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Management of Intestinal Obstruction

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<http://dx.doi.org/10.5772/63156>

Abstract

Objective: Intestinal obstruction is a blockage of the intestinal content through bowel. The block must be complete and permanent. Obstruction may be mechanical, simple or strangulated, and paralytic. The purpose of this chapter is to clarify, also evaluating our surgical experience, the steps to diagnose and the ways to treat intestinal obstructions.

Methods: In the period 2011–2015, we have treated 52 patients with the clinical presentation of intestinal obstruction. Acute mechanical small bowel obstructions were the most frequent (71.2%), whereas acute large bowel obstructions were the less frequent (28.8%). Some steps in the physical examination and plain radiography are as follows:

- Preliminary diagnosis: simple versus strangulated obstruction.
- First-step diagnosis: mechanical or paralytic obstruction.
- Second-step diagnosis (define the level of obstruction).
- Third-step diagnosis: kind of obstacle.

Results: In our experience, all complete intestinal obstructions have been treated with urgent surgery. Various types of surgical procedures have been employed based on intraoperative pathological findings

Conclusion: Surgical emergency is the first choice of treatment in strangulation obstruction and in simple (complete) mechanical obstruction of small and large bowel. Generally, paralytic ileus can be resolved with the treatment that caused it. Intestinal pseudo-obstruction is a syndrome characterized by a complete dilatation generally of large bowel without mechanical obstacle. The chronic pseudo-obstruction can be idiopathic or secondary to systemic disease.

Keywords: intestinal management, obstruction, pathophysiology, strangulation, intestinal pseudo-obstruction

1. Introduction

Intestinal obstruction is a syndrome characterized by a blockage of the intestinal content, gas and liquid, through small or large bowel. The block must be complete and permanent.

There are several data that can diversify the intestinal obstruction syndrome. First, the etiology based on a large number of factors that allow the subdivision into mechanical and functional/paralytic obstruction.

The other feature characterizes the syndrome: the seat of the obstruction along the bowel—upper small gut, distal small gut, and large bowel.

Finally, the cause of obstruction can involve the vascular supply of an intestinal segment, giving rise to strangulation obstruction that should be differentiated from simple obstruction.

The syndrome of intestinal obstruction with these various etiopathological and clinical features develops the same, overlappable, and pathophysiological alterations.

2. Etiology

Bowel obstruction can be caused by several factors.

The causes of mechanical obstruction can be divided into causes within the bowel lumen, causes in the intestinal wall, and extrinsic causes.

The causes within the bowel are infrequent. They can be due to large gallstones passed into the intestinal lumen by spontaneous bilio-digestive fistulas, most frequently cholecystoduodenal fistulas, very rarely phito-thricobezoar, masses of parasites, food bolus, concretions of barium following barium enema X-ray investigation or X-ray studies with opaque medium.

It is useful to point out that the fecaloma, fecal impaction in the rectal ampulla, based on the damage of the autonomic nervous system in the colorectal wall, can cause chronic alteration of intestinal transit with incomplete obstruction without the pathophysiological alterations of acute gut obstruction.

The causes in the gut wall include the neoplasms of small and large bowel, the congenital atresias, the stenosis due to chronic inflammatory disease (Crohn disease, diverticulitis, etc.), and postanastomotic or posttraumatic structures.

The extrinsic causes include a very large range of pathological conditions: compression by external masses, adhesions, bands, strangulated external or internal hernias, volvulus, and intussusception [1]. In the clinical practice, it should be valuable to distinguish between acute and chronic obstructions. Nevertheless to define both clinical pictures with accuracy is very useful. The acute intestinal obstruction, simple or strangulated forms, is characterized by complete and permanent blockage and consequently the acute pathophysiological syndrome of obstruction may develop with all clinical, laboratory, and instrumental features: bowel

dilatation, disturbances of fluids and electrolytes balance, congestion, and ischemic parietal damage, etc.

On the contrary, in the chronic obstruction, the blockage of gut transit is incomplete and the syndrome cannot develop completely and is characterized only by constipation.

3. Classification of intestinal obstruction

Intestinal obstruction may be mechanical or paralytic. Mechanical obstruction can be due to intraluminal, intrinsic to the intestinal wall and extrinsic.

Paralytic ileus due to reduction or the absence of peristalsis can be caused by peritoneal phlogosis, infection, abdominal surgery, pelvic surgery, and some medications such as antidepressant, pain medications, muscle and nerve disorders, and retroperitoneal hemorrhage.

The majority of patients have simple obstruction. On the contrary, there is also strangulation obstruction, usually due to complicated external hernia (abdominal wall) or internal (by congenital defects or postoperative adhesions): in these patients the vascular supply to a strangulated intestinal segment is compromised and consequently intestinal infarction. Strangulation obstruction leads to an increased risk of morbidity and mortality.

In the mechanical occlusion with strangulation, the vascular (arterial and venous) occlusion leads to bowel ischemia and necrosis. The evolution of strangulated bowel is the perforation and peritonitis. The occlusive syndrome becomes worse due to strangulation.

Intestinal pseudo-obstruction is a syndrome characterized by a complete dilatation generally of large bowel without mechanical obstacle. The intestinal pseudo-obstruction can affect small or large bowel and it may be possible to differentiate the syndromes with acute or chronic onset and evolution.

The chronic pseudo-obstruction can be idiopathic or secondary to systemic disease.

4. Epidemiology

The examination of homogeneous clinical cases of a single center allows us to clarify the epidemiological features.

In the period 2011–2015, 52 patients have been admitted in our service with the clinical presentation of intestinal obstruction.

Demographic data are as follows: 52 patients, 26 males, 26 females, and mean age 67 years (range 27–86 years).

Acute mechanical small bowel obstruction was the most frequent (71.2%) with various pathologies: adhesion-related obstructions, small bowel volvulus, gallstones ileus, malignan-

cies, abdominal wall hernias, internal hernias, carcinomatosis, and ileocecolic intussusception (**Figure 1**).

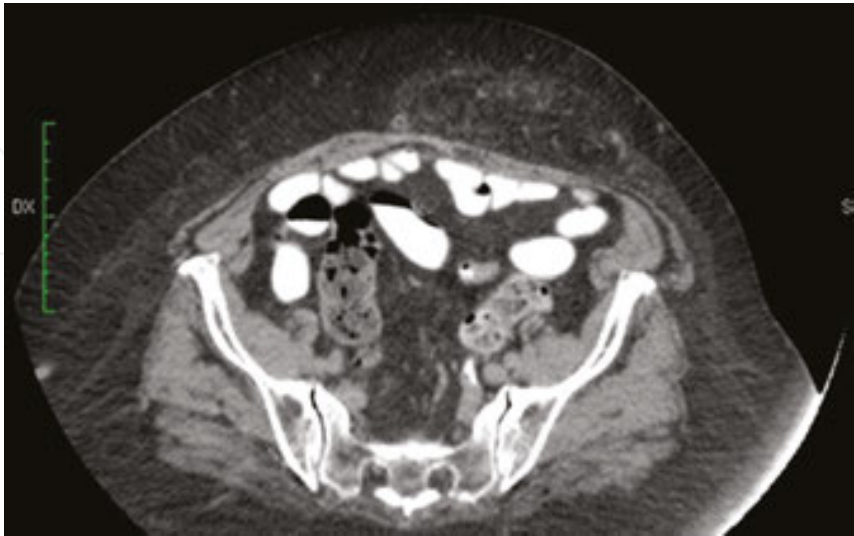


Figure 1. CT scan: ileocecolic intussusceptions.

