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Professional paper

Gastrointestinal Pseudo-Obstruction: Report of a Patient with Postoperative Pseudo-Obstruction

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A B S T R A C T

Postoperative pseudo-obstruction is a rare state of protracted gastrointestinal paresis that may progress to paralysis without the presence of obstructive lesions. Pseudo -obstruction is usually, but not exclusively, associated with an abdominal operative procedure (laparotomy), however, it may occasionally occur following extra-abdominal operations. As differentiated from the usual, 'physiologic' postoperative paresis, pseudo-obstruction persists for more than 7 days. The pathogenesis of postoperative pseudo-obstruction is complex and as yet partially unknown. Whereas the 'physiologic' postoperative gastrointestinal paresis includes short-term functional cholinergic depression of the visceral organs, in pseudo-obstruction focal lesions in the region of Auerbach's plexus, manifesting as visceral neuromyopathy, are involved. That is why the 'physiologic' postoperative paresis never transforms into paralytic ileus, while in pseudo-obstruction such a risk is potentially involved. The treatment for pseudo-obstruction is as a rule conservative. Surgical treatment (cecostomy) is rarely required. Colonoscopic decompresive suction is usually enough to eliminate the risk of colon rupture due to extensive distention by fast growing meteorism. A patient with postoperative pseudo-obstruction is presented.

Introduction

Pseudo-obstruction is a relatively new term for very rare gastrointestinal motility impairments, especially in the colonic region, manifesting as paresis or paralysis in the absence of obstructive lesion (Ogilvie's syndrome[1]). Pathophysiologically, it is a protracted peristalsis inefficiency which can be related to an abdominal[2], and less commonly extra-abdominal surgical procedure. However, it may occur in the course of various diseases, major medical insults, etc.[1], that give rise to the development of pseudo-obstruction (Table 1) as an influence of some medications. From the surgical point of view, pseudo-obstruction as a complication of

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TABLE 1
PROVOKING FACTORS FOR PSEUDO-OBSTRUCTION

Traumatic lesion
- abdominal, less frequently extra-abdominal (orthopedic, urologic) operative procedures
- accidental abdominal trauma
- craniocerebral injuries
- spinal and pelvic fractures

Homeostasis disorders
- hypo- and hyperkalemia
- hypo- and hypercalcemia
- hypo- and hypermagnesemia
- hypoxia

Infections
- sepsis
- pneumonia
- viral hepatitis
- adnexitis
- acute gastroenteritis (viral, bacterial)

Vascular disorders
- collagenous vascular diseases (periarteritis nodosa)
- mesenteric vascular insufficiency
- hypovolemia

Drugs
- anticholinergics
- antiparkinsonics
- $_1$ and $_2$ neural receptor antagonists
- antidepressants

Intoxication
- heavy metal intoxication (As, Hg)

Metabolic disorders
- diabetes mellitus
- hypothyroidism
- acute porphyria
- renal insufficiency
- Reflex provocation
- full urinary bladder
- gastric retention

Major medical insults
- stroke
- myocardial infarction

Neoplastic lesions
- malignant lesions infiltrating celiac ganglia

operative treatment (postoperative pseudo-obstruction) is of greatest importance, thus this paper will focus on this type of pseudo-obstruction.

Postoperative pseudo-obstruction is considered when postoperative 'physiologic' gastrointestinal paresis persists for more than seven days. Postoperative pseudo-obstruction begins in an acute form predominated by gastrointestinal paresis. It is especially pronounced in the colonic region and, if persisting, it may progress into the chronic form with the possible development of paralytic ileus. In this condition, gastrointestinal paresis is replaced by paralysis[3–8]. Postoperative pseudo-obstruction as well as a pseudo -obstruction of other than operative etiology can cause considerable differential diagnostic problems to the surgeon and anesthesiologist. It is not always easy to differentiate pseudo-obstruction from mechanical obstruction (mechanical ileus) or 'surgical' paralytic ileus. While postoperative pseudo-obstruction as a rule requires conservative treatment[2–6], the other two conditions demand surgical management.

