Management of Paralytic Ileus

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ABSTRACT

Ileus is a pathophysiologic state of inhibited motility in the gastrointestinal tract due to a physical/ anatomic obstruction in the lumen (obstructive ileus) or due to cessation of smooth muscle motor activity in the small intestine and colon. In Internal Medicine, paralytic ileus is most commonly caused by peritonitis, which is most often caused by acute pancreatitis.

To establish a diagnosis, several diagnosis evaluation procedures may need to be performed such as laboratory evaluation, radiologic examination, ultrasonography and CT examination. Management of paralytic ileus is aimed at the underlying disease and supportive therapy. With this management paralytic ileus will spontaneously remit.

***Keywords**: paralytic ileus, pathogenesis, diagnosis, management.*

INTRODUCTION

Gastrointestinal symptoms, particularly paralytic ileus, are not only found in primary gastrointestinal tract diseases, but also as a manifestation of other systemic ailments. In the field of internal medicine, in addition to peritonitis, many conditions could cause cessation of smooth muscle motor activity in the small intestines and colon, such as sepsis, as a side effect of certain medications, hormonal imbalance, electrolyte imbalance, and bowel ischemia.[1, 2, 3, 4]

Paralytic ileus is a clinical syndrome due to acute and transient disturbance of the transportation of the content of intestinal lumen due to cessation of smooth muscle motor activity in the small intestines and colon, with the potential to return to normal.[1, 2, 3]

This differs from physical bowel obstruction (obstructive ileus) caused by a physical/anatomic obstruction in the lumen of the small intestines or colon, which has a clear, simple, and comprehensible pathogenesis, and usually requires surgical intervention. Paralytic ileus usually does not have a clear mechanism, is complex, and is not clearly understood. Several factors, such as neurogenic, myogenic, and humoral factors, are suspected to be involved in the pathophysiology of paralytic ileus.[1, 2, 3, 4, 5]

Compared to obstructive ileus, paralytic ileus is more commonly encountered by clinicians. However, we still do not have the statistical incidence data on paralytic ileus, possibly because it is considered as a transient gastrointestinal syndrome with satisfactory prognosis.

Up to now, the management of paralytic ileus is aimed at the causative/ accompanying illness.[2,6,7,8] From this literature review, the author hopes that the management of paralytic ileus could be further improved in the future.

PATHOGENESIS/PATHOPHYSIOLOGY

The main function of the small intestines and the colon is to supply water, electrolyte, and nutrients to the body. Approximately 85% of chyme, consisting of 8 liters of fluid (1.5 liters of swallowed fluid and 6.5 liters of fluid from various digestive secretions), nutrients, vitamins, and minerals, is absorbed in the small intestines, while the remaining chyme, mainly consisting of fluid and electrolyte, is absorbed in the colon.[3, 9]

To be able to do so, food must be digested and moved along the lumen of the small intestines and the colon at an appropriate speed, to allow for digestion and absorption.

MOTILITY OF THE SMALL INTESTINES AND THE COLON

As chyme enters the intestines from the gaster, the proximal portion of the walls of the small intestines is stretched, which causes local concentric contraction at specific intervals along the intestines. This segmental contraction repeatedly divides chyme each minute, causing progressive mixing of solid food particles and the secretions of the small intestines. Simultaneously, there is also a peristaltic wave that pushes the chyme towards the anus with a speed of 0.5-2 cm/second. This peristaltic movement could arise from any part of the small intestines, far more rapidly at the duodenum compared to at the terminal ileum. Thus, under normal conditions, 3-5 hours is required for chyme to reach the ileocaecal sphincter from the pylorus. Concentric contraction also takes place in the colon, while at the same time; there is contraction of the three collections of longitudinal colon muscles on 3 longitudinal bands known as the teniae coli. This collective contraction causes the stimulated portion of the colon to bulge like a sac, so called haustration. This haustral contraction occurs every hour and lasts for 30 seconds, causing the content of the content to blend. This slow yet persistent haustral contraction is the main force that pushes the chyme from the ileocaecal sphincter to the transverse colon, in the caecum and ascending colon. From the transverse colon to the sigmoid, a peristaltic-like motion of the bowel, known as mass movement, replace the haustral contraction as the thrust force for bowel content. As it moves through the colon toward the anus, the fluid chyme gradually becomes more solid, until only approximately 80-200 cc of fluid remains in the feces.[9]

REGULATION OF SMOOTH MUSCLE AND COLON MOTILITY

An intramural nerve plexus resides within the walls of the small intestines and colon, known as the enteric nervous system, made up of two layers of neurons and relevant connector nerve fibers. The myenteric (Auerbach) plexus is located in the outer part between the longitudinal and circular layer, while the submucosal plexus (Meissner) is located in the submucosa. The myenteric plexus particularly controls motility, while the submucosal plexus particularly controls secretion, absorption, and intestinal blood flow.

