CASE REPORT CRSLS

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."[1] 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.

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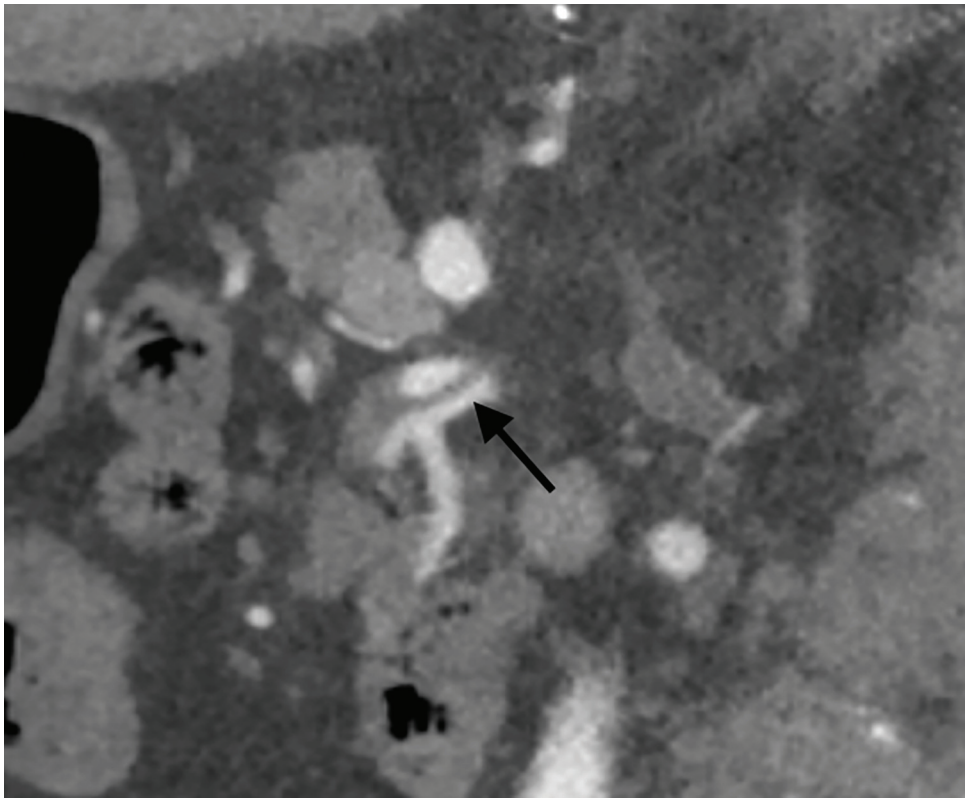

Figure 1. Computed tomography angiogram during acute presentation demonstrates dissection of superior mesenteric artery with flow in the true lumen inferiorly (***arrow***) filling the inferior pancreaticoduodenal artery. Some flow is evident superiorly in the false lumen above the dissection flap, with thrombosis distally.

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 hemarthrosis 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

