REVIEW Article

Diagnosis of Small Bowel Motility Disorders : Part II

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EXTRACT

Patients with small bowel motility disorder have a wide range of clinical manifestation from asymptomatic to severe symptoms similar to intestinal obstruction. However, between the two extremes, patients may have dyspeptic symptoms and were diagnosed as functional GI disorders because the organic causes can not be found by endoscopic or imaging studies. Thus, a subgroup of patients with functional GI disorders may have small bowel motility disorder.

Key words : Small bowel, motility, obstruction, functional

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SMALL BOWEL MOTILITY DISORDERS

Patients with small bowel motility disorder have a wide range of clinical manifestation from asymptomatic to severe symptoms similar to intestinal obstruction. However, between the two extremes, patients may have dyspeptic symptoms and were diagnosed as functional GI disorders because the organic causes can not be found by endoscopic or imaging studies. Thus, a subgroup of patients with functional GI disorders may have small bowel motility disorder.

The major clinical disorders related to small bowel motility disorders are pseudo-obstruction, bacterial overgrowth and the irritable bowel syndrome (some subgroups).

PSEUDO-OBSTRUCTION

Intestinal pseudo-obstruction is a clinical syndrome caused by ineffective propulsion of intestinal content in the absence of a mechanical obstruction. These patients usually present with episodes of nausea, vomiting, abdominal pain and distention. Patients are often initially suspected of having mechanical obstruction.

Intestinal pseudo-obstruction can be acute (ileus) or chronic.

1. Acute Intestinal Pseudo-obstruction (ileus)

Ileus is characterized clinically by acute symptoms of nausea, vomiting, abdominal distention and, in some patients, obstipation. Similar to the symptoms of acute mechanical obstruction but no obstruction was identified.

The most common cause of ileus is intraabdominal operation. However, there are other non-operative conditions associated with ileus as shown in Table 1.

When patients present with clinical symptoms of ileus, surgical conditions including mechanical obstruction, intraabdominal inflammation such as acute appendicitis or diverticulitis and intestinal ischemia should be considered and excluded.

Clinical presentations Non-surgical patient

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Table 1 Causes of ileus

Postlaparotomy
Electrolyte derangements (e.g., hypokalemia, hyponatremia, hypomagnesemia, hypermagnesemia)
Drugs (e.g., phenothiazines, opiate narcotics, diltiazem, verapamil, clozapine, anticholinergic agents)
Intra-abdominal inflammation (e.g., acute appendicitis, acute diverticulitis, perforated duodenal ulcer)
Retroperitoneal hemorrhage (e.g., ruptured abdominal aortic aneurysm, lumbar compression fracture)
Retroperitoneal inflamation/infection (e.g., acute pancreatitis , renal lithiasis, pyelonephritis)
Intestinal ischemic (e.g., mesenteric embolus, chronic mesenteric ischemia, mesenteric thrombosis)
Thoracic processes (e.g., lower rib fractures, lower lobe pneumonias, myocadial infarction)
Systemic sepsis

usually present with poorly localized abdominal discomfort due to distention. Severe, localized pain and tenderness suggest an inflammatory cause of ileus while abdominal cramping suggests mechanical obstruction.

Auscultation of the abdomen generally demonstrates decrease bowel sounds, in contrast to active bowel sounds in mechanical obstruction.

Diagnosis evaluation Plain abdomen usually demonstrates gas throughout the stomach, small intestine, colon or rectum.

CT abdomen and barium contrast study are useful in ruling out intraabdominal inflammation and obstruction.

Treatment

1. The underlying cause should be treated

2. In postperative ileus, the small bowel function usually return to normal within 24 hours after laparotomy, where as stomach and colon function return at 48 hours and at 3-5 days, respectively. If the recovery of bowel function was delayed, complications from surgery or other causes of ileus should be considered and corrected.

3. General treatment including limiting oral intake, maintenance of intravenous volume, and correction of electrolyte abnormalities especially hypokalemia should be employed.

