Superior Mesenteric Arterial Occlusion Following Laparoscopic Partial Fundoplication

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ABSTRACT
A 62-year-old male with history and endoscopic findings consistent with gastroesophageal reflux underwent elective laparoscopic fundoplication. He developed severe abdominal pain four days postoperatively, and computed tomography (CT) angiogram of the abdomen demonstrated occlusion of the superior mesenteric artery due to dissection. The patient was administered intravenous heparin following vascular surgical advice, resulting in resolution of the pain within an hour and no subsequent complications. Laparoscopy-associated mesenteric vascular events are rare but associated with very high morbidity and mortality. Mesenteric arterial occlusion is most frequently reported following laparoscopic cholecystectomy but may occur following many common laparoscopic procedures. Presentation generally occurs hours to days following the procedure, with severe abdominal pain out of proportion with physical signs. If left unrecognized, patients progress to bowel and visceral ischemia, necrosis, and multiorgan failure. Mechanisms postulated to cause these mesenteric vascular events involve changes in splanchnic blood flow, reduced cardiac output and systemic venous return, and hypercapnia related to carbon dioxide insufflation. Diagnosis may be made promptly with CT angiography, and potentially treated with intravenous heparin alone, avoiding a laparotomy or bowel resection. This is the first reported case of successful anticoagulation causing resolution of the occlusion sufficient to avoid reoperation or bowel resection. Once identified, this condition should be treated in liaison with vascular surgery colleagues, which may require anticoagulation, endovascular, or open intervention.

Key Words: Anticoagulation, Laparoscopy, Mesenteric ischemia.

INTRODUCTION
The first surgeons to report a laparoscopy-associated mesenteric vascular event wrote that, “particularly rare or devastating complications are unlikely to be reported in the medical literature because individual misjudgment is often implicated.” Although uncommon, this is a potentially catastrophic complication of commonly performed procedures, which all surgeons should understand. Severe abdominal pain following laparoscopic surgery should prompt rapid angiographic imaging of mesenteric vessels, as treatment and complete resolution of ischemia with no long-term complications is possible.

Case Details
A 62-year-old male was referred to the operating surgeon with symptoms consistent with esophageal reflux. He underwent endoscopy which demonstrated esophagitis and a hiatal hernia, and manometry studies found no other motility disorder. A barium swallow showed reflux of gastric contents to the midthoracic area. He was overweight with diet-controlled type two diabetes mellitus, with no preceding history of cardiac or mesenteric ischemia.
The patient underwent an elective laparoscopic fundoplication, in combination with a mesh umbilical hernia repair. Insufflation pressures of 17 mmHg were used to achieve adequate views, particularly while placing superior fundoplication sutures on the diaphragm and reconstructing the angle of His. These pressures are often required in an overweight patient or with a large left lobe of liver in a head up position, as occurred in this case. The patient was prescribed subcutaneous enoxaparin, compression stockings, and sequential calf compression devices intraoperatively. The procedure was uncomplicated and he was discharged home on postoperative day two. Subsequently four days postoperatively the patient developed severe abdominal pain and presented to the emergency department.

Initial plain x-rays did not show any free gas or other explanation for the patient’s symptoms, and blood tests showed only a mild leukocytosis. A portal venous contrast computed tomography (CT) scan of the abdomen demonstrated a segmental right renal infarct and slight dilatation of a segment of small bowel, but no explanation for the patient’s severe abdominal pain was identified. Subsequent CT angiogram of the abdomen was performed due to concern for gut ischemia, and this demonstrated occlusion of the superior mesenteric artery (SMA) and right renal artery of uncertain etiology, thought initially to be embolic. The superior mesenteric arterial occlusion was 5 cm in length and commenced 22 mm from the origin, with no filling defect more proximally in the coeliac trunk (see Figure 1).

Vascular surgical opinion was sought, and the patient was administered 5000 IU of intravenous heparin with resolution of the pain within several hours. No endovascular intervention was required. Following discussion with interventional radiology, appearances were thought more in keeping with arterial dissection rather than embolism. The patient was anticoagulated with enoxaparin at 100 mg twice daily for six weeks. He developed a hematoma of the knee which was aspirated and subsequently managed conservatively. Follow-up CT angiogram performed four weeks after the event demonstrated persistent proximal SMA dissection and proximal stenosis due to emboli.
to thrombus, with persistent jejunal and ileal arterial narrowing without occlusion. No arterial dissection flaps were evident and no aneurysmal change developed on follow-up ultrasounds five and nine months later (see Figure 2). He remained symptom free after the initial event requiring heparinization.