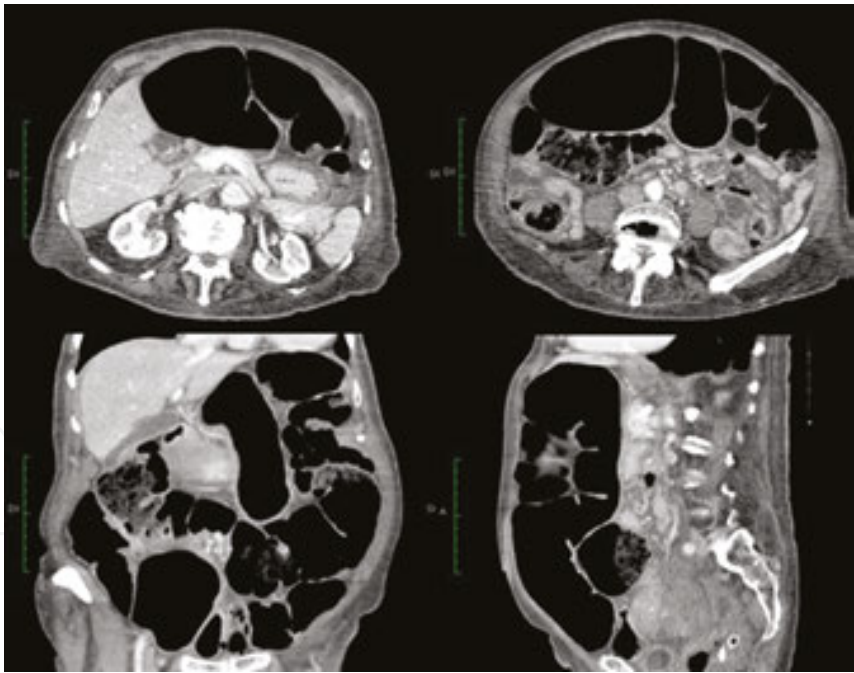


Figure 2. CT scan: sigmoid volvulus.

The less frequent in our experience were acute large bowel obstructions (28.8%). The more common pathologies were colon and rectal cancer, sigmoid volvulus (**Figure 2**). We have observed and treated only one patient with acute colonic pseudo-obstruction (Ogilvie’s syndrome) (**Table 1**).

	<i>n</i>	%
Adhesions	22	42.3
Small bowel volvulus	4	7.7
Sigmoid volvulus	1	1.9
Right colon cancer	3	5.8
Left colon cancer	5	9.6
Rectal cancer	5	9.6
Ogilvie's syndrome	1	1.9
Gallstones ileus	2	3.8
Carcinomatosis	1	1.9
Strangulated incisional hernia	3	5.8
Strangulated groin hernia	2	3.8
Strangulated umbilical hernia	1	1.9
Internal hernia	1	1.9
Ileoceocolic intussusception	1	1.9
Total	52	

Table 1. Fifty-two acute intestinal obstructions: pathologies.

	<i>n</i>	%
Adhesiolysis	18	34.6%
Small bowel resection	6	11.5
Right hemicolectomy	4	7.7
Left hemicolectomy	5	9.6
Intestinal derotation	4	7.7
Cecostomy	2	3.8
Enterotomy and gallstone removal	2	3.8
Anterior rectum resection	4	7.7
Total colectomy	1	1.9
Prosthetic mesh repair incisional hernia	3	5.8
Umbilical hernia repair	1	1.9
Prosthetic mesh repair groin hernia	2	3.8
	52	

Table 2. Fifty-two acute intestinal obstructions: surgical interventions.

In our experience, all intestinal obstructions have been treated with urgent surgery. Various types of surgical procedures have been employed based on intraoperative pathological findings. They should be useful to underline that the choice of the urgent treatment allowed the resolution of majority of adhesion-related obstructions and small bowel volvulus with the surgical procedure of adhesiolysis and intestinal derotation (42.3%). The intestinal resection has been performed in six cases (11.5%). Ogilvie’s syndrome required cecostomy; colorectal malignancies have been treated with primary tumors resection and delayed intestinal anastomosis. Our surgical interventions are reported in **Table 2**.

5. Pathophysiology of simple intestinal obstruction

In simple obstruction, important and progressive alterations take place and develop in the gut above the obstruction. Accumulation of gas and liquids with progressive distension in intestinal segments upstream the obstruction and the blockage of content progress change the bacteriological content. Also there are damages of blood circulation in the distended bowel wall. The accumulation of fluid and electrolytes in the obstructed gut and their loss in the general circulation take an important place in the pathophysiology of intestinal obstruction [2, 3].

5.1. Distension

The distension of obstructed bowel above the obstacle is due to the accumulation of fluid and gas. The intestinal gas that normally progresses by peristaltic movements shows the following composition at the start of occlusion: swallowed air, carbon dioxide (it comes from neutralization of bicarbonates), and later on gas bacterial fermentation. The carbon dioxide that forms in large quantities in the intestinal cavity participates minimally gaseous accumulation because it is largely absorbed by the intestinal mucosa.

Nitrogen N ₂	70%
Oxygen O ₂	10–12%
Carbone dioxide CO ₂	6–9%
Hydrogen H ₂	1%
Methane CH ₄	1%
Hydrogen disulphide	1–10%

Table 3. Intestinal obstruction: composition of intestinal gas.

On the contrary, the swallowed air significantly contributes to gaseous bowel distension because it contains a high rate of nitrogen content, which is not absorbed: in fact about 70% of the intestinal gas is constituted by nitrogen.

For this reason, the nasogastric aspiration in the patients with intestinal distension should be relevant and useful.

A little advantage can be added to gastric aspiration by the administration of pure oxygen to distended patients because increasing the pressure of a gas increases its solubility [4].

The common composition of intestinal gas in obstruction is reported in **Table 3** [5, 6].

5.2. Fluid loss

In the distended bowel above, the obstruction gastrointestinal secretions accumulate in large amounts. This occurs for two reasons: deprivation of the absorptive activity of intestine beyond the obstruction and also damage in fluid and electrolyte exchange in the wall of the obstructed and distended gut [7].

