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The Causes of Gastrointestinal Tract Perforation and its Management

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Abstract

Perforation of the gastrointestinal tract may be suspected based upon the patient's clinical presentation, or the diagnosis becomes obvious through a report of extra luminal "free" gas or fluid or fluid collection on diagnostic imaging performed to evaluate abdominal pain or another symptom. Clinical manifestations depend somewhat on the organ affected and the nature of the contents released (gas, succus entericus, stool), as well as the ability of the surrounding tissues to contain those contents. Intestinal perforation can present acutely or in an indolent manner (e.g., abscess or intestinal fistula formation). A confirmatory diagnosis is made primarily using abdominal imaging studies, but on occasion, exploration of the abdomen (open or laparoscopic) may be needed to make a diagnosis. Specific treatment depends upon the nature of the disease process that caused the perforation. Some etiologies are amenable toa nonoperative approach, while others will require emergent surgery.

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Introduction

Perforation requires full-thickness injury of the bowel wall; however, partial-thickness bowel injury (e.g., electrocautery, blunt trauma) can progress over time to become a full-thickness injury or perforation, subsequently releasing gastrointestinal contents. Full-thickness injury and subsequent perforation of the gastrointestinal tract can be due to a variety of etiologies, commonly instrumentation (particularly with cautery) or surgery, blunt or penetrating injury, and bowel obstruction. In addition to causing obstruction, neoplasms (particularly colon carcinoma) can also cause perforation by direct penetration of the tumor through the bowel wall. Other etiologies are less common (Khalid *et al.*, 2014). Spontaneous perforation can be related to inflammatory changes or tissues weakened by medications or connective tissue disorders. Esophageal, gastric, or duodenal perforations may also be associated with peptic ulcer disease, corrosive agents, or particular medications (Wain *et al.*, 2015).

With bowel obstruction, perforation occurs proximal to the obstruction as pressure builds up within the bowel, exceeding intestinal perfusion pressure and leading to ischemia and subsequently necrosis. When perforation is proximal to a colon obstruction, it usually occurs in the cecum in the presence of a competent ileocecal valve. Enteroliths and gallstones can also cause perforation by direct pressure or indirectly by leading to obstruction resulting in a proximal perforation (Eligijus Poskus *et al.*, 2014; Jain *et al.*, 2016). Alternatively, the excess pressure can cause the musculature of the bowel to fail mechanically; in other words, to simply split (diastatic rupture) without any obvious necrosis. Intestinal pseudo-obstruction can also lead to perforation by these mechanisms (Lob *et al.*, 2018). As free gas accumulates in the peritoneal cavity, it can compress intra- abdominal veins or lead to respiratory insufficiency by compromising diaphragmatic function (Agu *et al.*, 2014). Such a tension pneumoperitoneum (valvular pneumoperitoneum) can result from iatrogenic or pathologic processes. Perforation and subsequent inflammation can also cause abdominal compartment syndrome (Akinwale *et al.*, 2016).

Anatomic considerations

Knowledge of gastrointestinal anatomy and anatomic relationships to adjacent organs helps predict symptoms and to interpret imaging studies in patients with a possible gastrointestinal perforation. Whether or not gastrointestinal perforation leads to free fluid and diffuse peritonitis or is contained, resulting in an abscess or fistula formation, depends upon the location along the gastrointestinal tract and the patient's ability tomount an inflammatory response to the specific pathologic process (Singh *et al.*, 2008). As an example, retroperitoneal perforations are more likely to be contained. Immunosuppressive and anti-inflammatory medications impair this response. In brief, the relationship of the gastrointestinal tract to itself and other structures is as follows: The esophagus begins in the neck and descends adjacent to the aorta through the esophageal hiatus to the gastroesophageal junction. Perforations of the esophagus due to foreign body ingestion usually occur at the narrow areas of the esophagus, such as the cricopharyngeus muscle, aortic arch, left main stem bronchus, and lower esophageal sphincter. The stomach is located in the left upper quadrant of the abdomen but can occupy other areas of the abdomen, depending upon its degree of distention, phase of diaphragmatic excursion, and the position of the individual. Anteriorly, the stomach is adjacent to the left lobe of the liver, diaphragm, colon, and anterior abdominal wall. Posteriorly, the stomach is near the pancreas, spleen, left kidney and adrenal gland, splenic artery, left diaphragm, transverse mesocolon, and colon (Patel *et al.*, 2010).

The esophagus has three anatomical points of narrowing that are prone to perforation. These sites include the cricopharyngeus muscle, the broncho-aortic constriction, and the esophagogastric junction. The esophagogastric junction is the most common site of perforation. The relationship of the stomach to surrounding structures is depicted in the figure. The arterial supply to the stomach is derived primarily from the celiac axis. The celiac axis arises from the

proximal abdominal aorta and typically branches into the common hepatic, splenic, and left gastric arteries. The common hepatic artery usually gives rise to the gastroduodenal artery (in approximately 75 percent of people), which, in turn, branches off into the right gastroepiploic artery and the anterior and posterior superior pancreaticoduodenal arteries, which supply the pancreas. The right gastroepiploic artery joins with the left gastroepiploic artery, which emanates from the splenic artery in 90 percent of patients. The right gastric artery branches from the hepatic artery and anastomoses with the left gastric artery along the lesser curvature of the stomach. The small bowel is anatomically divided into three portions: the duodenum, jejunum, and ileum. The duodenum is retroperitoneal in its second and third portions and forms a loop around the head of the pancreas. The jejunum is in continuity with the fourth portion of the duodenum beginning at the ligament of Treitz; there are no true lines of demarcation that separate the jejunum from the ileum. The ileocecal valve marks the beginning of the colon in the right lower quadrant. The appendix hangs freely from the cecum, which is the first portion of the colon. Foreign bodies that perforate the small intestines most commonly occur at sites of gastrointestinal immobility (e.g., duodenum). The ascending and descending colon are retroperitoneal, while the transverse colon, which extends from the hepatic flexure to the splenic flexure, is intraperitoneal. The sigmoid colon continues from the descending colon, ending where the tinea coli converge to form the rectum. The anterior upper two-thirds of the rectum is located intra peritoneally and the remainder is extraperitoneal. The rectum lies anterior to the three inferior sacral vertebrae, coccyx, and sacral vessels and is posterior to the bladder in men and the vagina in women. Foreign bodies that perforate the colon tend to occur at transition zones from an intraperitoneal location to fixed retroperitoneal locations such as the cecum (Robles-Medranda *et al*., 2008).

Risk factors of perforation

Factors that increase the risk for gastrointestinal perforation are discussed below and are important to assess when taking the history of any patient suspected of having gastrointestinal perforation (Browning *et al*., 2007).

Instrumentation/surgery

Instrumentation of the gastrointestinal tract is the main cause of iatrogenic perforation and may include upper endoscopy (especially rigid endoscopy), sigmoidoscopy, colonoscopy (Souadka *et al*., 2012; Nassour and Fang, 2015) stent placement, endoscopic sclerotherapy (Schmitz *et al*., 2001) nasogastric intubation (Ghahremani *et al*., 1980) esophageal dilation, and surgery. The incidence of perforation related to endoscopy increases with procedural complexity. Perforation is less common with diagnostic compared with therapeutic procedures (Isomoto *et al*., 2009) A perforation rate of 0.11 percent for rigid endoscopy contrasts with a 0.03 percent rate for flexible endoscopy (Kavic *et al*., 2001). When iatrogenic perforation occurs, there is often significantly associated pathology. As an example, in the esophagus, there may be stricture, severe esophagitis (Eisenbach *et al*., 2006) or a diverticulum, and the presence of cervical osteophytes also increases the risk (Silvis *et al*., 1976)· The area of the esophagus at most risk for instrumental perforation is Killian's triangle (Brinster *et al*., 2004), which is the part of the pharynx formed by the inferior pharyngeal constrictor and cricopharyngeus muscle. During endoscopy, perforations are frequently recognized at the time of the procedure. At other times, the perforation remains occult for several days.