4. A nasogastric tube should be placed in the patient who has abdominal distention nausea or vomiting.

5. There has been reported that some medications may improve ileus such as neostigmine.

2. Chronic Intestinal Pseudo-obstruction (CIP)[3,4,12]

Chronic intestinal pseudo-obstruction is a clinical syndrome in which the patients have recurrent symptoms and signs of intestinal obstruction without detectable mechanical obstruction. This syndrome is

Table 2 Causes of chronic intestinal pseudo-obstruction [12]

I. Primary chronic intestinal pseudo-obstruction
A. Familial type
1. Familial visceral myopathies
2. Familial visceral neuropathies
3. Childhood visceral myopathies
B. Nonfamilial (or sporadic) type
1. Visceral myopathies
2. Visceral neuropathies
II. Secondary chronic intestinal pseudo-obstruction
A. Disease involving the intestinal smooth muscle
1. Collagen diseases: scleroderma, dermatomyositis, and systemic lupus erythematosus
2. Muscular dystrophies: myotonic dystrophy, Duchenne's muscular dystrophy
3. Amyloidosis
B. Neurologic diseases
1. Chagas' disease
2. Ganglioneuromatosis of the intestine
3. Paraneoplastic visceral neuropathy
C. Endocrine disorder
1. Myxedema
2. Hypoparathyroidism
D. Pharmacologic agents
1. Phenothiazines
2. Tricyclic antidepressants
3. Antiparkinsonian medications
4. Ganglionic blockers
5. Amanita (mushroom) poisoning
6. Narcotics (morphine and meperidine)
E. Miscellaneous
1. Nontropical sprue
2. Jejunoileal bypass
3. Small-intestine diverticulosis
4. Porphyria
5. Eosinophilic gastroenteritis
6. Radiation enteritis
7. Sclerosing mesenteritis
8. Diffuse lymphoid infiltration of the small intestine

caused by abnormalities of the intestinal smooth muscle or enteric nervous system which leads to abnormal gastrointestinal motility.

Causes of chronic intestinal pseudo-obstruction (CIP) The most common cause of CIP is secondary from underlying systemic disease. However, some patients have no underlying systemic disease (Chronic Idiopathic Intestinal Pseudoobstruction, CIIP) (Table 2).

Clinical manifestation of chronic intestinal pseudo-obstruction In both primary and secondary intestinal pseudo-obstruction, patients usually have similar gastrointestinal manifestations characterized by varying degree of recurrent symptoms and signs of intestinal obstruction indistinguish from those of mechanical obstruction such as nausea, vomiting, abdominal distention, bloating, abdominal pain and constipation. Some patients may have diarrhea. Patients who have CIP secondary from systemic diseases may have extragastrointestinal manifestation of the underlying disease. Tables 4 shows features differentiation chronic intestinal pseudoobstruction from true mechanical obstruction.

Diagnosis of chronic intestinal pseudo-obstruction The diagnosis of CIP can be made by ruling out mechanical obstruction and identifying

Patient presents with recurrent symptoms and signs of intestinal obstruction or ileus

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Documentation of signs of intestinal obstruction
plain abdomen x-rays to document intestinal obstruction findings such as gaseous dilatation of the intestine and air-fluid levels

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Rule out mechanical obstruction
BE, UGI and small bowel x-ray, enteroclysis to rule out mechanical obstruction.
When mechanical obstruction is ruled out, the diagnosis of intestinal pseudo-obstruction is made.

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Search for the cause of chronic intestinal pseudoobstruction
(1) Search for any systemic diseases (as listed in Table2) that may cause chronic intestinal pseudoobstruction by appropriate tests.
(2) The findings on gastrointestinal contrast roentgenograms may give clues to underlying diseases.
(3) Family history must be obtained.
(4) If the patient has surgery, a full thickness biopsy must be obtained to examine for smooth muscle and myenteric plexus abnormalities.

