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Intestinal Obstructions of Rare Occurrence: Intestinal Pseudo- Obstruction and Acute Small Bowel Obstruction

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Abstract

Intestinal pseudo-obstruction can be defined as an occlusive syndrome, with the signs and symptoms of intestinal obstruction, but without mechanical obstacle. The pathophysiological alterations causing the impairment of intestinal transit can be localized in different locations of the intestinal tract, such as the esophagus, stomach, duodenum, small intestine, colon, rectum. On the basis of the clinical manifestations, acute and chronic forms of intestinal pseudo-obstructions can be identified. In acute forms the alteration can be functional of a reflex nature or due to morpho-functional impairment of the intestinal autonomic nervous system. Chronic forms can be secondary, idiopathic and congenital. In all forms, differentiated on the etiological basis, the pathogenetic mechanism of chronic pseudo-obstruction is determined by the neuropathic or myopathic mechanism. The clinical examination allows us to formulate the suspicion of pseudo-obstruction and clinical-instrumental objectivity of acute and chronic pseudo-obstructions, must present the symptoms and signs of intestinal obstruction which must therefore be recorded, also by instrumental imaging tests (US, CT, MRI), but the diagnostic process must be completed by the exclusion of any mechanical obstacle. Acute forms from transient causes resolve quickly and completely following treatment of the underlying pathology. Chronic forms secondary to serious underlying diseases cause greater therapeutic problems. In chronic idiopathic forms, due to alterations of the autonomic nervous system, the use of acetylcholinesterase inhibitor drugs or metoclopramide (direct stimulation of smooth muscles) has in some cases been useful. Surgical intervention is justified as a measure of necessity in conditions not sensitive to medical therapy and, above all, when serious and continuous symptoms can be linked to a particular section of the digestive tract.

Conventional treatment of **small bowel occlusion** has been reviewed based on the recent literature acquisitions, followed by personal observations. Some small bowel occlusion syndromes require a different therapeutic approach and have a varying prognosis. Acute small bowel occlusion can be divided into the full blown cases, as complete small bowel obstruction (C-SBO), and also occlusions with probable strangulation requiring immediate surgical intervention, and the other group that includes the simple or partial small bowel obstruction (P-SBO) with the same, but moderate symptoms. In this second group indications for surgery are not urgent. Frequently a brief period of intestinal decompression and conservative medical treatment can achieve stable recovery.

Keywords

Intestinal pseudo-obstruction, Pathogenesis of psudo-obstruction, Management of pseudo-obstruction, Small bowel occlusion, Management SBO, Complete SBO, Partial SBO.

INTESTINAL PSEUDO-OBSTRUCTION

Introduction

Intestinal pseudo-obstruction can be defined as an occlusive syndrome, with the signs and symptoms of intestinal obstruction, but without mechanical obstacle.

The precisely defined clinical findings, which are more frequent today, are certainly related to more precise pathophysiological knowledge and the recognition of the determining causes allows, in practice, more rational therapeutic decisions, avoiding unnecessary laparotomies or those limited to intestinal biopsy or delays in surgical treatment of the mechanical forms. The difficulties of nosographic systematization of the intestinal pseudo-obstructions are intuitive and obvious, but it is possible to differentiate clinical forms with acute onset and evolution and so-called idiopathic chronic forms and those secondary to underlying systemic diseases (table 1).

TABLE 1

INTESTINAL PSEUDO-OBSTRUCTIONS

ACUTE

— hydro-electrolyte alterations
— anticholinergics, ganglioplegics
— laparotomy operations
— intraperitoneal diseases (perforation, appendicitis, cholecystitis, pancreatitis)
— extraperitoneal diseases (hematoma, vertebro-medullary trauma, pneumonia, myocardial infarction)
— esophageal sclerosing therapy
— idiopathic acute colon

CHRONICLES

SECONDARY/ACQUIRED
— endocrinopathies (diabetes, hypothyroidism)
— collagenopathies (scleroderma, dermatomyositis)
— neuropathies (m. Parkinson, multiple sclerosis)
— degenerative diseases (amyloidosis)
— elevated blood levels of prostaglandins A.E.F.
CONGENITAL
— familial hollow viscera myopathy
— familial visceral neuropathy
IDIOPATHIC
— sporadic visceral neuropathy
--- sporadic visceral myopathy

PATHOPHYSIOLOGY OF INTESTINAL PSEUDO-OBSTRUCTION

Acute intestinal pseudo-obstructions have sometimes been related to serious hydroelectrolyte alterations (hypokalemia, hypocalcemia) or to the intake of anticholinergic or ganglioplegic drugs; they have frequently been linked to surgical interventions, particularly laparotomy, and more often to serious intraperitoneal (perforation, appendicitis, cholecystitis, pancreatitis) or extraperitoneal (hematoma, vertebro-medullary trauma, pneumonia, myocardial infarction) diseases. The genesis, in these cases, is certainly of a reflex nature; paresis, linked to external stimuli of various kinds, is transitory and, generally, the regression of the adynamism of the intestinal muscles follows the treatment of the underlying pathology. The etiology of acute intestinal pseudo-obstruction, in particular in relation to the different, specific clinical manifestations with which it is associated, is not yet completely well defined. Some etiopathogenetic hypotheses are in evidence, such as the inhibition or reduction of parasympathetic activity, which leads to a decrease in the motor activity of the small intestine and in particular of the colon, the dysregulation of parietal stretch receptors and the decrease of nervous ganglion cells in the smooth muscles of the colon, ultimately all aspects that constitute the impairment of the autonomic nervous system (1). However, other possible factors that presumably intervene in the pathogenesis of acute intestinal pseudo-obstruction such as reduced splanchnic perfusion, anticholinergic drugs, opiates, hypokalemia and uremia must be considered,

also based on the contextual presence in the clinical picture, as trauma, spinal anesthesia and some drugs (1,2). In the context of acute pseudo-obstructions, the idiopathic one of the colon, known as Ogilvie's syndrome (3), should be considered, which perhaps deserves a clinical-diagnostic and therapeutic position in its own right. It has been reported in the absence of extra-colonic diseases and associated with numerous conditions such as cholecystitis and acute pancreatitis, traumatic retroperitoneal hematoma, lesions of the spinal cord and roots, serious lung diseases, heart failure, digestive hemorrhages , Parkinson's disease, multiple sclerosis. The alteration of the autonomic nervous system, with the inhibition of the parasympathetic, explains the pathogenesis of acute pseudo-obstruction of the colon, characterized by massive intestinal dilatation (4). Some cases of intestinal pseudo-obstruction have been reported, as a complication of esophageal sclerosing therapy. Transmural inflammation and periesophageal damage of the vagal trunks or, alternatively, dysfunction of the retroperitoneal autonomic ganglia caused by the passage of ethanolamine oleate through the diaphragm into the retroperitoneal space, can induce dysfunction in the distal areas of the enteric tube, simulating obstruction (5). The altered motor function, predominantly colic, would be triggered by an alteration of the balance between the sympathetic inhibitory system and the parasympathetic activator of motility. The prevalence of the sympathetic, well represented in the ascending colon, would lead to abnormal distention first in this tract, with liquid-gas accumulation and intraluminal hypertension. At the parietal level they are established, consequently, serious circulation disorders with alterations of venous return, edema and trophic changes that proceed from the mucosa to the serosa, sometimes leading to necrosis and perforation (6,4).

