desperate measure to control the bleeding. Doing such an extensive procedure for bleeding which could not be localized, ultimately saved the life of the patient. Mallory Weiss syndrome, Boerhaave syndrome, porto-systemic collaterals and aorta pulmonary collaterals eroding into posterior oesophagus were some of the possibilities considered but nothing could be proved.

Keywords: CHD, Cyanotic; Oesophagoscopy; Haemodynamics; Hypoxia; Surgery, emergency

Stress lesions of the upper gastro intestinal tract are well recognized complications after cardiac surgery in adults. Although there are no controlled studies about these lesions in paediatric cardiac surgical patients, the incidence is similar to high risk adult patients¹. Here we present a case of oesophageal haemorrhage following cardiac surgery for a complex congenital lesion in a five year old child.

The child was referred to us at the age of three years with a history of delayed developmental milestones, poor weight gain, and recurrent respiratory tract infections and increased sweating during feeding. She was a term baby, delivered normally with a birth weight of 2.6 kilograms. Clinically she was diagnosed to have complex congenital heart disease with increased pulmonary blood flow with congenital talipes equines varus. The echocardiogram revealed pulmonary atresia, sub aortic ventricular septal defect (VSD), large hypertensive multiple aorto-pulmonary communications (MAPCA'S), severe hypoplastic right pulmonary artery (RPA) and dilated left pulmonary artery (LPA). Angiogram showed two large MAPCA'S, pulmonary atresia and sub-aortic VSD. Coil embolization of MAPCA'S was tried, which was unsuccessful due to the large size. So modified pott shunt (descending aorta to LPA) and ligation of left MAPCA'S was done to improve the oxygenation and the child gained weight. Angiogram done one year later showed pulmonary atresia, ligated left MAPCA, aneurysmal right MAPCA and a well functioning modified pott shunt.

The child was planned for RV-PA conduit, ligation of aneurysmal right MAPCA and clipping of left modified Pott's shunt at the age of 5 years. Patient was receiving aspirin after modified pott shunt which was stopped one week before the planned procedure. Chest X-ray showed cardiomegaly with increased bronchovascular markings. Pre-operative echocardiogram showed mildly dilated left ventricle, large sub-aortic VSD, atresic pulmonary valve, closed ductus, hypoplastic pulmonary trunk and a functioning modified pott shunt with good flow. Intraoperative period was uneventful. Intubation of the trachea was smooth. The duration of the surgery was 180 minutes. Cardiopulmonary bypass was for 85 minutes and cross clamp was for 30 minutes. There no significant blood loss intraoperatively. Intraoperative transesophageal echocardiogram probe was not used. Activated clotting time post bypass was 126 seconds. Patient received glyceryl trinitrate 0.5 mcg/kg/min and dobutamine 10 mcg/kg/min.

Two hours post operatively, brown color aspirate was present in the orogastric tube once, which was treated with cold saline lavage, ranitidine 25mg intravenous twice daily and sucralfate 5ml eight hourly through orogastric tube. Patient was electively ventilated as the epicardial echocardiogram showed moderate RV dysfunction. Milrinone

0.375 mcg/kg/minute was added to the dobutamine and patient was atrially paced at the rate of 160/minute. All bleeding parameters and platelet count were within the normal limits. As the right ventricular function improved patient was on minimal ventilatory support.

On the 9th post operative day, patient vomited 200ml of fresh blood which was treated with hetastarch and packed red blood cells. Medical gastroenterologist's opinion was sought and a diagnosis of stress induced mucosal damage was made. Patient was put on intravenous omeprazole and sucralfate through orogastric tube. Gastroscopy revealed few erosions and a large clot in fundus. The next day patient had a massive haematemesis of about 400 ml and haemoglobin dropped to 4.7 gm%. The child was resuscitated with fresh blood, packed red blood cells and fresh frozen plasma. Since the bleeder was not localized by gastroscopy, interventional angiogram was done. Angiogram was negative and no bleeder was found. However considering the age of the child and massive haematemesis causing haemodynamic instability, empirical left gastric artery embolisation was done with poly vinyl alcoholoid particles and gel foam. Post angio there was no flow in the left gastric artery with a normal flow in hepatic artery.

