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Ischemic Necrosis of Small Bowel Following Laparoscopic Surgery

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ABSTRACT

Background and Objective: Small bowel ischemia following laparoscopy was described recently as a rare fatal complication of the CO_2 pneumoperitoneum. Of the 8 cases reported in the surgical literature, 7 were fatal, 1 was not. In this report, we describe the first gynecological case.

Methods: A 34-year-old woman who underwent laparoscopy with extensive adhesiolysis and myolysis was re-admitted with an acute abdomen on postoperative day 4. Immediate laparotomy revealed acute peritonitis, extensive adhesions, and a 3-cm defect in the small bowel. Tissue examination showed ischemic necrosis of edematous, but essentially normal, bowel mucosa. The postoperative course was extremely complicated. She was discharged after a 2-month hospital stay in the intensive care unit for rehabilitation.

Results: Data are available on 7 patients (including ours). All procedures were described as uneventful. The intraabdominal pressure was set at 15 mm Hg when specified. Some abdominal pain occurred in all, nausea and vomiting in 4, diarrhea in 2, abdominal distention in 1, fever in none. Quick reintervention laparotomy was performed in 2 and delayed in 5 (up to 4 days).

Discussion: The CO_2 pneumoperitoneum is a predisposing factor for intestinal ischemia as it reduces cardiac output and splanchnic blood flow. However, critical ischemia relies on underlying vasculopathy or an inciting event.

Conclusion: Patient selection, maintaining intraabdominal pressure at 15 mm Hg or less, and intermittent decompression of the gas represent the best options for preventing this complication.

Key Words: Small bowel ischemia, Pneumoperitoneum.

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INTRODUCTION

Laparoscopic surgery has become the standard of care for many gynecologic conditions, and the laparoscopic technique is recognized as being safe and effective. However, recent case reports in the surgical literature describe small bowel ischemia as a rare fatal complication of the CO_2 pneumoperitoneum.[1-7] Andrei et al[7] reviewed the literature on small bowel ischemia in 1999 and added 1 case that occurred following laparoscopic cholecystectomy. Their review yielded 5 cases following laparoscopic cholecystectomy[1-3,5,6] and 1 after laparoscopic Nissen fundoplication.[4] These authors also reported a personal communication of another case following laparoscopic cholecystectomy.[4] Of the 8 cases in the literature, 7 were fatal, 1 was not.

The CO_2 pneumoperitoneum appears to be a predisposing factor in the development of intestinal ischemia because it compromises the mesenteric circulation through decreased cardiac output and mechanical reduction of the blood flow due to increased intraabdominal pressure, and humoral vasoconstriction.[6-10] However, as we will describe, progression to critical ischemia seems to rely on underlying vasculopathy or an inciting event.

In this report, we describe the first case in gynecology of small bowel ischemic necrosis following laparoscopic surgery. Our intention is to increase awareness about this catastrophic complication and to examine the literature for possible causes and preventive measures.

CASE REPORT

A 34-year-old African-American woman, gravida 3, para 1, presented to the first author with a newly developed 4- to 5-cm left uterine mass associated with menorrhagia and pelvic pain of several months duration. The mass was thought to be a myoma. Ultrasound showed multiple small myomas. A diagnostic laparoscopy with possible myomectomy was planned pending a second opinion consult and medical clearance. Past surgical history included laparotomy with left oophorectomy for ovarian cysts and 2 elective terminations of pregnancy, the last of which may have been associated with an inflammatory process that contributed to development of the pelvic

mass not appreciated during previous pelvic examinations. Past medical history included asthma and use of nonsteroidal anti-inflammatory drugs for pain. Physical examination revealed bradycardia and a small thyroid nodule. A laboratory workup showed mild iron deficiency anemia (hemoglobin 11.6 g/dL; hematocrit 35.3%) and inconclusive thyroid studies with the patient being clinically euthyroid.

At laparoscopy, the open entry method was used. Adherent small bowel loops were noted at the entry site requiring careful dissection. With the establishment of an adequate pneumoperitoneum at a pressure of 15 mm Hg, exploration of the abdomen revealed extensive adhesions. Careful port placement and adhesiolysis using disposable scissors were performed to expose the pelvic organs. The uterus was intimately adherent to the sigmoid colon. Two uterine bulges consistent with myoma or adenomyoma were exposed and palpated. Because of safety considerations, myolysis was selected as the procedure of choice with serial bipolar needle punctures placed into the perceived junction between each lesion and uterus as well as in its dome. The abdomen was lavaged with copious amounts of saline, leaving behind a generous amount to minimize adhesion reformation. The operative time was 75 minutes, the anesthetic inhalation agents used were sevoflurane and nitrous oxide, and the estimated blood loss was <30 mL. The patient was discharged on the same day of surgery.