Chronic intestinal pseudo-obstruction (CIPO) can be acquired/secondary and congenital, idiopathic. In all forms, differentiated on the etiological basis, the pathogenetic mechanism of chronic pseudo-obstruction is determined by the neuropathic or myopathic mechanism. The most clinically defined and most common form of neuropathic chronic intestinal pseudo-obstruction is Hirschsprung's disease, caused by the congenital absence of the intramural nerve plexuses (agangliosis) which regulate the relaxation of the parietal muscles, determining the narrowing of a segment of the large intestine which compromises the progression of the intestinal contents and consequently causes dilation upstream. The deficit of the normal regulatory activity, carried out regularly by the interstitial cells of Cajal (ICC), can explain the obstruction (7). Congenital agangliosis is associated with other genetic disorders such as Laurence Moon syndrome and/or Bardert-Biedl syndrome (Laurence Moon syndrome and Bardert-Biedl syndrome, are two different names of the same syndrome or, according to a different evaluation, the two syndromes are different because Laurence Moon syndrome does not present polydactyly,

obesity and renal anomalies). Furthermore, Waardenburg-Shah syndrome (WSS) or Waardenburg-Shah syndrome type IV (WS4) must be considered, which is characterized by the association between Waardenburg syndrome (sensorineural deafness and pigmentation abnormalities) and Hirschsprung disease (agangliosis megacolon). Among the acquired conditions that generate CIPO, Chagas disease stands out. In this disease, the neurotoxin generated by Trypanosoma cruzii has a deleterious effect on the enteric nervous system. Also added to the neuropathic variants of CIPO are metabolic disorders such as diabetes mellitus and porphyria, as well as infiltrative diseases such as systemic sclerosis and amyloidosis. Even drugs of different types, such as antidepressants, anticholinergics, anti-tumor drugs of natural origin, the so-called vinca alkaloids, vincristine and vinblastine can interfere with the functional activity of the myenteric plexus. Finally, immune disorders must be evaluated which, however, can rarely cause CIPO, which has been diagnosed in a small number of cases associated with systemic lupus erythematosus (8) and, even more rarely, in cases of autoimmune enteric leiomyositis (9).
The myopathic variants of CIPO have been the subject of study and evaluation, even if this grouping of intestinal pseudo-obstructive

pathologies has remained heterogeneous and also they are the subject of non-unanimous acquisitions. From an anatomopathological perspective, visceral myopathy remains characterized by vacuolar degeneration, atrophy and fibrosis of the intestinal muscular layer without cellular inflammation (10). The familial form occurs in approximately 30% of cases having a mostly autosomal recessive transmission (11).

Chronic pseudo-obstructions, as both secondary or acquired and so-called idiopathic are less frequent. In secondary ones, the alteration of intestinal function is permanently part of the clinical picture of serious underlying diseases. In diabetes mellitus, the motility disorder is mainly characterized clinically by diarrhea alternating with constipation and only rarely takes the form of true pseudo-obstruction; furthermore, in the past, no anatomical alterations of the smooth muscles and myenteric plexus have been demonstrated (12).

More recently CIPO due to digestive neuropathy results from conditions that damage the extrinsic and/or intrinsic nervous systems of the intestine, as is the case with diabetic autonomic neuropathy (13).

CIPO has been associated with numerous diseases. Neuropathies such as Parkinson's disease and diabetes mellitus have been implicated in the degeneration of the enteric nervous system signaling pathway that causes CIPO. Scleroderma, lupus, and paraneoplastic syndromes have been associated with chronic immune-mediated pseudo-obstruction with injury to enteric nerves and smooth muscle cells of the gastrointestinal tract (14).

Already in the past, clinical-instrumental evidence has been identified relating to the role played by Herpes viruses, neurotropic viruses, in chronic intestinal pseudo-obstruction (15).

As already mentioned the most common infectious etiology is Chagas disease, in which inflammatory alterations of the enteric neural pathways cause pseudo-obstructive syndrome. (16). Furthermore, many genetic diseases (for example, Fabry disease, Ehler Danlos syndrome, Hirschsprung disease) are implicated in the etiopathogenesis of CIPO (17,18). In hypothyroidism, however, the myxedematous infiltration of the intestinal wall, with disturbance of the transmission of impulses through the neuro-muscular junction, can lead to intestinal paresis, clinically characterized by constipation with abdominal distension, up to true pseudo-obstruction (19). However, intestinal hypomotility has been emphasized as a characteristic manifestation in severe hypothyroidism which can progress to intestinal pseudo-obstruction, paralytic ileus and megacolon (20).

From an inverse clinical perspective, cases have been reported in which recurrent intestinal pseudo-obstruction was the clinical manifestation that led to the diagnosis of hypothyroidism, also highlighting the pathophysiological character of the intestinal adynamia that characterized the clinical manifestation (21,22).

In scleroderma, the altered motility can even involve the entire digestive tract, sometimes with prevalent esophageal involvement (LES incompetence, reflux esophagitis), sometimes with ileal or colonic prevalence (true pseudo-obstructive syndrome). In scleroderma, alterations of the gastrointestinal system frequently constitute the onset of the disease (23). In fact, motor disorders appear in the different segments of the gastrointestinal system, the esophagus, the stomach, the small intestine, the colon, with the clinical manifestation of various syndromes or diseases, such as gastro-esophageal reflux, delayed gastric emptying, diverticular disease of the colon, fecal incontinence (24). It is completely characteristic that scleroderma in the small intestine, in the course of intestinal pseudo-obstruction, induces an alteration of the intestinal bacterial flora with its abnormal excessive growth. Therefore in the course of this event the clinical manifestation in evidence is diarrhea with hydro-electrolyte abnormalities, which is added to the signs of altered intestinal function, such as pain and abdominal distension,

as for an actual pseudo-obstruction of the small intestine (25,24,26).

In amyloidosis, dermatomyositis, lupus erythematosus, Parkinson's disease and myotonic dystrophy, overt pseudo-obstructive changes have been reported more rarely or exceptionally.