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Treatment

Diagram 1

Table 3 Investigations for patients with intestinal pseudo-obstruction syndrome[12]

I. Document the obstructive episodes
1. History (including family history) and physical examination
2. Plain x-rays of the abdomen when the patients have obstructive symptoms

II. Rule out mechanical obstruction
a. Upper GI study, Barium enema, Abdominal CT Scan
b. Radionucline study (gastric emptying and small bowel transit scan)
c. Upper gastrointestinal endoscopy
d. Enteroclysis
e. Exploratory laparotomy if small-bowel x-rays and enteroclysis cannot rules out mechanical obstruction

III. Screening for associated systemic disease
1. Discontinue any medications that impair gastrointestinal motility
2. Antinuclear antibody
3. Thyroid functions: Thyroxin, T3 resin uptake
4. Fasting or 2-hr postprandial blood glucose
5. Serum creatinine phosphokinase and isoenzymes, and possible striated muscle biopsy
6. Urinary porphyrins
7. Hemagglutination and complement fixation for Chagas' disease
8. Anorectal manometry and rectal biopsy

IV. Other studies
1. Esophageal manometric study
2. Small-intestinal manometric study
3. Intravenous pyelogram, cystometrogram

V. Determination the underlying defect
Microscopic examination of the full thickness of bowel specimens
1. Examination of the smooth muscle
   a. Hematoxylin-eosin stain
   b. Trichrome stain
2. Examination of the intramural nerves and ganglions by parallel sectioning of the bowel wall
   a. Hematoxylin-eosin stain
   b. Silver stain
   c. Histochemical stains

whether patients have underlying systemic diseases, which can cause CIP. However, in chronic idiopathic pseudo-obstruction, no underlying disease is identified.

Work ups for patients with CIIP Diagnosis outlining the sequence of events in approaching chronic intestinal pseudo-obstruction. (Diagram 1 and Table 3)

1. Chronic Idiopathic Intestinal Pseudo-obstruction (CIIP) CIIP patients are the patients who have CIP without associated underlying systemic disease. CIIP is a rare condition. The literature about CIIP consists mainly of case reports. Three groups from both Europe and America, have reported on some aspects of CIIP in adults. The details of these reports are shown in Table 5

2. Chronic intestinal pseudo-obstruction secondary from other conditions[4]

2.1 Small Bowel Mooth Muscle Disorsers

2.1.1 Progressive systemic sclerosis (PSS) is a multisystem disorder characterized by obliterative small vessel vasculitis and proliferation of connective tissue with fibrosis of multiorgan. GI manifestations of scleroderma are found in 82% of patients involving from the mouth to the anus. The small bowel is the second most frequently involved gastrointestinal organ in PSS, after the esophagus. Small bowel hypomotility is present in as many as 88% of cases. The pathologic changes in the small bowel of PSS patients consist of smooth muscle atrophy and deposition of collagen in submucosal muscular and serosal layer. The

Table 4 Features differentiation chronic intestinal pseudo-obstruction from true mechanical obstruction[12]