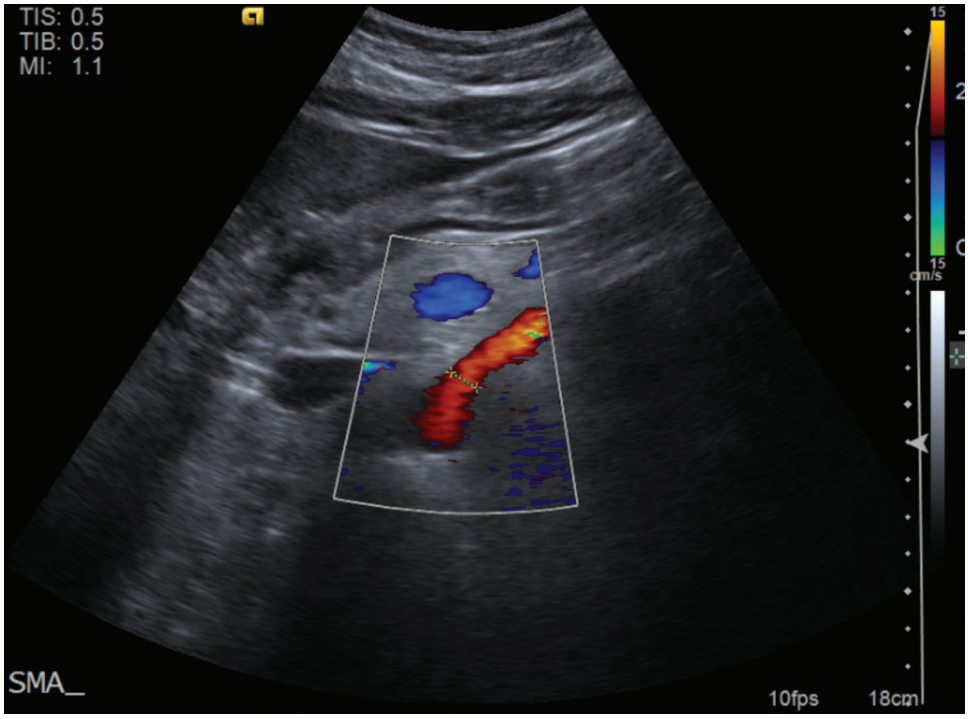

Figure 2. Follow-up ultrasound imaging of the same view of the superior mesenteric artery as figure 1. No dissection flap or thrombus is present. Superior mesenteric artery caliber measures 5.8-millimeters (***yellow ruler***).

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 arrythmia 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,[1–10] fundoplication,[11] inguinal[12] and midline hernia[13] repair, adhesiolysis and uterine myolysis,[14] gastric banding,[10] and retroperitoneoscopic lumbar sympathectomy.[15] 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.[4]

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,[10,14] one of whom subsequently died of sepsis in the setting of long-term parenteral nutrition.[4] 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.

Table 1.
Previously Documented Cases of Mesenteric Arterial Occlusion in the Literature

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	utcome	Coagulopathy	Other factors
Paul et al.	1994	British Journal of Surgery	Laparoscopic cholecystectomy	68	12 – 15	85	Unknown	Unknown	Laparotomy	Unclear	Ileum, right colon	Resection	Mortality	Unknown	HTN, weight loss
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, Jamieson	1994	Aust NZ J Surg	Laparoscopic Nissen fundoplication	55	Unknown	55	Heparin	24h	laparotomy	Coeliac trunk	Lower oesophagus, proximal stomach. Later stomach, gallbladder, spleen, small bowel, right colon	Resection	Mortality	No	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	30	15	Unknown	Unknown	< 12h	Laparotomy, angiography	SMA, IMA	Jejunum, proximal ileum	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	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	IMA	Colon	Unexpected death in hospital	Mortality	Unknown	Infrarenal aortic aneurysm with thrombotic plaque
Hasson et al.	2004	Journal of the Society of Laparoendoscopic Surgeons	Laparoscopic adhesiolysis and myolysis	34	15	Unknown	Unknown	4 days	Laparotomy	Unclear	Small bowel	Resection	Recovery	Unknown	
Rulli et al.	2006	Journal of Minimal Access Surgery	Right retroperitoneoscopic lumbar sympathectomy	88	15	120	Unknown	3 hours	Laparotomy	Unclear	Small bowel, ascending and transverse colon	Palliation	Mortality	Unknown	PVD, ex-smoker, HTN
Leduc, Mitchell	2006	Journal of the Society of Laparoendoscopic Surgeons	Laparoscopic cholecystectomy	57	12 – 15	Unknown	Unknown	1 day ('shortly afterwards')	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	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

Table 1. Continued

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	utcome	Coagulopathy	Other factors
Shaikh et al.	2011	Journal of Emergencies, Trauma and Shock	Laparoscopic cholecystectomy	43	12 – 15	45	LMWH	2 days	CT abdo	SMA	Terminal ileum	Resection	Mortality	Unknown	
Al-Khyatt et al.	2013	Journal of Medical Case Reports	Laparoscopic gastric banding	52	15	70	LMWH, SCDs	5 days	CT abdo	Unclear	Right colon	Resection	Recovery	Unknown	Obesity, diabetes, HTN, OSA
			Laparoscopic cholecystectomy	82	12	45	LMWH, SCDs	5 days	CT abdo	SMA	Stomach, small bowel, right colon	Palliation	Mortality	Unknown, on aspirin for TIA	HTN, TIA
			Laparoscopic cholecystectomy converted to open	58	12	50	LMWH, SCDs	3 days	Laparotomy	Unclear	Terminal ileum, right colon	Rsection	Recovery	Unknown	HTN, diabetes
Collinson et al.	2022	N/A	Laparoscopic partial fundoplication	62	17	120	LMWH, SCDs, enoxaparin	4 days	CT angiogram abdo	SMA	None demarcated	Intravenous heparin	Recovery	None	Obesity, diabetes

Abbreviations: HTN, hypertension; MI, myocardial infarction; SMA, superior mesenteric artery; Hx, history; CAD, coronary artery disease; CVA, cerebrovascular accident; IMA, inferior mesenteric artery; PVD, peripheral vascular disease; USS, ultrasound scan; LMWH, low molecular weight heparin; SCDs, sequential compression devices; CT abdo; computed tomography of the abdomen; TIA, transient ischaemic attack.