This enteric nervous system is able to maintain independent function (by reflex), and is thus called the mini gut brain, even though its mechanism is still influenced and modified by the central nervous system through the parasympathetic and sympathetic components of the autonomous nervous system. Stimulation of the parasympathetic component increases intestinal and colon motility, while stimulation of the sympathetic component inhibits intestinal motility. This can be seen from how several important reflexes mediated by the sympathetic component that cause inhibition of the motility of the small intestines and colon resemble one another, such as the intestino-intestinal reflex, which occurs if an intestinal portion is overly distended or its mucosa excessively irritated, inhibiting the motility of the other parts of the bowel; the peritoneo-intstinal reflex that occurs if there is iritation of the peritoneum; the reno-intestinal and vesico-intestinal reflex that occurs if the kidneys or bladder is irritated; and the somato-intestinal reflex that occurs if the abdominal skin is overly irritated.[9]

The parasympasympathetic component is located in the nucleus of the cranial division of the vagal nerve and the sacral segment of the medulla spinalis. The vagal nerve especially innervates the esophagus, gaster, and the pancreas, and only contributes little in the small intestines to the proximal portion of the colon, while the sacral divison goes through the cranial nerves to innervate the distal portion of the colon (the sigmoid colon, the rectum, and anus), which plays an important role in the defecation reflex. The sympathetic component is located on the thoraco-lumbal segment of the medulla spinalis, and the preganglionic nerve fibers leave the medulla spinalis towards the distant prevertebral ganglia, such as the celiac and mesenteric ganglia. Unlike the parasympathetic component, which innervates the oral and anal portions, the sympathetic component innervates the whole intestine and colon via its postganglionic nerve fibers. When stimulated, end fibers of the parasympathetic system releases acetylcholine, which increases the motility of the intestines and colon. On the other hand, stimulation on the sympathetic peripheral nerve fibers causes the release of noradrenaline, which inhibits peristaltic movement of the intestines and colon.[9]

Increased plasma cathecolamine due to post-operative stress is suspected to be closely related to post-operative paralytic ileus. In laboratory animals, adrenalectomy does not improve post-operative paralytic ileus, while splanchnicectomy returns a portion

of intestinal motility. This is important in demonstrating the role of the sympathetic component in the inhibition of the motility of the intestines and colon, while the role of adrenaline in post-perative paralytic ileus remains unclear.[5, 10]

The role of hormonal factors in the motility of the small intestines is still unclear, and is still being studied by many experts. Several hormones that are supposedly secreted during the process of digestion, such as gastrine, cholecystokinine, motiline, P substance, and insulin, increase intestinal peristalsis; while secretine, vasoactive intestinal polypeptide (VIP), and glucagon inhibit the intestinal peristalsis.[9,11]

Various clinical conditions in the field of internal medicine, such as peritonitis and retroperitoneal inflammation, hormonal and electrolyte imbalance, drug-induced conditions, blood-borne toxins, as well as disturbances in oxygen supply, are able to inhibit intestinal and colon motility (thus causing paralytic ileus). Several myogenic, neurogenic, and humoral factors are suspected to play independent or collective roles in the basic mechanism of the development of paralytic ileus under these conditions.[1, 2, 3 ,4]

ETIOLOGY[1, 2, 3, 4, 12,13]

Several conditions that could induce paralytic ileus in the field of internal medicine are as follows:

Intra-abdominal causes:

- Peritonitis, which is the most common cause of paralytic ileus due to peritoneo-intestinal reflex due to direct peritoneal iritation, is most commonly caused by:
  - acute pancreatitis,
  - acute cholecystitis,
  - peptic ulcer perforation,
  - bacterial peritonitis,
  - appendycitis.
- Retroperitoneal processes/extraperitoneal iritation:
  - ureteropelvic stone
  - pyelonephritis,
  - retroperitoneal hemorrhage.
- Disturbance in oxygen supply:
  - mesenteric artery insufficiency
  - mesenteric vein insufficiency