The first author kept in contact with the patient on a daily basis because of the extensive dissection. Her postoperative recovery appeared to be uneventful for the first 3 days. She was afebrile throughout, voiding well, and tolerating oral intake on the first postoperative day. She passed flatus on the second day and had a small bowel movement on the third day. She had a bloody drainage from the incision attributed to hydroflation on day 1 and a black and blue mark around the umbilicus on day 2, probably representing a bruise from surgical dissection. She also reported some abdominal pain and distention mostly during the first 2 days. However, on the morning of the fourth postoperative day, the patient reported that she had vomited and was told to go to the emergency room immediately.

The patient presented to the emergency room several hours later and was met by the first author as she was being admitted. An acute abdomen was diagnosed, and the surgical team mobilized for an immediate laparotomy. Shortly thereafter, the patient experienced a sudden onset of atrial fibrillation and hypotension. A triple lumen catheter was placed, and the patient was taken to the operating room for exploratory laparotomy. The second author performed surgery. Enteric fluid was noted to exit from the umbilical port site while the patient was being prepped and draped. A midline laparotomy was performed. Gross spillage of enteric fluid was noted as well as extremely dense adhesions between the bowel and anterior abdominal wall mostly in the midline. Extensive mobilization of the small bowel was carried out. An area of perforated bowel seeping fluid was noted along the midline. An incidental enterotomy occurred during the adhesiolysis. The affected bowel was resected and a Prasad double barrel ileostomy was done. Mobilization of the bowel was difficult and required repeated releasing incisions due to foreshortening of the mesentery.

The pathology report showed a 3-cm defect in the center of the resected bowel associated with changes consistent with ischemic necrosis. The bowel mucosa was slightly edematous but otherwise grossly normal. For instance, no coagulation necrosis, inflammatory reaction, or granulation tissue was present.

Following surgery, the patient was transferred to the intensive care unit (ICU) and treated with broad-spectrum antibiotics and activated protein C (Xigris). Her 2-month ICU course was extremely difficult. It was complicated by candidemia, unsuccessful extubation trials, profound multiple neuropathy, nosocomial pneumonia, pressure ulcer, drug-induced thrombocytopenia, and gastrointestinal bleeding, precipitating shock and 2 cardiopulmonary arrests. Gastrostomy and tracheostomy tubes were placed, as well as an inferior vena cava filter when she developed a deep-vein thrombosis. She is currently in rehabilitation.

DISCUSSION

This case represents a rare catastrophic complication that is not attributable to operative laparoscopic manipulations. As with all previously reported 6 cases for whom detailed information was available,[1,2,4,5-7] a seemingly successful operative outcome was interrupted by a devastating complication. None of the procedures lasted longer than 85 minutes; 4 took less than 1 hour to complete. The procedures were variably described as elective, routine, uneventful, or without apparent complications. Extensive lysis of adhesions was carried out in our

case; multiple adhesions were also noted in another report.[1] Different anesthetic agents were utilized, sevoflurane in our case, isoflurane,[2,7] deslfurane,[6] and halothane1 in others. The intraabdominal pressure was set at 15 mm Hg in our case as well as in all communications specifying this parameter.[1,5,7]

The case reports are from different countries: 3 (including ours) from the USA,[6,7] 2 from Germany,[1,3] 2 from the UK,[2,5] 1 from Australia,[4] and 1 personal communication from an unspecified source.[4] In the USA, the length of stay following the laparoscopic procedure was consistent with an outpatient approach as opposed to in-patient hospitalization considered standard elsewhere. Our 34-year-old patient was discharged the same day of surgery and returned to the hospital on day 4 after having vomited and reporting a few other symptoms. She was reoperated upon for an acute abdomen almost immediately thereafter.

The 72-year-old reported by Andrei et al[7] was discharged on day 1 and returned to the hospital on day 8. Her symptoms consisted of diffuse abdominal pain associated with nausea, vomiting, and bloody diarrhea that started 1 or 2 days after the operation. She was reoperated upon on day 9, following marked deterioration in her general condition. The entire small bowel was found gangrenous; she expired 48 hours later.

