Bilateral Tension Pneumothoraces and Subcutaneous Emphysema Following Colonoscopic Polypectomy: A Case Report and Discussion of Anesthesia Considerations

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A 78-year-old man presented preoperatively with severe abdominal pain, dyspnea, and subcutaneous emphysema in his face, neck, and chest approximately 8 hours after colonoscopy with a sigmoid polypectomy. A pneumoperitoneum, free air in the mesentery, pneumoretroperitoneum, pneumomediastinum, and bilateral pneumothoraces were diagnosed using radiography and computed axial tomography. He emergently underwent an exploratory laparotomy with colostomy following bilateral chest tube placement. At laparotomy, a perforation of the posterior sigmoid colon was identified at the site of earlier polypectomy. The patient remained intubated and mechanically ventilated for 3 days postoperatively. Perforations of the colon during colonoscopies are the most serious complication of the procedure. Continued insufflation of air or carbon dioxide into a perforated colon can result in extraluminal gas that can result in life-threatening tension pneumothoraces.

This case examines the consequences of colonic perforation and the anesthetic management for the definitive surgical treatment of a posterior sigmoid wall perforation. Anesthesia providers' awareness of the risk factors for colonic perforation due to colonoscopy, early signs and symptoms of perforation, and knowledge of the surgical and anesthetic management of perforation could lead to early recognition and intervention and likely to improved patient outcomes.

Keywords: Colonic perforation, pneumomediastinum, pneumoretroperitoneum, polypectomy, tension pneumothorax.

As colonoscopy use continues to dramatically increase, and as a sharp rise in the use of anesthesia services for such procedures continues, awareness of its potential complications become essential.[1,2] Specifically, anesthesia provider participation rates for colonoscopies have increased from 8.6% in 2003 to 23.3% in 2007 with a projected rate of 38% by 2011.[1] Colonoscopy has become one the most common medical procedures performed, in part because of the successful promotion of colorectal cancer (CRC) screening and prevention guidelines begun in 1980 by the American Cancer Society, in addition to the growing evidence for its efficacy.[3,4] New guidelines state that a 50% to 90% reduction in the incidence of CRC may be achieved with a colonoscopy; and a screening colonoscopy every 10 years for average-risk adults beginning at 50 years of age.[3] Considering that CRC is the third most common cancer diagnosed and the second leading cause of cancer-related death in the United States, and that all other positive CRC screening tests require a colonoscopy as a second procedure, the increase in its use becomes apparent.[3]

However, colonoscopy has a CRC false-negative rate of 5% and as an invasive procedure poses risks for serious complications.[3] Perforation of the colon is considered one of the most serious complications of colonoscopy, as it can lead to peritonitis, shock, sepsis, and death.[5-9] Other complications include hemorrhage, pneumothorax, septicemia, mesenteric tears, vasovagal reactions, endocarditis, colonic volvulus, and splenic trauma.[3,5,7,8,10,11]

The risk for perforation with a colonoscopy has been particularly consistent among various studies to be 1 in 1,000 (0.1%) in screened patients but was found to be somewhat higher in a Medicare population at 1 in 500 (0.2%).[12,13] In diagnostic colonoscopies, the rate of perforation is 0.2% to 0.5%, but therapeutic colonoscopies have been reported to have a perforation as high as 2%.[2,14] Some of the predisposing risk factors for perforation are advanced age, prior abdominal or pelvic surgery, and previous therapeutic colonoscopies.[6,9,14]

Conventional surgical treatment options for colonic perforation include suturing of the perforation and resection of the perforated bowel segment with either primary anastomosis or creation of a colostomy.[13] A retrospective review of 258,248 colonoscopies, performed between 1980 and 2006, found that primary anastomosis can be safely performed in up to 67% of patients if the perforation is diagnosed within 24 hours.[15] In contrast, primary anastomosis is successful in only 17% of patients pre-

senting later than 24 hours, which emphasizes the importance of early recognition and treatment.[15]

In contrast to surgical correction, a retrospective review of 134,383 colonoscopies found that the mortality rate was 3 times higher for patients who underwent surgery for perforations rather than electing for conservative treatment.[16] However, conservative, nonoperative, management of colonic perforation is usually reserved for the most stable patients who have no signs of peritonitis.[11,15] Conservative nonsurgical treatment includes intravenous hydration, nasogastric suction, strict fasting, antibiotics, and frequent reassessment.[11,15] Unfortunately, most patients suffering a colonoscopic perforation will require an emergent laparotomy, which has an associated 36% morbidity rate and 7% mortality rate and will leave 38% with a permanent stoma.[15]

This case examines the consequences of colonic perforation (pneumoperitoneum, pneumoretroperitoneum, and bilateral pneumothoraces) and the anesthetic management for the definitive surgical treatment of a posterior sigmoid wall perforation.

