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Digestive Fistulas: The Challenge Continues

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Abstract

The digestive fistulas correspond to abnormal communications between the digestive system and between this and the body surface. Even after many years of study on the subject, digestive fistulas continue to challenge surgeons and patients suffering harm, consuming fortunes in their treatment, as well as causing serious damage affected people, its due to high morbidity and mortality. This review aims to review the literature on the gastrointestinal fistula subject, focusing on etiology, pathophysiology, classification, diagnosis, current therapy and prognosis in order to revisit a highly important topic and continue helping clinicians, general surgeons and device digestive in combating this challenge for doctors and patients.

Keywords: Digestive system fistula; Intestinal fistula; Enterocutaneous fistula; Pathological conditions; Diagnosis; Therapy

Introduction

Dehiscence of anastomoses are the most feared complications in postoperative surgery of the digestive tract [1]. Consist of a structural defect of the intestinal wall, near the site of suture, presenting a communication between intra and extraluminal spaces. When the dehiscence is followed by formation of communication between two adjacent organs or between the organ and the external environment, we call digestive fistula (DF) [2].

Over the past four decades, the mortality associated with gastrointestinal fistulas has diminished from 40%-60% to approximately 15%-20% of patients. This improvement in prognosis is attributable to general advances in fluid and electrolyte/acid-base therapy, blood administration, critical care, ventilator management, antibiotic regimens, and nutritional management [3].

Formerly, malnutrition and electrolyte imbalance were the causes of death in the majority of these patients. In the present era of fistula treatment, mortality is largely

attributable to uncontrolled sepsis and sepsis-associated malnutrition. Sepsis is still responsible for almost 80% of all deaths in fistula patients [4].

Relatively infrequent, it can be associated with a diverse spectrum of presentation ranging from peritonitis and sepsis with radiological findings only, associated with symptoms. A more detailed definition encompasses clinical findings (pain, peritonitis, positive biochemical markers, fever, tachycardia), radiological findings showing fluid collections or containing gas and intraoperative finding [5].

The mechanism of fistula formation is varied. Acquired fistulas may occur as a result of inflammatory disease, abdominal trauma, surgical complications, radiation, and benign or malignant neoplasm. Spontaneous causes account for 15%-25% of gastrointestinal fistulas and include radiation; inflammatory bowel disease; diverticular disease; appendicitis; ischemic bowel; perforation of gastric and duodenal ulcers; pancreatic and gynecologic malignancies; and intestinal actinomycosis or tuberculosis [2,6]

The remaining 75%-85% of gastrointestinal fistulas are of iatrogenic origin and occur as a result of technical complications of surgical procedures and trauma. These include dehiscence of anastomoses; intraoperative injury to the bowel or blood supply; erosion from indwelling tubes; retention sutures or prosthetic mesh; and misplacement of a suture through the bowel during abdominal closure [7,8].

Other technical complications resulting in fistulas are those that occur at delayed periods after surgery, such as intraperitoneal bleeding and abscess formation with or without suture line dehiscence. Fistulas may also develop after drainage of a percutaneous abscess, with a connection created between the intestine and abdominal wall [9].

The fistulas can affect any part of the gastrointestinal tract, with an incidence of 1%-19%, occurring most commonly in esophageal and rectal anastomoses, as compared to other portions [10]. The DF is associated with high morbidity and mortality, resulting in longer hospital stays and costs and greater risk of re-operations and permanent stoma [11]. The purpose of this article is to review the literature on the subject, focusing on etiology, pathophysiology, classification, diagnosis, prognosis and current therapy.

Materials and Methods

References for this article were selected by searching in database sites using Scopus, PubMed, Web of Science and Scielo, using the following key words: digestive system fistula; intestinal fistula; enterocutaneous fistula; pathological conditions; diagnosis; therapeutics. Articles published in the last 10 years, about digestive fistulas and whose access to the full text was free were included. Of these, we selected articles that exposed definition, risk factors, classification, diagnosis and treatment of digestive fistulas. Articles in other languages and time greater than 10 years were excluded.

Etiology and Risk Factors

The anastomotic leakage has a multifactorial etiology, linking intrinsic patient factors and intraoperative factors, which include the technique and experience of the surgeon (**Table 1**). Among the patient-related factors, are nutritional status, steroid use, obesity, smoking, alcoholism, cardiovascular disease, scores of American Society of Anesthesia (ASA) >3, emergency surgery, male gender, advanced age, prior radiation rectal/anal anastomosis and primary disease of the digestive tract, such as Crohn's disease and diverticulitis [12-15].

Table 1 Risk factors for anastomotic fistula.

Patient factors	Nutritional status (malnutrition / obesity)
	Steroid use
	Smoking Alcoholism
	Cardiovascular disease
	ASA score > 3
	Emergency surgery
	Male
	Age
	Prior Radiation
	Anastomotic rectal / anal
	Primary disease of the digestive tract (e.g. Crohn's / Diverticulitis)
Intraoperative factors	Prolonged surgery
	Blood loss
	Drain use
	Vasopressors
	Proximal deviations
	Local blood supply
ASA: American Society of Anesthesiologists	

According to the American College of Surgeons, obesity is the major risk factor for wound dehiscence. Prospective studies showed that anastomotic dehiscence occurred in 33% of obese patients, compared with 15% of non-obese in

colorectal surgery with less than 5cm away from the anal verge. Smoking is responsible for microvascular disease, can cause secondary ischemia, favoring wound dehiscence [16].