Extra-abdominal causes:

- Metabolic disturbances:
  - electrolyte imbalance: particularly hypokalemia, hypocalcemia;
  - Uremia (acute or chronic renal failure);
  - Hormonal imbalance: diabetes melitus, hypoparathyroid, myxedema.
- Drug use: anticholynergic/spasmolytic agents, opiates, tricyclic antidepressants, phenotiazine.
- Sepsis and all infections of the body.

Clinical Approach

Acute bowel obstruction syndrome consists of abdominal pain, nausea and vomiting, gassiness (distention), and constipation. To enable clinical early detection of the type/kind of obstruction, we should pay attention to characteristic complaints and signs during history and physical examination, such as:

- Gassiness (distention), diminished bowel sounds, and tympany on percussion of the entire abdomen (meteorysmus), which are the chief classical clinical symptoms of paralytic ileus. Clinical symptoms of the underlying disease (etiology) may also be found, such as fever (in cases of infection or inflammation), or signs of shock, continuous abdominal pain, and local abdominal rigidity in cases of acute pancreatitis.[2, 6,7,13] (Table 1.)
- Abdominal pain in the form of intermittent colic with a certain interval, increased bowel sounds, and sometimes bowel contraction observed/felt on the abdominal wall are classic signs of obstructive ileus, due to increased contraction to push the content of the intestinal or colon lumen through the point of obstruction.[1,2,3,14] (Table 2.)
- Massive abdominal distention, generally accompanied by mild, continuous abdominal discomfort and normal or increased bowel sounds are found in idiopathic acute massive dilatation of the colon, known as the acute colonic pseudo-obstruction (Ogilvies syndrome). This condition is suspected to be due to an imbalance between the sympathetic innervation of the bowel and the sacral parasympathetic innervation, which innervates the distal colon.[14] This is a form of adynamic (paralytic) ileus, particularly occurring in the caecum or distal colon.[15]

Table 1 Clinical diagnostic criteria for Paralytic illeus [7, 8]

Clinical findings of paralytic illeus may take the form of:
- Gassiness, metereorismus (flatulence), reduced or diminished bowel sounds
- Nausea, vomiting, generally constipated, but diarrhea is possible
- May be accompanied by fever (sub-febrile or febrile)
- The patient's general condition mild to severely ill, may be accompanied by reduced consciousness.
- Shock may occur
- Accompanying disease/conditions that may cause increased risk: trauma, surgical procedures (particularly in the abdomen), acute pancreatitis, bile stone, diabetes mellitus, electrolyte imbalance, spasmolytic agents, pneumonia and all body infections.

Table 2 Clinical Symptoms of Paralytic Illeus Compared to Obstructional Illeus (According to Its Anatomical Location)[1, 2]

Clinical symptoms	Location of obstruction				
	Paralytic Ileus	Proxymal duodenum	Distal duodenum	Jejunoileal junction	Colon
Abdominal pain	absent/ discomfort	colic, often (with interval)	Colic, often	Colic, moderately	Colic rare
Gassiness	moderate-severe	Mild	Mild	Moderate	severe
Vomiting Frequency	little, rare	Voluminous, often	Voluminous, often	Little, rare	very rare
Characteristic	Acidic, bilious	Clear, acidic Cl, KCl	Greenish, bitter NaCl, $NaHCO_3$	Foul smelling, feculent	Foul smelling, feculent
Acid-base Imbalance	varies	Metabolic Alkalosis	Metabolic Acidosis	Dehydration	varies

DIAGNOSIS

To establish a diagnosis and to find the etiology of paralytic ileus, several diagnostic evaluation procedures may need to be performed, such as laboratory evaluation, radiological examination, electrocardiography, ultrasonography, and CT-scan imaging.[2, 6]

Not all of these evaluation methods need to be performed. We should weigh the benefits and limit it to examinations that are truly required for accurate determination of the underlying cause of the paralytic ileus (cost-effective).