When the normal anatomy of the esophagus or stomach has been disturbed, such as after Roux-en-Y gastric bypass, great care should be taken with nasogastric intubation (Lortie and Charbonney, 2016)· Other procedures can also be complicated with perforation, such as chest tube insertion low in the chest (Andrabi *et al*., 2007)' peritoneal dialysis catheter insertion, percutaneous gastrostomy (Covarrubias *et al*., 2013)' paracentesis, diagnostic peritoneal lavage, and percutaneous drainage of fluid

collections or abscess. With surgery, perforation can occur during initial laparoscopic access, during mobilization of the organs or during the takedown of adhesions, or as a result of thermal injury from electrocautery devices (Turrentine *et al.*, 2015). Gastrointestinal leakage can also occur postoperatively as a result of an anastomotic breakdown. Immunosuppressed individuals may be at increased risk for dehiscence and deep organ space infection following surgery. Medical illnesses such as diabetes, cirrhosis, and HIV are associated with an increased risk of anastomotic leak after colon resection for trauma (Stewart *et al.*, 1994).

Penetrating or blunt trauma

Traumatic perforation of the gastrointestinal tract is most likely a result of penetrating injury, although blunt perforation can occur with severe abdominal trauma acutely related to pressure effects or as a portion of the gastrointestinal tract is compressed against a fixed bony structure, or more slowly as a contusion develops into a full-thickness injury (Lortie and Charbonney, 2016).

Medications, other ingestions, foreign body

Medications or other ingested substances (caustic injury) and foreign bodies (ingested or medical devices) can lead to gastrointestinal perforation. Foreign bodies, such as sharp objects (toothpicks), food with sharp surfaces (e.g., chicken bones, fish), or gastric bezoar more commonly cause perforation, compared with dislodged medical implants (Shimizu *et al.*, 2010; Oestreich, 2009). Button batteries as an esophageal foreign body have a more pronounced perforation risk (Peters *et al.*, 2015). Surgically implanted foreign bodies such as hernia mesh (Ott *et al.*, 2005) and artificial vascular grafts (Fujihara *et al.*, 2013) can cause perforation with subsequent abscess and fistula formation or vasculoenteric fistulas. Aspirin and nonsteroidal anti-inflammatory drug (NSAID) use has been associated with perforation of the stomach and duodenum with diclofenac and ibuprofen being the most commonly implicated drugs (Morris *et al.*, 2003). Some disease-modifying antirheumatic drugs (DMARDs) have been associated with lower intestinal perforations (Strangfeld *et al.*, 2017). Rarely NSAIDs have produced jejunal perforations (Risty *et al.*, 2007). Glucocorticoids, particularly in association with NSAIDs, are particularly problematic (Aloysius *et al.*, 2006). Further, because steroids suppress the inflammatory response, detection of perforation can be delayed. NSAIDs, antibiotics, and potassium supplements are also common causative medications for pill-induced esophageal ulcers (Abid *et al.*, 2005). Othermedication-induced injury leading to perforation has been reported for immunosuppressive therapies, cancer chemotherapy in patients with metastases, and for iron supplementation causing esophageal injury (Corsi *et al.*, 2006).

Violent retching/vomiting

Violent retching/vomiting can lead to spontaneous esophageal perforation, known as Boerhaave syndrome. This occurs because of failure of the cricopharyngeal muscle to relax during vomiting or retching causing increased intraesophageal pressure in the lower esophagus (Wu *et al.*, 2007).

Hernia/intestinal volvulus/obstruction

Abdominal wall, groin, diaphragmatic, internal hernia, paraesophageal hernia, and volvulus (gastric, cecal, sigmoid) can all lead to perforation either related to bowel wall ischemia from strangulation, or pressure necrosis. Perforation can also occur with afferent loop obstruction after Roux-en-Y reconstruction. Crohn's disease has a propensity to perforate slowly, leading to the formation of entero-enteric or enterocutaneous fistula formation (Simillis *et al.*, 2008).

A portion of appendicitis results in perforation, which can lead to life-threatening complications if left untreated, including intra-abdominal infection, sepsis, intraperitoneal abscesses, and, rarely, death (Parks and Schroeppel, 2011). In adults, the risk of perforated appendicitis increases with the male gender, increasing age and comorbidity, and lack of medical insurance coverage (Drake *et al.*, 2013).

Peptic ulcer disease

Peptic ulcer disease (PUD) is the most common cause of stomach and duodenal perforation but occurs in a small percentage of patients with PUD (Behrman, 2005). In spite of the introduction of proton pump inhibitors, the incidence of perforation from PUD has not changed appreciably (Hermansson *et al.*, 2009). Marginal ulceration leading to perforation may also complicate surgeries that create a gastrojejunostomy (e.g., partial gastric resection, bariatric surgery.

Diverticular disease

Colonic diverticulosis is common in the developed world. All clinical cases of diverticulitis represent some degree of perforation of the thinned diverticular wall, leading to inflammation of the adjacent parietal peritoneum (West, 2008)· Perforation can also occur with duodenal or small intestinal diverticula (jejunal, Meckel's). These diverticula can become inflamed, much as in colonic diverticulitis, and perforate, which may lead to abscess formation.

Cardiovascular disease

Any process that reduces the blood flow to the intestines (occlusive or nonocclusive mesenteric ischemia) for an extended period of time increases the risk for perforation, including embolism, mesenteric occlusive disease, cardiopulmonary resuscitation, and heart failure that leads to gastrointestinal ischemia (Spoormans *et al.*, 2010).

Infectious disease

Typhoid, tuberculosis, and schistosomiasis can cause perforation of the small intestine (Tan *et al.*, 2009). With typhoid, the perforation is usually in a single location (ileum at necrotic Peyer's patches), but it can be multiple. Typhoid perforation is more common in children, adolescents, or young adults. Cytomegalovirus, particularly in an immunosuppressed patient, can cause intestinal perforation.

Neoplasms

Neoplasms can perforate by direct penetration and necrosis or by producing obstruction. Perforations related to the tumor can also occur spontaneously, following chemotherapy, or as a result of radiation treatments when the tumor involves the wall of a hollow viscus organ (Kang *et al.*, 2010). Delayed perforations of the esophagus or duodenum in patients with malignancy can be related to stent placement for malignantobstruction.

Connective tissue disease

Spontaneous perforation of the small intestine or colon has been reported in patients with underlying connective tissue diseases (e.g., Ehlers-Danlos syndrome), collagen vascular disease, and vasculitis(Nakashima *et al.*, 2006). This entity occurs in the neonate or premature infants. No demonstrable cause is appreciated (Farrugia *et al.*, 2003).

Clinical features of perforation

A careful history is important in evaluating patients with neck, chest, and abdominal pain. The history should include questioning about prior bouts of abdominal or chest pain, prior instrumentation (nasogastric tube, abdominal trauma, endoscopy), prior surgery, malignancy, possible ingested foreign bodies (e.g., fish or chicken bone ingestion), and medical conditions (e.g., peptic disease, medical device implants), including medications (nonsteroidal anti-inflammatory drugs [NSAIDs], glucocorticoids) that predispose to gastrointestinal perforation. Patients with perforation may complain of the chest or abdominal pain to some degree. Sudden, severe chest or abdominal pain following instrumentation or surgery is very concerning for perforation. Patients on immunosuppressive or anti-inflammatory agents may have an impaired inflammatory response, and some may have little or no pain and tenderness. Many patients will seek medical attention with the onset or worsening of the significant chest or abdominal pain, but a subset of patients will present in a delayed fashion (Aloysius *et al.*, 2006).

