Acute Bowel Ischemia after Coronary Bypass Surgery – A Catastrophic Event

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ABSTRACT

Acute bowel ischemia is a rare but often catastrophic event after coronary bypass surgery. We report three cases in our department and highlight the difficulty in making the diagnosis thus delaying timely intervention. We discuss why an aggressive approach in both investigating and managing this condition is warranted. We believe that early mesenteric angiogram and directed exploratory laparotomy are the preferred methods in managing this difficult condition.

Keywords: Mesenteric ischemia, mesenteric angiogram, exploratory laparotomy, risk factors

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INTRODUCTION

Acute bowel ischemia is an infrequent event (less than 1%) in patients undergoing coronary bypass surgery[1]. However when it occurs it is often lethal. Mortality rates range from 70 - 100%[2], The difficulty in making the diagnosis contributes heavily to the catastrophic end result. A high index of suspicion is important for an early diagnosis. This is based on noting inherent predisposing risk factors for bowel ischemia and recognizing ongoing clinical events. A mesenteric angiogram should immediately follow to confirm the diagnosis. Then, only with expedient surgery can the patient have any chance of survival. We report three cases in the last two years in our institution. We highlight the reasons for the uniformly dismal outcome and the difficulty in making the diagnosis. Finally, we state the preferred approach in dealing with this difficult condition.

PATIENT AND METHODS

Case 1

A 68-year-old Chinese female had a recent history of an extensive anterolateral myocardial infarction. Her co-morbid state included non-insulin dependent diabetes mellitus and hypertension. Elective cardiac catheterization revealed severe triple vessel disease, anterior wall hypokinesia and ejection fraction of 30%. She experienced severe chest pain and was started on heparin and glyceryl trinitrate without relief until insertion of an intra-aortic balloon pump (IABP). Emergency coronary bypass surgery with 3 bypass grafts was performed. Surgery was uneventful with a cross clamp time of 28 minutes and cardioplumonary bypass (CPB) time of 55 minutes. She was weaned off CPB with 6 micrograms of dopamine/kg/minute. She was extubated on the first postoperative day (POD-1) with same day removal of the IABP. On POD-8 she developed recurrent atrial fibrillation of 150 beats/min with a blood pressure of 100/80 mm Hg. Intravenous amiodarone was started. Her total white cell count (wbc) climbed to 19800/ml on POD-9 and she became febrile with a temperature of 38ºC. Blood and stool cultures were negative for bacterial growth. On POD-13 she complained of left flank pain. Investigations revealed a serum amylase level of 87iu/L and urine examination showed 100-200 red blood cells/ml. Her abdominal signs were unremarkable. Her white cell count was now 25900/ml. Later that night she became breathless with severe abdominal pain. Her abdomen was distended without guarding or rebound tenderness. Her plain abdominal films revealed only dilated loops of small and large bowel. She deteriorated and required intubation POD-14. Her pre-intubation blood gas was pH 7.45, P02 41.2 mm Hg, PC02 34.5 mmHg, standard HC03 24.4 mmol/L. She became comatose with a white cell count that was now 33,800/ml. Finally an exploratory laparotomy was made and this revealed hemopurulent fluid with small bowel necrosis from distal jejenum to terminal ileum and ischemia of the ascending colon. In view of extensive bowel infarction, the abdomen was closed and she died from multi-organ failure with intractable acidosis.