The alcohol worsens nutritional status. A multivariate analysis of dehiscence in 1417 colonic resections showed that ASA scores between 3 and 5 associated with emergency procedures are risk factors for its occurrence. Importantly, comorbidities such as diabetes mellitus, hypertension and cardiovascular disease, interfere with the ASA score and affect the microcirculation [1,2].

Among the intraoperative risk factors are prolonged operative time, blood loss, use of vasopressors and drain, proximal diversion and the local blood supply. The use of drains has been a subject of controversy for years [16]. It is believed that they play an important role in draining fluids peri-anastomotic, thereby reducing the formation of abscesses, however, are associated with high incidence of dehiscence [17].

The consensus in general leave the discretion of the surgeon's decision to use them or not. In relation to the use of vasopressors, the contraindication is given by virtue of causing vasoconstriction in the microcirculation, affecting the blood supply. In addition, it has been reported that the use of non-steroidal anti-inflammatory postoperatively, replacing simple analgesics, is associated with higher incidence of this complication [18]. Recent clinical and animal studies show that the effect of these drugs becomes detrimental to the healing process, whose inflammation is necessary and of importance in the early post-surgical. Furthermore, it is known that they have potential to attack the gastrointestinal mucosa and have anticoagulant effect [6,19].

As for proximal diversion, data not yet statistically validated show that the incidence of dehiscence are minor compared to those patients who have not undergone enterostomy deviations [20]. Like dehiscence, fistulas present as risk factors for its emergence whole factor that interferes with the organ vascularization and disrupt the normal healing process [21].

Classification

Gastrointestinal fistulas can be classified by their anatomic characteristics, and they are either internal or external (enterocutaneous). The actual anatomic course of the fistula should be defined. Typically, the name of a fistula is derived from the involved and connected organs or structures [1-3].

The intestinal fistulas can be classified according to the flow for 24 hours: high (greater than 500 mL), moderate (between 200 and 500 ml) and low (less than 200 ml). This classification is able to determine the patient's prognosis and differentiate treatment. They may also be classified according to their anatomic location and etiology (**Table 2**) [17].

Table 2 Classification of intestinal fistula.

Debt of drainage in 24 h	Anatomical location	Etiology
High >500 ml	Internal	Primary-Type I
Moderate 200-500 ml	External	Secondary-Type II
Low <200 ml		
ACS, American College of Surgeons		

The anatomical classification based on which gastrointestinal segment the fistula originated, may have direct communication with the skin (external) or with others adjacent organs (internal) [22]. Concerning etiology, the fistulas are classified into Type I or Primary fistulas resulting from an underlying disease affecting the gastrointestinal wall. Secondary or Type II are the result of the assault on the previously intact body wall, mainly represented by fistulas resulting from surgery with anastomotic dehiscence [17].

An accurate measure of fistula output, as well as the chemical makeup of the effluent, can provide assistance in preventing and treating metabolic deficits and correcting ongoing fluid, electrolyte, and protein losses. The anatomic and etiologic factors are much more important in predicting spontaneous closure than the actual output of the fistula. The underlying disease process helps to prognosticate both the closure rate and mortality [23].

Crohn's disease is one of the responsible for the onset of type I fistulas, one third of enterocutaneous fistulas secondary to it. The terminal ileum is the least affected part of the gastrointestinal tract [24]. The inflammatory process that affects the entire wall thickness promotes the emergence of an ulcer that later evolve into the formation of an abscess, which, upon rupture, creates the path of the fistula, either inside or adjacent viscera for skin [25]. The diverticular disease when complicated, gives rise to colon-bladder fistulas from the communication between the sigmoid colon and bladder. Complications of small and large intestinal fistulas occur with less frequency [26,27].

Massive gastrointestinal hemorrhage can result from the formation of a fistula between the small intestine and a blood vessel. One or more "herald bleeds" may be a prelude to hemorrhage. More commonly, anemia develops chronically and is associated with slow blood loss from a friable fistula tract [24]. Colonization and overgrowth of the small intestine by colonic bacteria can occur with enterocolic fistulas and may result in malabsorption and severe, malodorous diarrhea. Distal obstruction beyond the fistula tract from adhesions or other disease can develop and result in an increase in fistula output or failure of the proximal tract to close. Finally, carcinoma has been reported in chronic fistulas, especially those associated with Crohn's disease [25,26].

Colorectal tumors can develop fistulas to nearby organs such as bladder, vaginal canal, skin and even other bowel [27]. In postoperative colorectal surgery, fistulas are secondary to dehiscence of anastomoses, ranging from 0.5%-30% [28-30].