Postoperative pseudo-obstruction

'Physiologic' postoperative gastrointestinal paresis develops after operative procedures, mostly those including laparotomy, however, it occasionally occurs after some extra-abdominal operations (e.g., orthopedic, urologic, etc.). This type of gastrointestinal paresis usually lasts for 24 to 72 hours, never exceeding seven days in duration, whereafter a normal and efficient peristalsis is restituted spontaneously[2]. The reason for its occurrence is transient functional neurovegetative dystonia of Auerbach's plexus consequential to laparotomy, intraoperative visceral manipulation as well as possible biochemical homeostasis disturbance. General anesthesia as an additional etiologic factor cannot be excluded either. An

important role is thereby played by neuroleptics, ganglioblockers, and morphine with its derivatives. Therefore, the 'physiologic' postoperative paresis may also occur after extra-abdominal operations if performed in general anesthesia[2,9,10].

Postoperative 'physiologic' paresis should be distinguished from rare pseudo-obstruction. The two differ according to their pathogenesis, duration, and evolution. Pseudo-obstruction regularly lasts for more than seven days. Initially, it cannot be differentiated from 'physiologic' postoperative paresis. The later stage of pseudo-obstruction is predominated by protracted gastrointestinal paresis, which is most pronounced in the colon region, is accompanied by meteorism, and progresses to paralytic ileus in 0.5%–1.5% of patients. Pathogenetically, pseudo-obstruction is not underlain by functional lesions in the area of Auerbach's plexus, but is due to focal neuromuscular lesions in terms of visceral neuromyopathy[11]. Although the pathogenesis of pseudo-obstruction has not yet been fully clarified, generally the condition is successfully controlled by the elimination of particular provoking factors[12–16].

Clinical picture and pathophysiology of postoperative pseudo-obstruction

As mentioned above, in the initial stage of the disease it is impossible to differentiate pseudo-obstruction from 'physiologic' postoperative paresis. Pseudo-obstruction should only be considered when inefficient peristalsis persists for more than seven days. Abdominal distention gradually increases due to the rising meteorism primarily of the paretic colon. This is not accompanied by strong abdominal pain but rather by mild sensitivity and tension. More severe pain can only be present in the region of laparotomy wound as a consequence of abdominal wall distention. Gastric paresis results in retention of the gastric contents, accompanied by nausea and vomiting. On auscultation, weak peristaltic activity can only occasionally be heard, with some 'decanting' phenomena, exclusively at the more or less restituted small intestine peristalsis. Colonic meteorism accompanied by extreme distention of the intestinal wall can occasionally show rapid progression also in the region of cecum, which represents a 'blind sac', leading to its rupture with fatal outcome. However, this complication is extremely rare. A pseudo-obstruction persisting for more than ten days is called pseudo-obstruction.

The pathophysiologic events in pseudo-obstruction are based on prolonged gastrointestinal paresis, which is especially pronounced in the large intestine. The intestine responds to the retention of intestinal contents by enhanced secretory activity. This in turn results in the escape of extracellular fluid and electrolytes into the intestinal lumen. At the same time, intestinal flora flourishes in the stagnated intestinal contents, stimulating the processes of its putrefaction and fermentation. Elevated intraluminal pressure leads to intestinal wall distention, compromising its macro- and microcirculation. Water and electrolyte resorption is prevented, with the release of biological amines (e.g., serotonin, histamine) in the water. These changes in the intestinal wall lead to a damage to the 'mucosal barrier', a regulator of the intestinal functional activities. All these events allow for further uncontrolled loss of water and electrolytes, which in turn leads to dehydration of the body. As this condition is accompanied by concurrent elevation in the sympathetic tonus, additional damage to the micro- and macrocirculation as well as an inhibitory effect on circular smooth muscles of the intestine by blocking alfa and beta receptors of the myenteric plexus occur. This additionally potentiates paresis of the intestinal peristalsis

due to the preponderance of adrenergic over cholinergic activity[2]. If such a condition persists for a prolonged period of time, fluid and gases grossly accumulate in the intestinal lumen, with extreme distention of its wall, which eventually leads to complete cessation of peristaltic activity, and paralytic ileus develops. Maximal distention of the intestinal wall, especially in the region of cecum, may result in its rupture.