Case Summary

A 78-year-old man, with a height of 180 cm, weighing 70 kg, arrived at our emergency department (ED) by ambulance. The patient reported having had a colonoscopy with sigmoid polypectomy that morning at another institution. The patient was discharged home following colonoscopy while experiencing increasing abdominal pain, with accompanied swelling of the neck and anterior chest. The patient reported experiencing increasing abdominal pain and distension; swelling of his face, neck, and chest; and shortness of breath over the last 8 hours since his discharge and arrival home.

At admission, his vital signs were as follows: noninvasive blood pressure, 138/81 mm Hg; heart rate, 89/min; respiratory rate, 18/min. He was afebrile but reported feeling feverish. Oxygen saturation was 100% on a nonrebreather mask. On physical examination in the ED, the patient was found to be awake, alert, and oriented to person, place, and time. Auscultation of the lungs revealed diminished breath sounds on the right side with a few rales on the left. There was generalized subcutaneous emphysema that was most prominent in the pectoral, neck, and facial areas. No respiratory distress, cyanosis, or airway compromise was apparent, and the patient demonstrated good air movement. Auscultation of the heart rate revealed a regular rate with an audible S_1 and S_2. The patient denied chest pain. The abdomen was soft and tender to touch, with some guarding in the bilateral lower quadrants. The patient had normal speech and normal gross motor function, and cranial nerves II though VII were grossly intact. An 18-gauge peripheral venous catheter in the right antecubital vein had previously been placed by emergency medical service personnel. The patient was infused with a total of 1.5 L of intravenous (IV) normal saline, 4.5 g of IV piperacillin and tazobactam (Zosyn), and 1 g of IV vancomycin.

Emergency department laboratory results showed the following values: white blood cells, 8.8 k/µL; hematocrit, 44.9%; hemoglobin, 16.3 g/dL; platelets, 196 k/µL; neutrophils, 60%; bands, 38%; lymphocytes, 1%; sodium (Na), 134 mmol/L; potassium (K), 3.1 mmol/L; chloride, 98 mmol/L; carbon dioxide, 26 mmol/L; anion gap, 10 mmol/L; serum urea nitrogen, 12 mg/dL; creatinine, 1 mg/dL; and blood glucose, 151 mg/dL. Results of liver function tests and amylase and lipase levels were all within normal limits. The international normalized ratio was 1.1; prothrombin time, 11.4 seconds; and partial thromboplastin time, 24 seconds. A blood gas analysis was not ordered.

Computed axial tomography of the abdomen without contrast medium was ordered on a stat basis and read by both the ED physician and radiologist. There were bilateral pneumothoraces evident, with free air in the mediastinum and in the peritoneal cavity as well as air in the mesentery and retroperitoneum. The retroperitoneal free air also appeared to be tracking up to the mediastinum. Portable radiography was performed, and the film confirmed bilateral pneumothoraces.

The on-call pulmonologist, surgeon, and anesthesiologist were then contacted and consulted. The surgeon determined that the patient needed an emergent exploratory laparotomy for repair of colonic perforation. Our patient reported having been told in the past that he was difficult to intubate, and thus endotracheal intubation was deferred to the operating room, where the anesthesia care team could best ensure a safe and successful outcome.

The pulmonologist placed chest tubes bilaterally at bedside in the ED. After informed consent was obtained, and with the use of local anesthesia with 1% lidocaine, a 28-French trocar catheter was inserted in the right side of the chest. A "whoosh of air" was noted to have been released upon insertion. The patient tolerated the procedure without complications. A 20-French trocar catheter was then inserted using local anesthesia with 1% lidocaine in the left side of the chest. The patient again tolerated the procedure without complications. A chest radiograph obtained using portable equipment revealed that the bilateral pneumothoraces had resolved. A closed drainage apparatus with a sterile water seal was connected to each chest tube without suction.