External or enterocutaneous fistulas are by far the most common type of small intestinal fistula and are usually readily recognizable. In contrast, internal fistulas that communicate between the intestine and another hollow viscus or structure may not be suspected for some time because the symptoms may be minimal or may mimic the underlying disease process [31,32].

Pancreatic fistulas occur in pancreatic tumors, pancreatitis and by blunt or penetrating abdominal trauma [33]. The necrotizing pancreatitis can develop into isolated collections, multiple abscesses and pseudocysts, requiring percutaneous drainage or surgical necrosectomy involving the infectious focus and assisting in faster recovery of the patient. [34]. In cases of distal pancreatic fistulae the surgeon can use anastomoses between the pancreas and intestines or stomach, in order to drain the internal pancreatic secretion, avoiding the formation of collections, abscesses and therefore new fistulas [35]. Anastomosis that allows the escape of pancreatic enzymes out of the lumen is directly and indirectly linked to local and systemic damage as peritonitis, leukocytosis, increased serum amylase, fever, sepsis and organ dysfunction [36-38].

Complications

Fluid and electrolyte

Fluid and electrolyte disturbances occur commonly in patients with enterocutaneous fistulas. Secretions from the salivary glands, stomach, duodenum, pancreas, liver, and small intestine amount to 8-10 L/day, and this fluid is rich in sodium, potassium, chloride, and bicarbonate. The degree of volume depletion and electrolyte imbalance depends on the anatomic location of the fistula and can vary from 50-3000 mL/day [2].

The most common abnormalities seen are hypovolemia, hypokalemia, and metabolic acidosis. Hypokalemia occurs primarily from potassium loss in the fistula effluent, although hypovolemia also contributes by causing renal retention of sodium in exchange for potassium secretion. Sepsis contributes to the hypovolemic state by altering the metabolic rate and increasing insensible water loss [3]. Metabolic acidosis is caused by the loss of pancreatic juice rich in bicarbonate and is thus more common with proximal intestinal fistula [4].

Patients with fistulas causing fluid and electrolyte abnormalities have a higher mortality rate. Advances in critical care, invasive monitoring, and aggressive fluid and electrolyte management can reduce this early mortality considerably [5].

Malnutrition

The loss of luminal nutrients also has a major impact on gut growth and function. The direct effects of luminal nutrients include mucosal cell sloughing and provision of local nutrition to the enterocytes [39]. In addition, nutrients in the gut lumen are known to have trophic effects, such as increasing gastrointestinal hormone and growth factor release that, in

turn, stimulate the paracrine, endocrine, and autocrine effects of the growth factors [40]. Other indirect effects of intraluminal nutrients include increased motility and increased gastrointestinal secretions. Magnesium, selenium, and zinc depletion are common in patients with high-output fistulas and close monitoring is needed. Some individuals with high fistula loss may have high zinc loss in the effluent (12-15 mg/L), worsening output by decreased mucosal regeneration [41]. Nutritional deficiency may be exacerbated by the extra metabolic demands of sepsis or additional surgery. Thus, protein-calorie malnutrition and mineral and micronutrient depletion develop in almost all patients with a small intestinal fistula when a substantial absorptive surface area is bypassed or the enteric contents are lost externally [42].

Abdominal wall/Wound abnormalities

Skin erosion and excoriation commonly occur from an externally draining gastrointestinal fistula. The local digestive action of the gastrointestinal secretions, particularly pancreatic enzymes, can result in considerable discomfort to the patient [43]. The degree of local skin excoriation depends on the output and contents of the fistula effluent and is most severe with proximal intestinal fistulas. Malnutrition contributes to this process by delaying the formation of scar or granulation tissue [44]. Those fistulas that occur in large, open abdominal wall defects are particularly difficult to control as the effluent soils the entire gut surface [45]. Use of novel therapies to isolate these enteroatmospheric fistulas from the neighboring granulating loops of bowel, such as the use of ostomy appliances, duoderm, paste, and hemivacuum therapies, are valuable [46]. Effective isolation of the fistula may require coverage of the remaining wound with a split-thickness skin graft allowing expeditious correction of nutritional, fluid, and electrolyte deficits [47].

Diagnosis and Treatment

Acute intraoperative perforations are best handled by maintaining a strong index of suspicion for technical errors, recognizing the injury before the end of the procedure, and immediately repairing, suturing, or reinforcing weakened tissues [48]. Especially during prolonged laparoscopic procedures, the tendency for potential injuries must be recognized and overcome. Serosal injuries should be carefully examined [49]. Intraluminal instillation of methylene blue and saline or direct endoscopic examination can demonstrate a small perforation or provide reassurance that an area of concern is not a full-thickness injury. During repeat laparotomy for an open abdomen secondary to damage control laparotomy, the urge to break up interloop adhesions to search for interloop abscesses and reaffirm "normal" small intestinal anatomy should be suppressed, as the dense inflammation between the viscera leads to the development of serosal injuries and possible future fistulas [50].