Mention should be made of the rare form of so-called extensive pseudo-obstruction, which manifests with additional hypotonia and dilatation of extraperitoneal visceral organs such as renal pelvis and ureter, extrahepatic bile ducts, and parts of female reproductive organs. However, this type of pseudo-obstruction is rarely seen in postoperative pseudo--obstruction[10].

The diagnosis of postoperative pseudo-obstruction

It is of utmost importance to differentiate pseudo-obstruction from postoperative obstructive (mechanical) ileus or postoperative paralytic ileus as a complication of the operative procedure with development of local or general peritonitis. It has already been mentioned that the initial stage of postoperative pseudo-obstruction cannot be distinguished from 'physiologic' postoperative paresis. In the late stage, however, there may also be some differential diagnostic difficulties in differentiating pseudo-obstruction from a 'creeping' development of postoperative peritonitis or postoperative mechanical ileus (e.g., adhesive ileus). On making the diagnosis of pseudo-obstruction, the history and clinical finding are of paramount importance. After a normal immediate postoperative course, the expected spontaneous restitution of peristalsis fails to occur even after day seven postoperatively. A very low and very short peristaltic activity, occurring in very long intervals, can occasionally be detected by auscultation, however, it is inefficient. There is no defecation or flatulence. Inspection reveals distention of the abdominal wall; there is no »defance« on palpation, but there is diffuse, slight sensitivity. Abdominal percussion produces a clear, tympanic sound due to meteorism. There are no signs of free fluid accumulation in the peritoneal cavity. The patient does not suffer severe abdominal pain except for possible mildly painful tension, however, the laparotomy wound area may be more painful because of distention. Paresis and hypotonia of the stomach develop due to protracted peristaltic inactivity which partially involves the stomach. Large amounts of liquid gastric content are accumulated and retained in the gastric lumen. Digitorectal examination reveals an empty ampulla. A native abdominal roentgenogram in upright position is of great diagnostic help. If the patient cannot stand upright, roentgenogram should be taken while lying on his left side. In the initial stage of pseudo-obstruction (acute phase), roentgenogram will provide an insight into the extent and spread of meteorism without the presence of aeroliquid levels. In the late (chronic) stage of the disease, they are always present, yielding a typical x-ray pattern of intestinal obstruction. As differentiating from mechanical ileus, however, x-ray of the lower abdomen and pelvis in pseudo-obstruction shows air in the rectum. The terminal stage of pseudo-obstruction, which manifests as paralytic ileus, is characterized by auscultatory silence, along with extreme meteorism, abdominal distention, and x-ray presence of aeroliquid levels. Splashing of the fluid accumulated in the distended bowel is heard on shaking the patient's abdomen. The patient's general condition is deranged, including changes in homeostasis, threatening the patient's life. This terminal stage of pseudo-obstruction, if developed, resembles

the terminal stage of surgical paralytic ileus. Particular symptomatic parameters and diagnostic algorithms can be very useful to solve the difficult diagnostic dilemma between pseudo-obstruction, mechanical obstruction, and surgical paralytic ileus (Tables 2 and 3).

If, however, the diagnosis of pseudo--obstruction cannot be definitely made in spite of all endeavors, explorative laparotomy is indicated.

The treatment and prevention of postoperative pseudo-obstruction

Postoperative pseudo-obstruction can in a majority of patients be successfully controlled by appropriate therapeutic procedures that prevent the action of particular provoking factors (Table 1). These therapeutic procedures include: restitution of normal homeostasis; exclusion of drugs that modify intestinal motility; introduction of target antimicrobial therapy for inflammatory processes; prevention of urine and gastric content retention by placement of permanent catheter and nasogastric tube; and ensuring the required amounts of calories to the body, usually by parenteral nutrition.