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.[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,[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.[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.[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.[18] Varying clinical presentations exist, from minor lumen narrowing without bowel ischemia, to complete occlusion with bowel necrosis requiring resection.[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.[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.[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.[21,23]

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%.[23,24]

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.[21,23]

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 CO_2 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.[23,27]

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.[17,31] 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.[31] Endovascular intervention has the advantage of being minimally invasive and may be more appropriate for patients with subacute symptoms or significant medical comorbidities.[32] 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.[33] Open surgical intervention is necessary for any patients requiring assessment of bowel viability or resection of necrotic bowel.[32]

Regarding laparoscopy-associated mesenteric vascular events, improved survival is seen in patients < 60 years of age, and those who undergo bowel resection.[34] 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.[32,34] Richmond et al. advocate standardized reporting of all cases of "laparoscopy-associated mesenteric vascular events."[35]

References:

1. Paul A, Troidl H, Peters S, Stuttmann R. Fatal intestinal ischaemia following laparoscopic cholecystectomy. *Br J Surg.* 1994;81(8):1207–1207.

2. Jaffe V, Russell RCG. Fetal intestinal ischaemia following laparoscopic cholecystectomy. *Br J Surg.* 1994;81(12): 1827–1828.

3. Schorr RT. Laparoscopic upper abdominal operations and mesenteric infarction. *J Laparoendosc Surg.* 1995;5(6):389–392.

4. Richmond BK, Lucente FC, Boland JP. Laparoscopy-associated mesenteric vascular events: Description of an evolving clinical syndrome. *J Laparoendosc Adv Surg Tech A.* 1997;7(6): 363–367.

5. Sternberg A, Alfici R, Bronek S, Kimmel B. Laparoscopic surgery and splanchnic vessel thrombosis. *J Laparoendosc Adv Surg Tech A.* 1998;8(2):65–68.

6. Andrei VE, Schein M, Wise L. Small bowel ischemia following laparoscopic cholecystectomy. *Dig Surg.* 1999;16(6):522–524.

7. Leduc LJ, Mitchell A. Intestinal ischemia after laparoscopic cholecystectomy. *JSLS.* 2006;10(2):236–238.

8. Amulya CB, Rajagopalan V, Jyothiprakash AM, Chandrashekar R, Belagavi CS. Fatal small bowel ischaemia following laparoscopic cholecystectomy: report of a case. *BMJ Case Rep.* 2009; 2009.

9. Shaikh N, Rahman HA, Hanssens Y, John S. A rare complication of laparoscopic surgery. *J Emerg Trauma Shock.* 2011; 4(3):418–420.

10. Al-Khyatt W, Thomas JD, Humes DJ, Lobo DN. Intestinal ischemia following laparoscopic surgery: a case series. *J Med Case Rep.* 2013;7(25):25.

11. Mitchell PC, Jamieson GG. Coeliac axis and mesenteric arterial thrombosis following laparoscopic nissen fundoplication. *Aust N Z J Surg.* 1994;64(10):728–730.

12. Bandyopadhyay D, Kapadia CR. Large bowel ischemia following laparoscopic inguinal hernioplasty. *Surg Endosc.* 2003; 17(3):520–521.

13. Wassenaar EB, Raymakers JT, Rakic S. Fatal intestinal ischemia after laparoscopic correction of incisional hernia. *JSLS.* 2007;11(3):389–393.

14. Hasson HM, Galanopoulos C, Langerman A. Ischemic necrosis of small bowel following laparoscopic surgery. *JSLS.* 2004;8(2):159–163.

15. Rulli F, Galatà G, Micossi C, Dell'Isola C. Massive intestinal infarction following retroperitoneoscopic right lumbar sympathectomy. *J Minim Access Surg.* 2006;2(4):222–223.

