

**Case Report**

***Massive Volume Loss During Cardiopulmonary Bypass and its Association with Meckel's Diverticulum***

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**ABSTRACT**

The occurrence of any gastrointestinal (GI) complication concurrent with cardiac surgery may greatly increase a patient's morbidity and mortality. The most frequently discussed GI complication associated with cardiac surgery is hemorrhage from peptic ulcerations and the exacerbation of this condition through heparinization. However, there are consequences of other GI problems which the clinical perfusionist needs to be aware of. This paper presents a case of a 65 year old female undergoing routine coronary artery bypass grafting. Although this patient was anuric, an excessive amount of fluid loss and replacement occurred. This paper reviews the case management and the eventual diagnosis of acute peritonitis as a result of perforated Meckel's diverticulum and describes the subsequent surgical intervention for repair of the perforation.

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## INTRODUCTION

Sudden or excessive volume loss during cardiac surgery requiring cardiopulmonary bypass (CPB) is generally due to either a high urine output (Uo), impairment of the venous return to the extracorporeal circuit, hemorrhage, or extreme third spacing marked by generalized edema. In some cases, reservoir volume may be restored by repositioning a venous cannula or by surgically repairing the site of hemorrhage. In other cases, an underlying physiological cause may not be so apparent and may not be rectified so immediately. Whatever the cause, volume replacement is often necessary to restore an adequate and safe reservoir volume and maintain proper blood flow.

Meckel's diverticulum is a congenital anomaly occurring in an estimated 2% of the population. Clinical manifestations include hemorrhage, intestinal obstruction, fistulas, and diverticulitis with symptoms mimicking those of acute appendicitis. Perforation of the small intestine may occur where the diverticulum arises, possibly leading to peritonitis and eventually to substantial peritoneal fluid shifts due to the bowel's tremendous ability to sequester fluids. In most cases, Meckel's diverticulum is an incidental finding at postmortem or during laparotomy, and an accurate diagnosis is only made by exploratory surgery. Many patients with Meckel's diverticulum are asymptomatic and for these individuals the diverticulum is left alone. However, if the patient is symptomatic or if there is evidence of ectopic mucosa, the recommended treatment is prompt surgical removal of the diverticulum.

## CASE DESCRIPTION

A 65 year old 1.5 M<sup>2</sup> female presented to the operating room for coronary artery bypass grafting (CABG). Her cardiac catheterization results demonstrated severe three vessel disease with stenosis of the left anterior descending, first diagonal, obtuse marginal and posterior descending coronary arteries. Additional cardiac diagnostic data revealed a heavily calcified aortic root and decreased ventricular function. Past medical history was significant for hypertension, end stage renal disease requiring hemodialysis, chronic anemia, chronic relapsing pancreatitis, history of a duodenal ulcer, and a history of gastrointestinal (GI) hemorrhage presumed secondary to colonic diverticulosis. Of note, during her cardiac catheterization three days prior to surgery, the patient developed an allergic reaction to the contrast medium with swelling of the tongue and salivary glands but no rash or other anaphylactoid symptoms. She was treated with prednisone and her symptoms subsided.

Her preoperative blood work was essentially normal except for a chronically elevated BUN (39 mg/dl), creatinine (6.0 mg/dl), and amylase (151 U/l). Her hematocrit (Hct.) was 31.6% and her platelet count was 172 10<sup>9</sup>/L. She had been dialyzed the day prior to surgery. Her weight the morning of surgery was 53 kg.

## CASE MANAGEMENT

The patient arrived in the operating room in stable condition, was placed under general anesthesia, prepared for surgery, and a median sternotomy was performed. After two segments of saphenous vein were harvested and prepared, the patient was systemically anticoagulated with 16,000 IU of porcine mucosa heparin. A 36 Fr x 46 Fr two stage cannula<sup>a</sup> was placed in the right atrium and secured with purse string sutures. A 22 Fr aortic root cannula<sup>b</sup> was placed in the proximal aorta. The extracorporeal circuit consisted of a membrane oxygenator<sup>c</sup>, a hardshell venous reservoir<sup>d</sup>, an arterial filter<sup>e</sup>, and roller pumps<sup>d</sup>. The circuit was primed with 1500 ml balanced electrolyte solution, 100 ml 25% albumin and 8000 IU heparin. After an ACT of 400 seconds was obtained, the patient was placed on CPB and cooled to a target core temperature of 28°C. The aorta was cross clamped, and 500 ml of cold, oxygenated crystalloid cardioplegia was delivered into the aortic root via an 18 G needle. The myocardial temperature was measured<sup>f</sup> and an initial temperature of 9.8°C was achieved. The aorta was vented and the cardioplegia was returned to the circuit.

The first arterial blood gas (ABG) was drawn from the extracorporeal circuit 9 minutes after the initiation of CPB. The results were: pH 7.39, pO<sub>2</sub> 616 mmHg, pCO<sub>2</sub> 33 mmHg, base deficit -3.3, K 4.7 meq/l, Na 132 meq/l, and Hct 21%. After approximately 20 minutes on CPB, a gradual but significant reduction in reservoir volume was noted. There was no significant increase in central venous pressure, no observable obstruction to venous return, and no jugular distention. The patient was anuric due to her renal disease, and there was no obvious edema in the face or extremities. The anesthesiologist stated that based on her pre-bypass filling pressures she was not hypovolemic preoperatively from her hemodialysis the previous day. The abdomen was palpated at this time and appeared to be normal. The volume loss persisted, while the in-line monitor saturation/Hct showed the patient's Hct to be increasing. After 40 minutes on CPB, 1000 ml of electrolyte solution had been given in order to maintain a safe reservoir volume and appropriate blood flow and a total 1000 ml of cardioplegia had been administered.