Beside these therapeutic procedures, medicamentous therapy for the stimulation of peristalsis has a certain role in the management of pseudo-obstruction. Clinical experience has shown that erythro-

TABLE 2
HISTORY AND CLINICAL PARAMETERS HELPING IN DIFFERENTIAL DIAGNOSIS OF PSEUDO -OBSTRUCTION FROM MECHANICAL (SURGICAL) ILEUS

Pseudo-obstruction	Mechanical obstruction
pre-existing gastrointestinal symptoms (dyspepsia, dysphagia) frequently found in the history	usually no pre-existing gastrointestinal symptoms in the history
relapsing uroinfection may exist; urography shows dilated and atonic ureter and renal pelvis (extensive gastrointestinal pseudo-obstruction)	no symptoms of uropoietic involvement
retarded gastric emptying (x-ray); the stomach is hypotonic or atonic (gastrographin or diluted barium)	normal gastric emptying
irrigoradioscopy shows dilated colon without obstructive lesion (diluted barium is superior to gastrographin)*	irrigoradioscopy may show an obstructive lesion
native pelvis x-ray shows 'air' within the rectum	native pelvis x-ray does not show air in the rectum
intestinal passage (diluted barium) shows moderately dilated small intestine without obstructive lesion	intestinal passage may point to an obstructive lesion (small intestine ileus)
usually occurs during early postoperative period	generally does not occur immediately after the surgery
history data on the use of drugs that modify intestinal motility	no medication data
data on metabolic diseases	no data on metabolic diseases
explorative laparotomy reveals no obstructive lesion in addition to intestinal distention	explorative laparotomy reveals an obstructive lesion

*the examination is contraindicated in excessive colon distention

TABLE 3
ALGORITHMS USED FOR SUSPECTED MECHANICAL OBSTRUCTION

Abdominal x-ray suggesting large intestine obstruction irrigoradioscopy (diluted barium contrast) or colonoscopy (diagnostic and therapeutic)	
mechanical reason	functional reason
operation	conservative treatment according to etiology
Abdominal x-ray suggesting small intestine obstruction nasogastric tube with observation, possibly intestinal passage with diluted barium contrast, and possibly abdominal computed tomography	
mechanical reason	functional reason
operation	conservative treatment according to etiology

mycin, acting as a motilin antagonist, has a favorable effect on the peristaltic activity[16,18]. Mathias and Ducher[10] recommend parentheral administration combination of guanetidine or betanidine and neostigmine (2–4 amp/day) with the use of an enema. Guanetidine and betanidine act as adrenergic ganglioblockers, and neostigmine as an inhibitor of pseudo--cholinesterase. The beneficial effect of these pharmaceuticals usually results from excessive stimulation of the sympathicus. However, in patients with largely distended meteoristic colon, neostigmine should be avoided. These agents primarily act on the stomach and small intestine rather than large intestine. Therefore, their action can cause an abrupt elevation of intracolonic pressure and its possible rupture. Schumperlick and Hrynyschun[2] report on their favorable experience with the infusion of 10% low-molecular dextran in combination with 20% sorbitol. Their action results in partial tissue O_2 pressure increase, reduction of interstitial edema, and improved microcirculation, thus paving the way for restitution of peristaltic activity. Great expectations have recently been raised by ceruletide, a preparation releasing the smooth-muscle stimulating acetylcholine on myoneural synapses[10]. Ceruletide is contraindicated in patients with severe cardiac insufficiency. An administration of metoclopramide (Reglan) can sometimes be useful. If the aforementioned therapeutic procedures, including medicamentous therapy, fail to restitute efficient peristalsis, then endorectal suction is indicated. The aim of endorectal suction is to produce active decompression of the colon. It is performed by a colonoscopic technique allowing the introduction of an aspiration fenestrated

drain up to the transverse colon. Clinical experience has shown that there is no need for the drain to reach the cecum. Then, the drain is connected to a system for continuous suction of the content and gases from the colon. Endorectal suction should be carried out by a highly experienced endoscopist, because colonoscopy must be performed with very low and cautious air insufflation. Also, he must be able to observe any possible signs of intestinal wall ischemia (red and fragile mucosa), which requires an emergency operation (cecostomy). Such operation is also indicated by the development of extreme colon distention if the distended cecum diameter exceeds 10 cm, implying a pending risk of its immediate rupture.