16. Kang TL, Teich DL, McGillicuddy DC. Isolated, spontaneous superior mesenteric and celiac artery dissection: case report and review of literature. *J Emerg Med.* 2011;40(2).

17. Dou L, Tang H, Zheng P, Wang C, Li D, Yang J. Isolated superior mesenteric artery dissection: CTA features and clinical relevance. *Abdom Radiol (NY).* 2020;45(9):2879–2885.

18. Park YJ, Park CW, Park KB, Roh YN, Kim DI, Kim YW. Inference from clinical and fluid dynamic studies about underlying cause of spontaneous isolated superior mesenteric artery dissection. *J Vasc Surg.* 2011;53(1):80–86.

19. Caldwell CB, Ricotta JJ. Changes in visceral blood flow with elevated intraabdominal pressure. *J Surg Res.* 1987; 43(1):14–20.

20. Ishizaki Y, Bandai Y, Shimomura K, Abe H, Ohtomo Y, Idezuki Y. Safe intraabdominal pressure of carbon dioxide pneumoperitoneum during laparoscopic surgery. *Surgery.* 1993;114(3):549–554.

21. Ishizaki Y, Bandai Y, Shimomura K, Abe H, Ohtomo Y, Idezuki Y. Changes in splanchnic blood flow and cardiovascular effects following peritoneal insufflation of carbon dioxide. *Surg Endosc.* 1993;7(5):420–423.

22. Westerband A, Van de Water JM, Amzallag M, et al. Cardiovascular changes during laparoscopic cholecystectomy. *J Am Coll Surg*. 1992;175(6):535–538.

23. Sammour T, Mittal A, Loveday BPT, et al. Systematic review of oxidative stress associated with pneumoperitoneum. *Br J Surg*. 2009;96(8):836–850.

24. Schilling MK, Redaelli C, Krahenbuhl L, Signer C, Buchler MW. Splanchnic microcirculatory changes during CO2 laparoscopy. *J Am Coll Surg*. 1997;184(4):378–382.

25. Punnonen R, Viinamäki O. Vasopressin release during laparoscopy: role of increased intra-abdominal pressure. *Lancet*. 1982;319(8264):175–176.

26. Blobner M, Bogdanski R, Kochs E, Henke J, Findeis A, Jelen-Esselborn S. Effects of intraabdominally insufflated carbon dioxide and elevated intraabdominal pressure on splanchnic circulation: an experimental study in pigs. *Anesthesiology*. 1998;89(2):475–482.

27. Epstein RM, Wheeler HO, Frumin MJ, Habif DV, Papper EM, Bradley SE. The effect of hypercapnia on estimated hepatic blood flow, circulating splanchnic blood volume, and hepatic sulfobromophthalein clearance during general anesthesia in man. *J Clin Invest*. 1961;40(3):592–598.

28. Akbulut G, Polat C, Aktepe F, et al. The oxidative effect of prolonged CO2 pneumoperitoneum on renal tissue of rats. *Surg Endosc*. 2004;18(9):1384–1388.

29. Akbulut G, Serteser M, Polat C, et al. Changes in tissue-oxidative stress markers in an experimental model of laparoscopic donor nephrectomy. *Transplantation*. 2002;74(12):1768–1772.

30. Demirbas M, Samli M, Aksoy Y, Guler C, Kilinc A, Dincel C. Comparison of changes in tissue oxidative-stress markers in experimental model of open, laparoscopic, and retroperitoneoscopic donor nephrectomy. *J Endourol*. 2004;18(1):105–108.

31. Subhas G, Gupta A, Nawalany M, Oppat WF. Spontaneous isolated superior mesenteric artery dissection: a case report and literature review with management algorithm. *Ann Vasc Surg*. 2009;23(6):788–798.

32. Ryer EJ, Kalra M, Oderich GS, et al. Revascularization for acute mesenteric ischemia. *J Vasc Surg*. 2012;55(6):1682–1689.

33. Xu Y, Li X, Shang D, et al. Mid-term outcomes of symptomatic isolated superior mesenteric artery dissection with endovascular management. *Vascular*. 2021;29(2):301–310.

34. Park WM, Gloviczki P, Cherry KJ, et al. Contemporary management of acute mesenteric ischemia: factors associated with survival. *J Vasc Surg*. 2002;35(3):445–452.

35. Richmond BK, Thalheimer L. Laparoscopy associated mesenteric vascular complications. *Am Surg*. 2010;76(11):1177–1184.