Amyloidosis is a rare disease characterized by the extracellular deposition of the amyloid protein which is an abnormal fibrillar protein. The consequence of this accumulation is the structural and functional alteration of the tissues. The two most common forms are primary amyloidosis, AL Amyloidosis (light chain monoclonal immunoglobulin amyloidosis) and reactive or secondary amyloidosis, AA Amyloidosis. The first is associated with plasma cell dyscrasia, in which monoclonal light chains can be detected in serum or urine, while secondary amyloidosis, with serum deposits of amyloid A protein, is frequently reactive to chronic inflammation, infection and neoplasms (27,28). Other types of amyloidosis have also been described, such as the hemodialysis-related form, hereditary, the senile and the localized type (29). Amyloidosis can be systemic or localized to a single organ, such as the gastrointestinal tract (30). AA amyloidosis mainly affects (60%) the gastrointestinal tract, keeping in mind, however, that forms limited exclusively to the gastrointestinal tract are rare. Much less frequent is the involvement of the gastrointestinal tract in patients with AL amyloidosis (1%-8%) (31). The clinical picture of intestinal amyloidosis presents variable degrees of severity. Gastrointestinal symptoms may include abdominal pain (50%), change in bowel habits (47%), altered intestinal motility (39%), spontaneous intestinal perforation (16%) and gastrointestinal bleeding (39%) (32,29). Ultimately, it should not be forgotten that amyloidosis can affect the gastrointestinal system alone or present with multisystem involvement. From an anatomopathological perspective, the deposition of amyloid between the muscle fibers of the intestinal smooth muscles causes atrophy of the same fibers and deficit of the pressor function (myopathy). Furthermore, the infiltration of amyloid at the level of the myenteric plexus is associated with destruction of the interstitial cells of Cajal (neuropathy), which normally stimulate smooth muscle cells, and amyloidosis causes also vascular insufficiency. This results in hypomotility/aperistalsis of the affected intestinal segments. (33). It can be assumed that the usual clinical presentation of AL amyloidosis is characterized by signs of intestinal obstruction or intestinal transit difficulties with constipation, mechanical obstruction or chronic intestinal pseudo-obstruction, while AA amyloidosis presents more frequently with diarrhea and malabsorption. (34,29).

Connective tissue diseases (CTD) may present the onset of chronic intestinal pseudo-obstruction during their clinical evolution, although this is a rare occurrence. The impairment of intestinal transit in chronic pseudo-obstruction is underlying, in these pathologies, by the alteration/compromise of the peristaltic-motor function generally of a segment of the small intestine.

Dermatomyositis (DM) is a systemic connective tissue disease characterized primarily by proximal muscle weakness and myalgia with typical skin rash (35). The etiology of chronic pseudo-obstruction is not clearly defined in dermatomyositis, however in this clinical perspective, which is not often realized, the hypothesis has been put forward that, in dermatomyositis/polymyositis, during which chronic pseudo-obstruction develops , with the clinical evolution complicated by chronic hypoxia, hypoxia is precisely the cause of chronic intestinal pseudo-obstruction, although the hypoxic condition is not a stable alteration (36).

The pathogenesis of scleroderma is currently not completely clarified, however the main theories suggest that interactions between endothelial cells, lymphocytes, macrophages and fibroblasts determine the activation of myofibroblasts, leading to an overproduction of extracellular matrix and tissue fibrosis(37).

In the small intestine, scleroderma can usually cause small intestinal bacterial overgrowth (SIBO) and intestinal pseudo-obstruction (24).

The clinical presentation in patients often simulates true mechanical obstruction of the small intestine accompanied by abdominal pain, postprandial tenderness, abdominal distension and subsequent signs of malnutrition or malabsorption. When abnormal bacterial overgrowth develops in the small intestine, patients also present with diarrhea and develop electrolyte abnormalities (25). Intestinal pseudo-obstruction occurs in 3.7%–5.4% of patients with scleroderma and constitutes a complication with high morbidity and mortality (38,26). The pathogenesis of chronic intestinal pseudo-obstruction linked to systemic lupus erythematosus (SLE) remains unclear but the most likely indication is that the underlying pathophysiology is linked to intestinal vasculitis of the visceral smooth muscles leading to tissue alterations and parietal hypomotility (39,40). Parkinson's disease, among the neurological diseases, and diabetes, among the metabolic diseases, can present the complication of chronic intestinal pseudo-obstruction based on the appearance of intestinal motility disorders induced by the alteration of the parasympathetic and sympathetic autonomic nervous system (41).

Myotonic dystrophy (DM) is a progressive, autosomal dominant neuromuscular disease. Myotonic dystrophy alters the smooth muscles of various organs, including the gastrointestinal tract, inducing dystrophic alteration in smooth muscle cells which undergo severe impairment and progressive replacement with adipose tissue (42). Consequently, the pathological impairment of the intestinal smooth muscles induces serious alterations in motility. However, the simultaneous appearance of chronic intestinal pseudo-obstruction is an unusual event (43).

Another cause of chronic pseudo-obstruction, mainly ileal, has been hypothesized in the high blood levels of postaglandins A, E, F and the possibility of treatment with indomethacin has been suggested; the decrease in postaglandin levels was accompanied by a reduction or resolution of clinical and radiological manifestations (44,45). Prostaglandins (PGS) play an inhibitory role on intestinal motility.

Xuan-Zheng Shi *et al.* (46) have found, through experimental research, that the upregulation of the gene expression of cyclooxygenase-2 (COX-2) in intestinal obstruction is followed by the increase in prostaglandin E2 (PGE2), one of the most plentiful prostaglandins in the gastrointestinal tract and known to inhibit contractility of colonic smooth muscle cells (SMCs) in rats (47,48).

ELISA measurements in the external musculature of the colon showed that the level of PGE2 was significantly increased in the segment upstream of the obstacle with respect to time. In fact, PGE2 started to increase significantly on the first day, and its level continued to rise, on day 7, the PGE2 level was 120 times higher than the control. In contrast, there was no significant increase in PGE2 in the aboral segment (46).

Several metabolic factors can influence colonic motility, such as renal failure and electrolyte disturbances. Prostaglandins and proinflammatory cytokines, such as TNF-α, IL-6, IL-8 and IL-1β, are inflammatory mediators and implicated in gastrointestinal tract motility (49,50). Prostaglandins in particular have been implicated in POI (51), CIPO (52), acute dysmotility of the small intestine (53) and also influence ICC function and slow wave frequency [54]. However, the role of prostaglandins in the development of pseudo-obstructions has not yet been fully defined; they, and in particular prostaglandin E2, could be effective in reducing intestinal motility (55,56,2).

Pathogenesis of chronic idiopathic and congenital intestinal pseudo-obstructions

First of all, an evident familiarity has been recognized (57,58), with autosomal dominant transmission and the serious impairment of intestinal motility is linked either to alterations such as vacuolization and atrophy of the muscle fibrocells which can affect the entire digestive tract (hereditary myopathy of the hollow viscera (59), or to alteration of the transmission of the nervous impulse at the level of the myenteric plexus (non-hereditary sporadic

visceral neuropathy), due to suffering and reduction of the neurons (60) or due to a defect in the muscarinic function (61). A familial variety characterized by impairment (reduction in the number of neurons with rounded eosinophilic protein nuclear inclusions) of the central and peripheral nervous system associated with alterations of the myenteric nervous plexus (familial visceral neuropathy) has also been described (57), but in together this group still appears heterogeneous due to the variable impairment of the nervous system (62, 63). Chronic intestinal pseudo-obstruction (CIPO) is the classic clinical manifestation of severe intestinal dysmotility, to which the genetic alteration found in various patients may be underlying. Pseudo-obstructive syndrome is characterized by both extrinsic and parietal intestinal neurological alterations (neuropathies), of the interstitial cells of Cajal (mesenchymopathies) and sequentially of the parietal smooth muscle cells (myopathies) (64,65). In this clinical-pathological scenario, intestinal dysmotility syndromes are found with predominantly neuropathic alterations, mainly myopathic forms and forms in which the intestinal motor disorder is the clinical manifestation of mitochondrial alterations (66).