Acute pain

Inflammation of the gastrointestinal tract, because of perforation by a variety of etiologies, usually leads to

some degree of neck pain (or dysphagia) or chest or abdominal discomfort. The patient with a free perforation often notes with precision the time of the onset of the perforation. The patient may relate a sudden worsening of pain, followed by complete dissipation of the pain as perforation decompresses the inflamed organ, but relief is usually temporary. As the spilled gastrointestinal contents irritate the mediastinum or visceral peritoneum, a more constant pain will develop.

Acute symptoms associated with free perforation depend upon the nature and location of the gastrointestinal spillage (mediastinal, intraperitoneal, retroperitoneal). Cervical esophageal perforation can present with pharyngeal or neck pain associated with odynophagia, dysphagia, tenderness, or induration. Perforation of upper abdominal organs can irritate the diaphragm, leading to pain radiating to the shoulder. If perforation is confined to the retroperitoneum or lesser sac (e.g., duodenal perforation), the presentation may be more subtle. Retroperitoneal perforations often lead to back pain (Khalid *et al.*, 2014).

Because the pH of gastric contents is 1 to 2 along the gastric luminal surface, a sudden release of this acid into the abdomen causes severe and sudden peritoneal irritation and severe pain. The pH of the stomach contents is often buffered by recent food consumption. The leakage of small intestinal contents into the peritoneal cavity may also cause severe pain, and for this reason, any severe pain after, particularly a laparoscopic procedure, should cause the surgeon to suspect leakage (Isomoto *et al.*, 2009).

Abdominal/pelvic mass

It is not uncommon for perforation to lead to an abscess or phlegmon formation that can be appreciated on examination as an abdominal mass or with abdominal exploration. A pelvic abscess caused by a perforation can sometimes be felt on digital rectal examination. Diverticulitis is the most common etiology leading to intra-abdominal abscess formation (Brinster *et al.*, 2004).

Fistula formation

A fistula is an abnormal communication between two epithelialized surfaces. It can occur from bowel injury during instrumentation or surgery, anastomotic leak, or foreign body erosion. Fistulas are often related to inflammatory bowel diseases such as Crohn's disease. Rarely, perforated colon carcinoma can fistulize to adjacent structures or to the abdominal wall. The initial gastrointestinal perforation is contained between two loops of the bowel, and subsequent inflammatory changes lead to abnormal communication, which spontaneously decompresses any fluid collection or abscess that has formed. Patients who develop an external fistula will complain of the sudden appearance of drainage from a postoperative wound or from the abdominal wall or perineum in case of spontaneous fistulas (Farrugia *et al.*, 2003).

Sepsis

Sepsis can be the initial presentation of perforation, but its frequency is difficult to determine. The ability of the peritoneal surfaces to wall off a perforation may be impaired in patients with severe medical comorbidities, particularly frail, elderly, and immunosuppressed patients, resulting in free spillage of gastrointestinal contents into the abdomen, generalized abdominal infection, and sepsis (Moore and Moore, 2013). Sepsis in itself can contribute to the causation of perforation by reducing intestinal wall perfusion (Merrell, 1995). These patients are very ill-appearing, may or may not be febrile, and may be hemodynamically unstable with altered mental status. Anastomotic leak (e.g., colon surgery) can be associated with increased fluid and blood transfusion requirements(Behrman *et al.*, 1998). Organ dysfunction may be present, including acute respiratory distress syndrome, acute kidney injury, and disseminated intravascular coagulation. Timely and adequate peritoneal source control is the most important determinant in the management of patients with acute peritonitis/abdominal sepsis. In the Physiological Parameters for Prognosis in Abdominal Sepsis (PIPAS) study, the overall in-hospital mortality rate of 3137 patients was 8.9

percent. Ten independent variables were associated with mortality: malignancy, severe cardiovascular disease, severe chronic kidney disease, respiratory rate >22 breaths/minute, systolic blood pressure <100 mmHg, unresponsiveness, room air oxygen saturation level <90 percent, platelet count <50,000/microL, and serum lactate level >4 mmol/L. These variables were used to create the PIPAS severity score. The overall mortality was 2.9 percent for patients with scores of 0 to 1, 22.7 percent for 2 to 3, 46.8 percent for 4 to 5, and 86.7 percent for 7 to 8 (Sartelli *et al.*, 2019).

Physical examination of perforation

Physical exam should include vital signs; a thorough examination of the neck, chest, and abdomen; and rectal examination. In patients with gastrointestinal perforation, vital signs may initially be normal or reveal mild tachycardia or hypothermia. As the inflammatory response progresses, fever and other signs of sepsis may develop. Palpation of the neck and chest should look for signs of subcutaneous gas and auscultation and percussion of the chest for signs of effusion. Mediastinal gas might be heard as a systolic "crunch" (Hamman's sign) at the apex and left sternal border with each heartbeat. Palpation reveals crepitus in 30 percent of patients with thoracic esophageal perforation and in 65 percent of patients with cervical esophageal perforation (Sarr *et al.*, 1982). Patients with esophageal rupture caused by barotrauma can have facial swelling. The abdominal examination can be relatively normal initially or reveal only mild focal tenderness, as in the case of contained or retroperitoneal perforations. The abdomen may or may not be distended. Distention is common in those patients with perforation related to small bowel obstruction. When free intraperitoneal perforation has occurred, typical signs of focal or diffuse peritonitis are present. The rectal examination may be normal, as with contained upper abdominal gastrointestinal perforation or reveal a palpable mass in the cul-de-sac, representing a phlegmon or abscess. There may also be rectal tenderness as well as bogginess secondary to inflammation (Pieper-Bigelow *et al.*, 1990).

Laboratory studies of perforation

Laboratory studies are typically obtained in patients who present with acute abdominal pain including complete blood count (CBC), electrolytes, blood urea nitrogen (BUN), creatinine, liver function tests, lactate, amylase, and/or lipase. Serum amylase may be elevated in patients with intestinal perforation due to the absorption of amylase from the intestinal lumen (Pieper-Bigelow *et al.*, 1990). However, this finding is nonspecific. Alterations in serum amylase can be due to a variety of conditions, and many drugs affect serum amylase values. C-reactive protein levels may help to diagnose gastrointestinal leaks, particularly after bariatric surgery or colorectal surgery.

It has also been useful for diagnosing perforation associated with typhoid fever (Olubuyide *et al.*, 1989). Some inflammatory markers in drain fluid have also been associated with anastomotic leak following colorectal surgery. Although a diagnosis of the gastrointestinal leak was made in the APPEAL study, it was done in conjunction with imaging studies or because of stool in the effluent (Komen *et al.*, 2014). Drain studies are generally unnecessary. In addition, most surgeons do not routinely place drainage tubes in the abdomen.

Non-surgical related constraints

Lack of water and sanitation, overcrowding

More diffuse access to water safety and sanitation is fundamental for the control of typhoid fever, but the related huge economic costs and long timelines will not allow a short- to the middle-term solution.

Healthcare systems of poor resources countries, especially when affected by internal or external conflicts, may not afford the cost of these socioeconomic improvements. Conversely, targeted interventions on densely populated urban communities like slums, where typhoid fever is a serious problem, could be a possible way out. In the meantime, fewer resources could be directed towards rural areas with lower population density where enteric fever is less common (Mweu and English, 2008; Breiman *et al.*, 2012).