The diagnosis is early clinical, the first 5-10 days, and additional tests can help in the differential diagnosis. The most used image contrast exams are radiography and computed

tomography (CT), with an accuracy of 93% and 94%, respectively. However, both having a sensitivity of only 50% [51]. Postoperatively, unrecognized perforations caused during surgery or leaks that develop at suture or staple lines are manifested as instability or failure to improve as expected. A gastrointestinal fistula can be obvious in some patients and extremely difficult to identify in others. Fistula formation is frequently heralded by fever and abdominal pain until gastrointestinal contents discharge through an abdominal incision or the umbilicus [52]. Spontaneous fistulas from neoplasm or inflammatory disease usually develop in a more indolent manner. Enterocutaneous fistulas often have intestinal contents or gas exiting from a drain site or through the abdominal incision after an operation [53]. The drainage fluid is usually typical of intestinal contents, with obvious bile staining, and intestinal gas may accompany the effluent. At times the initial fistula drainage may appear clear rather than yellow or green, and the fistula may be misdiagnosed as a seroma or wound infection. At other times a heavy purulent component may also mask the enteric communication and instead suggest a wound infection. If the drainage persists and the diagnosis is uncertain, the patient may be given activated charcoal or indigo carmine by mouth and the drainage inspected for these substances [54].

The next phase of management is investigation. After stabilization is accomplished in the first 24-48 hours, investigation usually takes place over the next 7-10 days. Investigation implies a thorough evaluation of the gastrointestinal tract, definition of the anatomy of the fistula, and identification of any complicating features such as abscess, stricture, or distal obstruction. Investigative studies should be designed to determine the presence and location of the fistula and to provide information regarding its cause. This objective can be accomplished by several investigational methods [55].

Oral administration of indigo carmine or charcoal can be used to demonstrate the presence of a connection between the gastrointestinal tract and the abdominal wall or urinary bladder. These tests, however, prove only the presence of a fistula and do not identify its site or source. Probably the most important first test is a fistulogram, which will define the length and width of the fistula, as well as its anatomic location [52,55].

Fistulography performed early in the course of the disease will help determine (1) the site of the fistula, (2) intestinal continuity with the fistula, (3) the presence or absence of distal intestinal obstruction, (4) the nature of the intestine immediately adjacent to the fistula, and possibly (5) the presence or absence of an intraabdominal abscess. Performing the fistulogram first is prudent because contrast from an upper gastrointestinal series, contrast enema, or CT may make it difficult to interpret a fistulogram [55]. Fistulography should be followed by a complete contrast study of the gastrointestinal tract either orally or through existing intraluminal tubes. Such study is valuable both for identifying the internal source of the fistula and for defining its size and complicating factors such as

distal obstruction. Internal fistulas may be more difficult to evaluate [53].

Additional useful tests in the early stage of investigation are CT and ultrasonography. These tests can further define the anatomy of the vicinity of the fistula and evaluate for any ongoing or unrecognized intraabdominal processes or abscesses, as well as distal obstruction. A CT scan will be required in almost all patients for these reasons, especially to rule out any undrained collections [52]. CT scanning with oral and intravenous contrast media is highly sensitive and specific for intraabdominal free air and will assist in locating the fistula and identifying adjacent fluid collections and concomitant bowel obstruction. The use of CT, however, within the first week after surgery is associated with the expected presence of postoperative air within the abdominal cavity and thus may be difficult to interpret. Obviously, extravasation of intraluminal contrast on CT examination is diagnostic of perforation. CT and ultrasound are useful adjuncts when an intraabdominal abscess is suspected [51]. Significant fluid collections should be drained, preferably under CT or ultrasound guidance via a percutaneous route, and an indwelling catheter left in the cavity. This permits subsequent examination of the cavity under fluoroscopy with water-soluble contrast to assist in delineation of the fistula tract [50].

Therefore, the false-negative results must be considered as a possibility. In cases where no sepsis is found, the fistulography is an important test to determine the origin of the fistula, as yet undiscovered, documenting intestinal continuity and verify the presence of distal obstruction [53]. Additionally, ongoing losses must be anticipated and prevented. Malnutrition is easier to prevent than correct. Once established, malnutrition is difficult to correct, especially in the face of continuing sepsis. After the initial stabilization period, including control of sepsis and establishment of nutritional support, management can be divided into phases, starting with determination of the anatomy of the fistula and the likelihood of spontaneous closure [54]. This may then be followed by definitive surgical therapy for a fistula that does not close spontaneously, but a waiting period of at least 6 weeks is usually required. The final process is healing. The critical points in successful management of gastrointestinal fistulas are recognition of the fistula, control of infection and further contamination, restoration of fluid and electrolyte losses, and reestablishment of a positive nutritional balance before undertaking major definitive corrective procedures [56]. Management of a gastrointestinal fistula is a difficult and complex process. However, a systematic approach can lead to treatment that becomes manageable and potentially rewarding. In general, management can be compartmentalized into five stages: stabilization, investigation, decision, definitive therapy, and healing [57].