Investigations for Factor V Leiden mutation found no thrombophilia, nor any cardiac conditions to suggest an embolic source. Abdominal ultrasound performed five months later found the superior mesenteric artery to be narrowed to 7.2 mm proximally, and tortuous but patent. The patient has had no further symptoms of mesenteric ischemia. He continues taking clopidogrel 75 mg daily due to the possibility of this being an embolic event, although he has no arrhythmia or structural cardiac defect.

Review of Literature

Laparoscopy-associated mesenteric vascular events are described in the literature following various procedures, with both mesenteric arterial and portal venous occlusion described. Seventeen cases of mesenteric arterial occlusion following laparoscopic surgery are previously described in the literature (see Table 1). Mesenteric arterial events are described following laparoscopic cholecystectomy, fundoplication, inguinal and midline hernia repair, adhesiolysis and uterine myolysis, gastric banding, and retroperitoneoscopic lumbar sympathectomy. This condition is most common following laparoscopic cholecystectomy, such that some authors postulate that biliary colic could be a misdiagnosis in patients whose symptoms are due to chronic mesenteric ischemia.

Few cases have been recognized clinically within an appropriate timeframe to enable anticoagulation and/or endovascular intervention to avoid significant necrosis of bowel and other intra-abdominal organs. Outcomes vary from recovery without morbidity to fulminant organ failure and death. Only four cases of survival following this event are described in the literature, one of whom subsequently died of sepsis in the setting of long-term parenteral nutrition. The remaining cases died of sepsis and multiorgan failure, resulting in an 82% (14/17) mortality rate of this condition. The current case is the first documented incidence of mesenteric arterial ischemia where laparotomy and resection of necrotic bowel was not required, excluding those patients who were moribund and did not proceed to operation.

Figure 2. Follow-up ultrasound imaging of the same view of the superior mesenteric artery as figure 1. No dissection flap or thrombus is present. Superior mesenteric artery caliber measures 5.8-millimeters (yellow ruler).
| Authors | Year | Journal | Operation | Age (Yrs) | Insufflation pressures (mmHg) | Duration of operation (mins) | Prophylactic anticoagulants | Onset of pain | Investigation | Location of vascular occlusion | Ischaemic areas | Treatment | Outcome | Coagulopathy | Other factors |
|---------|------|---------|------------|-----------|-------------------------------|-----------------------------|-----------------------------|---------------|--------------|-----------------------------|----------------|-----------|---------|--------------|---------------|
| Paul et al. | 1994 | British Journal of Surgery | Laparoscopic cholecystectomy | 68 | 12 – 15 | 88 | Unknown | Unknown | Laparotomy | ileum, right colon | Resection | Mortality | Unknown | HTN, weight loss | Previous MI |
| Jaffe, Russell | 1994 | British Journal of Surgery | Laparoscopic cholecystectomy | 76 | Unknown | 70 | Unknown | 3 days | Laparotomy | SMA | Jejunum, ileum, ascending colon | Palliation | Mortality | Unknown | Previous MI |
| Mitchell, Jameson | 1994 | Aust NZ J Surg | Nissen fundoplication | 55 | Unknown | 55 | Heparin | 24h | laparotomy | Coeliac trunk | Resection | Mortality | Unknown | Narrow coeliac trunk ostium 2.5 mm |
| Schorr | 1995 | Journal of Laparoendoscopic Surgery | Laparoscopic cholecystectomy | 62 | Unknown | 40 | Unknown | 2 days | Autopsy | SMA | Jejunum, ileum, ascending colon | Unexpected death in hospital | Mortality | Unknown | Autoimmune haemolytic anaemia, splenectomy, hypothyroidism, Guillain Barre syndrome |
| Richmond et al. | 1997 | Journal of Laparoendoscopic and Advanced Surgical Techniques | Laparoscopic cholecystectomy | 90 | 15 | Unknown | Unknown | < 12h | Laparotomy, angiography | SMA, IMA | Resection | Late mortality | Unknown | Cocaine abuse, weight loss |
| Sternberg et al. | 1998 | Journal of Laparoendoscopic and Advanced Surgical Techniques | Laparoscopic cholecystectomy | 60 | 14 | Unknown | Heparin | 3 – 4 days | laparotomy | SMA | Fourth part of duodenum, jejunum, ileum, right colon | Palliation | Mortality | Unknown | Family Hx CAD and CVA |
| Andrei et al. | 1999 | Digestive Surgery | Laparoscopic cholecystectomy | 72 | 15 | Unknown | 50 | Unknown | 24 – 48h | Laparotomy | Unclear | Small bowel | Palliation | Mortality | Unknown | CAD, CKD on dialysis |
| Bandyopadhyay, Kapadia | 2003 | Surgical Endoscopy | Laparoscopic inguinal hernia repair | 78 | Unknown | Unknown | Unknown | 1 day | Autopsy | BMA | Colon | Unexpected death in hospital | Mortality | Unknown | Infrarenal aortic aneurysm with thrombotic plaque |
| Husson et al. | 2004 | Journal of the Society of Laparoendoscopic Surgeons | Laparoscopic cholecystectomy | 54 | 15 | Unknown | Unknown | 4 days | Laparotomy | Unclear | Small bowel | Resection | Recovery | Unknown | |
| Bull et al. | 2006 | Journal of Minimal Access Surgery | Right retroperitoneoscopic lumbar sympathectomy | 88 | 15 | Unknown | 120 | Unknown | 5 hours | Laparotomy | Unclear | Small bowel, ascending and transverse colon | Palliation | Mortality | Unknown | PVD, ex-smoker, HTN |
| Iedec, Mitchell | 2006 | Journal of the Society of Laparoendoscopic Surgeons | Laparoscopic cholecystectomy | 57 | 12 – 15 | Unknown | Unknown | 1 day | Autopsy | Unclear | Small bowel | Death at home | Mortality | Unknown | Obesity |
| Wassenaar et al. | 2007 | Journal of the Society of Laparoendoscopic Surgeons | Laparoscopic hernia repair | 47 | 12 | Unknown | 240 | Unknown | 3 days | USS, laparoscopy, colonoscopy, angiography | SMA | Ascending and transverse colon | Resection | Mortality | Unknown | Obesity, PVD, HTN |
| Amulya et al. | 2009 | British Medical Journal Case Reports | Laparoscopic cholecystectomy | 60s | 12 | 50 | Unknown | 2 days | USS, laparotomy | Unclear | Jejunum, ileum | Resection | Mortality | Unknown | CAD, ex-smoker |
In many cases, the diagnosis of bowel or other visceral ischemia and necrosis was made at laparotomy performed in the setting of acute deterioration and sepsis, and in several cases was deemed nonsurvivable at laparotomy.\textsuperscript{2,5,6,10,15} Table 1 outlines areas of ischemia at operation, with location of arterial occlusion if known. The location of occlusion was identified by angiogram in only two cases,\textsuperscript{4,13} in the remainder the location was judged based on pulsation of vessels at laparotomy. Pre-existing vascular risk factors of coronary or peripheral vascular disease were present in 6 of 17 previous cases. Weight loss prior to surgery was noted in two cases and has been hypothesized to suggest possible pre-existing chronic mesenteric ischemia.\textsuperscript{4,6}