Schorr's 62-year-old patient[6] was ready for discharge on day 1, but could not be because of family matters. While awaiting her family on day 2, she developed severe abdominal pain and tachycardia and was kept in the hospital. She suffered an unresponsive cardiac arrest while being investigated on day 3. Autopsy revealed infarction of the small bowel and colon to the hepatic flexure due to superior mesenteric artery thrombosis. The artery was not constricted by plaque, and no evidence was present of an embolic phenomenon anywhere.

The first case in the literature was reported by Paul et al[1] in Germany. Their 68-year-old patient, who was kept in the hospital, developed uncharacteristic abdominal pain and intermittent diarrhea. He was reoperated on on day 4 following increasing abdominal pain and general deterioration including renal failure. Gangrene of the ileum and right colon was managed with resection and primary anastomosis. Intraabdominal sepsis secondary to anastomotic breakdown led to several further reoperations. The patient died on day 45 from a septic complication.

The 76-year-old patient reported by Jaffe and Russell[2] from the UK was discharged on day 2 and readmitted on day 3 complaining of vague periumbilical discomfort. She was observed and investigated for 4 days and reoperated on on day 7 following rapid hemodynamic deterioration. The bowel was gangrenous from the duodenojejunal junction to the hepatic flexure. A diagnosis of superior mesenteric artery thrombosis was made, intestinal resection was not done, and the patient died 12 hours later.

The 55-year-old patient from Australia reported by Mitchell and Jamieson[4] developed on day 1 deep epigastric pain with nausea, retching, and itching. On day 2, she had fever and tachycardia with worsening pain and rash. She improved on day 3 but suffered sudden left subcostal pain at midnight and was operated on on day 4 following increasing abdominal pain and rigidity. The proximal stomach and esophagus were infarcted with full thickness necrosis of the esophagus. Resection and esophagogastric anastomosis was done. Subsequently, the patient underwent 7 reoperations, resecting ischemic tissue and ultimately died from overwhelming sepsis and hepatic infarction. Postmortem examination revealed congenital narrowing of the celiac artery, which contained an organized thrombosis. Other major vessels were normal without significant atheromatous disease.

The 36-year-old English patient reported by Dwerryhouse et al[5] was discharged on day 1. He returned to the hospital on day 2 complaining of colicky pain, abdominal distention, and vomiting. Ultrasound showed dilated fluid-filled loops of small bowel. Laparoscopy revealed dilated small bowel and a small amount of turbid peritoneal fluid. Laparotomy was performed. The terminal ileum was stenosed and thickened with associated fat wrapping suggestive of Crohn's disease. An ileocecal resection was performed, and the patient made an uneventful recovery. Histologic examination of the resected ileum showed features of a venous infarction with patchy mucosal necrosis.

The clinical presentations were similar in several aspects. Some abdominal pain was experienced by all patients, including ours, but was considered severe in only 2.[4,6] Nausea and vomiting occurred in 4 of the 7 patients, including ours,[4,5,7] diarrhea in 2,[1,7] and abdominal distention in 1 patient.[5] A febrile reaction was not present in any patient. Reintervention laparotomy was carried out in our patient as soon as she was admitted and on

the same day of readmission in Dwerryhouse et al's patient.[5] It was delayed in the other cases from 1 day[7] to 4 days[1,2] because of vague presentations and inconclusive investigations.

It is generally agreed that the CO_2 pneumoperitoneum is a predisposing factor for the development of intestinal ischemia because it causes the following physiologic alterations: a) decreased cardiac output due to increased systemic vascular resistance, decreased venous return, and elevated intrathoracic pressure,[8,9,11] b) significant reduction in the splanchnic blood flow, resulting from mechanical compression of the mesenteric veins, humoral vasoconstriction of the mesenteric bed and increased portal venous pressure caused by hypercapnia, local absorption of the CO_2, and increased release of vasopressin.[6-10] However, reduction in cardiac output and mesenteric blood flow associated with the CO_2 pneumoperitoneum should be insufficient to cause significant intestinal ischemia in healthy patients.