CIP	Mechanical Obstruction
1. Diarrhea or constipation	1. Constipation and obstipation
2. May have other gastrointestinal symptoms (e.g., dysphagia or symptoms of gastric atony)	2. No esophageal or gastric problems
3. Symptoms of abdominal pain, nausea, vomiting, or dysphagia between the attacks	3. Usually symptom-free between the attacks
4. Cachectic appearance	4. Seldomly cachectic
5. May have urinary retention and infection	5. No urinary symptoms
6. Symptoms and signs of systemic disease (e.g., scleroderma or muscular dystrophies) if it is secondary chronic intestinal pseudo-obstruction	6. No underlying systemic disease
7. May have family history to similar problems	7. No family history
8. Plain abdominal x-rays may show air throughout the small bowel and colon	8. Plain abdominal x-rays show no air beyond the point of obstruction
9. Esophagram may show esophageal aperistalsis and dilatation	9. Esophagogram is normal
10. Upper gastrointestinal roentgenograms may show gastric atony and megaduodenum	10. Uppergastrointestinal may show dilatation of proximal small bowel if the obstruction is in proximal bowel
11. Small-bowel roentgenograms may show dilatation of entire small bowel with or without multiple diverticula	11 Small bowel roentgenograms show dilatation of bowel proximal to the obstruction and obstructing lesion
12. Enteroclysis shows no obstrucing lesion	12. Enteroclysis may show obstructing lesion
13. Barium enema may show redundant colon or wide-mouthed diverticula	13. Barium enema may show obstructing lesion
14. Intravenous pyelogram may show megacystic or megaureter	14. Intravenous pyelogram is normal
15. Esophageal manometric studies may show diminished esophagogastric sphincter tone and low amplitude of contractions of lower two-thirds of esophagus	15. Esophageal manometric studies are normal
16. Jejunal manometric studies: Myopathic or neuropathic pattern	16. Jejunal manometric studies show obstructive pattern.
17. No obstructing lesion found during exploratory laparotomy	17. Obstructing lesion can be identified during exploratory laparotomy

Table 5 Clinical manifestations of CIIP

	Mann *et al* (1997)	**Stanghellini *et al* (1987)**	**Schuffer *et al* (1981)**
Inclusion criteria	1. Clinical and radiologic features of intestinal obstruction 2. A mechanical cause or systemic diseases know to cause gut dysfunction were excluded	1. Chronic obstructive symptoms 2. Both small bowel dilatation and non dilatation 3. No mechanical obstruction, systemic diseases 4. No visceral autonomic neuropathy 5. No degenerative or infiltrative diseases of small bowel smooth muscle	1. Chronic obstructive symptoms 2. Small bowel dilatation on barium contrast study 3. No mechanical obstruction
Number of CIIP patients	20	42	11
Age (mean)	22-67 year (43)	5-76 year	ND
M:F	11:9	14:28	ND
Clinical presentation (N)			
- Abdominal pain	80% (16)	74% (31)	85%*
- Vomiting	75% (15)	83% (35)	96%*
- Constipation	40% (8)	36% (15)	59%*
- Diarrhea	20% (4)	29% (12)	81%*
- Dysphagia	5% (1)	ND	36% (4)
Positive Family Hx	2 (VM)	ND	4 (2VM, 2VN)
Initial diagnosis			
- gut obstruction	45% (9)	ND	ND
- constipation	35% (7)	ND	ND
- SMA syndrome	0	ND	36% (4)
Number of full thickness biopsy	19	15	11
Biopsy result			
- visceral myopathy (VM)	13		4
- visceral neuropathy (VN)	3		2
- undetermine	3		5
Abnormal radiology	100%	57%	100%
Abnormal small bowel manometry	ND	100%	ND
Genitourinary involvement	20% (4)	17% (7)	18% (2)
No. of patient had surgery before diagnosis /number of surgery	19/59	33/ND	ND
Nutritionally dependence	56%	ND	ND
- enteral nutrition	12%		
- parenteral nutrition	44%		
Analgesic dependence	28%	ND	ND

*Note: *Include 14 PSS, 1 jejunal diverticulosis and 1 sclerosing mesenteritis (total N=27)*
VM = visceral myopathy, VN = visceral neuropahty

circular muscle is involved more often than the longitudinal muscle layer.

In early stage of the disease, small bowel hypomotility is due to neuropathic involvement. In advanced cases, this is more likely due to myopathic and fibrotic change. However, the submucosal and myenteric plexus appear normal by conventional staining. Manometrically the interdigestive motor complex is frequently absent and markedly diminished in amplitude in PSS patients with symptoms of small bowel dysmotility.

Small bowel radiographic abnormalities have been reported in about 60% of PSS patients and may not correlate with symptoms. The duodenum is often dilated with prolong retention of barium. A characteristic mucosal pattern of "valvular packing" and succulation may be found. Pneumatosis intestinalis has also been reported.