As outlined earlier, the first step in the management of any intestinal fistula is stabilization of the patient, to be accomplished within the first 24-48 hours of management. These patients are typically in a vulnerable state of health. Patients typically require correction of obligate third-space losses, as well as emesis, fistula output, urine output, or a

combination of these and other causes. Initial efforts should be directed toward intravenous fluid resuscitation, control of infection, ongoing measurement of fistulous and urine output, and protection of the surrounding skin [58]. For surgical treatment of patients with intestinal fistula must be carried nutritional recovery, replacement and stabilization of electrolytes, identification of the fistulous path, drainage adjacent and along the fistula and abscess to allow full closure thereof and the abdominal wall [59,60]. Depending on the site of the fistula, replacement of fistula output varies. High-output fistulas, those exceeding 500 mL/day, continue to result in the highest mortality rate, up to 35%, because of malnutrition, electrolyte imbalance, and sepsis [61]. Moderate-output and low-output fistulas are associated with low mortality rates and higher spontaneous closure rates. Small bowel, pancreatic, and biliary losses are isotonic. Colonic losses may be hypotonic, and gastric fistulas may be associated with the classic hypokalemic, hypochloremic metabolic alkalosis [62].

The natural course of an improperly managed high-output fistula is dehydration, electrolyte abnormalities, malnutrition, infection and sepsis, renal failure, and death. Initial management should address any existing hypovolemia; anemia; hypoalbuminemia; sodium, chloride, or potassium depletion; bile salt losses, and acid-base disorders [1].

Strict intake and output measurements are essential and central venous pressure monitoring and urinary catheterization are especially helpful with high-output fistulas. Invasive monitoring is often necessary because it is usually difficult to estimate antecedent fluid deficits accurately [2]. A central venous catheter can be extremely useful in this capacity and provides the additional benefit of supplying access for parenteral nutrition. The patient's urine output should be restored to greater than 0.5 mL/kg/h, assuming that renal function has not been impaired. Ongoing fluid losses should be fully replaced, and potassium, calcium, phosphorus, and magnesium deficits should be corrected [3]. These electrolyte deficits may take time to correct because the measured serum levels incompletely reflect massive depletion of intracellular ions. Sodium bicarbonate administration may be required to correct the metabolic acidosis that develops with a high-output or proximal fistula. There is no specific hemoglobin or hematocrit level that requires transfusion; rather, transfusion should be based on the patient's overall hemodynamic status, oxygen-carrying capacity, and oxygen delivery [4].

More importantly, however, the patient is in a state of nutritional emergency. For this patient to be stabilized and to potentially heal the fistula, positive nitrogen balance must be achieved. If nutritional therapy is not started early, these patients are at greater risk [14]. With the widespread advent of parenteral nutrition in the 1970s, the overall reduction in mortality to a range of 15%-20% was achieved consistently in a variety of reports, while improving the spontaneous closure rate. Parenteral nutrition, however, had no impact on fistula mortality; maintenance of adequate nutrition with more conventional methods was equally effective [17]. Despite aggressive nutritional support, malnutrition continues to be a

major clinical problem in 55%-90% of patients. Parenteral nutrition has greatly simplified the nutritional management of patients with gastrointestinal fistulas. Even though these patients often have abdominal abscesses and bacteremia, parenteral nutrition is safe and the overall incidence of catheter-related septic complications is no greater than that in other clinical situations [34].

Enteral nutrition is not without complications, however, and the process should be closely monitored. Complications such as diarrhea, aspiration, and bowel ischemia are not uncommon without careful clinical monitoring [20]. Enteral nutrition can be given for upper gastrointestinal fistulas, especially when the feeding tube can be placed beyond the fistula (e.g. a feeding tube placed beyond the ligament of Treitz for a gastric, duodenal, or pancreatic fistula). In general, when possible feeding tubes should be placed beyond the ligament of Treitz to decrease the potential risk for aspiration. If at least 4 feet of functional bowel exists between the ligament of Treitz and the external site of the fistula, enteral feedings of highly absorbable, low-residue nutrients may be administered [22]. Because both enteric and parenteral feeding has advantages and disadvantages, the source of nutritional supplementation should depend on the individual patient and the surgeon's preference and experience. In most cases, parenteral nutrition should be instituted as soon as possible [41]. Thereafter, steps to localize the fistula and control infection can be taken. Normal intestinal motility and function generally return once abdominal sepsis is controlled and fluid and electrolyte imbalances are corrected. If the fistula location is such that enteric access and alimentation are possible, enteral nutrition can be instituted and parenteral nutrition phased out [59]. By using a combination of approaches, adequate nutrition can be maintained throughout the patient's course [1].

It is advised to begin provision of nutritional support as soon as the patient is stabilized. Full caloric and nitrogen replacement can be provided within a few days of instituting nutritional support. Nutrition can be given by several routes [34]. Usually, either enteral tube feeding or parenteral nutrition will be required. The choice of which to use depends on the fistula anatomy. It is advantageous to provide at least a portion of the calories through the enteral route because the gastrointestinal tract is a much more efficacious way of providing nutrition, maintaining the intestinal mucosal barrier and immunologic integrity, and stimulating hepatic protein synthesis, which has been found to be essential in determination of the outcome in patients [3]. Thus, whenever possible, enteral nutrition is preferable to parenteral nutrition and probably decreases the incidence of multisystem organ failure and sepsis if administered appropriately [28].