The second ABG was taken after 39 minutes on CPB and showed all parameters were within normal limits, and the Hct was 25.2%. The volume loss persisted at a steady rate, and electrolyte solution was given for replacement. After 60 minutes, the cross clamp was removed and 100 mg of lidocaine was given via the extracorporeal circuit. The heart was defibrillated after three electrical shocks, and then paced at a rate of 30 beats per minute while the two proximal anastomoses were performed. The

- a Sarns 3M Corp., Ann Arbor, MI 48103
- b DLP, Inc., Grand Rapids, MI 49501-0409
- c AVECOR Cardiovascular, Plymouth, MN 55441
- d COBE Cardiovascular, Arvada, CA 80004
- e Terumo Corp., Tokyo, Japan
- f Electromedics Corp., Parker, CO 80134-9061

patient was weaned from bypass after 96 minutes and required minimal pharmacological support.

A total of 2000 ml of electrolyte solution and 1500 ml of cardioplegia was given during CPB, in addition to the 1608 ml of prime and 650 ml of IV saline administered by the anesthesiologist pre-bypass. This gave the patient a total hemodilution of 5758 ml. The last ABG drawn shortly before terminating CPB showed a Hct of 24.9%, demonstrating essentially no hemodilution. With the exception of a Na level of 128 meq/l all other parameters were normal.

As the chest was being closed, the patient's abdomen was observed to be distended and edematous. Intraoperative fluoroscopy showed some gas in her bowel, but was otherwise normal. A general surgeon was consulted and a diagnostic peritoneal lavage was performed using 1000 ml of normal saline. The effluent showed no signs of blood or intestinal contents and it was presumed that the patient's acute ascites was caused by an ileus. However, as the lavage catheter was being removed, a small amount of fecal material was observed. A laparotomy was immediately performed and the intestine was explored, revealing an acute perforation of the distal ileum approximately 60 cm proximal to the cecum. The location of the perforation correlated with that of a Meckel's diverticulum. It was also noted that the mesentery at this site was extremely edematous. The perforation was closed in transverse layers. Then the rest of the intestine was inspected and found to be normal. The small bowel was returned to the abdomen and the abdomen was closed. The remainder of the patient's hospital stay was uneventful, and she was discharged on the 11th postoperative day.

## DISCUSSION

Gastrointestinal complications represent a significant risk for post cardiectomy patients. Recent studies have shown that the overall incidence of GI complications following cardiac surgery is 1.4% with an associated mortality rate of 20% (1), and as high as 44% should GI surgery be required (2). One such GI complication is acute intestinal perforation with subsequent peritonitis. This peritonitis, caused by fecal irritation, may then lead to sequestration of fluid into the peritoneum via transudation or exudation, with the eventual development of ascites.

To fully comprehend these fluid shifts, one must appreciate the tremendous capacity the small intestine has to absorb water. For example, in a 70 kg adult, almost one half of the body's extracellular fluid volume is secreted into the upper GI tract daily. The jejunum alone receives 8-10 liters of isotonic fluid per 24 hours, and reabsorption in the proximal bowel will save between 75-80% of this fluid. However, under certain pathological conditions, such as an acute intestinal perforation, these fluid dynamics may be greatly altered leading to massive fluid shifts (3).

Meckel's diverticulum occurs from persistence of the omphalomesenteric duct connecting the lumen of the develop-

ing embryonic gut to the yolk sac. It is a true diverticulum in that it contains three layers of the normal intestinal wall (4); however, in about 25% of occurrences, heterotopic gastric or pancreatic tissue may be present (5). It usually occurs in the terminal ileum 45-90 cm proximal to the ileocecal valve (6). In autopsy studies, it has an incidence rate of .3%; however, it may be as high as 2% when surgical cases are reviewed (6,7). The most common complication associated with Meckel's diverticulum is bleeding from an ileal ulcer associated with heterotopic gastric tissue. However, in about 20% of patients, there is an occurrence of diverticulitis. Failure to properly diagnose this may lead to perforation of the diverticulum, peritonitis, and death.

The clinical dilemma with this case was trying to determine the cause for this patient's unusually high volume loss in conjunction with her rising Hct in spite of a relatively large volume of crystalloid administration. Based on this patient's 53 kg weight and BSA of 1.5 M<sup>2</sup>, her estimated circulating blood volume was 4.2 liters. Her initial Hct pre-bypass was 34.2%. Her hemodilution prior to the first ABG on bypass consisted of 1608 ml of prime, plus 500 ml crystalloid cardioplegia and 650 ml from intravenous saline. Using the formula for the estimated Hct following hemodilution:

$$\text{Est. Hct} = \frac{(\text{Circulating blood volume}) \times (\text{Pt. Hct})}{(\text{Volume added} + \text{Circulating blood volume})}$$

her initial Hct post dilution should have been about 20.6%, and in fact it was 20.7%. However, the fact that her Hct began to increase despite her anuria and no apparent third spacing was very suspicious. Using a total volume added during the procedure of 5.75 liters, her calculated post bypass Hct should have been 14.4%. Our measured result of 24.9% was rather baffling, until her ascites was discovered and her perforated Meckel's diverticulum with peritonitis was diagnosed.

Presumably this patient's preoperative treatment with steroids helped mask the symptoms of her recent perforation. Had she not undergone cardiac surgery and had the associated fluid shifts which dramatically accelerated her ascites, her contrast allergy and ensuing medical treatment may actually have led to her demise.

It is important that perfusionists consider and recognize GI complications as a source of fluid shifts during CPB, especially in the face of persistent volume loss, coupled with crystalloid fluid replacement and an unexpected lack of hemodilution based on Hct. The abdomen should be palpated and any sign of distention resembling acute ascites should trigger suspicion of a GI disorder.

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