The anesthesia care team then interviewed and assessed the patient. The patient's airway was determined to be a Mallampati class III with adequate neck extension and an estimated 3-cm thyromental distance. He was declared an ASA physical status 4, and general anesthesia with an endotracheal tube was planned. A rapid sequence intubation and use of a video laryngoscope (GlideScope, Verathon, Bothell, Washington) were planned because of the patient's pneumoperitoneum and self-reported diffi-

cult airway, respectively. The patient reported an allergy to morphine. He takes carbidopa-levodopa for his Parkinson disease. He takes metoprolol and lisinopril for hypertension; simvastatin for hyperlipidemia; and isosorbide dinitrate, aspirin, and clopidogrel for atherosclerotic heart disease. His cardiac history included an inferior wall myocardial infarction and percutaneous transluminal coronary angioplasty (PTCA) with stents 4 years earlier. A heart catheterization 2 years ago demonstrated patency of the stents. An echocardiogram obtained 1 year ago showed an ejection fraction of 55% to 60%. He had a prostatectomy because of prostate cancer. He was a former smoker and drank alcohol occasionally.

The patient was then given IV midazolam, 1 mg, for anxiolysis and was transferred to the operating room for an exploratory laparotomy with a possible Hartmann procedure. The team made 4 U of cross-matched packed red blood cells available. All noninvasive monitors (blood pressure cuff, electrocardiogram, and pulse oximeter) were applied, and baseline vital signs were as follows: noninvasive blood pressure, 150/85 mm Hg; heart rate, 75/min; respirations, 20/min; and oxygen saturation, 100% on 10 L/min of oxygen by mask. The patient was induced for general anesthesia with a rapid sequence induction technique. Following preoxygenation, a smooth IV push induction was achieved using etomidate, 20 mg; fentanyl, 100 µg; and succinylcholine, 120 mg. With the administration of cricoid pressure, the GlideScope video laryngoscope was employed to facilitate endotracheal intubation with an 8.0-mm cuffed endotracheal tube, secured at 22 cm to the lips. General anesthesia was maintained with varying concentrations of isoflurane in oxygen at 2 L/min. A right radial arterial catheter was placed to optimize hemodynamic monitoring and blood gas analysis. A right subclavian multi-lumen central venous catheter was placed to monitor hydration status and to provide long-term, secure IV access.

A baseline arterial blood gas analysis was then performed with the following results: pH, 7.44; $PaCO_2$, 33 mm Hg; PaO_2, 411 mm Hg; Na, 134 mmol/L; K, 3.2 mmol/L; calcium, 1.0 mmol/L; glucose, 176 mg/dL; hematocrit, 41%; and bicarbonate 22.4 mmol/L.

Vecuronium was given for muscle relaxation before surgical incision and at intervals throughout the case to maintain paralysis. The surgeon performed a midline laparotomy and discovered a perforation of the posterior sigmoid colon. A Hartmann procedure was performed, wherein the perforated portion of the sigmoid colon was resected, resulting in a proximal-end colostomy and closure of the rectal stump. The Hartmann procedure allowed time for the inflammatory process to resolve and for improvement of adhesions before a Hartmann reversal, in which intestinal continuity would be reestablished.[17]

The patient was transferred to the intensive care unit with portable monitoring and Ambu bag and remained sedated on a propofol IV drip. He was successfully weaned from mechanical ventilation and extubated on postoperative day 3. On postoperative day 4 his chest tubes were removed without complications. On postoperative day 13 he was transferred to an inpatient rehabilitation facility, and 16 days later he was discharged home with home nursing care.

Three months later he returned to the hospital for a Hartmann reversal procedure, which was successfully performed without sequelae. After a short inpatient observation period, he was discharged home.

Discussion

The types of colonoscopies performed are classified according to their purpose. Screening colonoscopies are used to screen for CRC in asymptomatic patients who do not have a history of polyps or CRC.[18] Surveillance colonoscopies are repeat procedures, usually at 3-year intervals, for patients who have a history of polyps or other high risk factors for CRC.[19] Diagnostic colonoscopies are for determination of symptom etiology.[7] Therapeutic colonoscopies involve the use of biopsy and techniques for polypectomy.[7,14]

At least 3 mechanisms have been postulated to explain perforation during colonoscopies; they are mechanical, pneumatic, and/or therapeutic related.[14,20] Mechanical injury has been thought to be the cause of 32% to 62.8% of colonic perforations.[5,13] This mechanism of perforation occurs from the direct manipulation of the flexible fiberoptic colonoscope in the colon. For example, perforation of the colon may occur when a colonoscope is forcibly passed through sections of bowel kinked by adhesions, when passed through a diverticulum, from lateral forces of a colonoscope curving against a stretched loop of bowel, or from the colonoscope retroflexing.[2,11]

Pneumatic perforation occurs because of increasing insufflation pressures that cause disruption of the serosa, splitting of the muscularis propria, mucosal herniation, and mucosal rupture.[20] Insufflation of the colon with air or carbon dioxide is necessary to provide adequate visualization of the colonic lumen by separating the colonic walls.[21] The pressure at which colonic mucosal rupture can occur has been found to be 226 plus or minus 14 mm Hg, and as little as 80 mm Hg for cecal rupture.[2,11,20] Although most colonoscope insufflating machines are designed with a 50-mm Hg pressure limit, the volume of gas insufflated is not limited and averages as much as 8 L for a typical colonoscopy.[22]