Inadequacy of immunization programs

Almost all public health typhoid vaccination programs in the groups of populations at greatest risk have been performed in Asia, with the strongest impact in endemic settings and in short- to medium-term. The oral vaccine was found to be highly cost-effective when targeting ages 1-14 years in high burden/high-risk districts, as well as urban slums and rural areas without improved water.

Remarkably, no vaccination experience has been reported from sub-Saharan Africa, where emerging threats, including multidrug resistance and increasing urbanization, would warrant concentration on immunization programs (Baker *et al.*, 2016).

The recently proposed Typhoid Risk Factor (TRF) index, which takes into account the drinking water sources, toilet facility types, and population density, seems a reliable tool to evaluate variations in the disease burden, helping decision-makers to identify high-risk areas and prioritize the right populations for vaccination (Behrman, 2005).

Delay in surgical treatment

Timely surgical treatment can prevent the severe peritoneal contamination observed in up to 70% of patients, associated with a high mortality rate. Moreover, early surgery might reduce the need for extensive surgical procedures, with their contribution to highmorbidity and mortality. From 30% to 100% of perforated patients may wait a long period before surgery, especially in rural areas and peripheral facilities. Indeed the diagnosis can be challenging in very young patients, in those who perforate while on medical treatment or in the presence of a generalized septic state, but if symptoms are evocative, diagnostic confirmation by either abdominal x-ray or ultrasound should not delay surgery. Similarly, adjustment of electrolytes and fluid imbalance or anemia correction should postpone surgery only for a short time as prolonged resuscitation can adversely affect the outcome (Hassan *et al.*, 2010)· Frequent causes of surgical delay are protracted or late referral from inadequate health facilities, difficult transport systems (both ambulances and roads), difficulties sourcing funds for treatment and diversion of patients to alternative medical therapies before consulting the hospital.

Surgical related constraints

Non-operative treatment has been proposed in the past in moribund patients or for long-standing perforations, but there is now uniform agreement that the ultimate treatment for TIP should be a surgical one, although the best surgical management remains controversial. The type of surgical technique might have limited influence on the outcome, which is likely more related to the preoperative clinical conditions of the patients, to the degree of abdominal contamination and to the quality of pre- and post-operative care (Mohil *et al.*, 2008).

Scarcity of prospective studies and guidelines

Several surgical solutions have been proposed for the treatment of TIP, with a consequent variability of morbidity and mortality. Indeed, explicit surgical guidelines, particularly aimed at resource-poor countries, are lacking. Most reports are retrospective, often including a small number of patients with incomplete data and poor statistical analysis.

Surgical morbidity and mortality are often reported without any risk adjustment based on the severity of the disease, delay of treatment, etc. The few available prospective studies highlight that patient's conditions have a more significant impact on patient's outcome than the type of surgical procedure (Tade *et al.*, 2011).

Unavailable appropriate postoperative care

Postoperative care may be quite complex in these very fragile patients, frequently presenting with a septic state, coexistent diseases and an impaired immunological status. Moreover, intensive care units supplied for possible renal or respiratory failures, with available appropriate antibiotics for overwhelming infections and with accessible tools for nutritional support, are found infrequently in resource-poor countries, especially in peripheral or rural settings (Ameh *et al.*, 1999).

Surgical technique

The type of the surgical procedure does not appear to influence the mortality of TIP; conversely, sound surgical judgment and experience are required to select the appropriate surgery according to the surgical findings and especially in advanced diseases (Haider *et al*., 2002).

Primary repair is usually performed for single or isolated perforations by single or two suture layers. Segmental resection and anastomosis are preferred in the presence of multiple adjacent perforations, while wedge resection is reported infrequently. Simple repair generally has a lower mortality rate than resection, although the death rate remains high when abdominal contamination is severe. The correlation between a high number of perforations, perhaps due to a highly virulent causative organism, and a poor surgical outcome is questionable. Enterocutaneous fistula is the most alarming complication, with a mortality up to 67%, that is likely underestimated because death can occur months after surgery (Memon *et al*., 2012).

An ileostomy is usually reserved for patients with severe disease, delayed presentation and very contaminated abdomen, with a high risk of suture leakage. Ileostomy has also been described as a routine primary procedure, although it is associated with a high morbidity rate and complications like prolapse, stricture, retraction, parastomal hernia, mainly when performed in patients with critical conditions (Chaudhary *et al*., 2015). Moreover, loss of intestinal fluids from ileostomy can be managed with difficulty in an austere environment and shortage of suitable ileostomy bags, with consequent skin damage around ileostomies, not rarely inducing the patient to a self-limitation of food intake.

Delayed primary closure of the abdominal wall has been recommended for heavily contaminated wounds for a long time, but to date, the optimal method of closure in such situations remains controversial. Vacuum-assisted closure appears promising but may not be feasible in peripheral facilities. Scheduled re-laparotomies, allowing early recognition of complications and a more appropriate cleaning of the abdominal cavity, have been performed with a positive impact on survival. However, this policy has the disadvantage of submitting the patients to multiple surgical trauma and increasing the workload of the operative theater (Pieper-Bigelow *et al*., 1990).

A laparoscopic approach to TIP has been occasionally carried out with acceptable results (Sinha *et al*., 2005). There is no evidence that laparoscopy is more advantageous than open surgery, although it could be considered as an advantageous diagnostic tool in doubtful abdomens. A concern is a need for highly technological equipment and appropriate maintenance, often lacking in poor resources countries.

Ileostomy care and complications

Ileostomy or colostomy creation may be required temporarily or permanently for the management of a variety of pathologic conditions, including congenital anomalies, colon obstruction, inflammatory bowel disease, intestinal trauma, or gastrointestinal malignancy (Doughty, 2005). The anatomic location and type of stoma construction have an impact on management. Loop colostomies tend to be larger and somewhat more difficult to manage than end colostomies. The type and volume of output (effluent), and therefore fluid loss, is determined by the location of the stoma relative to the ileocecal valve. Fluid loss is primarily a factor with end or loop ileostomies. With proper stoma care and attention to nutrition and fluid management, most ostomy patients are able to have full, healthy, active social and professional lives. In many cases, quality of life can be improved, even in the context of a permanent ostomy, with the treatment of the underlying disease (Andrews and Ryan, 2015).

Patient education

The patient must adapt to new patterns of fecal elimination and to their altered body and image of themselves. Successful adaptation requires the patient to master new skills and to deal effectively with the many emotional issues associated with their

altered anatomy and with altered continence. Interventions that promote adaptation include Preoperative stoma site selection by ostomy nurse specialist (enterostomal therapy nurse, wound ostomy continence nurse) or experienced surgeon. Preoperative stoma site marking has been associated with fewer ostomy-related complications (e.g., leakage, dermatitis), improved patients' ability to care for the ostomy independently, and reduced health care costs (Hendren *et al.*, 2015). Position papers by both the American Society of Colon and Rectal Surgeons (ASCRS) and Wound Ostomy Continence Nurse Society (WOCN) have been published to guide proper stoma site markings (Salvadalena *et al.*, 2015). A strong focus on individualized patient education, with a preoperative and postoperative component. Supportive counseling for all patients preoperatively and in-depth counseling for any patient who is having trouble adapting (White, 2004).

Several studies have demonstrated that involvement of an ostomy nurse specialist has a significant impact on long-term positive outcomes and reduced complication rates, as does involvement in ostomy support groups such as the United Ostomy Association of America (White, 2004).

Pouch system and routine ostomy care

Pouch systems

The main functions of ostomy pouches are to contain the ostomy effluent, contain the odor, and protect the peristomal skin. Many pouching systems are available, as either one- or two-piece systems: One-piece systems include a protective skin barrier with a tape border fused to an odor-proof pouch. Two-piece systems include a protective skin barrier with a tape border and flange or adhesive landing zone to which the patient attaches a separate odor-proof pouch. One-piece systems offer simplicity, and many of these systems provide flexibility, which is important for the patient whose stoma is located in a deep crease. Two-piece systems have the advantage that the pouch can be replaced without having to remove the protective skin barrier each time (Behrman *et al.*, 1998).