Case 2

A 65-year-old Chinese male had chronic stable angina for five years. His co-morbid state included hypertension, hyperlipidaemia and end stage renal

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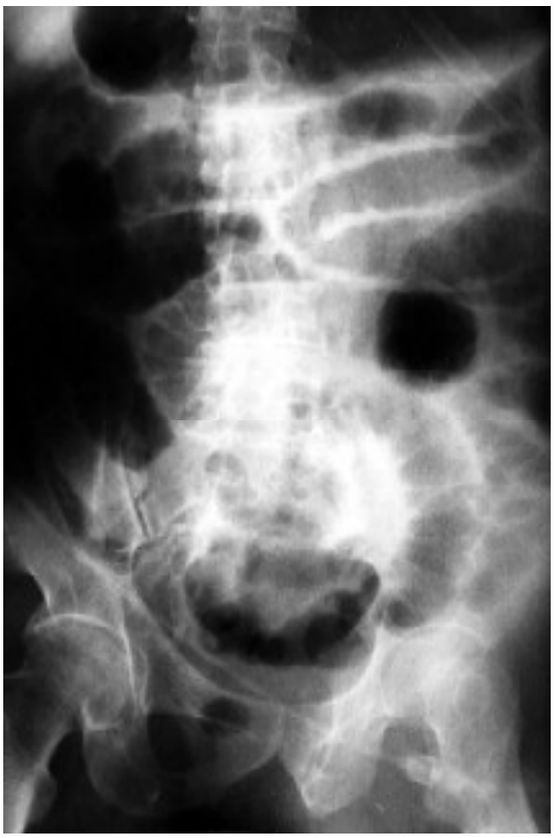

Fig. 1 Bowel Ischemia After Coronary Bypass Surgery.

failure due to chronic glomerulonephritis. The cardiac catheterization revealed severe triple vessel disease. The EF was 40%. The abdominal aorta was very tortuous. Whilst being prepared for surgery, he threw a generalized tonic-clonic fit. There was no residual neurological deficit. Electroencephalogram showed no epileptic activity and the CT scan of the brain was negative for infarction. He underwent coronary bypass surgery two days later with pre-operative haemodialysis. Operative findings included plaques at the innominate artery and aortic arch. Five bypass grafts were performed. Clamp time was 64 minutes and CPB time was 113 minutes. He was extubated on POD-1. On POD-2 he became confused and disorientated. Blood pressure was 166/84 mmHg. Heart rate was 90/min. The K+ level was 5.8. He improved with dialysis. He became febrile on POD-4 with rebound tenderness in the lower abdomen. Temperature was 38ºC. Per rectal exam was unremarkable. His white cell count climbed to 21560/ml. The plain abdominal film revealed loops of dilated small bowel with "thumb-printing" (Fig. 1) that was not recognised. Duplex exam of the abdominal aorta was inconclusive. He required intubation later in the day when he became drowsy, hypotensive and hypoxic. His blood gas revealed pH 7.35, HCO3 16mmol/L, BE -7.7 mmol/L. Serum amylase was 127 iu/L. Cardiac 2D-Echo was unremarkable. He had a cardiac arrest 2 hours later and was resuscitated. His chest was opened with no signs of pericardial effusion or pulmonary embolism. An immediate laparotomy was performed 15 cm of small bowel was necrotic. The SMA pulse was not palpable. An embolectomy of the SMA was successfully done. Non-viable ileum was excised. Unfortunately the patient had another arrest on the table and could not be resuscitated.