Source	Volume (ml)
Saliva	1500
Gastric secretion	2500
Bile	500
Pancreatic juice	700
Intestinal mucosa secretion	3000
Total	8200
Normal plasmatic volume	3500

Table 4. Volume of digestive secretion per day.

The saliva, gastric secretion, bile, pancreatic juice, and small intestinal secretion accumulate the total volume of about 8000 ml in 24 h as reported in more detail in **Table 4**. These fluid secretions are isotonic with the plasma, except for gastric secretion, which has minor sodium concentration (**Figure 3**) [8].

Therefore, normally water and electrolytes absorption is almost complete in the colon. In small bowel obstruction, the function of the colon cannot develop and the total intestinal secretions accumulate in the obstructed gut.

Beside the decreased absorption in the obstructed patients, there is also, above the obstacle, increased secretion into the bowel lumen.

In the pathophysiology of intestinal obstruction, the fluid and electrolytes loss plays a very important role. The progressive accumulation of gas and fluid in the intestinal lumen allows the increase in the endoluminal pressure to very high values: in the small bowel the pressure can reach 15 cm H₂O, whereas in the colon it can reach 25 cm H₂O. In the initial period of mechanical obstruction in the intestinal segments above the obstacle, active peristalsis causes further pressure rise to 20–30 cm H₂O [9]. In the large bowel, the intraluminal pressure can reach 50 cm H₂O because the pressure increase is based on the product of the pressure value

multiplied by bowel diameter. The major detrimental effect of the progressive gut distension and intraluminal pressure increase is the impairment of the intramural circulation of the bowel.

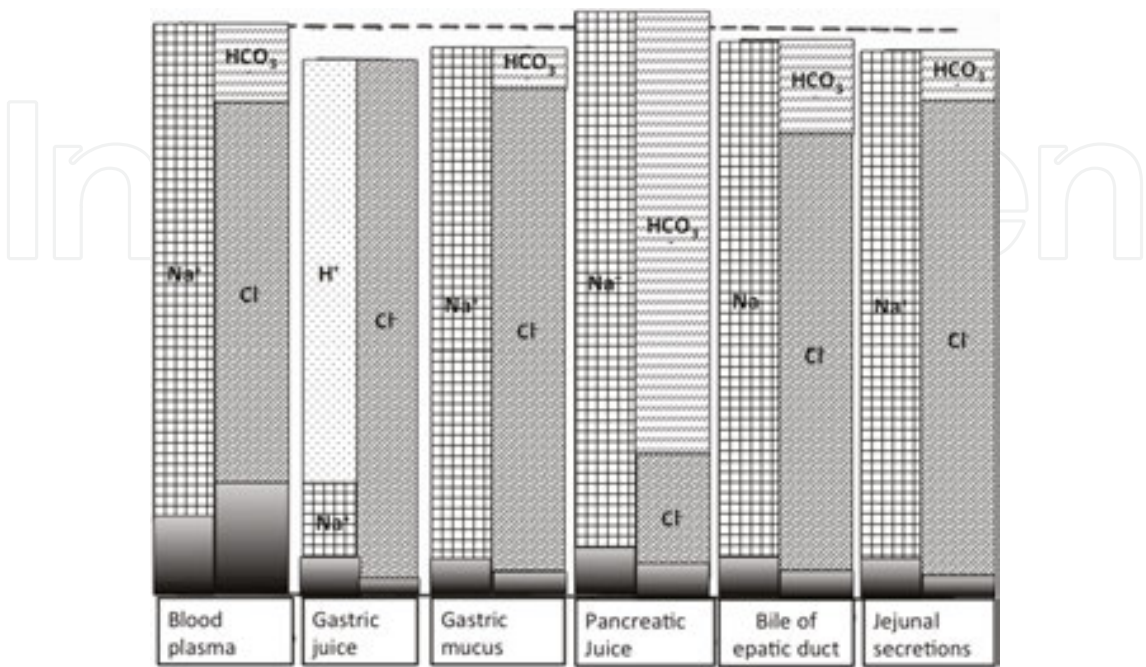


Figure 3. Electrolytes composition in blood plasma and digestive secretion.

5.3. Pathophysiology of circulatory changes in the distended bowel

The experimental studies demonstrated the linear connection between bowel obstruction, increase in intraluminal pressure, gut wall distension, changes in blood flow [10]. In the clinical situations, there are some difficulties to connect the degree of intraluminal pressure, intestinal distension, and the damage of parietal blood perfusion.

In the pathological setting, the increase in the intraluminal pressure should develop slowly and should not reach high degree [11, 12].

The intestinal wall distension in the obstructed patients causes increased distensibility of the gut wall that becomes more vulnerable to a further increment of distension. In this way, a small rise in the intraluminal pressure and the wall distension allows considerable tension in the intestinal wall and increased resistance in the capillaries with damage bowel blood flow.

The ischemic necrosis of obstructed bowel should be caused by progressive thinning of gut wall, reduction of the lumen of vessels, and finally interruption of the blood supply [4].

In the pathogenetic sequence, start a self-handing mechanism because the parietal distension increases the intestinal secretions with further intraluminal fluid accumulation, increased wall distension, impaired parietal blood flow, and finally hypovolemia.

The intestinal secretions enhance because the capillary leak increases the fluid flux to intestinal lumen [12, 13].

Beside fluid and electrolytes accumulation in the obstructed bowel, there is further fluid loss with vomiting. The metabolic effects of electrolytes and fluid loss, subtracted from the circulating blood volume and interstitial spaces, depend on the duration and site of obstruction. Proximal small bowel obstructions cause early and abundant vomiting with fluid, Cl, Na, and K loss, and consequently dehydration, hypokalemia, hyponatremia, hypochloremia, and metabolic alkalosis. In these proximal obstructions, the gut distension is less evident. On the contrary, the distal small bowel obstructions show more evident gut distension but the fluid and electrolytes depletion develop slowly, based on late vomiting and longer preserved resorption capacity. The dehydration causes hypovolemia, tachycardia, renal failure, decrease in the central venous pressure (CVP), and cardiac output, finally the hypovolemic shock. Moreover, the bowel distension may cause increase in the endoabdominal pressure with damage of venous return and pulmonary ventilation.

5.4. Bacteriology

Normally, the small intestine contains only transient bacterial flora with scanty growth because of the fluid content and fast transit. In fact, bacteria traverse the small intestine so rapidly that significant growth does not occur.

Instead in the case of obstruction and stasis, proliferation by geometrical progression results in rapid colonization of the intestinal lumen [14, 15].