There are certainly some cases of chronic intestinal pseudo-obstruction (CIPO) that can be considered hereditary with genetic characteristics of autosomal dominant and autosomal recessive chromosomal transmission. Pseudo-obstructive syndromes (CIPO) have been recorded with underlying congenital pathologies characterized by mitochondrial myopathies of the mitochondrial neurogastrointestinal encephalomyopathy (MNGIE) type, caused by a mutation in the TYMP gene (67,68). Furthermore, megacyst-microcolon intestinal hypoperistalsis syndrome (MMIHS) has been attributed to mutations in the ACTG2 gene (69). The syndrome of chronic atrial and intestinal dysrhythmia (CAID) caused by mutations in the SGOL1 gene can be classified among hereditary pseudo-obstructions (70). Chronic intestinal pseudo-obstruction has also been reported in association with Alpers' disease which is a mitochondrial disease/syndrome, included among the diseases characterized by mitochondrial DNA depletion disorders linked to mutations in the POLG gene (71). Finally, among the genetic pseudo-obstructions, the mitochondrial encephalopathy, lactic acidosis and stroke-like episodes (MELAS) syndrome must be considered, which is also caused by mitochondrial DNA depletion disorders (18,41).

DIAGNOSIS OF INTESTINAL PSEUDO-OBSTRUCTIONS

The diagnosis of intestinal pseudo-obstruction primarily requires the basic distinction from mechanical occlusions. Furthermore, there is the opportunity to identify and distinguish acute pseudo-obstructions from chronic forms. To this end, in the clinical picture, the onset is highlighted, which can be acute or progressive and which constitutes the characterizing element, albeit approximately, of the acute and chronic forms of intestinal pseudo-obstructions. A further discriminating element, always somewhat undefined, is the sector or segment of the digestive tract involved in the pathology. Acute pseudo-obstructions mainly affect the colon, while chronic forms mainly affect the small intestine.
However, it should not be forgotten that the pathophysiological alterations of pseudo-obstructions can involve all intestinal segments.

The clinical presentation of acute intestinal pseudo-obstructions, as widely reported in the specialist literature, shows the prevalent involvement of the colon. In many cases, concomitant pathological conditions are known in patients already hospitalized. In any case, abdominal distension is reported, with or without colicky pain, rapid and progressive; the closure of the bowel to feces and gas is complete. On physical examination, distension is notable; no localized or widespread defense contracture is detected; upon listening, peristalsis is present. The clinical picture is, therefore, comparable to that of mechanical occlusion. For differential purposes, significant characteristics are the sudden onset of any painful symptoms and, above all, the rapid worsening of abdominal distension. Gastric drainage of no or barely bilious fluid is minimal; leukocytosis, hyperglycemia and fluid-electrolyte imbalance, frequently present, are

not indicative. Imaging examinations of the abdomen show segmental overdistention of the colon, most evident in the right colon, i.e. with scan points at the right, left and sigmoid flexures. Fluid and air levels are absent, with evidence of parietal hypomobility.

The clinical picture of chronic pseudo-obstructions, must present the symptoms and signs of chronic intestinal dilatation which must therefore be recorded, also by instrumental imaging tests (US, CT, MRI) over a suitable period of time, such as at least a few months (72). In current clinical practice there is an indication of the maximum dilation, beyond which there is a risk of parietal injury, limited in particular to the right colon. In general, the colon is considered dilated if it is greater than 6 cm in diameter, with the cecum having an upper limit of 9 cm (73). A cecal diameter between 12 and 15 cm increases the risk of cecal rupture (74). However, caution/alarm guide criteria are available which refer to the dilation of various intestinal segments, summarized in the 3 – 6 – 9 rule: the 3-6-9 rule is a simple indication that describes the normal intestinal caliber: small intestine: <3 cm, large intestine: <6 cm, appendix: <6 mm, cecum: <9 cm. Above this size, the intestine is generally considered dilated and therefore opens up the possible diagnostic hypothesis of an obstruction or adynamic/paralytic ileus (75). CIPO, often lasting years, is characterized by alternating exacerbations and remissions. Abdominal pain, often crampy, is the most constant symptom; abdominal distension and sometimes fecal vomiting are episodic. Weight loss is more evident in the exacerbation phases. Diarrhea and constipation can alternate in each period of aggravation or characterize it individually. The clinical picture is therefore difficult to differentiate from that of mechanical occlusion, where some differences are perhaps in the often more intense pain and more constant closure of the bowel, but the isolated examination of these symptoms cannot lead to an effective diagnostic distinction. . It is therefore more useful to evaluate the onset and evolution of the clinical picture which generally, in mechanical forms, has a clear acute character, while in pseudo-obstructions it has a slower and even intermittent progression. Diagnostic perplexities obviously remain in cases of alternating symptoms from adhesions, internal hernias, substenosis due to Crohn's disease or anastomosis. The motility disorder can also be characterized by prevalent symptoms of a segment of the gastro-intestinal tube, such as distinguishable anatomical-clinical varieties: esophageal, gastro-duodenal, ileal and colonic (76). In the form with prevalent esophageal involvement, the dominant symptom is dysphagia; the gastro-duodenal variety manifests itself with epigastric distension, nausea and vomiting; the ileal one with pain and abdominal distension (the radiological examination demonstrates notable and widespread dilation of the small bowel loops); that of prevalent colonic impairment with crampy abdominal pain, notable abdominal distension, closure of the bowel to feces and gas, sometimes interrupted by diarrheal discharges and partial reduction of distention (direct radiographic examination highlights notable distension of the small intestine and colon). Laboratory investigations are always appropriate for the evaluation of the general conditions and, in particular, of the alterations of the hydro-electrolyte balance, alterations that occur constantly, worsening with the persistence of the intestinal obstruction condition, both in mechanical occlusions and in the dynamic and pseudo -obstructive forms. The preliminary exclusion of any mechanical obstruction is essential in the diagnosis of the obstructive syndrome, for which the suspicion of pseudo-obstruction has been raised. Imaging tests are necessary and obligatory references, such as radiological exams, such as standard abdominal x-ray, or with oral or enema contrast, abdominal magnetic resonance imaging (MRI) or computed tomography (CT). These instrumental investigations have the aim of excluding, in a completely reliable manner, an intrinsic or extrinsic occlusive lesion in the lumen of any intestinal segment. Imaging tests should be integrated, in some cases, with the endoscopic evaluation of the site of "pseudo-obstruction" downstream of the distended segment of the intestine, to exclude various possible pathologies such as organic stenosis, congenital occlusive membrane, or atresia. The abdomen imaging exams do not offer many differential diagnostic possibilities; any serious