Case 3

A 66-year-old Malay male had a history of previous coronary bypass surgery in 1993. The co-morbid state included hypertension, hyperlipidaemia and chronic renal failure. He developed recurrent angina with repeated hospital admissions. The cardiac catheterization revealed only one patent graft ie LIMA to LAD. The EF was 50%. Due to refractory angina, he required an IABP insertion and emergency coronary bypass surgery. The abdominal aorta was noted to be tortuous at the time of IABP insertion under fluoroscopy. He had three venous bypass grafts with a cross-clamp time of 49 minutes and a CPB time of 3 hr and 32 minutes. The IABP position was not optimal. An attempt at adjustment was made but failed due to the tortuous nature of theaorta. Later it was noticed that he had developed trash feet and the IABP was removed immediately with good dorsalis pedis pulses felt bilaterally. Later he complained of abdominal pain but examination was unremarkable. Serum amylase was 240 iu/L. On POD-2, left lower quadrant tenderness developed. Per rectal examination was unremarkable. The arterial blood gases revealed pH 7.23, BE -9.3 mmol/L, HCO3 17mmol/L. A mesenteric angiogram was immediately ordered. This revealed a patent proximal main SMA with a dearth of ileal branches (Fig. 2). The right colic and middle colic arteries were absent. The superior mesenteric vein was patent 120 mg of intra-arterial papaverine was infused with some improvement (Fig. 3). Emergency laparotomy revealed the following: proximal jejunum had patchy bruising and petechial staining. 4 cm of distal ileum was infarcted. Segmental arterial pulsations were weak. Resection of non-viable bowel was done. The next day he developed ventricular tachycardia requiring electrical defibrillation. He remained on adrenaline 0.2 ug/kg/min and dopamine 7 ug/kg/min. His blood pressure remained 80 to 90 mmHg systolic. The drain revealed bloody fluid. A planned second look was not done due to the reluctance of the anaesthetist and general surgeon to proceed in view of his poor haemodynamic state. He developed

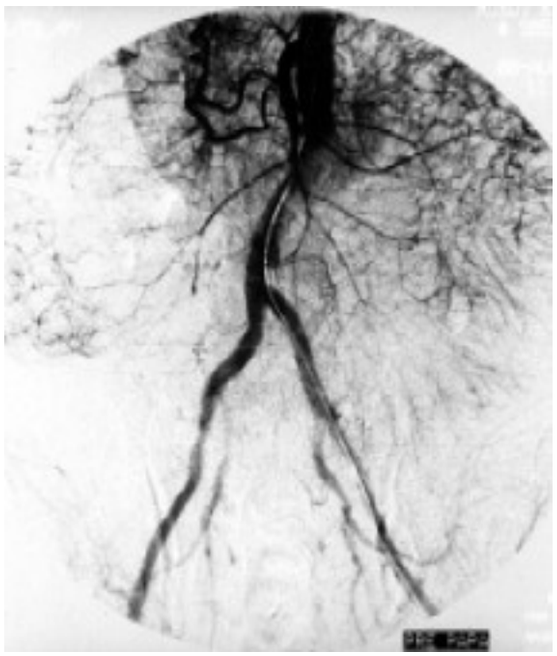

Fig. 2 Bowel Ischemia After Coronary Bypass Surgery.

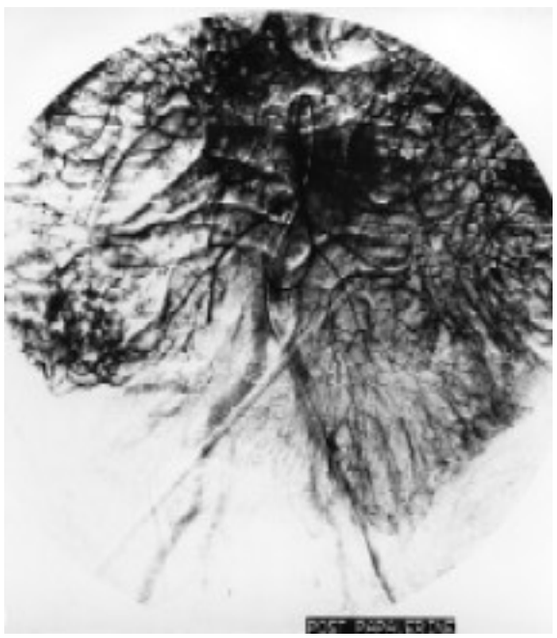

Fig. 3 Bowel Ischemia After Coronary Bypass Surgery.

intractable acidosis and succumbed on POD-4.