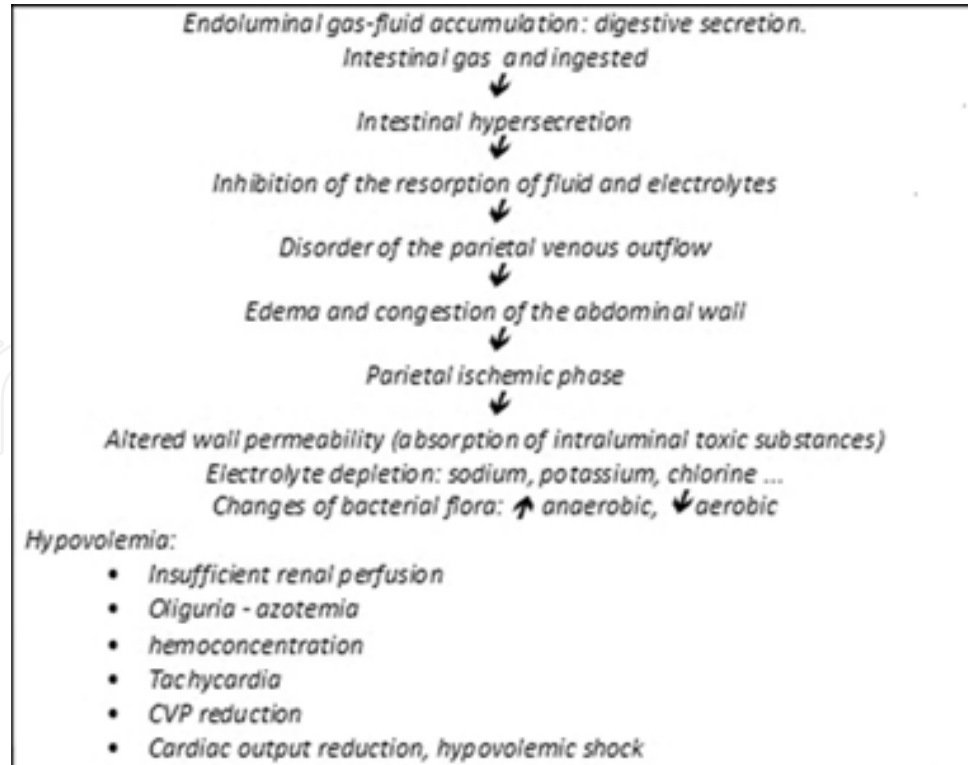


Figure 4. Intestinal obstruction: pathophysiological features.

Moreover, the increase in bacterial flora is particularly evident in the anaerobic organisms such as *Bacteroides*, *Coliforms*, and *Clostridia* [16]. The considerable increase in bacteria proliferation does not show clinical effects in the first phase of simple intestinal obstruction before the anatomical and functional impairment of intestinal wall.

The pathophysiological features in intestinal obstruction are summarized in **Figure 4**.

6. Pathophysiology of strangulation obstruction

The interruption of blood flow in an intestinal segment beside the lumen obstruction characterizes the strangulation obstruction. The most frequent causes of strangulation obstruction are incarcerated external hernias (abdominal wall: inguinal, femoral, umbilical, and incisional hernias), internal hernias (fibrous band, paraduodenal, foramen of Winslow, pericecal, intersigmoid, transmesenteric, and retroanastomotic), volvulus, and intussusception.

The pressure in the obstructed intestinal segment exceeds very quickly the pressure venous in the bowel wall and in the corresponding mesentery. The following step is the venous blockage in these vessels, then capillary rupture with hemorrhagic infarction in the submucosa, mucosa, and finally in all layers of intestinal wall.

The ischemic evolution of this condition is preceded by intramural thrombosis veins and is completed with the necrosis that proceeds from the mucosa to the serosa.

In the necrotic intestinal segment, the perforation can occur followed by severe septic peritonitis. Before this final lethal conclusion, the severe septic-toxic conditions can develop in the strangulation occlusion.

The damage of the blood supply and the normal function of intestinal wall allow serious consequences with transudation of toxic materials from Gram-positive and especially Gram-negative anaerobic organisms of intestinal obstructed lumen across the bowel wall in the peritoneal cavity. The systemic effects of the absorption by peritoneal serosa of toxic material are serious hemodynamic alterations, hypovolemia, hypotension, and septic shock. The role of intestinal bacterial flora in the production of the toxic transudation has been demonstrated in the past by Cohn, based on the protective action of antibiotics [17]. Beside the toxic systemic compromissions, in the strangulation obstruction, the metabolic consequences of fluid and electrolytes loss also develop such as in the simple intestinal obstruction.

7. Pathophysiology of large bowel obstruction

The pathophysiological syndrome of large bowel obstruction in general terms can be overlapped by small bowel obstruction but differs in the time in which it develops. The obstruction of the right colon is quite similar to distal small bowel obstruction in the pathophysiological evolution.

The distal large bowel obstructions (left, sigmoid colon, and rectum) instead show characteristic pathophysiological and clinical data.

Large bowel obstruction follows a slower course. The symptoms of dehydration are less severe. In the early stages, the colon retains the absorption capacity of fluid and electrolytes. For this reason, it preserves normal blood electrolyte concentration; therefore, the isotonic loss of water and electrolytes is associated with decreased plasma volume: hemoconcentration, decrease of CVP, and oliguria.

In the obstructed colon, the gaseous distension and the endoluminal pressure increase progressively. Consequently, it can develop damage of the blood flow in the parietal vessel earlier and more evident than in the small gut because the colon has the lowest blood flow across the abdominal viscera. The ischemia interests before the mucosa, impairing its functions; furthermore, this condition points out that the intraoperative evaluation of colonic blood perfusion cannot be based only on an external examination of serous membrane.

The increasing endoluminal pressure in the intestinal segment with thin walls, such as cecum, can cause perforation and septic peritonitis. Also in large bowel obstruction, the blockage of intestinal content increases the growth of bacterial flora, and the damage of intestinal wall functions allows the absorption of septic-toxic fluid intestinal content. In the beginning, only the colon is distended but usually the ileocecal valve becomes incompetent and allows the dilatation to progress proximally into the small gut. The presentation of the clinical picture of “closed-loop obstruction” without distension of small bowel that preserves its functions for a short period is quite unusual. This condition is due to competent ileocecal valve with a double obstruction, the valve and colonic obstruction, and the risk in the closed loop with an increase in the intraluminal pressure, obstruction of blood supply, gangrene, perforation, and peritonitis.

The obstructions of large bowel in the majority of cases are due to neoplastic diseases, adhesions, and volvulus.

In the large bowel obstruction, the cramping pain occurs longer than small bowel occlusion.

Colonic volvulus (cecal or sigmoid) are characterized by a great dilatation of cecum or sigmoid colon on imaging exams (plain radiography—CT scan). The neoplastic obstruction of distal colon or rectum shows great distension of the colon above the obstacle and chronic and progressive symptoms of constipation with the change of regular bowel function toward constipated bowel function, changes of stool caliber, long-term cathartic use. Abdominal CT scan can demonstrate a mass as a cause of large bowel obstruction and synchronous lesions as metastases and enlarged lymph nodes.