Spontaneous isolated superior mesenteric artery dissection (SIS-MAD) is described in the literature, with risk factors including hypertension, tobacco smoking, connective tissue disorders, vasculitis, and atherosclerosis.\textsuperscript{16,17} Due to its typical location 1–3 cm from the ostium of the SMA, some authors suggest that shear stress at the transition point between fixed retropancreatic and mobile mesenteric segments contributes to the intimal tear of dissection.\textsuperscript{18} Varying clinical presentations exist, from minor lumen narrowing without bowel ischemia, to complete occlusion with bowel necrosis requiring resection.\textsuperscript{17}

Our patient’s very recent laparoscopic surgery suggests that his SMA dissection was likely related to pathophysiological mechanisms during laparoscopy as described below, rather than those underlying SIS-MAD. Regardless of the exact cause, whether due to dissection or thromboembolism, this patient experienced acute SMA occlusion, with risk of bowel necrosis if not recognized and treated appropriately.

**Mechanisms**

Proposed mechanisms underlying mesenteric vascular events during laparoscopy include changes in splanchnic blood flow due to mechanical compression from pneumoperitoneum, reduced cardiac output and systemic venous return, and hypercapnia causing mesenteric vasoconstriction.

At laparoscopy pressures of 14–16 mmHg, cardiac output may be decreased by 30\%, and systemic vascular resistance increased by 65\%, thus increasing afterload.\textsuperscript{4,19–23} In a canine model using pneumoperitoneum at a pressure of 16 mmHg, a reduction in blood flow of up to 30\% was seen in the superior mesenteric artery and portal vein.\textsuperscript{21,23} This reduction was noted to be gradual, progressive over
time, and greater than that expected for the decrease in cardiac output alone, suggesting other local factors affecting blood flow.21,25

Decreased splanchnic blood flow is postulated to be caused in part by direct mechanical compression of veins by insufflation pressures, causing increased vascular resistance in splanchnic veins, with an upstream effect on arterial flow.21

Human studies using a laser Doppler flow probe to measure organ perfusion during laparoscopic cholecystectomy found that increasing insufflation pressure from 10 to 15 mmHg reduced blood flow to the stomach by 40%, duodenum by 11%, jejunum by 32%, colon by 44%, and liver by 39%.23

Splanchnic vasoconstriction has been shown to be mediated by vasopressin causing mesenteric and renal vascular constriction in response to increased intra-abdominal pressure and peritoneal stretch.21,23,25 Compression of venous outflow in mesenteric vessels triggers an intrinsic myogenic response resulting in vasoconstriction.21,25