DISCUSSION

It is a well known that acute bowel ischemia is often a lethal event. The patho-physiology of bowel ischemia is complex and multifactorial. Acute bowel ischemia, of arterial origin, falls into two broad categories: occlusive ischemia due to thrombosis or embolism (70%) and non-occlusive ischemia (30%) seen in "shock-bowel" syndrome and "low-flow" syndrome where high dosages of vasoconstricting agents are often used in haemodynamically unstable patients. Conditions that are predictors of susceptibility to thrombo-embolism include diffuse arteriosclerotic disease (the arteriopath), hypertension, diabetes mellitus, hyperlipidaemia and apoplexy. The risk of embolization is also high with interventions in an atherosclerotic or tortuous abdominal aorta. The use of the intra-aortic balloon pump is known to cause embolic showers especially if placement is difficult, requiring excessive manipulation in a diseased aorta. Cardiac catheterization too may effect embolic showers causing trash feet and mesenteric vascular occlusion. Commonly used drugs such as digoxin, epinephrine, norepinephrine and high doses of dopamine are known to cause or potentiate mesenteric vasoconstriction. Chronic dialysis patients[3] are known to be susceptible to non-occlusive mesenteric ischemia.

In case 1, the predisposing risk factors were age, a moderately impaired ejection fraction, diabetes and hypertension. Ongoing clinical events that predisposed to thrombo-embolism were the use of an IABP and recurrent atrial fibrillation with hypotension on POD-8. Fever, severe leukocytosis and flank pain with minimal abdominal signs on POD-13 should have raised the suspicion of bowel ischemia. Delaying the decision to explore her abdomen until acidosis and collapse not surprisingly proved fatal. Frank bowel necrosis had already occurred. The plain abdominal film and serum amylase levels were not helpful.

In case 2, the predisposing risk factors were age, hypertension, hyperlipidaemia, end-stage renal failure, a tortuous abdominal aorta and atherosclerotic plaques in the arch of the aorta. Ongoing clinical events such as cannulation of the atherosclerotic aorta with possible "sandblasting" effect could have caused intra-and peri-operative dislodgement of atherosclerotic debris. On POD-4, significant abdominal signs had developed with a temperature of 38ºC, leukocytosis of 21560/ml and acidosis. Exploratory laparotomy was again delayed. Despite a successful embolectomy of the SMA, the patient succumbed to intractable shock.

In case 3, the predisposing risk factors were age, hypertension, hyperlipidaemia and chronic renal failure. Ongoing clinical events were the IABP insertion in a tortuous aorta, a complex redo cardiac operation with a long CPB time and the attempt to adjust the position of the IABP postoperatively. The latter caused dislodgement of atherosclerotic debris as evidence from the trash feet. Further, he developed abdominal pain after balloon removal

with a raised serum amylase. This should have raised the suspicion that mesenteric embolism had occurred. A one day delay in doing the mesenteric angiogram proved fatal. The angiographic picture was consistent with embolization to the SMA and its distal circulation. Intra-arterial papaverine was partly successful in relieving the associated vasospasm but laparotomy revealed infarcted distal ileum. Unfortunately the patient was not brought back for a second look operation. The second operation was certainly indicated in view of the uncertain viability of unresected bowel. The patient succumbed to intractable acidosis. The 3 cases demonstrate that without a timely diagnosis, this is often a lethal event. Unfortunately establishing an early diagnosis is difficult. Apart from abdominal pain, there are usually no abdominal signs till established bowel infarction has occurred. The pain however is characteristically out of proportion to the physical signs. In the plain abdominal film, apart from non-specific dilated loops, there are no other signs until pneumotosis, frank perforation or portal venous gas develop. "Thumb-printing" and formless loops of small bowel can sometimes be seen due to mucosal edema and haemorrhage. However this usually signifies infarction. The serum amylase is not always raised and is non-specific. A leukocytosis exceeding 15,000/ml and severe metabolic acidosis may occur in only 50-70% of patients with acute ischemia. In view of the lack of characteristic clinical symptoms and signs in the early phase of bowel ischemia, the only way to come to a diagnosis is to have a high index of suspicion. Each patient should be looked at from the point of having any predisposing factors and then taking notice of significant ongoing clinical events that may precipitate bowel ischemia. Once suspicion is aroused, it is mandatory to be aggressive in pursuing a mesenteric angiogram. The angiogram may be therapeutic when coupled with intra-arterial papaverine for non-occlusive ischemia. If occlusive, it may reduce concomitant vasospasm, limiting the extent of infarction. Immediate selective laparotomy can be planned. The alternative to an aggressive approach usually meets with a lethal outcome. Most literature reports cite a mortality of close to 90% when the diagnosis is delayed[5]. Thus in order to salvage these patients, early mesenteric angiogram should be done, accepting a fair number of negative studies. Boley has shown[6] that the mesenteric angiogram is the preferred investigative modality. He achieved a 54% survival rate in 35 cases of bowel ischemia. The survival rate in non-occlusive ischemia was 60%. In another review[7] he achieved a 55% survival in cases of embolism to the SMA where angiogram was followed by intra-arterial papaverine and selected laparotomy. The alternative "traditional" methods resulted in 80% mortality. Other diagnostic modalities have been advocated. Duplex examination is not always possible as distended bowel prevents adequate visualization. Peritoneal lavage for white and red cell counts and amylase levels gives positive results only in the late phase[4]. Laparoscopy may be helpful but raising the intra-abdominal pressure beyond 20 mm Hg is detrimental and worsens bowel ischemia. Also dilated loops may preclude adequate examination.