8. Pathophysiology of paralytic obstruction of the intestines (adynamic ileus)

There are distension and vomiting in this form of obstruction of the bowel but no mechanical obstruction. The adynamic ileus is due to a paralysis of the musculature of the bowel. Hypo-

kalemia causes intracellular reduction of potassium that is replaced by sodium and consequently depolarization of electric potentials of membranes of muscle and nerve cells, which aggravates the intestinal paralysis.

Intestinal motility has dual adjustment, central and peripheral or autonomous: this explains the variability of causes and stimuli that provoke a reflex paresis of the intestinal musculature.

In fact, we have to consider peritonitis, retroperitoneal hemorrhage, renal trauma, renoureteral colic, lesions of dorsal-lumbar spine, pneumonia, and pleurisy basal, some neurological drugs, and finally the laparotomy causing a transient disturbance of gastrointestinal motility (postoperative dynamic ileus).

Distension is associated with altered motility to stabilize the occlusive syndrome. In the beginning, the distension stimulates the peristalsis, but, settled the occlusive conditions, with greater distension the inhibitory effect is largest. In fact, the gut distension causes the inhibitory reflex of intestinal motility by receptors of longitudinal musculature of the bowel.

The accumulation of fluid and gas is accompanied by altered functions of intestinal mucosa. Clinical presentation of adynamic ileus is usually less severe than mechanical obstruction.

Clinical findings are abdominal distension, the absence of flatus and bowel movement, and vomiting. There are no colicky pain and peristalsis because of the intestinal paralysis [18].

The basic radiologic examination shows intestinal distension and some air-fluid levels, i.e., messy. The radiological finding that can confirm the diagnosis of ileus is the air in the colon and rectum, and on abdominal computed tomography (CT), there is no demonstrable mechanical obstruction [19]. Usually, the therapeutic approach is conservative based on the control and improvement of fluid and electrolytes disorders, particularly hypokalemia. It can also be useful in some patients in controlling particular medication as opiates or anticholinergics.

9. Diagnosis

Different clinical forms are included in the generic diagnosis of “intestinal obstruction”, which are to be distinguished from each other. Therefore, we propose a diagnostic course divided into sequential steps.

First, a preliminary diagnosis with distinction between simple versus strangulation obstruction is performed.

Then, in the first step of the diagnosis, the distinction of mechanical or paralytic obstruction is performed.

In the second step of diagnosis, we assess the level of obstruction, such as high small bowel obstruction, low small bowel obstruction, and large bowel obstruction.

Finally, in the third step of diagnosis, the type of obstacle is defined based on imaging examinations.

Each data (history, physical examination, laboratory, and instrumental) of the diagnostic evaluation could take on particular characteristic useful for a precise differential diagnosis, such as simple obstruction, strangulation, large bowel obstruction, and so on.

In the history, some data are relevant to the risk of bowel obstruction. Prior abdominal or pelvic surgery and peritoneal sepsis can cause adhesions and bands following any operations or septic process in the abdomen.

The evaluation should be made of inflammatory bowel disease (IBD) or other intestinal inflammation based on the previous diagnosis, therapy, and evolution of the disease.

History and current evolution of gastrointestinal or gynecologic neoplasms previously treated with surgery, chemotherapy, and irradiation can be risk factors for intestinal obstruction.

Most relevant in the history should be the communication of the change in regularity and frequency of bowel movement by an elderly patient due to possibility of undiagnosed colorectal cancer. Clinical features of intestinal obstruction are especially focused on two symptoms and two signs: colicky abdominal pain, absence of flatus or bowel movements, abdominal distension, and vomiting.

Abdominal pain is colicky, cramping, due to increased peristalsis, with paroxysms occurring every 4–5 min. In the first phase of obstruction, the abdominal pain should be more severe but if it is prolonged the occlusion for a serious delay in therapy can reduce the intensity of pain because peristalsis stops, so its disappearance should be a bad sign. In the later phase, it also increases abdominal distension and fluid—electrolytes loss [20, 21].

Periumbilical and cramping pain can be due to distal small bowel obstruction. Proximal small bowel obstruction could develop with less pain and distension but severe vomiting.

Large bowel obstruction (especially distal colon) may show pain below umbilicus and the paroxysms may occur longer for intervals of 6–10 min.

Severe and continuous pain should suggest strangulation obstruction. The absence of flatus and bowel movement in the true intestinal obstruction is complete. If there is small bowel obstruction, colon may take 1 or 2 days to empty. Indeed, the obstructive syndrome starts with the absence of flatus. The vomiting in the high, proximal small bowel obstruction is profuse and frequent. The higher is the obstruction, the worse is the vomiting. In the large bowel or distal small bowel obstruction, the vomiting can be delayed. After about 3 days of complete obstruction, the vomiting becomes feculent because the change in the intestinal bacterial flora causes a significant increase in anaerobic organisms. In the large bowel, obstruction may appear early vomiting reflex type based on intestinal distension. Abdominal distension should be considered the most frequent physical sign of intestinal obstruction [22–24].

The degree of abdominal distension varies depending upon the site of the obstacle or the extension of the obstructed bowel. In the proximal, small gut occlusion could occur at a lower degree of abdominal distension or no distension: intestinal occlusion without distension. In distal small bowel or large bowel obstruction, the abdominal distension is the most obvious clinical relevance. Abdominal distension is also present and obvious in the patients with

dynamic obstruction or intestinal pseudo-obstruction. If distension is conspicuous and other signs are minimal, there is probably large gut obstruction. Sigmoid volvulus can cause extreme distension. On the other hand, in the first phase of obstructive syndrome, the patients with a “closed-loop obstruction” or intestinal hernias or small bowel volvulus with short intestinal segment, abdominal distension can be minimal.

Beside the abdominal distension, the physical examination can point out hyperresonance, obstructive gut sounds, and visible peristalsis.

In the obstructed patients, it is possible to hear some characteristic sounds by abdominal auscultation: runs of borborygmi, chorus of tinkling high pitched musical sounds at the same time of peristaltic waves, and colicky pain. These data by auscultation are absent in the patients with abdominal distension by dynamic occlusion. Distended bowel results in hyperresonance or tympany to abdominal percussion, but fluid-filled loops can result in dullness. The visible peristalsis can be seen in very thin patients.

Simple obstruction	Strangulation obstruction
	Evidence of abdominal wall incarcerated hernias (groin, femoral, and obturator incisional)
Colicky pain Absent	Fast onset of abdominal pain. Constant pain, not colicky. Abdominal tenderness localized or
abdominal tenderness	diffuse
	Finally, peritoneal signs due to peritonitis (bowel ischemia, perforation, and peritonitis)

Table 5. Intestinal obstruction: preliminary differential diagnosis.