hydro-electrolyte imbalances, gaseous distension of an isolated district or multiple intestinal segments, with air-fluid levels and disorder in the arrangement of the loops can equally characterize the pseudo-obstructive and mechanical occlusive forms. In the latter, only sometimes can the arrest of gaseous distension near the obstacle be identified. In some cases of colonic distension diagnostic clarification is therefore reliable when carrying out endoscopic or contrastographic radiological tests of the digestive tract; in particular, in the cases of colonic pseudo-obstruction, the use of endoscopic procedure, can add to the diagnostic role of possible obstructive pathologies, also a therapeutic role through the decompression of the dilated colonic segment. The motility disorder can also be characterized by prevalent symptoms of a segment of the gastro-intestinal tube, such as distinguishable anatomical-clinical varieties: esophageal, gastro-duodenal, ileal and colic (76). In the form with prevalent esophageal involvement, the dominant symptom is dysphagia; the gastro-duodenal variety manifests itself with epigastric distension, nausea and vomiting; the ileal one with pain and abdominal distension (imaging tests demonstrate notable and widespread dilatation of the small bowel loops); that of prevalent colic impairment with crampy abdominal pain, notable abdominal distension, closure of the bowel to feces and gas, sometimes interrupted by diarrheal discharges and partial reduction of distention (even the simple standard radiographic examination highlights notable distention of the small intestine and colon). Distinctive elements of the so-called idiopathic chronic pseudo-obstructive forms can be obtained by esophageal manometry, since both the alteration of the smooth muscles and that of the autonomic nervous system generally affect the entire gastro-intestinal tract. In the forms caused by alteration of the smooth muscles, a reduction in esophageal peristalsis is detectable with normal functionality of the LES; in the event of neurological disorder of the myenteric plexus, absence of esophageal peristalsis and reduced relaxation of the LES. Recent clinical-experimental investigations have demonstrated, in subjects with recognized chronic idiopathic pseudo-obstruction, a clear reduction in the electrical activity of the colon after a meal (alteration of the gastro-colic reflex) (77,78).

MANAGEMENT OF INTESTINAL PSEUDO-OBSTRUCTIONS

The exact diagnostic recognition of the pseudo-obstructive forms and the determining causes entails rational therapeutic implications. The pathophysiological sequence that leads to acute pseudo-obstruction of the colon is attributable to the alteration of the normal balance between the sympathetic inhibitory system and the parasympathetic activator of motility. The inhibitory activity of the sympathetic, highly represented in the proximal colon, would be prevalent and can motivate the maximum distension at the level of the ascending colon and cecum (79). In many cases, concomitant pathological conditions are known in patients already hospitalized: advanced age, comorbidities caused by electrolyte imbalance, altered basic functional status, immobility, non-operative trauma, serious infections and hospitalizations for cardiovascular diseases, as conditions predisposing the onset of acute colonic pseudo-obstruction (ACPO) have been considered. On physical examination, abdominal distension is notable, with or without colicky pain, rapid and progressive; no localized or widespread defense contracture is detected; upon listening, peristalsis is present; the closure of the bowel to feces and gas is complete. The clinical picture is, therefore, comparable to that of mechanical occlusion. For differential purposes, significant characteristics are the sudden onset of any painful symptoms and, above all, the rapid worsening of abdominal distension. Gastric drainage of non- or barely bilious fluid is minimal; leukocytosis, hyperglycemia and fluid-electrolyte imbalance, frequently present, are not indicative. Standard radiological examination of the abdomen does not always demonstrate overdistention of the colon in all sections; more often, breakpoints at the right, left, and sigmoid flexure, with relative greater segmental distension, ultimately prevalent in the right colon. The absence of air-fluid levels, the flattening of the colonic wall with immobile and non-edematous haustra may constitute further differential elements (80). The sequence of standard radiographs allows

the increase in colonic distention to be assessed. CT with oral and intravenous (IV) contrast is the appropriate diagnostic procedure: it can exclude the mechanical causes of intestinal obstruction and highlight any concomitant pathologies (retroperitoneal hematoma or abdominal abscess), and signs of ischemic suffering of the intestinal wall. The barium enema with contrast, in the absence of supervening complications, performed urgently, unequivocally demonstrates the absence of organic lesions; it is also useful in differentiating other mechanical causes, such as cecal volvulus, cecal "bascule" and other cholectasis from ischemic colitis and toxic megacolon in Crohn's and idiopathic rectocolitis. Diagnostic colonoscopy is a procedure to avoid. In the absence of recognized causes of ACPO, emergency treatment, together with the restoration of possibly abnormal blood chemistry parameters, is aimed at immediate decompression of the colon, the wall of which is suffering from ischemic conditions, especially at the level of the cecum, is more likely to rupture when diameter exceeds 9 cm. The laparotomy, despite the diagnostic doubt, and the cecostomy of necessity allowed appreciable results (81). The bloodless method of endoscopic decompression has been found to be effective in various experiences (82,83,79)

Acute forms from transient causes resolve quickly and completely following treatment of the underlying pathology (most often limited or diffuse peritoneal or pulmonary inflammation, taking anticholinergics or ganglioplegics). Chronic forms secondary to serious underlying diseases cause greater therapeutic problems. The treatment of myxedema, Parkinson's disease and multiple sclerosis generally allows only slight improvement in intestinal transit; in diabetes, collagenopathies and amyloidosis, the pseudo-obstructive syndrome is difficult to modify in the treatment of the underlying disease. In chronic idiopathic forms, due to alterations of the autonomic nervous system, the use of acetylcholinesterase inhibitor drugs or metoclopramide (direct stimulation of smooth muscles) has in some cases been useful.

Surgical intervention is justified as a measure of necessity in conditions not sensitive to medical therapy and, above all, when serious and continuous symptoms can be linked to a particular section of the digestive tract. The dysphagic symptoms, simulating achalasia, can be relieved with instrumental esophageal dilations or with extramucous cardiomyotomy. The gastro-duodenal prevalence, reported by epigastric pain, vomiting and radiographic demonstration of megaduodenum, may justify gastro- or duodeno-jejunostomy interventions or simple duodenoplasty to reduce its lumen. The forms with obstructive symptoms of a predominantly ileal type (abdominal pain, notable distension of the loops) are generally not indicated for surgery and exploratory laparotomy can initiate cycles of relaparotomies due to the recurrent course of the pseudo-obstructive syndrome and because it will subsequently be increasingly difficult to differentiate a mechanical obstacle, from a bridle for example, in a previously laparotomized patient. In these cases, full-thickness biopsies of the intestinal wall will also be very useful. When the notable abdominal distension and the complete closure of the bowel to feces and gas is indicative of prevalent colonic concern, the bloodless method of aspiration via colonoscopy has proven sufficient in some experiences (84,85,79). The simple cecostomy, however, is almost always curative; segmental or total colectomy with intraperitoneal ileo-proctostomy in cases of local or complete impairment of viscera viability (86,82).