Laboratory evaluation

Particularly for paralytic ileus, laboratory examination plays a very important role, since many metabolic and electrolyte disturbances as well as infectious or inflammatory diseases commonly cause paralytic ileus.[2]

Several laboratory procedures that need to be conducted in cases of paralytic ileus:

- Complete blood check: Hb, Ht, erythrocyte, leukocyte, differential count, to identify infection/ inflammation.
- Serum electrolyte level evaluation: K, Ca, Mg, to identify electrolyte imbalance.
- Blood chemistry:
  - Renal function evaluation: blood urea-nitrogen, and creatinine
  - Hepatobiliary and pancreatic function: blood sugar, bilirubin, SGOT and SGPT, cholesterol, triglyceride, amylase, and lypase.

Radiological examination

It is important to conduct a chest x-ray to detect pneumonia and the presence of free air in the subdiaphragmatic space (due to perforation of the gastrointestinal tract).[3] Radiological examination of the abdomen without contrast in 3 positions (upright, supine, and lateral) is crucial in establishing the clinical diagnosis of paralytic ileus.[1,2,3,4,6,13,14]

The accuracy of diagnosis based on 3 position abdominal radiology is approximately 85%.[3]

- Under normal conditions, generally no gas is found in the intestines, and only little gas bubbles are found in the feces in the colon.[2]
- In cases of paralytic ileus, there is an evenly spread accumulation of air in the gaster, intestines, and colon,[2,4] and the herring bone image is found, which is actually the colon haustra, visualized due to excessive distention by air.[6]

- In cases of obstructive ileus, there is a deposit of air and fluid at the proximal of the location of obstruction, causing distention of this portion, as well as a stepladder air-fluid pattern, while at the distal of obstruction, no air is found.[2,4]

Radiological imaging of the abdomen using barium contrast agents are sometimes needed, if we cannot differentiate paralytic ileus or acute colonic pseudo-obstruction with mechanic obstruction of the colon on plain abdominal imaging. Using contrast agents, we can identify narrowing of the lumen and the site of obstruction in cases of mechanical bowel obstruction.

Electrocardiography

Electrocardiography is useful to detect hypokalemia in paralytic ileus.

ULTRASONOGRAPHY

If pancreatitis, cholecystitis, or liver abscess is suspected, ultrasonography of the upper abdomen may be performed.[6,7,8]

Special Imaging Devices

Special imaging devices (MRI, etc.) may be used if other methods of evaluation cannot identify the cause of paralytic ileus.

Management

In essence, management of paralytic ileus directly aims to address the etiology, without the need of surgical management.[2, 3, 16] Up to now, there is no drug/pharmacotherapy that has been proven to be beneficial to restore intestinal or colon motility in patients with paralytic ileus.[2, 5, 10]

Determining the underlying disease in paralytic ileus is not simple. Thus, conservative therapy could immediately be administered when clinical symptoms give strong hints of paralytic ileus, while identifying the etiologic diagnosis, while causal therapy can immediately be commenced when the underlying disease/condition is determined (Figure 1.).