Critical intestinal ischemia following CO_2 pneumoperitoneum may be triggered by thrombosis of an arterial trunk,[1,2,4,6] acute compromise of the mesenteric circulation of undetermined nature,[7] or a focal lesion caused by a local insult[5] (present report). Arterial thrombosis or acute insufficiency causes gangrene or infarction, with or without full thickness necrosis, of the gastrointestinal segment supplied by the compromised arterial trunk. Superior mesenteric artery thrombosis was diagnosed clinically in 2 instances[1,2] and acute insufficiency in one.[7] These were attributed to atherosclerosis on the basis of age and history of cardiovascular disease. Arterial thrombosis noted at autopsy was deemed to be due to unknown causes in 1 patient,[6] and to congenital stenosis of the artery in another.[4] In our case and that of Dwerryhouse et al,[5] the triggering mechanism appears to have been related to a local insult: dense adhesions pulling against a foreshortened mesentery in our case and abnormal ileum suggestive of Crohn's disease in theirs. The severity of insult in our case caused transmural necrosis, consistent with the description of severe focal segmental ischemia provided by Greenwald et al.[12] The gravity of the clinical situation resulting from a 3-cm open defect in the small bowel may be compared to that associated with a grossly ruptured appendix. Free flow of intestinal contents into the peritoneal cavity would soon lead to generalized peritonitis and major third space losses into the peritoneum promptly resulting in dehydration, hemodynamic instability, and toxic shock.

The profound multiple neuropathy noticed in our patient represents a peculiar phenomenon that has been linked to bowel ischemia. Woodward et al[13] recently described a previously fit 23-year-old man who presented with a sudden onset of profound autonomic neuropathy of unknown origin and subsequently developed ischemic enterocolitis requiring colectomy and subtotal enterectomy.

The approach to this rare catastrophic complication should be directed to prevention rather than treatment. Analysis of the reported cases indicate that signs and symptoms existing prior to rapid patient deterioration were generally insufficient to warrant intervention by the reporting teams, and surgical management after the fact was frequently futile. However, a high index of suspicion and early evaluation by ultrasound followed by laparotomy may be helpful in improving survival rates. It is generally agreed that periodic decompression of the pneumoperitoneum gas while maintaining the intraabdominal pressure at 15 mm Hg or less represents the best available option for preventing this complication.[1,2,4,5,7,9] Patients in whom the splanchnic circulation is suspected of being impaired, based on preexisting atherosclerosis or other risk factors, should be handled with great care or offered an open laparotomy rather than the laparoscopy approach.

References:

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

2. Jaffe V, Russel RCG. Fatal intestinal ischemia following laparoscopic cholecystectomy [comment]. *Br J Surg.* 1994;81: 1827-1828.

3. Thiel H, Lang RD. Komplikationen nach 1000 laparoskopischen cholecystecktomien. Chirurg. 1994;65:795-800. Quoted by: Andrei VE, Schein M, Wise L. Small bowel ischemia following laparoscopic cholecystectomy. *Dig Surg.* 1999;16:522-524.

4. Mitchell PC, Jamieson GG. Coeliac axis and mesenteric arterial thrombosis following laparoscopic Nissen fundoplication. *Aus N Z J Surg.* 1994;64:728-730.

5. Dwerryhouse SJ, Melsom DS, Burton PA, Thompson MH. Acute intestinal ischaemia after laparoscopic cholecystectomy. *Br J Surg.* 1995;82:1413.

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

7. Andrei VE, Schein M, Wise L. Small bowel ischemia following laparoscopic cholecystectomy. *Dig Surg.* 1999;16:522-524.

8. Caldwell CB, Ricotta JJ. Changes in visceral blood flow with elevated intrabdominal pressure. *J Surg Res*. 1987;43:14-20.

9. Ishizaki I, Bandai I, Shimomura K, Abe H, Ohtome I, Idezuki I. Changes in splanchnic blood flow and cardiovascular effect following peritoneal insufflation of carbon dioxide. *Surg Endosc*. 1993;7:420-423.

10. Eleftheriadis E, Kotzampassik K, Botsios D, Tazartinoglou E, Farmakis H, Dadoukis J. Splanchnic ischemia during laparoscopic cholecystectomy. *Surg Endosc*. 1996;10:324-326.

11. Schein M, Wittmann DH, Aprahamian CC, Condon RE. The abdominal compartment syndrome: the physiologic and clinical consequences of elevated intra-abdominal pressure. *J Am Coll Surg*. 1995;180:745-753.

12. Greenwald DA, Brandt LJ, Reinus JF. Ischemic bowel disease in the elderly. *Gastroenterol Clin North Am*. 2001;30:445-473.

13. Woodward JM, Sanders DS, Keighley MR, Allan RN. Ischemic enterocolitis complicating idiopathic dysautonomia. *Gut*. 1998;43:285-287.