Pouch placement

The patient should be taught strategies that can help promote pouch adherence to the skin, minimize leakage, and protect peristomal skin. These include: Selecting a pouching system that conforms to the abdominal contour at the stoma location. For example, a flat pouch with a rigid flange requires a relatively flat, at least 4 cm pouching surface that is distant from scars, skin creases or folds, and bony prominences (Suwanabol and Hardiman, 2018).

A stoma located in a concave abdominal plane may be best managed with a convex pouching system, which can increase protrusion of the stoma and improve drainage of effluent into the pouch. Transverse loop colostomies are typically large stomas in the upper quadrants that are difficult to conceal, and prolapse is more common. Cecostomies, now rarely performed, are typically skin-level stomas located adjacent to the groin crease, which compromises pouch adherence sizing the opening of the protective skin barrier to minimize the amount of exposed skin. Stomas often change shape and size in the postoperative period. After the stoma has assumed its final appearance (usually several weeks after construction), a precut protective skin barrier may be supplied, so the patient or their caregivers do not need to cut out the barrier ring each time a new appliance is placed. Using adjunctive products to improve the fixation of the pouch (adhesive agents, Skin Prep) and to prevent irritation and injury to the peristomal skin (skin barrier paste, skin barrier powder, skin barrier ring) (Colwell, 2004). Loop ileostomies are typically more difficult to manage than end ileostomies because the stoma frequently empties close to the skin surface.

Because the small bowel effluent is rich in proteolytic enzymes, the patient with an ileostomy must exercise particular caution in managing the peristomal skin.

These patients should routinely use barrier wafers, rings, and/or paste to assure that their skin is not exposed to the drainage and must treat any minor skin damage aggressively to prevent progression (White, 2004).

Pouch emptying and care

Odor and gas are common concerns for any individual with an ostomy (Annells, 2006).

The patient should be assured that ostomy pouches are odor-proof, but when the pouch is emptied, the odor is normal. Simple strategies can help reduce odor. These include the following:

Emptying the pouch when it is approximately one-third full will prevent disruption of the pouch seal from excess weight.

Changing the pouch one to two times weekly, and as needed, for any signs of leakage or for itching/burning of the peristomal skin.

It was keeping the tail of the pouch clean so that it does not become a source of the odor. This can be accomplished by everting the tail of a pouch prior to emptying it. For some pouches that have an integrated closure mechanism, tail eversion is not required.

Using a room spray or pouch deodorant to minimize odor associated with emptying.

If the odor is a particular concern for the patient, bismuth subgallate or chlorophyllin copper complex effectively reduces stool odor when taken routinely.

Bismuth subgallate tends to thicken the stool, so it may best be used for the patient with an ileostomy or proximal colostomy.

Chlorophyllin may have a slight diarrheal effect and is more appropriate for the patient with a descending/sigmoid colostomy.

Diet and control of gas

Many patients assume that they will have to adhere to a special diet because of their ostomy. Dietary modifications are usually minimal, but specific foods can influence the amount of the gas and the consistency and odor of the effluent (Floruta, 2001).

Patients should be given a general list of gas-producing foods. The carbohydrate raffinose is poorly digested and leads to gas production by the action of colonic bacteria. Common foods containing raffinose include beans, cabbage, cauliflower, brussel sprouts, broccoli, and asparagus. Starch and soluble fiber are other forms of carbohydrates that can contribute to gas formation. Potatoes, corn, noodles, and wheat produce gas, while rice does not. Soluble fiber (found in oat bran, peas and other legumes, beans, and most fruit) also causes gas. Patients should also be given an explanation that the usual "lag time" between ingestion of a gas-producing food and actual flatulence is between two to four hours for ileostomy and six to eight hours for distal colostomy.

In addition to dietary modifications, ileostomy patients should be taught to avoid drinking carbonated drinks, drinking through straws, chewing gum, and smoking since these measures tend to increasegas ingestion.

Strategies to control gas include measures to reduce the volume of gas produced or to affect the "timing" of flatulence, "muffling" measures and "venting" strategies. Dietary modifications and over-the-counter gas-reducing agents (e.g., Beano and Gas-X) help reduce the volume of gas. "Muffling" measures include layers of clothing and light pressure exerted against the stoma with the hand or arm when flatulence is anticipated.

For patients with large volumes of gas, there are pouching systems with filters, which vent and deodorize flatus; there are also "add-on" flatus filters that can be used with any pouching system (Keenan and Hadley, 1984).

Ileostomy patients

The type and volume of output (effluent), and therefore fluid loss, is determined by the location of the stoma relative to the ileocecal valve. Ileostomies, cecostomies, and ascending colostomies typically produce output (effluent) >500 mL per day that contains digestive enzymes, which is irritating to the mucosa and skin.

Dehydration

Dehydration affects up to 30 percent of patients after loop ileostomy creation and is the most common cause for hospital readmission after ileostomy surgery (Hayden *et al.*, 2013).

How much fluid should an adult drink each day to prevent dehydration? It is a simple question with no easy answer, as studies have produced varying recommendations. As examples:

The United States National Academies of Sciences, Engineering, and Medicine (formerly the Institute of Medicine) have recommended adequate intake (AI) values for total water at levels to prevent dehydration. The AI for men aged 19+ is 3.7 liters each day, 3 liters (13 cups) of which should be consumed as beverages. The AI for women aged 19+ is 2.7 liters, about 2.2 liters (9 cups) of which should be consumed as beverages each day.

The Dietitians of Canada have recommended 3 liters (12 cups) for men 19 years old and over each day and 2.2 L (9 cups) for women 19 years old and over each day.

The Eatwell guide of the British National Health Service (NHS) has recommended drinking six to eight cups or glasses of fluid a day.

Each cup or glass generally contains eight ounces of fluid. Thus, eight cups of fluid would add up to 64 ounces or approximately 1.9 liters.

Regardless, patients with an ileostomy should be instructed to increase their daily fluid intake beyond the recommended AI for the general population by at least 500 to 750 mL and to drink even more during periods of high-volume output or heavy sweating. Preferred fluids include water, broth, vegetable juices, and some sports drinks, but patients should be advised that certain sports drinks may not be absorbed and may even exacerbate stoma output and dehydration. The use of pediatric electrolyte solutions (e.g., PediaLyte, Emergen-C) is preferable to the use of sports drinks. Ileostomy patients and patients with ascending colostomies should be taught the importance of adequate daily fluid intake. The average output for the ileostomy patient ranges from 500 to 1300 mL a day; during the early postoperative period and episodes of gastroenteritis, daily output can be 1800 mL or even higher (McCann, 2003). A loop ileostomy performed in conjunction with a pelvic pouch procedure is located more proximally in the ileum and is associated with even more fluid and enzymatic output. This daily fluid loss places the ileostomy patient at greater risk for dehydration, especially during episodes of increased output or heavy perspiration.

Ileostomy effluent contains significant amounts of sodium and potassium. Patients should also be taught the signs and symptoms of fluid-electrolyte imbalance and the importance of prompt treatment should these symptoms occur. These include dry mouth, reduced urine output, dark, concentrated urine, feelings of dizziness upon standing, marked fatigue, and abdominal cramping (Carmel and Goldberg, 2004).
Protocols and pathways have been proposed to minimize dehydration and readmission in patients with new ileostomies. Using combinations of preoperative teaching, in-hospital engagement of the nurses and Wound, Ostomy and Continence care (WOCN) teams, in-hospital involvement and encouragement of patients and families in stoma care, postdischarge counseling, and tracking of intake and output has effectively reduced hospital readmission while maintaining an appropriate hospital length of stay (Nagle *et al.*, 2012).