In some cases of PSS, gastrointestinal symptoms may precede skin manifestation and differentiating from CIIP is difficult.

In the Table 6 are features differentiating CIIP from gastrointestinal involvement of PSS. There have been reported that treatment with Cisapride has improved gastric emptying and upper abdominal symptoms in scleroderma patients[11] and Octreotide induces phase III activity in patients with previous complete absence of MMCs and also reduces bacterial overgrowth and symptoms[12].

2.1.2 Dermatomyositis and polymyositis The small bowel may be involved. Megaduo-denum and delayed intestinal transit of barium are prominent features. Pathologically, edema of the bowel wall, muscle atrophy, fibrosis, suggesting a visceral myopathy, mucosal ulceration or perforation due to vasculitis may be seen.

2.1.3 Amyloidosis The gastrointestinal tract is frequently affected in systemic amyloidiosis either by direct deposition, by involvement of the autonomic nerves, or by vascular insufficiency resulting from deposition of amyloid in the blood vessels, leading to intestinal pseudo-obstruction.

2.1.4 Myotonic dystrophy Myotonic muscular dystrophy is a slow progressive disease characterized by myotonia, or difficulty in muscle relaxation.

Dysphagia is the most common gastrointestinal symptom resulting from esophageal involvement. Diarrhea and abdominal cramping are common and occur in up to one third of those affected. However, intestinal pseudo-obstruction is rare. Radiologic examination of the small bowel may demonstrate abnormal but non-specific, including dilation, diminished motility and delayed barium transit. Manometric abnormality is commonly found in patients with symptoms of intestinal dysmotility, including low amplitude contractions in fasting and fed state, retrograde propagation of phase III, interruption of phase III, and increase incidence of tonic contraction.

Histologically, both intestinal smooth muscle and myenteric plexus abnormalities have been described, indicating that intestinal dysmotility may be caused by both smooth muscle and enteric nerve

Table 6 Features differentiating CIIP from gastrointestinal involvement of PSS

	CIIP	PSS
1. Family Hx	+ or -	-
2. Autonomic, central or peripheral NS abnormalities	+ or -	-
3. Esophageal manometry	- Hyperactive, continuous simulataneous esophageal contraction, normal LES pressure - May has incomplete or no relaxation of LES	- Absent or low amplitude contraction of lower esophagus - Low LES pressure, normal LES relaxation
4. Small bowel barium study		
*Duodenal	Moderate-markedly dilatation	Normal - mild dilatation
*Small bowel	Hyperactive but poorly propulsive small bowel content in visceral neuropathy	Succulations and packed vulvulae
5. Urologic abnormalities	+ or -	-

dysfunction.

2.1.5 Duchene's Muscular Dystrophy In Duchene's muscular dystrophy, smooth muscle of the gastrointestinal tract is also involved. However, gastrointestinal symptoms are usually related to dysmotility of the esophagus and stomach, which are more severely affected than the small bowel.

Histological changes comprise of degeneration and atophy of smooth muscle fibers, and separation of fiber by connective tissue. The myenteric plexus is not involved. Acute and chronic pseudo-obstruction have been reported in this disease.

2.2 Disorder of enteric nervous system

2.2.1 Chagas' disease Chronic infection with Trypanosoma cruzi leads to destruction of the submucosal and myenteric plexuses along the entire length of the gastrointestinal tract. Megacolon and megaesophagus are the most common presentations. Small bowel may be involved, leading to megaduodenum and megajejunum. Normal frequency but slow velocity of propagation of the MMC has been reported. In severe case, intestinal pseudo-obstruction may be developed.

2.2.2 Neurofibromatosis or Von Reklinghausen's disease Gastrointestinal neurofibromas are most commonly found in the small bowel. They are reported to occur in up to 10% of patients with neurofibromatosis. Most of them are asymptomatic. Mechanical small bowel obstruction or intussusception may happen.