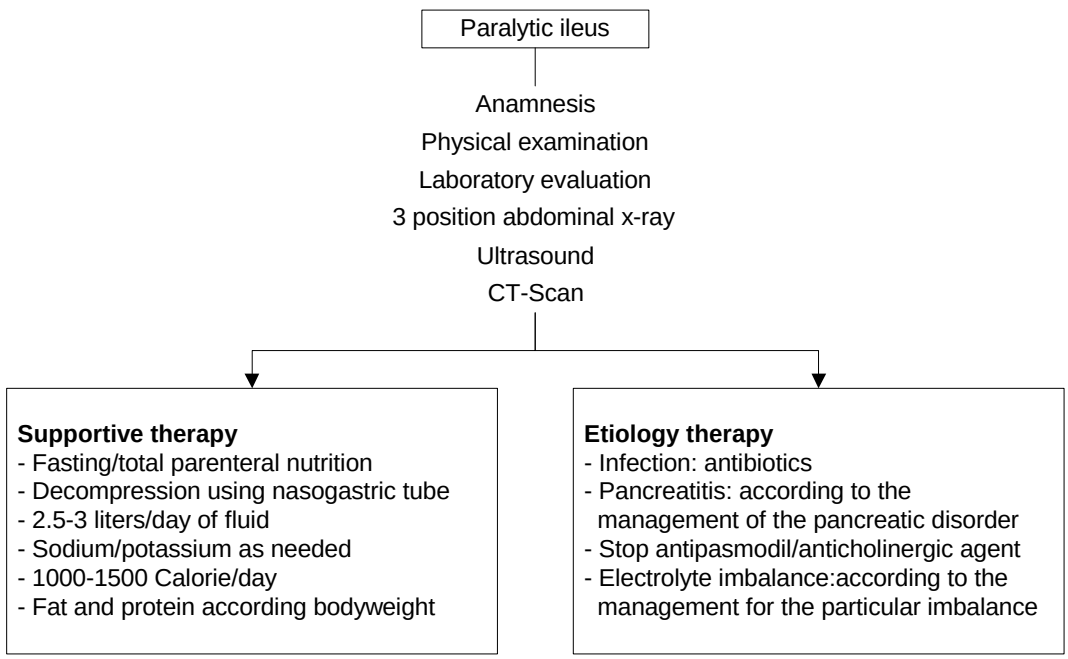

Figure 1. The Algorithm for The Management of Paralytic Ileus

Supportive Therapy

Supportive therapy for paralytic ileus consists of:

- General care
- Correction of fluid, electrolyte, and acid-base imbalance
- Abdominal decompression
- Total parenteral nutrition

General Care

- Information and education on the patient's illness and management to the patient and family is very important, since therapeutic success is very much determined by the patient's cooperation, such as fasting, the need to insert a nasogastric tube, further investigation, and drugs that need purchasing.
- Bed rest.
- Fasting.
- Monitor general condition and vital signs (consciousness, blood pressure, pulse rate, temperature, and respiratory rate) intermittently every 6-8 hours for the initial 24-48 hours.
- Insert an IV line and administer crystalloids (0.9% NaCl or lactic ringer fluid) for emergency. Adjust the volume and rate of administration according to the patient's condition.
- Conduct the following laboratory evaluations: complete peripheral blood check, ureum and creatinine, blood sugar, serum electrolyte, blood gas analysis.
- Insert a urinary catheter to determine 24 hour urine output.
- Monitor using ECG to identify hypokalemia.
- Evaluate laboratory findings (complete peripheral blood check, ureum and creatinine, blood sugar, serum electrolyte, blood gas analysis) every 6-8 hours for the initial 24-48 hours.[17]

CORRECTION OF FLUID, ELECTROLYTE, AND ACID-BASE IMBALANCE

If the patient's general condition and vital signs are satisfactory, fluid requirement is generally 2.5-3 liters / day.[6,8] If there are signs of hypovolemic shock, administer fluid (Ringer lactic / NaCl 0.9%) as quickly as possible until the blood pressure rises over 100/60 mmHg, then adjust the IVFD administration according to intermittent physical and laboratory evaluation.[2,6,8]

The amount of fluid required is determined from the estimated fluid deficit, daily maintenance requirement, and expected fluid loss. Fluid deficit could be calculated by comparing the patient's current weight to that prior to the time of illness. The level of dehydration is estimated from the weight loss during illness, ranging from 4% in patients with mild dehydration, to 8% in patients with severe dehydration. Half of the fluid deficit is administered in the initial 24 hours, while the remaining volume is administered in the next 24 hours. Fluid loss from vomiting and the nasogastric tube, as well as 24-hour urine output should be continuously monitored to determined total daily fluid requirement.[2]

Metabolic alkalosis could occur due to HCl loss from vomiting and intermittent suction of nasogastric fluid. This condition can be corrected by the administration of the isotonic crystalloid agent NaCl 0.9% to replace extracellular fluid volume loss. After adequate urine output, intravenous administration of KCl (5-10 mEq/ hour) should be administered, since potassium loss through the kidneys is great. Twenty-four hour evaluation of urine potassium is very beneficial for the evaluation of therapy.[2,17]

Metabolic acidosis particularly occurs due to intake disturbance (patients are not able to tolerate food/oral drinks). This condition can be corrected by the administration of sodium bicarbonate. Bicarbonate correction must be administered if the arterial pH level is below 7.1. Correction of pH level to 7.4 should be conducted progressively, because too rapid correction causes the intracellular fluid to enter the extracellular space, and the pH level of cerebrospinal fluid to drop drastically, aggravating existing neurological symptoms. Half of bicarbonate deficit should be administered to increase plasma HCO_3 level to 16 mEq/L in 12 hours, while the remaining should be administered in the consecutive 12 hours.[2]

Abdominal decompression

Decompression to reduce accumulation of gas in the gastrointestinal tract is useful to:

1. Reduce complaints of abdominal discomfort/pain
2. Reduce difficulty breathing
3. Reduce nausea and vomiting
4. Prevent aspiration of vomit substance into the respiratory tract.