	Mechanical	Dynamic
History	Previous abdominal or pelvic surgery, radiation therapy, history of abdominal malignancy	Every risk factors of dynamic occlusion (causes of reflex paresis)
Pain	Present, colicky	Absent or due to abdominal distension
Vomiting	Present	Present
Abdominal distension	Present	Present
Absence of flatus or bowel movement	Present	Present
Plain radiography	Bowel distention Evident air-fluid levels differential height, regular arranged disposition	Bowel distention. Few air-fluid levels—somewhat messy

Table 6. Intestinal obstruction: first steps differential diagnosis.

The clinical examination should evaluate systemic compromission of intestinal obstruction syndrome. It should be highlighted dehydration, tachycardia, hypotension, reduced urine output, fever, electrolytes alterations, and dry mucus membranes. The physical examination

of abdomen will be completed with control of old laparotomy scar, any abdominal wall, or groin hernias.

The examination can identify the abnormal masses, such as abscess, volvulus, and tumor, which can be the cause of obstruction. Abdominal tenderness is not a characteristic feature of uncomplicated obstruction. Obvious tenderness localized or diffuse suggests complicated obstruction: strangulation, perforation, etc.

Rectal examination is an integral part of a clinical examination. Usually, this examination cannot add further information but it can find rectal neoplastic lesion or mucus or blood that probably suggests a strangulating lesion higher up, intussusception, or inflammatory intestinal lesion such as IBD.

Based on the clinical appearance and basic radiological examinations, the first steps of the diagnosis are shown in **Tables 5–7**.

	High small bowel occlusion	Low small bowel occlusion	Large bowel occlusion
Pain	Absent	Evident	Evident
Vomiting	Early, copious, continuous	Late	Very late
Abdominal distension	Absent	Present	Present
Absence of flatus or bowel movement	Present	Present	Present
Plain radiography	Is it possible follow the distended bowel segments and hypotize the site of obstruction (transition between dilated proximal and non-dilated distal bowel)		

Table 7. Intestinal obstruction: second-step differential diagnosis.

9.1. Laboratory studies

Routine laboratory studies are not specific for a diagnosis of intestinal obstruction. The laboratory data should evaluate hypovolemia, initial renal failure, hemoconcentration, metabolic abnormalities (hyponatremia, hypokalemia), and leucocytosis.

Neutrophilic leucocytosis can signalize complications such as strangulation or ischemic lesions. On the other hand, the anemia can indicate intestinal tumor or IBD.

In the obstructed patients with appearance of systemic compromission (hypothermia, tachycardia, fever, and renal failure), the complete clinical assessment requires arterial blood gas (ABG) and serum lactate. These evaluations can show some different details. Metabolic alkalosis follows severe vomiting. Metabolic acidosis takes place in the case of severe hypovolemia, hypoperfusion, organ failure, and ischemic bowel lesions [25]. The laboratory markers of ischemia to differentiate simple bowel obstruction from strangulation obstruction have been long searched [26]. First, elevated serum lactate (metabolic acidosis) with not very high specificity (sensitivity 90%, specificity 87%) can be used [27, 28]. Cronk et al. has suggested

the use, as marker of ischemia with valuable results, of intestinal fatty acid binding protein, connected to necrotic enterocytes [29].

The third step of our assessment of intestinal obstruction can allow the achievement of more defined diagnosis.

9.2. Plain radiography

About some imaging modalities, plain radiography is mostly employed. This is very practical and useful because it can confirm basic diagnosis of intestinal obstruction [30]. Usually, there are only few data useful for the distinction between mechanical or dynamic obstruction. Moreover, the plain radiography is widely available and less expensive; its regular performance requires an upright position of patients; the lateral position is a makeshift solution. The supine position shows insufficient results for diagnosis, only bowel distension. The findings of plain radiography in bowel obstruction are as follows:

- Multiple air fluid levels, more evident based on upright position;
- Dilatation of intestinal segments proximal the obstacle and collapse in distal bowel.



Figure 5. Plain radiography: small bowel obstruction.

These can be useful for the diagnosis of mechanical obstruction: the remark of a regular arranged disposition of multiple air fluid levels with evident size increases from each other (**Figure 5**) [31]. The plain radiography can detect the pneumoperitoneum by intestinal perforation. We have to remember that the remark of less gaseous distension (gasless abdomen) of intestinal loops in obstructed patients can be possible because of a complete fluid filling of loops. Mullaw suggests the evaluation of the string of pearls sign: fluid-filled bowel loops with small amounts of intraluminal gas [32]. Certainly, plain abdominal radiography in upright position can confirm the basic diagnosis of intestinal obstruction with high enough sensitivity (80%) and specificity (75%) [33]. The use of the examination in detecting the site of obstacle and in differentiating the small from large bowel obstruction is very limited.

9.3. Abdominal CT

Plain abdominal films, normally upright position if possible, should be the first imaging examination in the diagnostic program of suspected intestinal obstruction because it is readily available and in some cases it is resolvable if the doubt of obstruction is not confirmed. However, normally we must complete the diagnosis with abdominal CT scan. The performance of CT in the diagnostic plan is now valuable. Some data are similar to the findings of plain radiography as bowel dilatation above the obstruction and air-fluid levels. CT scan usually can identify the specific site of obstruction clarifying the transition point of distended and empty loops and also the complete intestinal occlusion. This examination should detect the etiology of obstruction by identifying internal or parietal hernias, neoplastic or inflammatory masses, and recognize the complications such as ischemic/necrotic evolution over all by strangulation obstruction and finally the perforation [32, 34–36]. CT scan can provide other diagnostic information: ascites, rotation of mesentery (whirl sign), mesenteric edema, bowel wall thickening >3 mm, submucosal edema-hemorrhage, venous cutoff sign by venous thrombosis, poor segmental bowel wall enhancement, pneumatosis intestinalis, edematous mesentery, and hemorrhage in the mesentery. All these findings can suggest complications that have vascular involvement in intestinal obstruction [37–39].

9.4. Abdominal ultrasonography

Abdominal US is now considered as an integral part of clinical examination and consequently it is currently performed in the patients with abdominal pains. Finally, it can be employed in the patients with contraindications to CT, pregnant patients, and patients with very severe systemic impairment. The contribution of abdominal US to intestinal obstruction diagnosis should be limited to identify intestinal distension, abdominal masses, and internal hernias, which can be site of incarcerated intestinal loops. Abdominal US can provide very few findings about air-fluid levels, the site, etiology, and complications of intestinal obstruction [40].

9.5. MRI

The accuracy of magnetic resonance imaging (MRI) is almost similar to CT scan for the confirmation of basic diagnosis of obstructions, location, and etiology of the obstruction. This examination shows poor detection of masses and inflammation [41–43].

Contrast studies, such as water soluble contrast material or contrast fluoroscopy, have indications and purposes rather limited in the obstruction diagnosed as complete and persistent.