Uncontrolled sepsis remains the major factor contributing to mortality in patients with small intestinal fistulas. Aggressive management of all ongoing infections and careful surveillance for new septic foci are necessary for successful management. Tachycardia, persistent fever, and leukocytosis usually portend inadequate control of the fistula or abscess formation. Frequent physical examination and judicious use of

ultrasonography and computed tomography (CT) are mandatory [33].

Malnutrition in the presence of uncontrolled sepsis cannot be treated without effective surgical drainage of the septic source. As long as uncontrolled sepsis persists, the patient's condition will continue to deteriorate [39]. The stabilization phase often involves control of a septic source. Typically, drainage of an intraabdominal abscess is required, which is ideally accomplished in an image-guided, percutaneous fashion. In addition, fistula drainage must be controlled and the skin of the abdominal wall protected. Local control is an extremely important component of the early management of a fistula [40].

The best treatment is prevention of complications with any surgical procedure. Intraoperative risk factors and factors related to the patient intervention is possible and better control to avoid deiscences [2]. As a general rule, the type I fistulas require resection of the diseased segment, while the type II fistulas have potential spontaneous closure as conservative treatment [17]. Conservative treatment for late dehiscence and low output fistulas includes adequate percutaneous drainage replacement of fluids and electrolytes, nutritional support and antibiotic therapy for patients with signs of systemic or local inflammation with pain. About a third of enterocutaneous fistulas will close spontaneously with care [3].

The insertion of a catheter allows control of debt and exchange, in the case of fistula, catheter progressively smaller caliber with treatment, follows the path of healing and the evolution of the case [58]. Enteral nutrition should be initiated as soon as possible in patients with a risk factor for fistula, since it constitutes a protective factor [59]. For early anastomotic dehiscence and great debt fistulas, the surgical management is the best option. Surgery gives a definitive diagnosis and institute treatment before a new complication develops [60-62].

Prognosis

The main causes of morbidity following the dehiscence and intestinal fistulae are malnutrition, electrolyte disturbance and sepsis. Nutritional problems are present in more than half of patients and significant loss of bile secretions, pancreatic and/or intestinal exerts considerable impact on treatment outcome. Fistulas of the small intestine have higher rates of mortality and complications compared with colonic fistulas, due to greater debt and association with sepsis and malnutrition [2-4]. Patients who develop wound dehiscence postoperatively present lower functional outcomes to other patients undergoing digestive surgeries with losses in their quality of life. Some patients progress to sepsis, others require ostomies, sometimes permanent, when further surgery is indicated. After the appropriate conservative treatment, it is expected that the digestive fistula close between 4-6 weeks. When it does not, is indicated surgical treatment, which is a real challenge for the surgeon, as this will come across a hostile abdomen, almost impenetrable, full of adhesions

between intestinal loops, which can rupture during the procedure, as well a latent infection, pseudotumor of skeins bowel loops that require drying, which can cause new fistula due to new anastomoses, and wound infection, new adhesions of intestinal obstruction syndrome, among other complications [3].

The patient care should be individualized according to the underlying disease, age, comorbidities and treatment response. It requires a lot of dedication and time commitment of the multidisciplinary team to ensure the best results when treating.

Conclusion

Digestive fistulas have multifactorial etiology and can affect any part of the gastrointestinal tract. Digestive fistulas are a common, complex problem in the surgical patient population. Diligent control of sepsis, electrolyte imbalance, fistula output, and nutrition management can significantly reduce the patient's morbidity and mortality. A patient's course can be optimized through definitive surgical repair when spontaneous closure does not occur. They are expected to have diverse manifestations among patients, since these can also have varying comorbidities. Thus, these factors hinder the cohesion of measures in a rigid protocol, able to cover all diversities. Therefore, digestive fistulas are a vast field of research to improve, not only the diagnosis and treatment, but especially its prevention.