Long-standing clinical experience has shown that the risk of the development of postoperative pseudo-obstruction can be significantly reduced by appropriate preoperative preparation. Bowel emptying on the day preceding the surgery (enema, contact laxative, 10% mannitol, orthograde lavage) will not only reduce the operative risk, but will at the same time decrease the risk of postoperative pseudo-obstruction. Furthermore, preoperative correction of the acid-base dysbalance, anemia, and metabolic catabolism has a very important role in the prevention of pseudo--obstruction. Surgical technique is of utmost preventive importance. Gentle and careful manipulation of the viscera reduces the risk of gastrointestinal neuromyopathy with consequential pseudo-obstruction. Finally, the reflex factor of impact on the occurrence of postoperative pseudo--obstruction is avoided by the placement of nasogastric tube in the immediate postoperative period after laparotomy, and of a permenant catheter to prevent retention of urine with distention of urinary bladder, and retention of gastric content with distention of the hypotonic stomach.

Patient with Postoperative Pseudo-Obstruction: Case Report

A man aged 65 was admitted to the Department of Surgery, Zabok General Hospital, for verified (biopsy) adenocarcinoma of the rectum ampulla, without evidence of distant metastases. Upon preoperative preparation, which included intestinal lavage by mannitol solution (10%) and usual antibiotic prophylaxis, the operative procedure in general endotracheal anesthesia was performed on June 11, 1999. The procedure consisted of abdominoperineal extirpation of the rectum with left-sided terminal colostomy (PHD: adenocarcinoma, Dukes A). Upon the operation, the patient was transferred to the Intensive Care Unit. During the first three postoperative days, the patient was free from any major subjective and objective difficulties, and had normal laboratory findings. On day 4 postoperatively, however, abrupt abdominal distention due to gastrointestinal paresis occurred instead of the expected efficient peristalsis. On auscultation, a low 'decanting' phenomenon was heard. Abdominal x-ray (in upright position) showed a strongly pronounced meteorism of the gastrointestinal tract without aeroliquid levels (Figure 1). Five hundred mL of liquid gastric content were drained by a nasogastric tube. Neostigmine (4 0.5 mL/day) and an enema were introduced. On day 5 postoperatively, a small amount of mashy stool was evacuated via artificial anus, with considerable flatulence. Abdominal distention and nasogastric tube activity (50 mL) decreased. Such a relatively satisfactory condition persisted on the next, sixth postoperative day, however, without restitution of efficient peristalsis. The patient had neither stools nor flatulence in spite of prokinetic therapy continuation. On day 7, exacerbation of the patient's condition with severe abdominal distention, mild dyspnea, and abdominal sensitivity recurred. Mild, diffuse sensitivity of the abdomen was recorded, with occa-

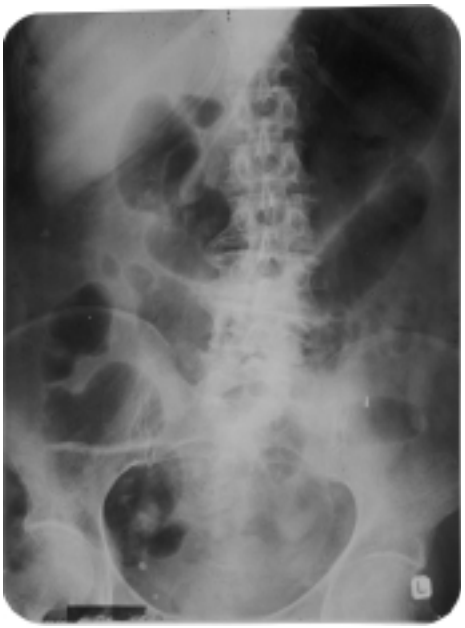

Fig. 1. Early stage of pseudo-obstruction: pronounced meteorism and gastrointestinal tract distention without the presence of aeroliquid levels.