Decompression is mainly conducted by inserting a nasogastric tube, to remove gas from the upper gastrointestinal tract, and in certain conditions, such as acute colon pseudo-obstruction, endoscopy/ colonoscopy-guided rectal tube insertion may be performed to remove gas from the colon.[15]

Total Parenteral Nutrition

Sufficient nutritional intake is crucial in maintain metabolism and repairing damaged cells in the body. No paralytic ileus patient is able to tolerate any oral intake of food or drink, and thus there needs to be a consideration of whether the patient needs to fast or should receive total parenteral nutrition, to ensure normal body metabolism. Total parenteral nutrition should immediately be administered if signs of protein-calorie malnutrition (weight loss, hypoalbuminemia) is observed, or if oral intake intolerance in cases of paralytic ileus is estimated to last for 7 days or more.[3, 18]

The patient's calorie requirement could be calculated from the basal energy expenditure plus other requirements. According to the metabolic-endocrine sub-department of the department of internal medicine of Cipto Mangunkusumo Hospital, the basal energy expenditure is 25 calories/kg ideal body weight/day for females, and 30 calories/kg ideal body weight/day for males. This number should be added by additional calorie requirement due to other complications, such as infection (20-30%), malnutrition (20-30%), fever (10% for every 1 degree Celsius). Simplified, total parenteral nutrition is estimated to be 1000-1500 calorie/day.[7,13,20] The composition of parenteral nutritional fluid should be as follows: carbohydrate : lipid : protein = 50% : 30% : 20%.[22] Amino acid fluid (3-4%), and 5-10% dextrose may be administered simultaneously with fat/lipid emulsions.[6,19]

Once bowel sounds are heard or the patient has passed gas through the anus, fluid feeding may be initiated through the nasogastric tube (enteral feeding), combined with parenteral nutrition, progressively adjusted to the patient's tolerance.[6,20]

Medications

Even though sympathetic activation is suspected to be responsible for the development of paralytic ileus, the administration of sympathetic inhibitors such as guanetidine, dihydroergotamine, or reserpine has little effect in post-operative paralytic ileus, and their use is limited by cardiovascular side effects (hypotension) that occur when the medicine is administered.[5,10,16]

The use of prokinetic agents such as metochlopramide or cisapride has not been proven to be effective in cases of paralytic ileus.[2, 5, 21]

Even with the lack of well documented proof of efficacy, neostigmin has long been used to prevent/shorten the length of post-operative paralytic ileus.[11, 14] In the field of internal medicine, immediately after the etiology of paralytic ileus is managed accordingly, 3 x 1 vial of neostigmin/prostigmin may be administered to enhance bowel motility.[6, 8]

MANAGEMENT OF UNDERLYING DISEASE/ CONDITION

Paralytic Ileus Due to Acute Pancreatitis

Conservative treatment is still considered the basic therapy for any stage of acute pancreatitis. Patients are asked to fast; a nasogastric tube is inserted for suction of gastric fluid. Pethidine is administered several times a day to ease the pain. Correction of fluid and electrolyte is achieved trhough parenteral administration of fluid and electrolytes and total parenteral nutrition.

To reduce pancreatic secretion, aside from gastric fluid suction through the nasogastric tube, administer 1 vial of somatostatin IV per hour per drip inside dextrose 5% or octrotide S.K. 3 x 1 vial for 3-5 days, if the patient can afford it.[6, 8]

Paralytic Ileus Due to Electrolyte Imbalance

Clinical manifestations of electrolyte disturbance are common, and can take the form of paralytic ileus, particularly in cases of hypokalemia, or other electrolyte imbalance such as hypokalcemia or hypermagnesemia. Electrolyte imbalance is believed to influence the transport of calcium ion into the smooth muscle cells of the small intestines and the colon, which is required for the contraction of the smooth muscle.[1, 4, 22]