10. Management

The first approach in the management of intestinal obstruction includes the correction of physiologic impairment caused by obstruction. Some measures can be required: the use of a bladder catheter to monitoring urine output, adequate intravenous access, arterial canalization, and CVP monitoring.

The purpose of the therapeutic approach is the correction of hypovolemia and electrolytes depletion with volume resuscitation. The development of fluid-electrolyte replacement and the adequacy of resuscitation should be guided by the degree of systemic impairment and the reaction of the patient to therapy. Therefore, aggressive replacement of fluid and electrolytes can be employed after restoration of renal function. The use of nasogastric tube for the control of severe intestinal distension can be helpful.

Antibiotics should be started at the confirmation of diagnosis of intestinal obstruction, mostly if fever, and leucocytosis is present.

The aim of the use of antibiotics is based on the control and treatment of intestinal overgrowth of bacteria and their translocation across the bowel wall [44].

Antibiotics, based on the particular type of bacterial overgrowth in the obstruction, could have more coverage against anaerobes and Gram-negative bacteria. The main objective of the therapeutic program of bowel obstruction is to remove the obstacle. Surgery is the leading option.

Three criteria guide this therapeutic choice:

- Degree of impairment of general conditions due to complications: intestinal ischemia, necrosis, perforation, and peritonitis;
- Etiology of obstructive syndrome (hypothesized or confirmed);
- Type of intestinal obstruction diagnosed (hypothesized or confirmed):
- Complete versus incomplete
- Small or large bowel obstacle site
- Strangulation occlusion

Peritonitis and abdominal sepsis caused by complications of obstructive syndrome (perforation, ischemia, necrosis, etc.) prescribe urgent surgical intervention. The choices of the surgical procedures are conditioned by pathological findings, sometimes intraoperative.

Clinical instability, diagnostic uncertainly, unexplained leucocytosis and metabolic acidosis, and consequently the doubt of perforation or abdominal sepsis justify the abdominal surgical

exploration. Also the suspicion of strangulation occlusion, based on continuous and severe abdominal pains, should prompt surgery.

The surgical choice for irreducible or strangulated hernia of abdominal wall is very obvious.

In the case of high suspicion for digestive malignancy, most frequently in large bowel, surgical intervention should be performed. In these cases, surgical procedures should contemporary treat both diseases, intestinal obstruction and digestive neoplasm: primary resection followed by temporary diversion (Hartmann procedure) or immediate reconstruction.

In summary, for the complete and permanent intestinal obstruction, the surgical intervention should be the first-line option.

In the management of intestinal obstructions, there are some issues under discussion with no simple solution.

The treatment of acute small bowel obstruction should be a common clinical challenge. The choice of operative management within the first 12–24 h from the onset can be followed by nontherapeutic laparotomy with the unfortunate results of further adhesions and postoperative morbidities [45]. Nasogastric decompression, fluid-electrolytes replacement, and careful clinical reassessment can have a considerable success rate in the approach of small bowel obstruction. Unfortunately, failure to acknowledge or late recognition of strangulation obstruction cause increased morbidity and mortality [46–48]. In this complicated setting, the solution is the selection of the patients. Of course, as stated previously, the cases with clinical and/or instrumental evidence of peritoneal phlogosis or perforation are excluded from this evaluation. Some criteria have been proposed to identify the patients with alleged simple small bowel obstruction for immediate operative treatment. Clinical appearance of fast onset of abdominal pain, continuous pain, not colicky, abdominal tenderness localized, or diffuse on physical examination suggest the choice of immediate surgical approach. There are also specific findings on abdominal CT: free intraperitoneal fluid, mesenteric edema, thickened wall, pneumatosis intestinalis, “small bowel feces signs” (gas bubbles and debris within the lumen of obstructed small bowel) [49]. On the other hand, the selected patients’ choice for nonoperative management should be characterized by the following criteria: the absence of abdominal wall hernias, previous abdominal pelvic surgery, previous abdominal malignancies, history and diagnosis of IBD (especially Crohn disease), colicky pain, absent abdominal tenderness on clinical assessment, and finally hemodynamic stability and absence of impairment of general conditions.

In summary, clinically stable patients with partial obstruction can be treated by conservative management [50].

Conservative management includes intestinal intubation and decompression, aggressive intravenous rehydration, and antibiotics [51].

The results of the conservative management of acute mechanical small bowel obstruction are uncertain and not conclusive, from the data of literature [52]. There are high success rates in the stable patients with incomplete obstruction [53, 54]. Among the patients with adhesive small bowel obstruction, 24.6% of patients are treated with nonoperative management,

without surgery or readmission [55]. On the other hand, high rates of recurrence and the risk of complications including vascular impairment were reported [47]. Therefore, among the patients managed conservatively to start with, the operative rates were very high because of the diagnostic difficulty to distinguish simple from strangulation obstruction on clinical and instrumental examinations [56].

The international guidelines [34, 57] for the evaluation and management of small bowel obstruction confirm and summarize the data from the literature.

The guidelines in evidence are as follows:

- The instrumental diagnosis should be based on the CT scan of abdomen because it can clarify the grade, severity, and etiology of small bowel obstruction.
- Urgent surgical approach is the first option for small bowel obstructions with evidence of peritonitis or clinical deterioration (fever, tachycardia, leucocytosis, and metabolic acidosis).
- Patients with partial or complete small bowel obstruction and stable general conditions and without physical and instrumental signs of peritoneal phlogosis can undergo initial nonoperative management.
- Water-soluble contrast study can be useful in partial small bowel obstruction not resolved within 48 h based on the improvement of water-soluble contrast on bowel function.
- After 3–5 days of conservative management, the patients with small bowel obstruction should undergo surgery;
- Laparoscopic treatment can be a safe and possible procedure for small bowel obstruction, but not commonly employed. In fact, its use requires some selection criteria: proximal obstruction, localized distension on radiography, no sepsis, and mild abdominal distension [58–60].

Large bowel obstruction due to colorectal cancer requires the treatment of malignancy and abdominal urgency. The first objective should be the control and management of malignancy. Several factors may influence the therapeutic choice: the location of the tumor (proximal-distal colon), the degree of colonic distension and the impairment of blood flow of intestinal wall, the involvement of the general conditions of the patient with organ failure, dehydration, hypovolemia, and sepsis.

In this scenario, variable and complex, it is very difficult to establish a well-defined and unequivocal line therapy in relation to the surgical procedure to be used. There are several proposals: at the beginning resolution of the occlusive complication only with colostomy (two-stage procedure) followed, sometime later, by resection of neoplastic lesion (with radical surgical criteria or palliative). On the other hand, a one-stage procedure with resection of the tumor (radical or palliative), followed by temporary colostomy (Hartmann's procedure) or primary anastomosis, can be employed.

Endoscopic colonic stents have been proposed in neoplastic obstruction of distal colon for palliation or as a bridge to surgery [61]. With the palliative intent, the colorectal stent can be

used as an alternative to colostomy, whereas in the hospitals it can be employed as a bridge to elective surgery with specific expertise [62].