sionally heard low 'decanting' phenomenon on auscultation. Native abdominal x-ray revealed a series of aeroliquid levels in moderately dilated curvatures of the small intestine and more strongly dilated left colon, in the region of its left flexure (Figures 2 and 3). The patient's hemogram, and water and electrolyte status showed no major changes. Operative treatment was indicated for suspicion of mechanical intestinal obstruction (adhesive ileus?), and the patient was reoperated on in endotracheal anesthesia on day 9 postoperatively.

On exploration, distention of the small intestine and especially left colon with accumulated gases and liquid content, without the signs of organic obstruction, was observed. At the same time, however, there was a high mobility of the descending and sigmoid colon due to the failure of coalescence between their mesocolon and posterior abdominal wall. Upon enterotomy, 2500 mL of liquid content were aspirated from the intestinal lumen. The operation was completed by the descending colon colopexy. The reoperation was followed by normal postoperative course. Peristalsis was restituted on day 3 postoperatively, or day 12 after the first operation, with regular evacuation *via* colostomy, and with flatulence. On day 12 post-reoperation, the patient was discharged for home care, with normal colostomy function and normal operative wound healing.

Discussion and Conclusion

Although pseudo-obstruction is by no means a frequent event, it presents a complex diagnostic and therapeutic problem. This especially holds for so-called postoperative pseudo-obstruction which occasionally may cause serious complications during the postoperative course and even lead to a life-threatening state. In a majority of cases, though, pseudo-obstruction does not show such an aggressive course and is usually successfully controlled by appropriate therapy. The reason for pseudo-obstruction should be looked for in visceral neuromyopathy consequential to neuromuscular lesions in the area of Auerbach's plexus. Pseudo-obstruction may involve the entire digestive tract (stomach, small intestine, large intestine), but is most pronounced in the region of left colon characterized by the rich network of Auerbach's plexus. The onset of postoperative pseudo-obstruction cannot be differentiated from the 'physiologic' intestinal paresis. Therefore, prokinetic agents (e.g., neostigmine) should not be administered during the first three postoperative days. It is desirable for the postoperative paresis to recover spontaneously rather than by medicamentous load[17]. Pseudo-obstruction should only be considered if paresis of the gastrointesti-

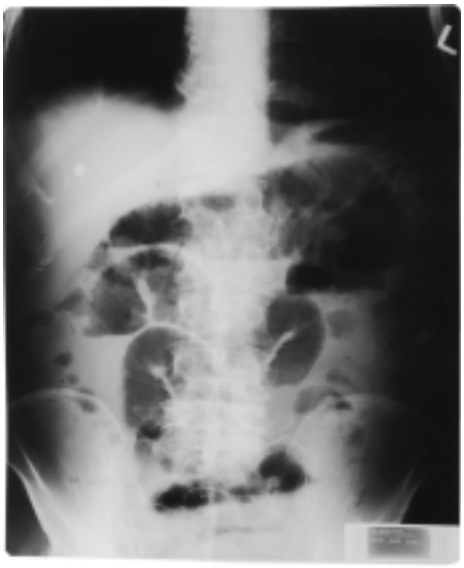

Fig. 2. Moderate distention of small intestine curvatures with strong dilatation of the descending colon in its oral segment; visible aeroliquid levels.