Finally it is imperative that the surgeon takes precautionary measures. Quick surgery (shorter CPB times) should be performed with higher perfusion pressures in patients who have predisposing risk factors especially advanced age and a heavy atherosclerotic load. Maintaining optimal haemodynamics, correcting aggressively arrhythmias especially atrial fibrillation, use of systemic vasodilators such as calcium channel blockers and nitrates and expeditious removal of balloon pumps are important postoperative measures.

As Singapore's population rapidly ages and with the ready availability of cardiovascular services, more elderly patients will undergo coronary bypass surgery. Many will have risk factors that when combined with a triggering event result in acute intestinal ischemia. Some of these patients will even have stormy postoperative courses and require extraordinary cardiopulmonary support only to present with acute bowel ischemia. It is therefore imperative that the attending cardiac surgeon have in place, a working algorithm, to make an early diagnosis thus allowing rapid intervention.

CONCLUSION

In conclusion, only 3 factors can make a difference in the outcome of acute intestinal ischemia after coronary bypass surgery. They are a high index of suspicion, an aggressive investigative protocol utilizing early mesenteric angiograms and timely intervention involving a combination of intra-arterial papaverine and exploratory laporotomy.

REFERENCES

1. Albes JM, Schistek R, Baier R, Unger F. Intestinal ischemia associated with cardiopulmonary bypass surgery: a life threatening complication. J Cardiovasc Surg 1991; 32:527-33.
2. Huddy SPJ, Joyce WP, Pepper JR. Gastrointestinal complications in 4473 patients who underwent cardiopulmonary bypass surgery. Br J Surg 1991; 78:293-6.
3. Dahlberg PJ, Kisken WA, Newcomer KL, et al. Mesenteric ischemia in chronic dialysis patients. Am J of Nephrology 1985; 5:327-32.

4. Richardson J, Flint LM, Polk LM. Peritoneal lavage: a useful diagnostic adjunct for peritonitis. Surgery 1983; 93:826-9.
5. Kayela RN, Sammartano RJ, Boley SJ. Aggressive approach to acute mesenteric ischemia. Surg Clin N Ame Vol 72; 1:157-82.
6. Boley SJ, Sprayregen S, Siegelman SS, Veith FJ. Initial results from an aggressive roentgeonological and surgical approach to acute intestinal ischemia. Surgery 1977; 82:848-55.
7. Boley SJ, Feinstein FR, Sammartano R, Brandt LJ. New concepts in management of emboli of the superior mesenteric artery. Surg Gynecol Obstet 1981; 153:561- 9.