References

1. Kingham TP, Pachter HL (2009) Colonic anastomotic leak: risk factors, diagnosis and treatment. *J Am Coll Surg* 208: 269-276.
2. Girard E, Messager M, Sauvanet A (2014) Anastomotic leakage after gastrointestinal surgery: diagnosis and management. *J Visc Surg* 151: 441-450.
3. Kwon SH, Oh JH, Kim HJ (2008) Interventional management of gastrointestinal fistulas. *Korean J Radiol* 9: 541-549.
4. Tong CY, Lim LL, Brody RA (2012) High output enterocutaneous fistula: a literature review and a case study. *Asia Pac J Clin Nutr* 21: 464-469.
5. Kulu Y, Buchler MW, Ulrich A (2015) Perioperative complications of the lower gastrointestinal tract : Prevention, recognition and treatment. *Chirurg* 86: 311-318.
6. Yauw ST, Wever KE, Hoesseini A (2015) Systematic review of experimental studies on intestinal anastomosis. *Br J Surg* 102: 726-734.
7. Genser L, Manceau G, Karoui M (2013) Postoperative and long-term outcomes after redo surgery for failed colorectal or coloanal anastomosis: retrospective analysis of 50 patients and review of the literature. *Dis Colon Rectum* 56: 747-755.
8. Williams LJ, Zolfaghari S, Boushey RP (2010) Complications of enterocutaneous fistulas and their management. *Clin Colon Rectal Surg* 23: 209-220.
9. Njeze GE, Achebe UJ (2013) Enterocutaneous fistula: a review of 82 cases. *Niger J Clin Pract* 16: 174-177.
10. Aurello P, Magistri P, D'Angelo F (2015) Treatment of esophagojejunal anastomosis leakage: a systematic review from the last two decades. *Am Surg* 81: 450-453.
11. Cadeddu F, Salis F, Lisi G (2015) Complex anal fistula remains a challenge for colorectal surgeon. *Int J Colorectal Dis* 30: 595-603.
12. Tirumani SH, Baez JC, Jagannathan JP (2013) Tumor-bowel fistula: what radiologists should know. *Abdom Imaging* 38: 1014-1023.
13. Ribièrè S, Bouhnik Y (2014) Management of intestinal complications in inflammatory bowel diseases. *Rev Prat* 64: 1249-1255.
14. Juncadella AC, Alame AM, Sands LR (2015) Perianal Crohn's disease: a review. *Postgrad Med* 127: 266-272.
15. Pigot F (2015) Treatment of anal fistula and abscess. *J Visc Surg* 152: S23-S29.
16. Llop JM, Cobo S, Padullés A (2012) Nutritional support and risk factors of appearance of enterocutaneous fistulas. *Nutr Hosp* 27: 213-218.
17. Schechter WP, Hirshberg A, Chang DS (2009) Enteric fistulas: principles of management. *J Am Coll Surg* 209: 484-491.
18. Slade D, Scott N (2008) Intestinal Fistulas. *Surgery* 26: 343-346.
19. Fujita F, Torashima Y, Kuroki T (2014) Risk factors and predictive factors for anastomotic leakage after resection for colorectal cancer: reappraisal of the literature. *Surg Today* 44: 1595-1602.
20. Martinez JL, Luque-de-León E, Ballinas OG (2012) Factors predictive of recurrence and mortality after surgical repair of enterocutaneous fistula. *J Gastrointest Surg* 16: 156-163.
21. Pfeifer J, Tomasch G, Uranues S (2011) The surgical anatomy and etiology of gastrointestinal fistulas. *Eur J Trauma Emerg Surg* 37: 209-213.
22. Hirst NA, Tiernan JP, Millner PA (2014) Systematic review of methods to predict and detect anastomotic leakage in colorectal surgery. *Colorectal Dis* 16: 95-109.
23. Hyman NH (2008) Managing anastomotic leaks from intestinal anastomosis. *J Royal Coll Surg Edinb Ireland* 7: 31-35.
24. Ong EM, Ghazi LJ, Schwartz DA (2015) Crohn's & Colitis Foundation of America, Inc. Guidelines for imaging of Crohn's perianal fistulizing disease. *Inflamm Bowel Dis* 21: 731-736.
25. Pellino G, Selvaggi F (2014) Surgical treatment of perianal fistulizing Crohn's disease: from lay-open to cell-based therapy--an overview. *Scientific World Journal* 2014: 146281.
26. Wise PE, Schwartz DA (2012) The evaluation and treatment of Crohn perianalfistulae: EUA, EUS, MRI, and other imaging modalities. *Gastroenterol Clin North Am* 41: 379-391.
27. Fujita F, Torashima Y, Kuroki T (2014) Risk factors and predictive factors for anastomotic leakage after resection for colorectal cancer: reappraisal of the literature. *Surg Today* 44: 1595-1602.
28. Boccola MA, Lin J, Rozen WM (2010) Reducing anastomotic leakage in oncologic colorectal surgery: An evidence-based review. *Anticancer Res* 30: 601-608.
29. Davis B, Rivadeneira DE (2013) Complications of Colorectal Anastomoses: leaks, strictures, and bleeding. *Surg Clin North Am* 93: 61-87.