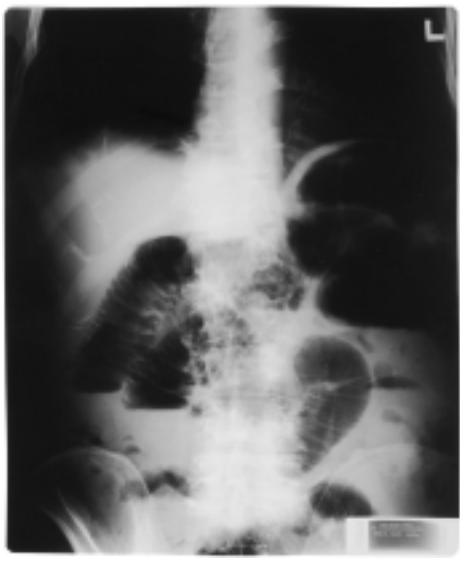

Fig. 3. In addition to distention of the small intestine curvatures with aeroliquid levels, progressing meteorism in the oral segment of the left colon.

nal tract persists for more than 7 days postoperatively. In such a case, previously mentioned therapeutic measures should be introduced. We should correction all conditions that may give rise to pseudo-obstruction (e.g., water and electrolyte dysbalance, hypoxia, urine retention, and gastric content). As there is no obstructive lesion in pseudo-obstruction, it requires conservative treatment including colonoscopic decompresive aspiration. Operative procedure is indicated rare. In clinical practice, however, it is not always easy to distinguish pseudo-obstruction, especially the postoperative one, from mechanical ileus or surgical paralytic ileus. In this case, explorative laparotomy is indicated if the diagnosis of pseudo-obstruction cannot be definitely made. It is unjustifiable to insist on the diagnosis of pseudo-obstruction, if the patient then dies from mechanical ileus or from sequels of diffuse peritonitis.

In our patient, a congenital defect underlied the development of pseudo-obstruction. Unlike most people, in our patient the left colon was not attached to the posterior abdominal wall but remained fully mobile. Under our oppinion the reason for this anomaly was the failure of the mesocolon coalescence with the posterior abdominal wall. In the phase of postoperative peristalsis 'wakening', the left colon could therefore be transferred to the medial line, whereby the vascular structures in the mesocolon were folded, which in turn resulted in intermittent hypoxia that generated pseudo-obstruction. For such a cascade of events, the potential development of postoperative ileus could not be ruled out. Therefore, explorative laparotomy was definitely indicated. Colopexy was performed in the same act to prevent the possible risk of colon volvulus.

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GASTROINTESTINALNA PSEUDOOPSTRUKCIJA: PRIKAZ BOLESNIKA S POSLIJEOPERACIJSKOM INTERMITENTNOM PSEUDOOPSTRUKCIJOM

SAŽETAK

Postoperativna pseudoopstrukcija predstavlja rijetko stanje protrahirane gastrointestinalne pareze koja može prijeći u paralizu, a bez nazočnosti opstruktivne lezije. Najčešće je iako ne isključivo, povezana s abdominalnim operacijskim zahvatom (laparotomijom), ali se katkada može pojaviti i nakon ekstraabdominalnih operacija. Za razliku od uobičajene »fiziološke« poslijeoperacijske pareze pseudoopstrukcija traje duže od sedam dana. Patogeneza postoperativne pseudoopstrukcije je složena, djelomično još nepoznata. Dok se kod »fiziološke« poslijeoperacijske gastrointestinalne pareze radi o kratkotrajnoj, funkcionalnoj kolinergičnoj depresiji organa utrobe, kod pseudoopstrukcije se radi o fokalnim lezijama u području Auerbachovog pleksusa u smislu visceralne neuromiopatije. Zbog toga »fiziološka« poslijeoperacijska pareza nikada ne prelazi u paralitički ileus, a koja opasnost u pseudoopstrukciji je potencijalno moguća. Terapija pseudoopstrukcije u pravilu je konzervativna. U liječenju pseudoopstrukcije rijetko je potrebno primjeniti kirurško liječenje (cekostomija). Kolonoskopska dekompresijska sukcija obično zadovoljava da bi se spriječila opasnost rupture kolona zbog ekstenzivne distenzije naglo rastućim meteorizmom. U nastavku je prikazan bolesnik s poslijeoperacijskom pseudoopstrukcijom.