

Turkish Journal of Anaesthesiology & Reanimation

# Bowel Oedema Necessitating Urgent Abdominal Decompression Following Cardiopulmonary Bypass: An Exaggerated Presentation of a Recognised Complication

Rudrashish Haldar 🕩, Aanchal Dixit ២

Department of Anaesthesiology, Sanjay Gandhi Postgraduate Institute of Medical Sciences, Lucknow, India

Cite this article as: Haldar R, Dixit A. Bowel Oedema Necessitating Urgent Abdominal Decompression Following Cardiopulmonary Bypass: An Exaggerated Presentation of a Recognised Complication. Turk J Anaesthesiol Reanim 2020; 48(4): 328-30.

#### Abstract

Gastrointestinal complications after the termination of cardiopulmonary bypass are uncommon; however, they can lead to serious consequences. We encountered an unusual case of an 11-month-old infant who developed gross abdominal distention, leading to ventilatory and haemodynamic embarrassment following separation from the cardiopulmonary bypass. This presumably was a severe manifestation of the inflammatory response observed with extracorporeal circulation, which manifested as bowel oedema and was diagnosed using point-of-care ultrasound. As a rescue measure, urgent abdominal decompression was performed by cutting open the abdominal wall, which restored the ventilator and haemodynamic parameters to almost normal values.

Keywords: Bowel oedema, cardiopulmonary bypass, extracorporeal circulation, systemic inflammatory response

# Introduction

Cardiopulmonary bypass (CPB) provokes an acute inflammatory response, the effects of which may be unpredictable and often associated with serious morbidity and mortality. Existing literature is replete with instances where the complex innate inflammatory response initiated by the operative trauma, CPB and ischaemic reperfusion injury has resulted in the serious dysfunction of multiple organs. Cardiac abnormalities such as atrial fibrillations, (1) renal dysfunction in the form of acute kidney injury, (2) neurological sequelae such as delirium (3) and pulmonary abnormalities and decrease in platelet count (4) are commonly observed following the termination of CPB. Paediatric patients, particularly infants and newborns, have a small circulating plasma volume and immature organs; therefore, the effects of hypothermia, circulatory changes, haemodilution on acid–base balance and systemic inflammatory response (SIRS) caused by extracorporeal circulation are more profound than those observed in adults. After obtaining written and informed consent from the parent of the patient, we report an interesting case of post-CPB bowel oedema that increased intraabdominal pressures to substantially harmful levels, necessitating emergency abdominal decompression.

### **Case Presentation**

An 11-month-old male infant weighing 9 kg and diagnosed with perimembranous ventricular septal defect (VSD) with patent foramen ovale was admitted for repairing the defect. Following the induction of anaesthesia and establishment of invasive lines for monitoring, the surgery was commenced. Cardiopulmonary bypass was initiated, and cardioplegia was delivered. Dacron patch closure of the VSD was performed. Gradual rewarming was initiated, and attempts were made to wean the patient from CPB. However, it failed, and hypotension persisted despite high ionotropic support. CPB had to be re-established, and the surgical site was reopened for inspection. Assessment of the aorta revealed a subvalvular membrane beneath the aortic cusps, obstructing the left ventricular outflow tract. This membrane was

excised, and the aorta was repaired. Rewarming and weaning from CPB was re-attempted. Separation from CPB was successful in this attempt. Because the patient was placed on CPB for the second time, the total duration of the patient's exposure to extracorporeal circulation was approximately 70 min. Few minutes after the patient was separated from CPB, we observed that the patient was developing visible abdominal distention inexplicably along with simultaneous abnormal rise in the peak airway pressures (approximately 26 cm H<sub>o</sub>O). Persistent hypotension developed despite high ionotropic support (noradrenaline at 0.1 mcg kg<sup>-1</sup> min<sup>-1</sup> and adrenaline at 0.12 mcg kg<sup>-1</sup> min<sup>-1</sup>). Moreover, urinary output started decreasing (<0.5 mL kg<sup>-1</sup> hr<sup>-1</sup>). Although a Ryle's tube was placed to decompress the abdomen, the distention did not considerably reduce, and respiratory and haemodynamic alterations persisted. An analysis of preoperative albumin levels and intraoperative serum electrolytes revealed normal values. To investigate the unexplained abdominal distention, an urgent ultrasound examination of the abdomen was performed on the operation table. Ultrasound examination revealed gross bowel oedema and dilated bowel loops distending the entire abdomen (Figure 1). This distention was being transmitted to the thoracic cavity, causing respiratory and haemodynamic compromise along with a decreased urinary output. Considering the urgency of the situation, it was decided to seek the assistance of gastrosurgeons to cut open the abdominal cavity for releasing the abdominal pressure and drain the oedema fluid. The abdominal cavity was opened, and the distended bowel loops were decompressed. Thereafter, 110 ml of oedema fluid was drained, following which a Bogota bag was used to close the abdomen to avoid further precipitous increase of intraabdominal pressures. Following abdominal decompression, the peak pressures immediately reduced and the haemodynamic parameters restored (to almost normal values with ionotropic support).