High ostomy output

First-line management of patients with elevated ileostomy output (defined as >1.5 L/day) should include soluble fiber supplementation, also referred to as bulk-forming laxatives, since they are primarily used to treat constipation (e.g., Metamucil, psyllium, Konsyl, FiberCon, Fiber Gummies). Patients may slowly increase supplementation up to four times daily and also at double doses. Medical management of patients with inappropriately elevated ileostomy

outputs is required in patients who do not respond to fiber supplementation and includes antimotility agents (e.g., loperamide [Imodium], diphenoxylate and atropine [Lomotil], octreotide, cholestyramine, and, rarely, tincture of opium). Rarely, patients with difficult-to-control ileostomy output may need to be maintained on intravenous hydration via a long-term indwelling venous access cannula. Those with persistent, recurrent, or difficult-to-manage complications from loop ileostomy creation should be considered for early reversal of the stoma and restoration of intestinal continuity, when feasible.

Ileostomy patients with high output may need to change to a protective skin barrier that is extended wear and/or the addition of a skin barrier ring to prevent washout of the skin barrier. Also, changing to a high-output pouch would be beneficial for patient management (Covarrubias *et al*., 2013).

Food blockage

Ileostomy patients should also be taught strategies to prevent food blockage proximal to the ostomy site, which can occur because theileal lumen is <1 inch (2.5 cm) in diameter. There is also the potential for further narrowing at the point where the bowel passes through the fascia/muscle layer. If the patient consumes large amounts of insoluble fiber, the undigested fiber may create an obstructing mass (bezoar). Common "offenders" include popcorn, coconut, mushrooms, black olives, stringy vegetables, corn, nuts, celery, foods with skins, dried fruits, and meats with casings. Food blockage is easily prevented by instructing the patient to consume potential offenders one at a time in small amounts, to chew thoroughly, and to monitor their response (Carmel and Goldberg, 2004).

Drug malabsorption

Because the small bowel is the most important site of drug absorption, patients with an ileostomy are at risk for suboptimal drug absorption. The patient with an ileostomy must be taught to take medications in dosage forms of quick dissolution, such as liquids, gelatin capsules, and uncoated tablets, and avoid time-released and enteric-coated medications as well as very large tablets since these forms of medication, are likely to be incompletely absorbed. Consult with a pharmacist regarding medications that may not dissolve or be absorbed appropriately. They must also avoid laxatives due to the risk for acute dehydration (Erwin-Toth and Doughty,1992).

Physical activity

A common concern for many patients is the impact of the stoma on activities of daily living. The patient can be reassured that most activities can be safely resumed with minimal if any, modifications. As an example, bathing and showering can be performed with the pouch on or off, and clothing modifications are generally not required. Most sports activities can be resumed as well, with the exception of extreme contact sports, which could potentially damage the stoma. The addition of a belt or binder is helpful in maintaining a pouch seal during vigorous activity and with perspiration (Erwin-Toth and Doughty,1992).

Sex

Sexual activity is a particular concern for many patients with an ostomy. Patient counseling should address questions regarding sexual activity and partner response (Manderson, 2005). The ostomy does not affect organic sexual function. However, if the ostomy was placed due to some form of pelvic surgery or prior radiation treatment, it is possible that the autonomic nerves controlling sexual function, which are located adjacent to the rectum and the pelvic sidewall, may have been injured.

It is helpful for the patient to empty the pouch and assure an intact pouch seal before engaging in sexual activity. In addition, many patients and their partners find it helpful to use lingerie or a cummerbund to conceal and secure the pouch. Commercial pouch covers, lingerie, and undergarments specifically designed for ostomates are available (White, 2004).

Travel

Patients who are traveling should be advised to:

Take extra ostomy supplies, and if flying, place them in carry-on luggage.

Some airports offer private pre-screenings upon request.

Avoid exposing ostomy pouches and adhesive adjuncts to extreme temperatures, which may alter the adhesive quality.

Drink only bottled water if local tap water is not known to be safe.

Ostomy complications

The incidence of stomal complications ranges from 14 to 79 percent (Persson *et al.*, 2010), nearly half of all stomas eventually become "problematic" due to pouching and peristomal skin issues (Caricato *et al.*, 2007). Complications vary with the type of ostomy, with lower complication rates for those with end colostomy and end ileostomy (Güenaga *et al.*, 2007). Loop ileostomies have the highest complication rates.

The most common problems of end and loop ileostomies are dehydration and skin irritation (related to the high-output, high alkaline enzymatic effluent), and small bowel obstruction.

Although prolapse can occur in all types of stomas, it is more prevalent in loop colostomies, particularly those constructed using the transverse colon. Parastomal hernia and retraction are the most common complications for end and loop ileostomies and colostomies (Güenaga *et al.*, 2007). Stomal and peristomal complications can occur in the early postoperative period or many years following stomal construction (Nastro *et al.*, 2010). Although profiles of early and late ostomy complications do overlap (e.g., stomas can retract early or late), they are often quite different:

Very early complications (days)

Complications that occur very early in the postoperative course (days) are often related to technical issues (Cottam *et al.*, 2007) and often require a return to the operating room. Examples include large bowel obstruction due to a twist in the bowel leading to the stoma.

Early complications (<3 months)

Early complications, defined as those occurring within three months of stoma construction, are often related to suboptimal stoma site selection but are heavily influenced by patient factors (e.g., old age, poor nutritional status, higher American Society of Anesthesiologists [ASA] class, comorbidities, obesity, tobacco use, and underlying malignancy) (Cottam *et al.*, 2007). Early complications include stomal ischemia/necrosis, stomal bleeding, stomal retraction, and mucocutaneous separation.

Stomal necrosis

The incidence of stomal necrosis in the immediate postoperative period is as high as 14 percent. Adequate mobilization of the bowel, preservation of the blood supply to the stoma, and an adequate trephine size are important factors for avoiding this complication. Independent risk factors for stomal necrosis include emergency surgery, obesity, and inflammatory bowel disease, in particular Crohn disease (Leenen and Kuypers, 1989).

Stomal necrosis most commonly occurs in the early postoperative period as a result of venous congestion or arterial insufficiency (e.g., tight fascial opening, excessive mesenteric stripping). The most critical assessment is to determine the extent of necrosis, which can be performed by inserting a lubricated test tube into the stoma and using a flashlight to visualize the proximal mucosa. An alternative is to use a flexible sigmoidoscope or a lighted anoscope. Management is based upon the clinical scenario:

If the necrosis extends to the proximal bowel below the anterior fascia, immediate revision is required.

If the proximal bowel is viable and the necrosis is limited to the stoma (superficial to the anterior fascia), observation may be appropriate.

If necrosis progresses, a revision is required.

If sloughing occurs, only gentle debridement may be necessary. Sloughing of the stoma can result in stomal

retraction and pouching challenges but may not require surgical intervention.

In acute settings, stoma ischemia is more often limited to the mucosa above the fascia and thus can be observed, albeit with a possible resultant stricture in the future (Paquette, 2018).