A case of chronic intestinal pseudo-obstruction with abnormal small bowel manometry and delayed small bowel transit in neurofibromatosis has been reported.

2.2.3 Visceral Neuropathy of carcinomatosis Chronic intestinal pseudo-obstruction has been reported in small cell carcinoma of the lung and epidermoid carcinoma of the lip, representing a paraneoplastic syndrome caused by visceral neuropathy.

Histologically, the small bowel shows widespread neuronal and axonal degeneration of the myenteric and submucosal plexuses as well as mononuclear cell infiltration and Schwann's cell proliferation.

The lung cancer is usually occult when patients present with intestinal pseudo obstruction. The small bowel is usually not dilated but there is delay of barium passage through the small bowel. Interdigestive MMCs may be not found, manometrically.

2.2.4 Pakinson's disease Gastrointestinal dysmotility symptoms including dysphagia, nausea, bloating and constipation are common in patients with Parkinson's disease.

Small bowel manometric studies in Parkinson's disease patients reveal infrequent or absent MMCs, hypomotility in the fed state; and an increased incidence of retrograde and tonic contraction compared to controls.

The pathogenesis of small bowel dysmotility is unknown. Lewy's bodies, which are neurons containing cytoplasmic hyaline inclusions, have been found in the myenteric plexus of the esophagus and colon. However, Lewy's bodies have not been reported in the small bowel.

2.2.5 Hypothyroidism Constipation is the most common gastrointestinal symptom in hypothyroid patients. Intestinal pseudo-obstruction and paralytic ileus may develop in severe hypothyroidism (myxedema). Small bowel transit is significantly slowed and manometric studies reveal decreased amplitude of small bowel contraction and decreased motility index.

2.2.6 Hypoparathyroidism Intestinal pseudo-obstruction and malabsorbtion have been reported in hypoparathyroid patients. Barium studies reveal dilated loops of small bowel and prolong transit time. Symptoms improve with calcium administration. Glucocorticoids may be useful in some patients with severe ileus and steatorrhea.

2.3 Celiac disease

Intestinal pseudo-obstruction has been reported in patients with Celiac disease. Dilated small bowel loops, with delayed passage of barium were observed. Small bowel manometry studies in patients with celiac disease have not been reported.

2.4 Jejunal diverticulosis

Diverticula of the small intestine are most common in the jejunum. Jejunal diverticulosis is associated with many diseases, including scleroderma, celiac disease, Fabry's disease, visceral myopathy (type II), and Cronkhite-Canada syndrome.

Chronic intestinal pseudoobstruction have been reported in patients with jejunal diverticulosis. Small bowel dysmotility is most likely to be the underlying of the formation of diverticula. Small bowel manometry show that phase I of the MMC is dominated by spastic contractions. These contractions may be antegrade, simultaneous, or retrograde. Histology

of the bowel shows both characteristic of visceral myopathy and neuropathy.

2.5 Irradiation

Ionizing radiation damages all structures of the small intestine, including mucosa, blood vessels, connective tissue, enteric nerves and smooth muscle. Radiation damage to the bowel can be separated into acute and chronic injury. Chronic intestinal pseudo-obstruction has been described in two patients many years after they received abdominal radiation. Small bowel studies show dilated loops of bowel with air-fluid levels and thickened bowel wall. One patient had manometric study showed normal MMCs, decreased amplitude and frequency of contractions in the distal duodenum and absence of peristalsis contractions in the jejunum and subsequent histologic examination of the bowel suggested visceral myopathy as the cause of dysmotility.

2.6 Spinal cord injury

Intestinal pseudo-obstruction has been reported in a patient with spinal cord injury. In addition, chronically, postprandial abdominal distention and discomfort are common, occuring in more than 40% of spinal cord injury patients.