A porcine study demonstrated that pneumoperitoneum performed with carbon dioxide initially caused mesenteric hyperemia at lower pressures, but at pressures of 8–12 mmHg mesenteric arterial and venous blood flow decreased as a result of arterial resistance and reduced cardiac output.23,26

Carbon dioxide insufflation causes an increase in arterial pressure of CO2 due to diffusion across the peritoneum and absorption into the systemic circulation. This results in decreased tissue pH, causing vasoconstriction, increased splanchnic vascular resistance, and reduced mesenteric and hepatic perfusion.23,26

Varying measures of oxidative stress have been investigated in human and animal studies to determine the effect of pneumoperitoneum in causing ischemia-reperfusion injury. There is no consensus on the validity of different markers of oxidative stress or measurement of these, so it is unclear whether pneumoperitoneum for laparoscopy causes more oxidative stress than similar open procedures. Animal studies have suggested that pneumoperitoneum causes end-organ reperfusion injury in livers and kidneys of rats.23,28,29 Other animal studies in rabbits showed similar markers of oxidative stress following both open and retroperitoneoscopic procedures.23,30 Some human studies demonstrated higher markers of oxidative stress following open cholecystectomy than laparoscopic cholecystectomy, conflicting with animal model results.23

**DISCUSSION**

This case is the first reported instance of mesenteric arterial ischemia following laparoscopic surgery where early intervention avoided laparotomy and bowel resection, or mortality. This demonstrates that even in cases of significant vascular occlusion with bowel ischemia, prompt recognition and anticoagulation can prevent bowel necrosis.

Strategies to decrease organ hypoperfusion and oxidative stress in laparoscopic procedures are numerous with varied evidence for their success. These include use of lowest possible insufflation pressure, starting with 12–15 mmHg and decreasing to the lowest pressure where visibility is still adequate.23 However, there are reports of vascular occlusion occurring even with insufflation pressures of 12–15 mmHg.4 Intermittent desufflation of the abdomen during the procedure has been suggested.1,4,23 Where possible, reverse Trendelenburg or other head-up positions should be minimized to avoid further reduction in venous return.23 Intermittent pneumatic calf compression devices increase venous return in addition to decreasing venous thrombosis risk.23 Maintaining adequate ventilation during anesthesia may reduce the effect of hypercapnia on splanchnic blood flow.4

Due to the absence of previously reported instances of provoked arterial dissection following laparoscopy, there are no recommendations for its prevention. Indeed, prevention of all future laparoscopy-associated mesenteric vascular events is likely unrealistic, due to the paucity of evidence that cases are limited to those patients with coagulopathy or vasculopathy risk factors, undergoing particular procedures, or with use of particularly high insufflation pressures.

If it is accepted that rare instances of laparoscopy-associated mesenteric vascular events will continue to occur, attention must then shift to early recognition to enable prompt treatment and prevent bowel necrosis and multiorgan failure. We advocate the use of early CT angiography to investigate this possible complication in patients with pain that is greater than expected after laparoscopic surgery.11 If mesenteric arterial ischemia is diagnosed, treatment should be undertaken in consultation with a vascular surgical service. Due to its rarity, no recommendations exist for management of laparoscopy-provoked SMA dissection, therefore we may look to treatment of SIS-MAD for guidance. Previous recommendations on treatment of laparoscopy-associated mesenteric vascular events are based on the paradigm that most patients already have necrotic bowel requiring resection, possibly with associated multiorgan failure.
Conservative management has been successful in many cases of SIS-MAD and consists of heparin anticoagulation, bowel rest, and antihypertensives as required. This is appropriate if there is no evidence of ruptured SMA branches or bowel ischemia. Indications for endovascular or open revascularization for SIS-MAD include evidence of intestinal ischemia; progression of dissection and aneurysm size; narrowing or thrombosis of the true lumen; or saccular aneurysm formation at risk of rupture or embolization. Endovascular intervention has the advantage of being minimally invasive and may be more appropriate for patients with subacute symptoms or significant medical comorbidities. Patients who undergo endovascular stenting may require anticoagulation in addition to dual antiplatelet therapy for a minimum of six months, with single agent antiplatelet cover thereafter, although consensus is lacking. Open surgical intervention is necessary for any patients requiring assessment of bowel viability or resection of necrotic bowel.

Regarding laparoscopy-associated mesenteric vascular events, improved survival is seen in patients <60 years of age, and those who undergo bowel resection. Second-look laparotomy is advocated by several authors, with 38%-39% of patients undergoing further bowel resection at second laparotomy indicating significant delay in demarcation of viable bowel. Richmond et al. advocate standardized reporting of all cases of “laparoscopy-associated mesenteric vascular events.”

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