30. Tsiftsis DD, Christodoulakis M, Taflampas P (2009) Anastomotic Leakage after Low Anterior Resection for Rectal Cancer: Facts, Obscurity, and Fiction. *Surg Today* 39: 183-188.
31. Cirocchi R, Cochetti G, Randolph J (2014) Laparoscopic treatment of colovesical fistulas due to complicated colonic diverticular disease: a systematic review. *Tech Coloproctol* 18: 873-885.
32. Tirumani SH, Baez JC, Jagannathan JP (2013) Tumor-bowel fistula: what radiologists should know. *Abdom Imaging* 38: 1014-1023.
33. Malleo G, Pulvirenti A, Marchegiani G (2014) Diagnosis and management of postoperative pancreatic fistula. *Langenbecks Arch Surg* 399: 801-810.
34. Hackert T, Büchler MW (2015) Management of postoperative pancreatic fistula. *Chirurg* 86: 519-524.
35. Peng SY, Wang JW, Hong DF (2011) Binding pancreatocenteric anastomosis: from binding pancreaticojejunostomy to binding pancreaticogastrostomy. *Updates Surg* 63: 69-74.
36. Jin K, Zhou H, Zhang J (2015) Systematic review and meta-analysis of somatostatin analogues in the prevention of postoperative complication after pancreaticoduodenectomy. *Dig Surg* 32: 196-207.
37. Varadarajulu S, Rana SS, Bhasin DK (2013) Endoscopic therapy for pancreatic duct leaks and disruptions. *Gastrointest Endosc Clin N Am* 23: 863-892.
38. Tu Y, Jiao H, Tan X (2013) Retroperitoneal laparoscopic debridement and drainage of infected retroperitoneal necrosis in severe acute pancreatitis. *Asian J Surg* 36: 159-164.
39. Polk TM, Schwab CW (2012) Metabolic and nutritional support of the enterocutaneous fistula patient: a three-phase approach. *World J Surg* 36: 524-533.
40. Sepehrpour S, Papagrorgoriadis S (2010) A systematic review of the benefit of total parenteral nutrition in the management of enterocutaneous fistulas. *Minerva Chir* 65: 577-585.
41. Rodríguez Cano AM (2014) Nutrition therapy in enterocutaneous fistula; from physiology to individualized treatment. *Nutr Hosp* 29: 37-49.
42. Wang Y, Kang Y, Zhou JR (2013) Comparison of two types of enteral nutrition in patients with intestinal fistula. *Zhonghua Yi Xue Za Zhi* 93: 2364-2366.
43. Friese RS (2012) The open abdomen: definitions, management principles, and nutrition support considerations. *Nutr Clin Pract* 27: 492-498.
44. Butler CE, Baumann DP, Janis JE, Rosen MJ (2013) Abdominal wall reconstruction. *Curr Probl Surg* 50: 557-586.
45. Gomes CA Jr, Andriolo RB, Bennett C (2015) Percutaneous endoscopic gastrostomy versus nasogastric tube feeding for adults with swallowing disturbances. *Cochrane Database Syst Rev* 5: CD008096.
46. Lee SH (2012) Surgical management of enterocutaneous fistula. *Korean J Radiol* 13: S17-S20.
47. Curtis K, Judson K (2014) Management of fistulae in patients with open abdomen. *Nurs Stand* 28: 56-58.
48. Rahman FN, Stavas JM (2015) Interventional radiologic management and treatment of enterocutaneous fistulae. *J Vasc Interv Radiol* 26: 7-19.
49. Tirumani SH, Shinagare AB, Jagannathan JP (2014) Multidetector-row CT of tumour-bowel fistula: Experience at a tertiary cancer centre. *Clin Radiol* 69: e100-e107.
50. Joyce MR, Dietz DW (2009) Management of Complex Gastrointestinal Fistula. *Curr Probl Surg* 46: 384-430.
51. Khati NJ, Sondel Lewis N, Frazier AA (2015) CT of acute perianal abscesses and infected fistulae: a pictorial essay. *Emerg Radiol* 22: 329-335.
52. Baudin G, Chassang M, Gelsi E (2012) CT-guided percutaneous catheter drainage of acute infectious necrotizing pancreatitis: assessment of effectiveness and safety. *AJR Am J Roentgenol* 199: 192-199.
53. Siddiqui MR, Ashrafian H, Tozer P (2012) A diagnostic accuracy meta-analysis of endoanal ultrasound and MRI for perianal fistula assessment. *Dis Colon Rectum* 55: 576-585.
54. Vanbeckevoort D, Bielen D, Vanslebrouck R (2014) Magnetic resonance imaging of perianal fistulas. *Magn Reson Imaging Clin N Am* 22: 113-123.
55. Faccioli N, Foti G, Molinari E (2012) Role of fistulography in evaluating pancreatic fistula after pancreaticoduodenectomy. *Br J Radiol* 85: 219-224.
56. Manta R, Magno L, Conigliaro R (2013) Endoscopic repair of post-surgical gastrointestinal complications. *Dig Liver Dis* 45: 879-885.
57. Terzi C, Egeli T, Canda AE (2014) Management of enteroatmospheric fistulae. *Int Wound J* 11: 17-21.
58. Vakalopoulos KA, Daams F, Wu Z, Timmermans L, Jeekel JJ, et al. (2013) Tissue adhesives in gastrointestinal anastomosis: a systematic review. *J Surg Res* 180: 290-300.
59. López J, Rodríguez K, Targarona EM (2015) Systematic review of cyanoacrylate embolization for refractory gastrointestinal fistulae: a promising therapy. *Surg Innov* 22: 88-96.
60. Rahman FN, Stavas JM (2015) Interventional radiologic management and treatment of enterocutaneous fistulae. *J Vasc Interv Radiol* 26: 7-19.
61. Cirocchi R, Trastulli S, Morelli U (2013) The treatment of anal fistulas with biologically derived products: is innovation better than conventional surgical treatment? An update. *Tech Coloproctol* 17: 259-273.
62. Bada YO, Téllez-Avila FI, Takahashi-Monroy T (2009) Surgical and medical treatment outcome of enterocutaneous fistulas. *Acta Gastroenterol Latinoam* 39: 273-277.