Stomal bleeding

Major bleeding from the stoma (as opposed to a gastrointestinal bleed) is uncommon and usually indicates either a stomal laceration from a poorly fitting appliance or the presence of peristomal varices in the patient with portal hypertension. Minor bleeding from the stoma can also occur early in the postoperative period related to the creation of the stoma or later with overly vigorous stomal cleansing. Initial management of stomal bleeding involves direct pressure and local cauterization (handheld cautery, silver nitrate) or suturing of the bleeding vessel is easily identified. Peristomal varices are most frequently seen in patients who underwent a colectomy for ulcerative colitis in the setting of primary sclerosing cholangitis. Peristomal varices can also develop in patients with other causes of portal hypertension. Initial management consists of direct pressure followed by injection sclerotherapy or direct suture. However, recurrence is frequent, and medical therapy or intervention (e.g., transjugular intrahepatic portosystemic shunting) may be needed to reduce portal pressures (Harris *et al* ., 2005).

Stomal retraction

Stomal retraction is defined as a stoma that is 0.5 cm or more below the skin surface within six weeks of construction, typically as a result of tension on the stoma. Stomal retraction leads to leakage and difficulties with pouch adherence, resulting in peristomal skin irritation. The incidence of stomal retraction ranges between 1 and 40 percent. The most common risk factors are obesity due to the thickness of the abdominal wall and foreshortened mesentery and initial stoma height <10 mm (Cottam *et al*., 2007). Proper stoma height and minimizing tension are important factors for preventing this complication. Management also depends upon the clinical scenario:

If the stoma retracts below the fascia, immediate operative revision is required to prevent intra-abdominal contamination from the stoma output.

A stoma that has retracted but stays above the fascia can be managed with local wound care, a convex pouching system, and the use of a belt or binder. If these measures fail to provide a secure pouch seal, surgical revision may be needed. However, revision is appropriate only when an improved outcome can be expected and is not appropriate when the cause of the problem has not been addressed. Approximately 1 percent of patients experience stomal retraction as a result of postoperative weight gain. Overweight patients should be encouraged and assisted to lose weight prior to surgical revision.

If nonoperative management of a retracted stoma fails, operative revision or re-siting of the stoma is necessary. Re-siting the stoma to the upper abdominal wall, which is usually thinner, may be helpful.

Mucocutaneous separation

Mucocutaneous separation refers to the separation of the ostomy from the peristomal skin. Mucocutaneous separation results in leakage and skin irritation. It occurs in 12 to 24 percent of patients early in the postoperative period (Cottam *et al*., 2007).

The best approach to preventing this complication is the meticulous technique when approximating the bowel to the skin. Mucocutaneous separation can be partial or circumferential; if circumferential, stomal stenosis can occur as the tissues heal by secondary intention. Circumferential separation of the suture line with retraction of the stoma should be revised immediately. For less severe separations, the defect can be filled with absorptive material, such as calcium alginate, skin barrier powder, paste, or hydrofiber. Covering the area with the protective skin barrier with a barrier ring will help protect the wound from effluent and facilitate healing.

Late complications (>3 months)

Late stomal complications are generally described as permanent ostomies since many temporary stomas are reversed within three months. Risk factors for late complications include duration of stoma, increases in intra-abdominal pressure (obesity, chronic obstructive pulmonary disease), emergency surgery (Harris *et al.*, 2005). Inadequate mobilization of the bowel with the resultant height of stoma <10 mm, and inappropriately sized aperture.

The most common late complications include parastomal hernia, stomal prolapse, and stoma stenosis. A closed stoma site following ostomy reversal can also be associated with complications such as wound infection, delayed healing, and hernia formation.

Parastomal hernia

Parastomal hernia formation is a common complication, especially among colostomy patients. Risk factors include obesity and poor abdominal muscle tone, conditions producing a chronic cough, placement of the stoma outside of the rectus muscle, and a large fascial opening. Most parastomal hernias are asymptomatic and do not progress to complications (e.g., incarceration, strangulation, bowel obstruction) (Colwell, 2005).

Stomal prolapse

Prolapse is the telescoping of the intestine out from the stoma and can occur with any type of stoma. Prolapse can make appliance placement and adherence difficult, and prolonged prolapse causes intestinal edema and, if significant, can lead to intestinal incarceration or strangulation. The incidence ranges from 7 to 26 percent, with the highest rates associated with a loop transverse colostomy and end descending colostomies (Cheung, 1995). Risk factors for prolapse may include a large abdominal trephine, increased intra-abdominal pressure, and a redundant loop of bowel proximal to the stoma. Alternative fixation techniques during ostomy construction have been proposed to prevent prolapse (Maeda *et al.*, 2003). However, there are no data to support these approaches.

Uncomplicated prolapse can be managed conservatively with cool compresses and/or application of an osmotic agent (e.g., table sugar or honey) to reduce edema, followed by manual reduction of the prolapse and application of a binder with a prolapse over-belt to keep the bowel recued into the abdomen, or by pouching modifications to accommodate the prolapsed bowel when reduction cannot be established or maintained (Shapiro *et al* ., 2010). The manual reduction should be initiated at the very tip of the prolapse (beehive) or lumen, and then gentle, slow invagination should proceed. In this way, the prolapsed bowel will intussuscept back into the abdomen. Complicated prolapse or prolapse producing ischemic changes or severe mucosal irritation and bleeding usually requires surgical intervention. Local revision of the prolapse is accomplished by performing a full-thickness resection of the prolapsed intestinal segment with the construction of the stoma at the original site. In the event of a further recurrence, additional bowel resection and relocation of the stoma may be necessary (Paquette, 2018).

Stomal stenosis

Stomal stenosis refers to a narrowing of the stoma sufficient to interfere with normal function. The incidence ranges from 2 to 15 percent and is more common with an end colostomy (Beraldo *et al.*, 2006). Stomal stenosis can occur in the early postoperative period but is more likely to develop months later. Early stenosis of ileostomy, due to edema at the fascial and more superficial levels (assuming appropriate skin opening), can be conservatively managed with gentle insertion of a large 36 French soft-tipped Foley bladder catheter just proximal to the fascial level. The balloon should not be inflated. If there is significant resistance upon intubation of the stoma, the procedure should be abandoned. Care must be taken to avoid perforation. Stenosis can occur later at the skin or the fascia level, or stoma outlet secondary due to scarring or tightness of the mucocutaneous junction. Stenosis may be

attributable to peristomal sepsis, retraction, an ill-fitting pouching system, or suboptimal surgical technique. The patient should be evaluated for other processes that could contribute (e.g., Crohn's disease, primary or recurrent malignancy).

Mild stenosis may be identified only by digital examination of the stoma, with few symptoms, and can usually be managed by dietary modifications (e.g., avoidance of insoluble fiber); gentle routine dilatation of the stoma may also be helpful but is not evidence-based (Harris *et al*., 2005).

Clinically significant stenosis usually causes cramping pain followed by explosive output and usually requires surgical correction. For these, local revision may be preferred over dilation, which can be complicated by peristomal bleeding, tissue injury, fibrosis, and further stenosis. Local repair involves the excision of scar tissue with adequate mobilization and creation of a new tension-free stoma at a new or relocated site. Enlargement of the skin opening via a double Z-plasty technique may be useful in some situations (e.g., limited number of sites for ostomy relocation, limited scarring) (Lyons and Simon, 1960).

Peristomal skin problems (any time)

The most common ostomy complication is peristomal skin breakdown, with varying severities from minor skin trauma to dermatitis, to ulceration, to pyoderma gangrenosum (in Crohn patients). Peristomal skin breakdown can occur early or late and is more prevalent with ileostomies than colostomies (Shapiro *et al*., 2010).

Mechanical trauma

Mechanical trauma typically presents as patchy areas of irritated, denuded skin that result from repeated removal of adhesive products or overly aggressive cleansing techniques. Patients with peristomal hair should be taught to clip the hair to prevent mechanical trauma to the hair follicles. Patients should be taught to use plasticizing skin sealants to help prevent skin damage (skin sealants are optional and specific to the manufacturer) with pouch removal and should be cautioned to use a gentle technique when cleaning the peristomal skin.