Figure 1. Ultrasonography image showing bowel oedema and free fluid in the abdomen

Thereafter, the patient was shifted to the intensive care unit on ionotropic support (noradrenaline at 0.04 mcg kg<sup>-1</sup> min<sup>-1</sup> and adrenaline at 0.04 mcg kg<sup>-1</sup> min<sup>-1</sup>). His urine output gradually improved to approximately 1.5 mL kg<sup>-1</sup> hr<sup>-1</sup>. After 18 hr, the patient's ionotropic support was tapered off, and the patient was extubated. Postoperatively, the patient showed a transient increase in creatinine levels on the 3<sup>rd</sup> day, which normalised on the 7<sup>th</sup> day. On the 8<sup>th</sup> day, the Bogota bag was removed, and abdomen was uneventfully closed in layers under general anaesthesia. Finally, the patient was safely discharged on the 21<sup>st</sup> postoperative day.

### Discussion

Numerous patients undergoing cardiac surgery experience difficulty while weaning from CPB, which necessitates the reestablishment of CPB and urgent therapeutic measures. Gastrointestinal complications are the sporadic but serious consequences of cardiac surgery involving CPB. They may vary severity; therefore, their treatment strategies also differ. Previous researchers have identified certain independent predictors for the development of postoperative gastrointestinal complications, such as intestinal ischaemia, paralytic ileus, gastrointestinal bleeding and liver dysfunction. These include age of >70 years, prolonged CPB, postoperative blood transfusion and re-exploration for bleeding (5).

Gastrointestinal ischaemia following CPB occurs due to low cardiac output, hypotension following blood loss and splanchnic atheroemboli. Moreover, recurrent periods of hypotension occur during surgery, causing the redistribution of blood flow toward the kidneys, brain and heart at the expense of the visceral organs. In addition, aortic cross clamping and CPB with extracorporeal circulation during cardiac surgery significantly decrease the mesenteric blood flow, resulting in intestinal hypoperfusion (6, 7). The gastrointestinal system is particularly vulnerable to ischaemia due to the absence of autoregulation and preferential shunting of blood away from the gastrointestinal circulation during periods of hypotension.

Gut ischaemia extending for a sufficient duration impairs the gastrointestinal tract barrier function and promotes bacterial translocation from the intestinal lumen due to the loss of enterocyte integrity and breakdown of tight junctions (8). This increase in mucosal permeability, proinflammatory cytokine and systemic endotoxin concentration, compounded by exposure to the extracorporeal circuit, provokes a severe immune response causing intestinal damage.

Infants and newborns, owing to their small circulating plasma volumes and immature organs, experience the exaggerated effects of hypothermia, circulatory changes, and haemodilution on acid–base balance, which are inherent to the CPB exposure and SIRS caused by extracorporeal circulation, which is more intense than the one in adults. Because the effects of postoperative inflammatory response correlate with the duration of extracorporeal circulation, (9) if the exposure to CPB is prolonged or repeated, these effects are amplified. Although gastrointestinal complications following CPB are well recognised, they are typically delayed and manifest on the 2<sup>nd</sup> or 3<sup>rd</sup> postoperative day (10). Severe intraoperative manifestation of the same has not yet been reported. Filtration during CPB (conventional or zero-balance ultrafiltration) may be used to remove inflammatory mediators and vasoactive substances, whereas modified ultrafiltration is performed after CPB to reverse haemodilution and decrease tissue oedema. In our patient, ultrafiltration was not used, and although it could have reduced the inflammatory response to some extent, this reduction cannot be accurately predicted (11, 12). In the absence of any other plausible reason, we postulate that the immaturity of the infant's organ system and the need for reestablishment of CPB, which increased the overall exposure and duration of bypass (approximately 70 min), contributed to the development of gross intestinal oedema and abdominal distention, thereby resulting in ventilatory and haemodynamic disturbances.