Hypokalemia

Hypokalemia often occurs or should be suspected in cases of diarrhea, chronic/excessive use of non potassium saving diuretic agents, respiratory alkalosis in patients with hypokalemic periodic paralysis.[23, 24]

Administer KCl intravenously, since patients are unable to tolerate any oral administration in cases of paralytic ileus. The dose of KCl is generally no more than 10 mEq/hour per infusion. Administration of large doses causes venous iritation and phlebitis. In life threatening situations, large doses of KCl (20-40 mEq/hour) may be administered through large peripheral veins under close ECG monitoring. Serum potassium should be evaluated every 3-6 hours, due to the risk of cardiac arrhythmia.[23, 25]

Hypocalcemia

Hypocalcemia is often encountered in cases of hypoparathyroidism/pseudohypoparathyroidism, acute pancreatitis, or carcinoma of the thyroid gland medulla, prostate cancer, breast cancer with osteoblastic metastasis, chronic renal failure (phosphate retention, and

vitamin D metabolism disturbance), administration of cytotoxic agents in lymphoma or leukemia.[24, 25]

Administer 1-2 vials of 10 ml of 10% gluconic calcium or 10% calcium chloride intravenously at a slow rate (over 5 minutes) or dilute 3-6 vials of gluconic calcium in 500 ml Dextrose 5% per infusion with a rate of 0.5-2 mg/kg bodyweight per hour, or 30-100 ml/hour (depending on the degree of blood calcium insufficiency) to increase calcium level to 7-8.5 mg/dl. Intermittent blood calcium level monitoring is very useful for the evaluation of therapy.[24, 25]

Hypermagnesemia

Hypermagnesemia is often encountered in chronic renal failure, in the treatment of ecclampsia with $MgSO_4$, and in chronic or excessive use of laxatives or antacids that contain $MgSO_4$, Mg-citrate.[22, 24]

Depending on the patient's renal function, the following therapy may be performed: [22, 25]

- Administration of physiologic NaCl and diuretics (furosemide) to increase magnesium excretion, or
- Intravenous administration of calcium (as an Mg antagonist): 500 mg Calcium chloride at a rate of 100 mg/minute,
- Hemodialisis, in cases of severe renal dysfunction.

Paralytic Ileus due to Infection

All kinds of body infection, particularly septicemia, could cause paralytic ileus. Antibiotics should be administered parenterally according to the type of infection and the results of the antibiotic sensitivity test to achieve satisfactory outcome.

Prognosis

Several conditions may create problems in the management of paralytic ileus, such as hypovolemic shock, septicemia, even septic shock and malnutrition. However, in general, the prognosis for paralytic ileus is satisfactory, even if sometimes the etiology cannot be identified. With supportive care, paralytic ileus may spontaneously abate. [2]

The length of care depends on the cause of paralytic ileus: [7]

- If it is caused by acute pancreatitis:
  - in mild-moderate acute pancreatitis (edema): 2-3 weeks
  - in severe acute pancreatitis (hemorrhagic/ abscess): 3-5 weeks
- If it is caused by spasmolytic agents: 1 week
- If it is caused by infection: 1-3 weeks

CONCLUSION

- Paralytic ileus is never a primary disorder, but instead is a clinical syndrome due to acute and temporary loss of smooth muscle motor activity (motility) in the small intestines or colon.
- In the field of internal medicine, paralytic ileus is most commonly caused by peritonitis, which is most often caused by acute pancreatitis.
- Several other cases that could cause paralytic ileus include electrolyte imbalance, drug use, sepsis, and all body infection/inflammatory processes.
- The clinical diagnosis of paralytic ileus is made based on characteristic clinical findings as follows: nausea, vomiting, gassiness (distention), meteorismus, diminished bowel sounds.
- Identification of the underlying disease is the key to success in the management of paralytic ileus.
- To identify the etiology of paralytic ileus, several diagnostic investigative methods must be performed, such as: laboratory evaluation, ECG, and 3 position radiological examinations. Abdominal ultrasound or imaging may be performed if necessary.
- In managing paralytic ileus, the basic treatment is aimed at the underlying disease of paralytic ileus, and does not require surgical procedures. With suportive therapy and management of the underlying/accompanying disease, paralytic ileus will spontaneously remit.

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