Investigation

1. Radiologic studies[5]

The most important role of radiology in the diagnosis of CIP is in the exclusion of mechanical obstruction. The radiologic findings depend on the anatomical regions affected, and may suggest whether the disorder is due to neuropathy or myopathy. In visceral myopathy, pronounced duodenal enlargement, lack of haustration, increase colonic calibre and poor-to-absent contractions may be seen. On the other hand, visceral neuropathy is mainly characterized by disordered smooth muscle contractility which is best appreciated on fluoroscopy. Packed valvula in the small bowel and wide-mouthed sacculation in the colon may be seen in PSS.

In a minority of patients (17-20%), dilatation of renal pelvis, ureter or urinary bladder is identified.

Other radiological studies may be indicated to discover the etiology of the syndrome including brain CT or MRI for neuropathic CIP patients with extrinsic autonomic neuropathy, chest x-ray or CT scans for CIP patients with risk factors of lung cancer.

Radiologic identification of the diffuse nature of a motor disorder in any patients is very helpful in establishing the diagnosis of chronic intestinal pseudoobstruction and to differentiate it from true obstruction. In addition, radiography can often differentiate patients with progressive systemic sclerosis from those with visceral myopathy or visceral neuropathy, if typical features are recognized.

2. Motility studies[2,6-11,13]

Small bowel manometry is necessary to confirm the clinical impression of pseudo-obstruction, after excluding the possibility of mechanical obstructionby radiological and/or endoscopic methods.

Esophageal manometry often demonstrates esophageal motility abnormalities in patients with CIP.

3. Gastrointestinal transit studies

Mouth-to-caecum transit of solids and liquids, and transit of solid radiolabel through the small bowel, are significantly prolonged[1].

4. Histological analysis

If the radiographs or manometric findings are consistent with the diagnosis, there usually is no need for confirmatory biopsy. If the patient undergoes exploratory laparotomy to exclude an obstruction, and none is found, full thickness biopsy, several centimeters long, of dilated and non dilated small intestine segments should be taken.

Full thickness biopsy may enable the pathologist to identify the neuropathic or myopathic abnormalities.

Rectal biopsy may be of value when the diagnosis of intestinal neuronal dysplasia is considered.

Management

In mild cases of small bowel motility disorders, patients are usually diagnosed and treated as functional gastrointestinal disorders. In severe cases, especially in chronic intestinal pseudoobstruction, the goals of treatment are restoration of normal intestinal propulsion, adequate nutrition, and treatment of complications such as bacterial overgrowth.

In patients with small bowel motility disorder secondary from other conditions, the underlying condition should be treated or corrected properly such as electrolyte imbalance, hyper or hypothyroid, hypoparathyroid and drug induced bowel dysmotility.

General Management

Symptomatic and supportive management should be considered in every patients. Oral or intravenous fluids and electrolytes should be administered in pa-

tients with dehydration and/or electrolyte imbalance. Gastric and intestinal decompression by nasogastric suction is important in patients with abdominal distention, especially in acute stage. Abdominal pain should be treated appropriately.

Nutritional Support

Dietary manipulations can be helpful in relieving symptoms and encouraging weight gain. In patients with CIP, gastrointestinal transit is usually impaired; thus, liquid food is preferred. Low fat, low fiber and lactose free diets have been advocated. A high dietary load of fat worsens existing steatorrhea and diarrhea. Lactose and fiber can increase the amount of gas and distention, the result of bacterial breakdown. In addition, dietary fiber reduction may reduce the risk of obstruction from bezoar formation.

Vitamin, folate, iron and trace elements supplements should be used as needed. However, dietary manipulations alone is successful only in a minority of patients usually in those with mild to moderate symptoms and is less likely to be effective in patients with severe obstructive symptoms.

Many patients with CIP require home enteral or parenteral nutrition. A trial of enteral nutrition should be attempt before parenteral nutrition. Percutaneous endoscopic gastrostomies (PEG) should be placed in CIP patients who do not have significant gastroparesis. In patients with gastroparesis, jejunal feeding should be employed.