EPIDEMIOLOGY – CLINICAL PRESENTATION

In our personal experience we have evaluated 14 patients, with acute and chronic intestinal pseudo-obstruction, aged between 26 and 92 years, 9 male and 5 female.
In every case the "occlusive" clinical appearance was acute and only in 5 there were precedents of recurrence of obstructive syndrome. In 8 cases of pseudo-obstruction with a predominantly colonic location, for diagnostic purposes, peculiar and distinctive characteristics from true mechanical occlusions were the acute onset of the symptoms, painful, tensive or cramp-like, rapid and worsening distension, minimal gastric

drainage of non-bile fluid. Abdominal examination without preparation rarely demonstrated overdistention of the entire colon; more often points of interruption at the level of the right, left, sigmoid flexure, with relative greater segmental distension, ultimately prevalent in the right colon; the absence of air-fluid levels, the flattening of the colonic wall with immobile and non-edematous haustra constituted further differential elements. The barium enema, performed urgently, has always unequivocally demonstrated the absence of organic lesions and has been useful in differentiating other mechanical causes, such as volvulus of the cecum and cecal "bascule" and other colectasias from ischemic colitis and toxic megacolon in Crohn's and idiopathic recto-colitis.

The anamnestic evaluation of previous or currently connectable diseases revealed sclerotic-hypertensive heart disease in 4 patients, diabetes in 5 and serious hydro-electrolyte imbalances in 4.

In only one case the pseudo-obstructive syndrome was resolved by simple perendoscopic decompression; in seven, cecostomy was indicated as necessary; in one of these, due to subsequent and serious ischemic suffering, right colectomy was performed. Two patients died in the immediate postoperative course.

The clinical manifestations (vomiting, pain and distention limited to the upper abdominal quadrants), often of a recurrent nature, were also sufficiently indicative in the forms with a prevalent gastro-duodenal localization (in 2 patients) or jejuno-ileal (in 4 patients). The imaging exams always allowed distinctive elements, but in a diabetic patient, submitted twenty days before to anterior resection of the rectum for carcinoma, the diagnostic orientation for ab extrinsic mechanical causes again led to blank laparotomy, however avoided in three other observations, one of which had an unrecognized etiology and two certainly linked to the intake of antidepressant drugs, and treated with metoclopramide. The radiological demonstration of megaduodenum in one patient and notable gastro-duodeno-jejunal ectasia in other, was indicative, albeit in the absence of familiarity, for idiopathic visceral degenerative forms, later confirmed by histology. The simple gastro-jejunostomy operation was decisive in the first of these; in the other, the partial gastric resection with gastro-jejunostomy was followed by progressively worsening pseudo-obstructive episodes, certainly not subject to further surgical measures, until the unfortunate outcome/exitus ninety days after the operation.

ACUTE SMALL BOWEL OBSTRUCTION

INTRODUCTION

Acute small bowel obstruction is a common surgical emergency due to mechanical blockage of the bowel by intra-peritoneal adhesions in most cases. After the improvement in results which in the last two decades has followed a more aggressive therapeutic approach, more recently, no further decreases in morbidity and mortality have been recorded which remain high for a pathology which is in itself benign, as intra-abdominal adhesions, along with other pathological processes. It therefore seemed advisable to reconsider the usual diagnostic-therapeutic schemes, based on the study of literature acquisitions and some personal experiences. These evaluations suggest that it is possible to identify, beyond the generic label of acute small bowel occlusion, dissimilar occlusive syndromes with different treatments and prognoses. Overcoming the therapeutic choice of emergency surgery in all acute small bowel occlusions, it is possible to propose, for the clearly identified patientes, a correctly delimited period of conservative treatment with intestinal decompression which, in various experiences, has not only served to postpone the surgery but was followed by stable resolution of the occlusive syndrome.

EPYDEMIOLOGY – CLINICAL PRESENTATION

As part of our personal observations we evaluated 30 patients with acute small bowel occlusion. This retrospective examination of some cases is characterized by uniform clinical

judgment and therapeutic orientation. As usual, the largest share of small bowel occlusions is attributable to simple adherent bridles, without any vascular compromise, in patients who have previously undergone laparoscopies/laparotomies for different causes; with less frequency, occlusions with strangulation from parietal or internal hernia follow and finaly a heterogeneous group whose etiology is mostly unusual. There is no evidence of age difference between patients with different occlusive causes; however, in advanced age there is a greater incidence of occlusions due to neoplastic causes even in the small intestine. Age as a generic index of progressive impairment of general conditions can lead to an increase in morbidity. The different etiologies have influenced some peculiarities in the predominance and duration of the symptoms: the rapid clinical progress of occlusions due to hernia strangulation is characteristic, as opposed to the slow evolution of those due to adhesion syndrome.

The surgery was the first therapeutic procedure applied urgently for hernia occlusions (11 patients) with the risk of strangulation. In the remaining 19 cases, with well-founded hypothesized adhesion bridle occlusion, secondary to previous surgery, characterized by recent onset and minor distension of the intestinal loops, the observational procedure with conservative therapy was performed. The conservative therapeutic approach was interrupted within 18/24 hours in 14 cases for the absence of signs of resolution of the occlusive syndrome; only in 5 cases was the conservative approach successful. Therefore it is possible to propose a therapeutic approach that involves the identification of those cases in which it is reasonable to start a period of observation since some of them could experience a stable resolution, with the appropriate hydroelectrolytic rehydration therapy and nasogastric aspiration. without surgery.

The general postoperative complications recorded in our 25 patients, undergoing surgery, were bronchopneumonia in 3 (12%), urinary infections in 6 (24%), lower limb phlebitis in 2 (8%); the specific complications were postoperative mechanical reocclusion in 2 (8%), laparotomy incision infection in 3 (12%), postoperative ileus in 2 (8%). Post-treatment morbidity in the 5 patients successfully underwent decompression therapy did not present any significant elements; mortality is absent in both patient groups. In this experience, no correlation was found between a presumed operative delay and an increase in morbidity, confirming that, within certain time limits and for some causes of occlusion, a delay in surgical treatment, whether planned from the beginning or occurred with the change in therapy, has no significant consequences. Ultimately, apart from the causes of occlusion which lead to ischemic suffering of an intestinal loop in a short space of time (hernial strangulation, volvulus, intussusception, etc.), simple mechanical occlusion of the small bowel offers a sufficiently long period (24-42 hours) in which the start of hydroelectrolyte reintegration therapy, accompanied by effective careful intestinal aspiration (nasogastric or jejunal tube) could constitute a useful preparatory phase for surgery, but in some cases (incomplete occlusion from bridles for example) could be followed by stable resolution of the occlusive syndrome.