The tumor site along the colon is an important factor for the therapeutic choice. Right colectomy can be defined as the treatment of choice for right-sided colon cancer in obstruction setting. It is a safe technique for one-stage resection and anastomosis [63]. The guidelines of World Society of Emergency Surgery suggest some recommendations on obstructive left colon carcinoma [62]. Hartmann’s procedure should be the preferred choice in the patients with impaired blood supply of intestinal wall and high surgical risk compared to loop colostomy. Hartmann’s procedure shows overlapped survival results compared to segmental colonic resection with primary anastomosis.

The patients submitted to primary resection and anastomosis have similar mortality-morbidity rates with total or subtotal colectomy and segmental colectomy. The immediate results are similar in these patients (with primary resection and anastomosis) related to the choice of intraoperative colonic irrigation or manual decompression.

11. Intestinal pseudo-obstruction

Intestinal pseudo-obstruction is a syndrome characterized by a complete intestinal dilatation, generally of large bowel, without mechanical obstacle. The proposal nosography of intestinal pseudo-obstruction is reported in Table 8.

Intestinal pseudo-obstruction
Acute
Hydro electrolytes alterations
Anticholinergic, ganglionic blocker drugs
Laparotomy
Intraperitoneal diseases (perforation, appendicitis, cholecystitis, pancreatitis)
Extraperitoneal diseases (hematoma, vertebral trauma, pneumonia, myocardial infarct)
Sclerosing therapy of esophagus
Acute colonic pseudo-obstruction
Chronic
Secondary
Endocrine disease (diabetes, hypothyroidism)
Collagenopathies (scleroderma, dermatomyositis)
Neuropathies (Parkinson’s disease, multiple sclerosis)
Degenerative diseases (amyloidosis)
Elevated blood levels of prostaglandin A, E, F
Idiopathic
Hereditary hollow visceral myopathy
Familial neuronal disease

Table 8. Intestinal pseudo-obstruction: nosography.

The intestinal pseudo-obstruction can affect small or large bowel and it may be possible to differentiate the syndromes with acute or chronic onset and evolution.

Paralytic ileus can be inserted in the broader field of acute pseudo-obstructions that are based on pathophysiological impairment of intestinal peristalsis (intestinal paresis). The acute pseudo-obstruction can be caused by severe fluid electrolytes disorders (hypokalemia, hypocalcemia), medications with anticholinergics or opiates, abdominal interventions (postoperative ileus), inflammatory and septic abdominal diseases as peritonitis, pancreatitis, perforations, intestinal ischemia, and retroperitoneal trauma.

In this area, the acute colonic pseudo-obstruction (ACPO) (Ogilvie's syndrome) should be highlighted. The Ogilvie's syndrome has been diagnosed in association with various pathologies such as cholecystitis, acute pancreatitis, retroperitoneal traumatic hematoma, Parkinson disease, and so on. The impaired colonic motility should be caused by imbalance in the autonomic nervous system: increase in sympathetic tone and decrease in parasympathetic tone [64]. Increased sympathetic tone to the colon results in the inhibition of colonic motility [65]. The pathologic findings, more evident in the right colon, are distension, fluid-gaseous accumulation, and increased endoluminal pressure. Severe blood circulatory impairment can occur in the large bowel wall with damage of venous return, edema, trophic mucosal alteration to serous, sometimes necrosis, and perforation. The cecum is more dilated colonic section. According to Laplace's law, the cecum, with its larger diameter, requires less pressure to increase in size and in wall tension. Ischemia, longitudinal splitting of serosa, and herniation of the mucosa and perforation, the so-called "diastasis breaking" of cecum, is caused with the increased wall tension.

Clinical features are obvious abdominal distension as earliest sign, no tenderness, hyperresonance, or tympany to percussion throughout the abdomen, and no peristalsis and bowel sounds to auscultation. The symptoms are similar to large bowel obstruction and develop over 3–7 days.

Imaging examinations, plain radiography, CT scan should help to exclude mechanical bowel obstruction. The size of the cecum (more than 8–10 mm) on abdominal films could be useful to decide colonic decompression, surgical or endoscopic, because of the risk of perforation [66].

In the ACPO, conservative treatment can be proposed: no oral intake, nasogastric decompression, correction of fluid and electrolytes disorders, and discontinuance of drugs that inhibit gastrointestinal motility [67].

In the conservative option, the use of pharmacologic agents can be added to increase colonic motility. Several drugs have been employed with nonunique and uncertain results, erythromycin, cisapride, metoclopramide, and neostigmine [68, 69]. The invasive therapeutic approach of Ogilvie's syndrome includes colonoscopic decompression and surgical intervention. The endoscopic decompression should be a safe and effective procedure for ACPO and has been associated with high success rates (77–86%) [70].

Recurrence rates of colonoscopy decompression are also high, ranging from 20 to 60% [71]. Clinical signs of ischemia, abdominal sepsis, perforation, or failure of conservative manage-

ment require surgery. The choice of surgical procedure is indicated by intraoperative pathological findings: tube cecostomy, subtotal colectomy, etc.

Chronic intestinal pseudo-obstruction can be subdivided into secondary and idiopathic. In the first cases, the chronic pseudo-obstructions are part of severe systemic diseases: endocrine diseases (diabetes and hypothyroidism) and collagenopathies (scleroderma, amyloidosis, dermatomyositis, and lupus erythematosus). The chronic intestinal idiopathic pseudo-obstructions are frequently familial diseases. Pathophysiology is not completely defined and should be based on derangement of autonomic nervous system with alteration of intestinal motility. The pathophysiological findings are hereditary hollow visceral myopathy, familial neuronal visceral disease, lesion of myenteric plexus, and alimentary tract ganglioneuromatosis.

The role of interstitial cells of Cajal has been hypothesized in the pathogenesis of idiopathic chronic intestinal pseudo-obstruction. Electron microscopy and immunochemistry studies showed a decreased number of interstitial cells of Cajal in the intestinal wall and alterations in interstitial cells of Cajal network [72].

According to clinical features, the cramping abdominal pain is more frequent; nausea, vomiting, and abdominal distension are occasional, sporadic. There are also alternating diarrhea and constipation and weight loss. Finally, the clinical evolution of the disease is chronic.

The results of the therapy of secondary forms are scanty and uncertain; usually, the therapy is connected with the treatment of the serious systemic diseases. Drugs that stimulate the smooth muscles (acetylcholinesterase inhibitors) can be employed in the idiopathic forms. Surgical procedures are indicated for the treatment of severe and continuous symptomatology that can be related to a portion of the digestive tract or for complete failure of conservative therapy [73].

Acknowledgements

The author is very grateful to Drs Libero Luca Giambavicchio and Francesco Lapolla for their valuable assistance in the typographical transcription of the manuscript.

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