To determine patients suitable for enteral nutrition, nasojejunal or a combination of nasogastric and nasojejunal feeding tubes should be tried before the long term enteral tube placement . A combined nasogastric-nasojejunal tube allows both gastric decompression and simultaneous jejunal feeding. An isotonic, low residue, fiber-free formula should be used in patients with CIP.

In the patients with failure enteral feeding, parenteral nutrition is a mainstay treatment. Central parenteral nutrition is successful in maintaining the patient's weight and reversing trace element and vitamin deficiency. However, this treatment is associated with significant morbidity and mortality and is costly.

Restoring of Normal Intestinal Motility

Prokinetic drugs

1. Metoclopramide is a dopamine antagonist with central and peripheral effects. In normal subjects and disease states, metoclopramide has been shown to promote esophageal periatalsis, increase LES pressure and accelerate gastric emptying but has limited effect on small bowel motility. Metoclopramide has been used in the treatment of familial visceral myopathy, idiopathic intestinal pseudoobstruction and scleroderma, but its overall efficacy has been disappointing.

2. Domperidone is a dopamine antagonist that operates primarily through peripheral receptors. It does not cross the blood-brain barrier and is free from CNS side effect. The effects of domperidone on gastrointestinal tract are similar to those of metoclopramide. It appears to be ineffective for the treatment of CIP.

3. Cisapride is a nondopaminergic, prokinetic agent that acts by releasing acetylcholine from enteric nerve endings. In an uncontrolled study of 106 intestinal pseudoobstruction patients, cisapride improved symptoms in the majority of patients. However, a controlled trial failed to show improvement of symptoms in patients with intestinal pseudoobstruction.

Because cisapride appears to be safe, it should be tried in all patients. The dosage is 20 mg three times daily for adults.

4. Erythromycin, a macrolide antibiotic, acts as a motilin agonist, has been used in just a few patients with CIP. Erythromycin may be useful in the acute setting of CIP where symptoms primarily resulted from delay gastric emptying. The recommendation dose is 3mg/kg every 8 hours intravenously followed by oral administration (250 mg three times daily) for 5-7 days. There is no evidence that, use alone, it is effective beyond 2-3 weeks.

5. Octreotide, a synthetic analogue of somatosatin, can induce migrating motor complexes in the small intestine. A bedtime dose of 50 (g, with or without a daily dose of erythromycin suspension, 200 mg t.i.d, can sometimes result in clinical improvement, especially in patients with progressive systemic sclerosis. It is important to use small doses of octreotide (50 (g) and to give the medication at night. Larger doses can impair small bowel motility and pancreatic secretion, causing malabsorption. Nighttime dosing is used to prevent inhibition of the postprandial motor activity.

Surgical treatment

Surgical therapy for severe small bowel motility disorders has, in general, proved disappointingly. The results of segmental resections and bypass procedures in CIP have been almost not helpful. However, surgi-

cal bypass of affected segments might be beneficial in highly selected patients whose only a short segment of the bowel is involved. It is important to remember that small bowel motility disorders, especially CIP, often be generalized process. Once any abdominal surgery is performed, it may be difficult to exclude a mechanical obstruction from adhesion when the patient returns with obstructive symptoms.

Small bowel transplantation

Small bowel transplantation may be a life saving procedure in patients with severe small bowel dysmotility. Patients who are dependent on TPN and suffer from its complication may have benefit from small bowel transplantation.

Treatment of Complication

Bacterial overgrowth

Treatment of bacterial overgrowth and secondary fat malabsorption with broad-spectrum antibiotic are useful in a small number of patients. A 10 day-course of antibiotic such as tetracyclin, doxycycline, ampicillin, ciprofloxacin, or metronidazole may induce weight gain and remission of diarrhea.

In patients with demonstrated steatorrhea, rotating antibiotics for 1 week every month may be tried.

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