Discussion

In most case reports, small bowel occlusions are largely linked (over 50% of cases) to adhesions in an almost constant relationship with previous intraperitoneal interventions. Other well-represented causes (30%) of jejuno-ileal occlusion are parietal hernias or internal hernias and neoplastic pathology; the remaining cases (20%) constitute a heterogeneous group in which disparate causes converge as inflammatory bowel disease (Crohn disease), intraluminal obstruction by foreign bodies, or stool blockage and volvulus. In the pediatric population, common causes include congenital atresia,

intussusception and other congenital anomalies. Some patients also present, the observation is almost always intraoperative, dual cause, both valid, of intestinal obstruction. The attempt at conservative therapy of a simple small bowel obstruction, or also partial small bowel

obstruction (P-SBO) due to a presumed adhesion bridle, requires accurate and careful etiological identification, the evaluation of the general conditions and the possible hydro-electrolyte depletion, but above all the evaluation, as precise as possible, of the eventual negative effect of delaying surgery. A certain number of small bowel obstructions are resolved through a "rest" phase with the brief suspension (24/42 hours) of feeding, and depletion with nasogastric aspiration, thus allowing the intestinal loops to return to normal size; in this way adhesions and malpositions reduce or lose the possibility of constituting a mechanical obstacle to intestinal transit. Usually, small bowel obstruction resolves after a few days (87). Some SBOs due to particular pathological conditions such as obstructions by advanced tumors with intra-abdominal metastases, whose initial treatment should be non-surgical, fall within the initial conservative therapeutic indication; also SBO (partial or total) from inflammatory bowel diseases, in which non-surgical treatment, in combination with high-dose steroids and parenteral therapy for prolonged periods of intestinal rest, is indicated to reduce the inflammatory process, adopting surgical therapy if the conservative treatment fails; as well SBOs subtended by circumscribed peritonitis (intra-abdominal abscess), in which conservative thepapeutic option with CT– guided drainage of the abscess is usually sufficient to resolve the obstruction.

On the contrary complete mechanical obstruction of the intestine (C-SBO) leads to distension of the proximal segments and decompression of the distal intestinal segments. Initially, peristalsis remains active and increases, leading to frequent bowel movements. Vomiting may occur due to distension of the stomach and intestines proximal to the obstruction. The distension of the intestinal wall, progressive and persistent upstream of the occlusion, will first interrupt the venous blood flow, causing edema and inflammation of the intestinal wall. Third spacing may also occur, so that excessive fluid moves from the intravascular space into the interstitial space, the third space, which is the nonfunctional area between cells. This can cause potentially serious problems such as edema, reduced cardiac output and hypotension (88). In the thickened and inflamed intestinal wall, arterial hypoperfusion leads to the onset of ischemic suffering and bacterial translocation. Bacterial translocation can cause peritonitis and bacteremia, most commonly by Escherichia coli. As severe parietal distention persists, hypoperfusion is followed by complete interruption of arterial flow, leading to intestinal ischemia and finally to perforation and peritonitis.(89).

Actually many patients with small bowel obstruction require surgical treatment.
In all cases, the initial treatment of acute small bowel obstruction, before further distinguishing between complete or partial occlusion, involves hydroelectrolyte restorative therapy, pain control, antibiotics and nasogastric decompression. Antibiotics of choice for small bowel obstruction should target the intestinal flora and cover both gram-negative and anaerobic bacteria (90).

As already mentioned, partial obstructions of the small intestine can often be treated conservatively with nasogastric decompression and the infusion of fluids and electrolytes, while surgery may not be necessary. (91,92).

There is considerable controversy regarding the management of patients with SBO. Some surgeons believe, without making a distinction between partial or incomplete small bowel occlusion and complete occlusion, that patients with SBO who undergo surgery have a lower frequency of recurrence and a longer time interval before eventual recurrence (93).

However, since the accurate identification of patients with incomplete SBO who could avoid operation by intestinal decompression is difficult, it may be reasonable to propose an initial therapeutic phase with nasogastric or nasointestinal decompression tube, in the absence of clinical evidence of intestinal strangulation (94, 95).
At least two different modalities for implementing intestinal decompression have been proposed, but no firm and reliable conclusion has been reached as to whether nasointestinal decompression (NIT) is superior

to nasogastric decompression (NGT) in the treatment of patients with SBO.

There is little information in the literature regarding NIT versus NGT in the management of SBO.
As regards the comparison between naso-intestinal and naso-gastric decompression in the literature, through meta-analysis studies, no different therapeutic efficacy is reported in the management of both P-SBO and C-SBO (92,96).

There is little information in the literature regarding NIT versus NGT in the management of SBO.
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Overall, Xian-Wen Dong *et al.* (6) argue, based on their experience, that partial small bowel obstruction (P-SBO) was more likely to respond to nasointestinal decompression, whereas patients with complete small bowel obstruction (C-SBO) required surgery. Over four-fifths of C-SBO patients required surgical therapy, while four-fifths of P-SBO patients were treated with intestinal decompression, followed by resolution of the obstruction syndrome.

The recommendation that the operative procedure to treat C-SBO should not be delayed is highlighted, while patients with P-SBO who did not show clinical, laboratory or instrumental signs of strangulation were more likely to have a positive response to treatment with decompression tube (97).

Wolfson *et al.* (98) demonstrated that in 64% of patients with C-SBO while only in 17% of patients with P-SBO an operation was necessary. They therefore stated that most patients with clinical features of C-SBO would ultimately require a surgical procedure, whereas patients with P-SBO had a greater likelihood of success with decompression via the nasointestinal tube (NIT).

Some studies have evaluated the use of water-soluble oral contrast medium (WSCM) as a tool in the management of SBO and as a predictive tool for the non-operative resolution of adhesive SBO. Although it does not cause resolution of SBO, WSCM offers a consistent diagnostic possibility and can reduce hospital stay in patients who do not require surgery. (99)

Furthermore, a systematic review of retrospective data from 242 patients in 10 studies with acute uncomplicated adhesive SBO indicated no statistically significant differences between the administration of water-soluble contrast versus saline in reducing the need for surgery (24% vs 20%), respectively) or intestinal resection (8% and 4%) (100).

The final evaluation which reports no mortality due to the possible delay in surgical intervention following the application of intestinal decompression procedures in SBO is certainly relevant (95,101).
Although a prolonged interval of time between the onset of symptoms and the start of direct surgical treatment could be considered a prognostically unfavorable element for a possible progressive hydro-electrolyte imbalance, despite the contextual correct infusion therapy and endoluminal decompression, however, in some experiences (102), the importance of the delay in carrying out the surgical intervention, which proved to be unavoidable, was not statistically significant in the various etiological groups of occlusion, with the exception of occlusions with strangulation, which absolutely do not fall within a wait-and-see therapeutic approach. Alongside the clinical examination, which retains a decisive role, laboratory and instrumental investigations deserve careful evaluation in order to identify occlusions due to strangulation, to confirm the site of the occlusion, and finally to choose the cases that can be started for conservative treatment initial and assessable within a limited period of time.