Next day haematemesis and melena persisted. Surgical gastroenterologist started somatostatin 1 mcg/kg/min and gastric lavage with adrenaline. Patient again had a massive haematemesis of 400-500 ml. Patient was resuscitated with boluses of adrenaline, fresh blood and fresh frozen plasma. Emergency laparotomy was done to find out the bleeder. Patient was shifted to operation theatre with blood pressure of 60/40 mm Hg, central venous pressure of 3 mm Hg and heart rate of 160/ min. Even after opening the stomach, cause of bleeding could not be ascertained. Intra operatively retrograde gastroscopy was done which showed oesophagus and fundus filled with blood and it was inconclusive about the bleeder. However blood was seen coming from the lower gastro oesophageal junction. The surgeon went above into the oesophagus and could not locate the bleeder which was becoming uncontrollable. Desperate were the situation, transhiatal oesophagectomy with a gastric pull through and a feeding jejunostomy was done.

Patient had unstable haemodynamics throughout the procedure which was treated with fresh blood, packed red blood cells, crystalloids and boluses of adrenaline. Later adrenaline 0.1 mcg/kg/min infusion was started. The total duration of the surgery was about 5 hours. Dobutamine 10 mcg/kg/min, milrinone 0.375 mcg/kg/min and adrenaline 0.1mcg/kg/min were on flow. Pathology report which was collected a day after showed a diagnosis of reflux oesophagitis. Jejunostomy feeding was started on the second day. Iotropes were

tapered and patient was weaned off the ventilator. Two units of platelets were transfused as the platelet count was low. Since patient's respiratory mechanics were inadequate aminophylline infusion was started. However, patient's respiratory mechanics improved over a period of time and we were able to taper aminophylline gradually. Finally patient was extubated on 18th post operative day.

Sternal dehiscence developed for which sternal rewiring was done under general anaesthesia. Procedure took about 45 minutes which was uneventful. Patient was extubated next day morning. Finally after 30 days of intensive unit care, patient was shifted to the ward. The patient was eventually discharged from hospital at the end of 40 days.

Discussion

The incidence of stress lesions of the upper gastrointestinal tract in children after cardiac surgery resembles that in high risk adult patients¹. The major factor implicated in the pathogenesis of gastrointestinal lesions post cardiac surgery is reduced systemic blood flow, which leads to inappropriate oxygen delivery and energy deficit. The gastrointestinal tract in children does not have the ability to autoregulate to compensate for the reductions in blood pressure. Moreover due to persistent vasoconstriction, splanchnic hypoperfusion may continue even after the haemodynamic instability has been regained¹. Behren R et al found that although the incidence of GI inflammation and ulceration was more or less similar regardless of the prophylaxis, the incidence of severe inflammation or ulceration of the upper GI tract was significantly less (18% Vs 44%) in children given prophylaxis. The total incidence resembled that of high risk adult patients. We had started the child on ranitidine prophylaxis on the zero post operative day. The other group of drugs like sucralfate and pantoprazole were added later.

There is a positive correlation between the incidence of the upper GI tract lesions and the severity of the underlying disease¹. Severe change occurs in only 17% of children with an uncomplicated post operative course whereas 50% of the children in whom recovery took longer had pronounced inflammation or ulceration. Gastrointestinal haemorrhage in infants and children is a catastrophic event but is not associated with significant mortality except those with a severe primary illness². This child had a complex cyanotic congenital heart disease resulting in multiple procedures and stressful post operative period. The high risk group were children with unstable haemodynamic circulation who required high doses of intravenous catecholamines, children after cardiac arrest and resuscitation, repeat thorotomies and children with renal failure requiring dialysis. Our patient had all the criteria for high risk group except renal failure. The

mean interval between surgery and upper gastrointestinal haemorrhage was 9.6 days (1-30). Early intervention would be life saving in patients who are too ill to compensate for the haemodynamic disturbances which follow³. Even though torrential haemorrhage occurred only on the 9th post operative day in this case, altered blood was aspirated from the orogastric tube as early as the zero post operative day. As the patient was mechanically ventilated and under sedation, the usual signs and symptoms were not obvious thereby misleading us about the gravity of the situation. Aggressive management at that stage would have prevented the catastrophic events.