Visceral hypotension is the most significant factor in the development of gastrointestinal complications following cardiac surgery; the effects of intraoperative hypothermia and non-pulsatile flow (which are detrimental for mucosal perfusion) along with hypotension contribute to the generation of intestinal oedema. This oedema leads to the compression of the major vessels, causing haemodynamic aberrations and reducing renal perfusion, consequently decreasing the urinary output. Urgent diagnosis aided by point-of-care ultrasound and expeditionary decompression of the abdominal cavity and drainage of the oedema fluid provided dramatic relief from a near-catastrophic condition.

# Conclusion

Through this report, we endeavour to make the clinicians cognizant regarding the possibility of development of such complications intraoperatively and the emergent measures undertaken to counter them. Further, we reiterate the vital role played by ultrasound as a point-of-care standard for clinical diagnosis and decision-making during emergency situations.

**Informed Consent:** Written informed consent was obtained from patients' parents who participated in this case.

**Peer-review:** Externally peer-reviewed.

Author Contributions: Concept – R.H., A.D.; Design – R.H., A.D.; Supervision – R.H.; Resources – R.H., A.D.; Materials – R.H.,

A.D.; Data Collection and/or Processing – A.D.; Analysis and/or Interpretation – R.H.; Literature Search – R.H., A.D.; Writing Manuscript – R.H.; Critical Review – A.D.; Other – R.H.

**Conflict of Interest:** The authors have no conflicts of interest to declare.

**Financial Disclosure:** The authors declared that this study has received no financial support.

## References

- Zakkar M, Ascione R, James AF, Angelini GD, Suleiman MS. Inflammation, oxidative stress and postoperative atrial fibrillation in cardiac surgery. Pharmacol Ther 2015; 154: 13-20. [CrossRef]
- Lagny MG, Jouret F, Koch JN, Blaffart F, Donneau AF, Albert A, et al. Incidence and outcomes of acute kidney injury after cardiac surgery using either criteria of the RIFLE classification. BMC Nephrol 2015; 16: 76. [CrossRef]
- O'Neal JB, Billings FT 4th, Liu X, Shotwell MS, Liang Y, Shah AS, et al. Risk factors for delirium after cardiac surgery: an historical cohort study outlining the influence of cardiopulmonary bypass. Can J Anaesth 2017; 64: 1129-37. [CrossRef]
- Luo S, Wang Y, An Q, Chen H, Zhao J, Zhang J, et al. Platelets protect lung from injury induced by systemic inflammatory response. Sci Rep 2017; 7: 42080. [CrossRef]
- Zacharias A, Schwann TA, Parenteau GL, Riordan CJ, Durham SJ, Engoren M, et al. Predictors of Gastrointestinal Complications in Cardiac Surgery. Tex Heart Inst J 2000; 27: 93-9.
- Ohri SK, Somasundaram S, Koak Y, Macpherson A, Keogh BE, Taylor KM, et al. The effect of intestinal hypoperfusion on intestinal absorption and permeability during cardiopulmonary bypass. Gastroenterology 1994; 106: 318-23. [CrossRef]
- Straub U, Winning J, Greilach P, Isringhaus H, Kalweit G, Huwer H. Alterations of mesenteric blood flow after cardiopulmonary bypass: a Doppler sonographic study. J Cardiothorac Vasc Anesth 2004; 18: 731-3. [CrossRef]
- Piton G, Capellier G. Biomarkers of gut barrier failure in the ICU. Curr Opin Crit Care 2016; 22: 152-60. [CrossRef]
- Habes QLM, Linssen V, Nooijen S, Kiers D, Gerretsen J, Pickkers P, et al. Markers of intestinal damage and their relation to cytokine levels in cardiac surgery patients. Shock 2017; 47: 709-14. [CrossRef]
- Vermeulen Windsant IC, Hellenthal FA, Derikx JP, Prins MH, Buurman WA, Jacobs MJ, et al. Circulating intestinal fatty acid-binding protein as an early marker of intestinal necrosis after aortic surgery: a prospective observational cohort study. Ann Surg 2012; 255: 796-803. [CrossRef]
- Gaynor JW. Use of ultrafiltration during and after cardiopulmonary bypass in children. J Thoracic Cardiovasc Surg 2001; 122: 209-11. [CrossRef]
- Thapmongkol S, Masaratana P, Subtaweesin T, Sayasathid J, Thatsakorn K, Namchaisiri J. The effects of modified ultrafiltration on clinical outcomes of adult and pediatric cardiac surgery. Asian Biomedicine 2017; 9: 591-9. [CrossRef]