Treatment involves the elimination of the causative factors and application of skin barrier powder to the involved areas, followed by blotting with a skin sealant or moistened finger to provide a nonpowdery pouching surface (Colwell, 2004).

Dermatitis

Peristomal skin irritation is more common for patients with an ileostomy due to the nature of the effluent. It is characterized by severely denuded skin along the inferior aspect of the stoma. Creating a protuberant spout for the ileostomy approximately 2 to 3 cm high to optimize pouch fit is the best method to minimize contact of effluent with the skin (Colwell, 2004). Peristomal skin irritation can also result from mechanical trauma, an allergic reaction to a pouching product, or peristomal fungal infection, which is more common in warm and humid climates or related to antibiotic therapy (Shabbir and Britton, 2010). Fungal infections present as a maculopapular rash with distinct satellite lesions. Allergic reactions can occur with any of the products used to protect the peristomal skin or to assure adhesion of the pouching system; allergic reactions are characterized by pruritus, erythema, and blistering in the area corresponding to and demarcated by contact with the offending agent (Nastro *et al*., 2010).

Treatment involves identification and correction of the causative factors, elimination of any allergens, and treatment of the denuded areas with skin barrier powder or an antifungal powder (e.g., nystatin or miconazole). If necessary, the powder can be lightly blotted with a moist finger or skin sealant to assure a pouchable surface (Erwin-Toth, 2000). Topical steroids may sometimes be required for a severe reaction. Whenever possible, the patient with peristomal skin problems should be seen by an ostomy nurse specialist. Refractory peristomal skin breakdown should prompt a referral to a surgeon with experience and expertise in stoma management.

Parastomal ulceration

Parastomal ulceration, defined as discontinuity of peristomal skin with adjacent inflammation, is usually the result of an infected postoperative hematoma or intestinal fistula (Akinwale *et al*., 2016).

Granulomas

Granulomas are red, moist, elevated lesions at the mucocutaneous border. Often they are a result of retained suture or other extraneous material. Granulomas bleed easily and may be tender. They may become infected. Maintaining barrier seals can be a problem. Treatment consists of examining and removing any extraneous material and eliminating the lesion. Silver nitrate should be applied to remove the elevated tissue. Several treatments may be necessary. After treatment and until healed, the area should be crusted with barrier powder and a skin sealant. If extensive, the pouching system may need to be adjusted until the area heals. Failure of lesions to heal or frequent reoccurrence should be evaluated for other pathology (Carmel *et al*., 2016).

Peristomal pyoderma gangrenosum

Pyoderma gangrenosum (PG) is a neutrophilic dermatosis with unclear etiology. Peristomal pyoderma gangrenosum (PPG) is a subtype of PG that occurs at the stoma site, typically in patients with inflammatory bowel disease (IBD). PPG occurs in 0.5 to 1.5 per million people annually, which accounts for 15 percent of PG cases (Afif *et al*., 2018). According to a 2019 systematic review (79 studies; 335 cases), most PPG patients are female (67 percent), with a mean age of 48 years and a diagnosis of IBD (81 percent; Crohn's disease 50 percent, ulcerative colitis 31 percent)· PPG has also been identified in patients with intra- abdominal malignancies (Hughes *et al*., 2000). Ileostomies, colostomies, and other stomas (urostomies) are involved in 78, 16, and 6 percent of patients.

PPG can develop within weeks to years after stoma construction, with an incidence of <1 percent of stomas (2 to 4 percent in those with IBD) (Walling and Sweet,1987; Lyon *et al*., 2000; Callen 1998). Sixty-nine percent of patients reported an IBD flare with the onset of PPG.The lesions typically present as full-thickness ulcers, and pain and pathergy are dominant characteristics. The diagnosis of PPG is clinical and usually one of exclusion since there is no definitive diagnostic test. The lesion is frequently misdiagnosed as a stitch abscess, contact dermatitis, urinary or fecal fistula, an extension of Crohn's disease, or a wound infection. Patients with suspected PPG should be referred to a dermatologist for possible biopsy (Güenaga *et al*., 2007).

Biopsy of the skin lesion is nondiagnostic but does help to exclude certain pathologies (e.g., cancer, Crohn's disease) (Hughes *et al*., 2000). If a skin biopsy is performed to rule out other pathology, acute and chronic inflammation is the typical finding. The biopsy should be performed with caution as this may increase the size of the wound. Granulomas may be noted on microscopic examination. Biopsies should be performed at the leading edge of the ulceration with 4 to 6 mm punch biopsies. Similarly, cultures should also be obtained to evaluate for any infectious etiologies; PPG ulcers are sterile or grow commensal skin or gut flora. Selecting an unaffected segment of the bowel for the stoma is the best way to prevent this complication. However, once occurring, these lesions are managed by systemic, intralesional, and/or topical anti-inflammatory agents, depending on severity (Afif *et al*., 2018).

Mild cases of PPG without the active systemic disease can be managed with topical agents. Topical therapies for PPG include corticosteroids and calcineurin inhibitors (e.g., tacrolimus) with similar clinical efficacies (62 versus 56 percent). Intralesional injection of corticosteroids is less effective and, in some cases, worsened PPG.

More severe or rapidly evolving cases of PPG require systemic medication or even surgical intervention. Corticosteroids are the most commonly used systemic treatment, with a complete response rate of 52 percent in 146 patients. Other systemic options with a similar (50 percent) response rate include

cyclosporine and dapsone. Systemic metronidazole, azathioprine, sulfasalazine, and tacrolimus are less commonly used. Tumor necrosis factor alpha inhibitors such as infliximab and adalimumab are favorable therapeutic options for PPG concomitant with active IBD and can be used for refractory PPG regardless of whether IBD is present.

Wound care is critical for PPG management. The overall goals are to provide a clean wound environment, absorb exudate, maintain moisture, and prevent further skin damage (Stamm *et al.*, 1995) Care must be taken to minimize trauma since pathergy is a prominent feature of these lesions. Dressings should be nonadherent, and stoma management should be modified to minimize trauma to the affected area (Kiran *et al.*, 2005).

Surgical management may be required for intractability (most common), severe colitis, or complications (e.g., perforation, stenosis, obstruction, herniation). The success rate depends on surgical technique (Afif *et al.*, 2018).

Stoma closure or resection of bowel with active IBD completely healed PPG with few recurrences in small case series.

Stoma relocation resolved PPG in almost all patients, but 67 percent recurred at the new stoma location.

Wound debridement was less effective, associated with only 26 percent complete response.

Continent ileostomy complications

Patients with a Kock pouch or Barnett Continent Ileostomy Reservoir can develop nipple valve slippage, which presents as obstruction or inability to appropriately intubate and evacuate intestinal contents. Surgical management is indicated.

Revision may be possible; however, some situations require resection and reconstruction of a new continent-type ileostomy or end-ileostomy (Beraldo *et al.*, 2006).

Conclusion

Most gastroduodenal perforations are spontaneous from peptic ulcer disease. The management is not standardized as it essentially depends on the clinical scenario and the surgeon's experience. A perforated peptic ulcer is an indication for operation in nearly all cases except when a patient is unfit for surgery. Surgical techniques are varied, but laparotomy and omental patch repair remain the gold standard, while laparoscopic surgery should only be considered when expertise is available. This must be followed by *H. pylori* eradication therapy to prevent a recurrence. Gastrectomy is recommended in patients with large or malignant ulcers to enhance outcomes. Primary closure is achievable in traumatic perforation, but with the exsanguinating critically ill patient in severe major trauma, damage limitation surgery to correct physiology prior to a later anatomical reconstruction is the principle of management.

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