Therapeutic-related perforation relates to the use of technological modalities to remove polyps or other intraluminal abnormalities during a colonoscopy. Some reports suggest that polypectomies confer the highest risk for perforation[2,5]; however, a recent retrospective study found that only 25.6% of perforations were attributed to polypectomy.[13] Perforations due to polypectomy are

Immediate decrease in arterial and mixed venous SpO_2	Pleuritic chest pain
Rapid reduction in cardiac output	Dyspnea
Tachycardia	Progressive respiratory failure
Increased peak inspiratory pressures	Tachypnea
Subcutaneous emphysema[a]	Tachycardia
Hypotension[b]	Decreased SpO_2
Sudden cardiac arrest[c]	Agitation

Table 1. Signs and Symptoms of Tension Pneumothorax in Ventilated Patients[23,24,27]

SpO_2 indicates oxygen saturation as measured by pulse oximetry.
[a] Subcutaneous emphysema was seen in 100% of cases of advanced tension pneumothorax in a case series of 71 ventilated patients.[27]
[b] Hypotension is considered a pre-terminal sign.[24]
[c] Sudden cardiac arrest is more common in ventilated patients than in awake patients.[23]

Table 2. Signs and Symptoms of Tension Pneumothorax in Awake and Spontaneously Breathing Patients[23,24]

SpO_2 indicates oxygen saturation as measured by pulse oximetry.

usually at the site of polyp removal, as is suspected to have occurred in our patient.[11]

Exploratory laparotomy is recommended for colonic perforations if peritoneal signs are present, the history is consistent with a large defect, and/or there is suspicion of poor bowel preparation.[11] Predictors of stoma formation are severe peritoneal contamination and the presence of malignant colonic neoplasms.[13] Additionally, the sigmoid colon is the most common site of perforation, and perforations are typically larger and more likely to require a colostomy because of the high risk for peritoneal contamination.[6,11,13,15] Our patient experienced a sigmoid colon perforation and had peritoneal signs of colonic perforation. Although he was hemodynamically stable, the extent of the pneumoperitoneum, pneumoretroperitoneum, and subcutaneous emphysema indicated a large colonic perforation with potential contamination, thus justifying his need for a laparotomy with colostomy.

The occurrence of a pneumoretroperitoneum and the resulting bilateral tension pneumothoraces in this case likely resulted from continued air insufflation following perforation of the posterior wall of the sigmoid colon at the site of polypectomy.[5] It is reasonable to postulate that the insufflated air traveled extraluminally and into the mesentery and then into the adjacent retroperitoneal space, and continued to travel along the tissue planes to the mediastinum, eventually rupturing the mediastinal pleura and resulting in intrapleural air causing bilateral tension pneumothoraces.[5,14] Alternatively, the high pressure of air in the peritoneal cavity could have passed through diaphragmatic fenestrations and entered the pleural spaces more directly.[14]

Tension pneumothorax (TPT) is typically thought to occur when a 1-way valve exists between the lung parenchyma (alveolar space) and the intrapleural space; in this scenario, air is added to the intrapleural space upon inspiration but is not released during expiration

with resulting tension.[23] However, Leigh-Smith and Harris[24] state, in their extensive literature review on TPT, that using intrapleural pressure rather than clinical definitions is more accurate. Their intrapleural pressure definition of TPT is particularly relevant to this case.[24] The tension pneumothoraces in our patient were in fact due to extraluminal air from insufflation of a perforated sigmoid colon that made its way to the negative pressure (–5 to –8 cm H_2O) of the intrapleural space, and not from air entering through damaged lung parenchyma to the intrapleural space. Such distinction of TPT etiology is an important anesthesia consideration. For example, if induction, intubation, and positive pressure ventilation were to precede decompression of a typical 1-way valve-type TPT, the TPT could be made immediately worse by adding air with positive pressure ventilation to the intrapleural space, causing life-threatening sequelae (Table 1).[23] One-way valve-type tension pneumothoraces can be very serious in ventilated patients, with mortality rates as high as 91%.[24] Such high mortality is thought to result from sudden cardiac arrest.[24-26] The elevated intrathoracic and intrapleural pressures result in compression of major vessels, leading to sudden and catastrophic preload and cardiac output reductions (see Table 1).[23,24,27]