No laboratory investigation can resolve these questions. Marked leukocytosis may lead to suspicion of occlusion with vascular compromise, but the data remains nonspecific

and inconsistent. Alterations in the hydro-electrolyte balance are characteristic of all occlusive syndromes and therefore non-specific for the purposes of a specific diagnosis and require prompt and accurate correction. The most frequent symptoms are colicky abdominal pain, nausea and vomiting; the sign of abdominal distension is characteristic, not very evident in the initial phase; similarly, the closure of the bowel, for the early diagnosis of small bowel obstruction, is not reliable, as there may be residual emptying of the colon. The finding on objective examination of the abdomen of an area, even limited, of defense contracture gives rise to the well-founded suspicion of occlusion with strangulation. A reasonable hypothesis of the location of the intestinal obstruction can be advanced with the usual imaging tests such as US, standard Rx, CT Abdomen. The standard x-ray of the abdomen, obviously in orthostasis, although not highly sensitive, helps to validate the diagnosis if the presence of air-fluid levels or a shortage of gas is observed. (103) Multislice CT has been shown to be a particularly effective imaging tool for evaluating patients with suspected SBO, with a sensitivity greater than 95%. Furthermore, CT imaging is also able to detect C-SBO and its complications, which require urgent surgical procedure, generally not detected with standard x-ray, including ischemia, perforation, mesenteric edema and pneumatosis (104). Ultrasound is less expensive and less invasive than CT and can reliably exclude SBO in 89% of patients; specificity is reportedly 100%. It may represent a useful alternative imaging modality in children and pregnant women.

The choice of the abstentionist approach towards small bowel occlusion requires the acquisition of some clinical "certainties", some steps in the selection phase of patients with occlusion to initiate the conservative approach. The distinction with mechanical occlusion of the colon must be established with sufficient certainty, because it has a different etiology, pathophysiological basis and clinical evolution and a different therapeutic program. Cases in which there is even the slightest suspicion of occlusion with strangulation must be quickly identified, since in this condition surgical intervention cannot be postponed. Finally, even in the context of ileal occlusions without strangulation, it is necessary to select cases with complete occlusion already established for some time, in which the surgical intervention should not be further delayed. In fact, procrastinating a necessary intervention can have negative repercussions whose extent varies depending on the cause of the mechanical occlusion. Therefore the therapeutic approach must be differentiated in relation to the different etiologies: the suspicion of postlaparoscopic-laparotomic bridles or adhesion malpositions can rightly lead towards an abstentionist choice at least in the first hours. This hypothesis can also be considered in patients with advanced abdominal tumors.

The obvious advantage of avoiding surgery is combined with a lower risk of reocclusion since every relaparoscopy-laparotomy for lysis of adhesions leaves open the possibility that these will reform again in an unfavorable position without having concretely effective means of prevention. However, outside of the etiological connotation of initial/incomplete occlusion due to adherence/malposition and a reasonable amount of time for spontaneous resolution, in the face of an uncertain and incomplete therapeutic response, further delay of surgical intervention, later proved necessary, is a questionable option.

In fact, in the patient in whom an adhesion syndrome is unlikely or in whom there is no sign of release in approximately 24-36 hours, the conditions for the stable success of expectant medical therapy are lost, while the fundamental role of surgical intervention is reaffirmed.
Some experiences in the literature (105,106, 107,108,109) report stable solutions, without complications, with medical abstention therapy, prolonged up to 48-72 hours, for partial or simple acute small bowel occlusions in very high percentages (over 65% /81% of cases). Ultimately, current and widely shared clinical experience demonstrates that the non-operative management is effective in approximately 70–90% of patients with SBO by adhesive syndrome (110,111, 112,113).

However, it can currently be considered that incomplete intestinal occlusion has a real anatomical-clinical significance (particularly in those who have previously had abdominal surgery). On a pathophysiological level, the distinction between complete and incomplete occlusion appears evident: the difficult progression of the intestinal contents, not its blockage, despite causing the appearance of some clinical appearances and characteristic signs (intestinal distension which can be assessed clinically and radiologically, with some air-fluid levels, reduction/ temporary suspension of the normal channeling of feces and gas, modest colic-type abdominal pain), does not give rise to the onset of complete occlusive syndrome, with all its serious hydro-electrolyte and anatomopathological alterations affecting the intestinal walls. On the level of clinical practice, this differentiation is certainly more indefinite and difficult as it involves giving a "quantitative" evaluation, so to speak, of the same symptoms that make up the clinical picture of both complete and partial occlusion. A rational therapeutic protocol does not include immediate surgical intervention in all cases with abdominal distention and air fluid levels. The emergency operative indication must not ignore rapid and accurate hypotheses of etiological diagnosis, medical treatment, even prodromal to surgery, procedures limited in time, such as clinical examination, haematochemical and instrumental investigations, the correction of any deficits of vital parameters, hydroelectrolyte reintegration, intestinal decompression. No sign of resolution within 24-36 hours or a worsening of the general clinical picture requires surgical intervention. In conclusion, it can be considered that each case of small bowel obstruction must be considered individually, separating the full-blown and complete cases and those with suspected strangulation requiring emergency intervention, from those with moderate clinical manifestations, which are not clearly and immediately "surgical". which could benefit from a short period of medical therapy for a stable resolution.

CONCLUSIONS

Intestinal pseudo-obstructions can be defined as an occlusive syndrome without mechanical obstacle. The clinical findings, which are more frequent today, are certainly related to more precise pathophysiological knowledge and the recognition of the determining causes allows, in practice, more rational therapeutic decisions, avoiding unnecessary laparotomies or those limited to intestinal biopsy or delays in surgical treatment of mechanical forms. In conclusion, in personal experience, intestinal pseudo-obstructions constituted a not unusual pathology, the diagnostic recognition of which was possible through clinical-anamnestic evaluation and was confirmed in many cases by radiological imaging investigations without preparation and contrast graphics, carried out in the first instance. The pathophysiological reasons, although certainly not defined, were adequate for rational surgical implications and indications.

Acute small bowel obstruction presents, in a considerable number of cases, characteristics of the pathology underlying the obstructive syndrome which determine possible clinical manifestations of the onset and above all the evolution which require an adequate and appropriate therapeutic approach. The efficient obstacle to the progression of the intestinal contents always constitutes the central nucleus of the occlusive syndrome at all gastro-entero-colic levels. At the level of the small intestine, some pathological conditions determine pathophysiological situations of incomplete intestinal obstruction or with the possibility of regression with the simple decompression of the intestinal segment lying upstream of the obstacle. Adhesive or cicatricial syndromes secondary to previous intraperitoneal accesses of surgical procedures are involved, or also the inflammatory processes of IBD, susceptible to even partial post-therapeutic regression. The immediate diagnostic confirmation of cases of occlusion with strangulation, of cases with complete occlusive syndrome (C-SBO) established for some time (a few days) and of occlusions of any etiology that do not respond to

a very short period of conservative therapy forces urgent surgery.

The pathophysiological characteristic of partial occlusion (P-SBO) and adhesive small bowel obstruction (A-SBO) has introduced, after the necessary clinical, laboratory and instrumental imaging diagnostic identification, in these simple or incomplete small bowel occlusions, inflammatory and from adhesions which are largely post-operative, the initial therapeutic approach with a conservative procedure, through intestinal "rest", decompression upstream of the obstacle, antibiotic and hydro-electrolyte reintegrative therapy, parenteral nutritional support. As reported by the examination of the literature, albeit unstructured and unsystematic, the non-surgical therapeutic approach in correctly identified cases has provided favorable results of stable resolution of the occlusive syndrome.

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