The importance of early recognition of GI complication with a low threshold for proceeding to exploratory laparotomy has been stressed⁴. The heart surgery would have improved the cardiac status in these patients, so that they would be able to withstand the general anaesthesia and abdominal surgical interventions. Several of the independent risk factors contributing to gastrointestinal complications were related to splanchnic hypoperfusion. This may be due to vasoconstriction, which often correlates with low cardiac output. It will be aggravated by the use of vasopressors during extracorporeal circulation and in the post operative period⁴. During reperfusion oxygen combines with hypoxanthine in a reaction catalysed by xanthine oxidase to form superoxide radicals. These superoxide radicals exert direct toxic effects on mucosal cells and should be usually scavenged by superoxide dismutase. Allopurinol, a xanthine oxidase inhibitor which is very helpful in adults may have a role to play in these paediatric set of patients though randomized controlled studies not available. The pathogenesis of early bleeds were related to acute haemodynamic disturbances due to hypovolemia or low cardiac output, whereas late onset bleeds were mostly related to focal infections⁵. Hypovolemia, prolonged CPB time and mechanical ventilation have been implicated in the pathogenesis of gastrointestinal complications after cardiac surgery⁶. In our case extensive haemorrhage made the child hypovolemic and right ventricular dysfunction made us to ventilate for a longer period of time. CPB time was 85 minutes which is slightly longer due to the complexity of the procedure. Upper gastroscopy determines the location and nature of the bleeding lesion, but also provides prognostic information regarding the risk of further bleeding. Moreover, therapeutic interventions can be performed during the initial endoscopy. It is seen that early endoscopy is not only safe and effective, but also decreases the transfusion requirement and the length of hospital stay⁷. Although the gastroenterologist could not visualize the location of the bleeder because of un-cleared blood or clots, it is not infrequent⁸. There is

approximately 15-20% chance of re-bleed with in the next 72 hours after the first episode. The main clinical and endoscopic factors associated with failure of endoscopic therapy include (1) active, spurting haemorrhage, (2) ulcer size >2 cms, (3) shock, (4) low Hb level and (5) ulcer location on lesser curvature of stomach or the posterior duodenal bulb⁷ all of which was possible in our case except the last factor.

Interventional radiology provides a non-operative modality to control bleeding when therapeutic endoscopy is unsuccessful, thereby avoiding the high morbidity and mortality associated with emergency surgery. Empirical left gastric artery embolisation was done as the angiogram was negative and the radiologist had no other go than take this drastic measure in view of the condition of the child. Arterial embolisation is generally safe in the upper GI tract because of its rich collateral supply⁷. Y. Eroglu et al evaluated outcome of the octreotide, a somatostatin analogue in children with GI bleeding with or without portal hypertension and found that it has a less clear role in GI bleeding unrelated to portal hypertension⁸. The cause of haemorrhage in our patient was unlikely portal hypertension. This might explain the inability of octreotide to stop the torrential bleeding. No matter how hopeless the situation looks persistent and patient efforts by the treating physician's team had resulted in successful management and eventual discharge of this child from the hospital. Prophylaxis against stress ulcers with appropriate drug therapy and timely detection and treatment should bring down the incidents of these kinds of catastrophic events. Doing such an extensive procedure for bleeding which could not be localized ultimately save the life of the patient. Mallory Weiss syndrome, Boerhaave syndrome, porto-systemic collaterals, preoperative undiagnosed oesophageal pathology and aorta pulmonary collaterals eroding into posterior oesophagus were some of the possibilities considered but nothing could be proved.

References

1. Behrens R: Frequency of stress lesions of the upper gastrointestinal tract in paediatric patients after cardiac surgery: effects of prophylaxis. *Br Heart J*; 72(2):186-9, 1994.
2. Hillemeier C, Gryboski JD: Gastrointestinal bleeding in the paediatric patient. *Yale J Biol Med*; 57(2):135-47, 1984.
3. Jayaprakash A, McGrath C: Upper gastrointestinal hemorrhage following cardiac surgery: a comparative study with vascular surgery patients from a single centre. *Eur J Gastroenterol Hepatol*; 16(2):191-4, 2004.

4. Andersson B, Nilsson J, Brandt J, Hoglund P, Andersson R: Gastrointestinal complications after cardiac surgery. *Br J Surg*: 92(3):326-33, 2005.
5. Van der Voort Peter HJ, Zandstra Durk F: Pathogenesis, risk factors, and incidence of upper gastrointestinal bleeding after cardiac surgery: Is specific prophylaxis in routine bypass procedures needed? *J Cardiothorac Vasc Anesth*: 14(3): 293-299, 2000.
6. D'Ancona G, Baillot R, Poirier B: Determinants of Gastrointestinal complications in Cardiac surgery: *Tex Heart Inst J*; 30(4):280-285, 2003.
7. Haung CS, Lichtenstien DR: Non variceal upper gastrointestinal bleeding. *Gastroenterol Clin N Am*: 32: 1053 -1078, 2003.
8. Eroglu Y, Emerick KM, Whitingon PF: Octreotide therapy for control of acute gastrointestinal bleeding in children. *J Pediatr Gastroenterol Nutr*. 38:41-47, 2004.