The manifestations of a TPT in awake and spontaneously breathing patients (Table 2) and those in ventilated patients (see Table 1) are different and should be recognized early to avoid delay in treating life-threatening sequelae.[23,24,27]

Treatment of a TPT during a colonoscopy may include administration of 100% oxygen, discontinuation of any further gas insufflation of the colon, and, if the patient is hemodynamically unstable, immediate needle decompression followed by chest tube placement even before chest radiographs are obtained.[24] However, if the awake and spontaneously breathing patient is stable, chest radiographs are recommended for confirmation before decompression or chest tube placement.[24] For mechanically ventilated patients, it is recommended that immediate decompression should occur before radiography.[24]

Increased intra-abdominal pressure from a pneu-

moperitoneum can cause decreased compliance, increased airway pressures, V/Q mismatch, endobronchial intubation, subcutaneous emphysema, pneumomediastinum, pneumopericardium, and TPT.[28] A laparotomy will immediately decompress a pneumoperitoneum.[29] However, at surgical closure, increasing intra-abdominal pressure can manifest early as hypercarbia, high peak inspiratory pressures, and increased central venous and pulmonary occlusion pressures; late signs are hypotension and oliguria.[29,30] Anesthesia considerations entail measures to monitor and limit intra-abdominal pressure, including inserting a nasogastric tube for gastric decompression, avoiding steep Trendelenburg positioning, avoiding hypervolemia, and maintaining adequate muscle relaxation from incision to after closure.[29,31]

Additionally, avoiding the use of nitrous oxide (N_2O) should be an anesthetic consideration in patients with multiple pathologically air-filled spaces because of N_2O's potential to worsen such conditions.[32-34] Additionally, avoiding the use of N_2O during prolonged bowel procedures has been shown to limit bowel distension.[32]

Severe abdominal pain following a colonoscopy with pain radiating to the patient's shoulder should be carefully assessed because it could indicate perforation and the presence of free air in the peritoneum.[9,15] Dismissing abdominal discomfort or distension as expected symptoms inherent with colonoscopies could delay diagnosis and treatment of a possible perforation for several days.[11] It is important to note that less than 50% of perforations cause symptoms during the colonoscopy procedure.[5] For example, 8 hours elapsed before our patient experienced dyspnea with further spread of subcutaneous emphysema and increasing abdominal pain. Patients should be assessed closely for signs of severe abdominal pain or discomfort following a colonoscopy because it may be an early sign of perforation.[9,11,15] Additionally, the presence of nausea and vomiting following a colonoscopy using propofol, which has substantial antiemetic effects, is likely related to insufflation but could also be a sign of perforation.[10] Abdominal, upright radiographs are 87% diagnostic of perforations, but computed axial tomography is recommended if findings are not definitive or if the presence of free air in the abdomen cannot be ruled out by radiographs alone.[15] Fever or leukocytosis are other objective findings of perforation, but 10% of patients may not exhibit symptoms.[11]

Air is still the most commonly used insufflating gas for colonoscopies in most parts of the world.[22] Studies comparing the use of air versus carbon dioxide insufflation during colonoscopies have shown that the use of carbon dioxide leads to less pain and discomfort during and after the procedure without causing a rise in end-tidal carbon dioxide ($ETCO_2$).[21,22] Additionally, carbon dioxide is rapidly absorbed by the tissues and excreted by the lungs.[21] Furthermore, carbon dioxide may decrease the occurrence of abdominal pain after colonoscopy, which might allow easier detection of suspected perforations. However, specific research regarding whether the use of carbon dioxide insufflation can more promptly detect perforation is lacking.

Each endoscopy unit should have a written policy for discharge criteria.[35] Typical discharge criteria from an endoscopy unit include responding appropriately to questions, sitting upright in a chair for 5 minutes, and dressing with little or no assistance.[36] Additionally, written discharge instructions should be provided to all patients that include instructions on diet restrictions, resumption of medications, activity restrictions, procedure-specific signs and symptoms of potential complications, and the steps to follow if such complications were to occur.[35,36] Such written discharge instructions should be provided in compliance with guidelines promulgated by the American Society of Gastrointestinal Endoscopy.[35] It is unknown what discharge instructions were given to our patient because his colonoscopy was performed at another institution. Patients and their family members or other caregivers should be encouraged to report any adverse symptoms immediately to the attending physician, and/or call emergency medical service to proceed to the nearest ED for further evaluation.

Anesthesia providers' awareness of the risk factors for colonic perforation due to colonoscopy, early signs and symptoms of perforation, and knowledge of the surgical and anesthetic management of perforation could lead to early recognition, intervention, and likely improved patient outcomes.

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