Paralytic Ileus in Vegetarian with Pneumonia Infection

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ABSTRACT

Paralytic ileus which is commonly found in clinical practice is referred to clinical syndrome of transportation disturbance of the intestinal lumen content due to various etiology and underlying condition. It has been considered a transient gastrointestinal syndrome with good prognosis. Most cases respond well to conservative management. However, inappropriate diagnostic approach and management will result in severe complication leading to death such as septicemia and perforation. We reported a case of paralytic ileus in young male who is vegetarian with pneumonia infection as the suspected underlying etiology. Radiological examination of the abdomen in three position (upright, supine and lateral) showed dilated gaster and duodenum with minimal air fluid level, no herring bone appearance and absent of free intraperitoneal air. The laboratory result also showed low level of vitamin B_{12} which might be due to his lactovegetarian diet habit. Management including supportive therapy such as decompression, fasting, adequate parenteral nutrition, fluid balance and treatment of pneumonia as the underlying cause of paralytic ileus had been resulted in good clinical response.

***Keywords**: Paralytic ileus, pneumonia, treatment*

INTRODUCTION

Paralytic ileus is a clinical syndrome caused by acute and transient disturbance of transportation of the intestinal lumen content due to cessation of smooth muscle motor activity in the small intestines and colon, with is potentially be returned to normal.[1,2] Compared to obstructive ileus, paralytic ileus is more common encountered by physicians. However, we still do not have statistical data on incidence of paralytic ileus, possibly because it has been considered a transient gastrointestinal syndrome with satisfactory prognosis.[2] Acute intestinal pseudo-obstruction consists of massive dilatation of the intestine in the absence of mechanical obstruction.[3,4] The approximate risk of spontaneous perforation is 3 percent, with an attendant mortality rate of 50%. Most cases respond to conservative management.[4,5] However, diagnostic and management approach based on guidelines have not been available yet.

Underlying conditions that could induce paralytic ileus are intraabdominal (peritonitis, retroperitoneal processes, disturbance in oxygen supply) and extra abdominal causes (metabolic disturbances such as ele ctrolyte imbalance, drugs, pneumonia, sepsis and whole body infection).[2,3,6] Several conditions may create problems in the management of paralytic ileus such as hypovolemic shock, septicemia, septic shock and malnutrition.[1,2]

Careful history and physical examination are important initial steps in assessing peritoneal signs and other evidence of a primary intra-abdominal inflammatory process or mechanical obstruction that may require surgical intervention.[3] Clinical findings of paralytic ileus may take form of:

- gassiness, metereorismus (flatulence), reduced or di minished 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 loss of 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 whole body infections.[2,7]

Decreased or absent of bowel sound indicates acute intestinal obstruction, pseudo-obstruction or paralytic ileus. Further examinations that support the diagnosis are very important. Conventional laboratory studies may help to assess electrolyte abnormalities or intra-abdominal infectious/inflammatory process. Supine, upright and lateral abdominal X-rays determine the distribution of intestinal gas and asses for the presence of free intraperitoneal air.[3]

Most cases respond to conservative management, non operative continuous decompression and adequate treatment of the primary disease.[4,5] In protracted cases, gastric decompression eliminates upper GI secretions and decreases vomiting and gastric distention. Rectal tubes necessitate colonoscopic decompression, especially when the cecal diameter approaches 9-10 cm. In most patients with acute colonic pseudo-obstruction, conservative management will result in the resolution of colonic distention within three days.[3] Basic supportive measurements consist of fasting, fluid replacement, and correction of electrolyte imbalance. Prompt antimicrobial therapy is indicated if infection is suspected. Medications that slow GI motility (adrenergic agonists, sedative, narcotic analgesics) should be withdrawn or dose reduced. Total parenteral nutrition may be required in protracted cases.[3]

Ponec RJ et al., found that treatment with neostigmine was effective to decompress the colon in patients with acute colonic pseudo-obstruction. The use of neostigmine should be considered before colonoscopy is performed in patients with acute colonic pseudo-obstruction who have not had a response to conservative management.[4] Surgical exploration is reserved for acute cases with peritoneal sign, ischemic bowel or other evidence of perforation.[4]

Vegetarian diets can be classified as either lactovegetarian, ovovegetarian, lactoovovegetarian, or vegan if they include, respectively, dairy products, eggs, both dairy product and eggs, or no animal products at all. Vegan diets have a very low cobalamin (vitamin B_{12}) content.[8]

The main source of dietary cobalamin for humans is food of animal origin.[9] Vitamin B_{12} is produced in nature only by vitamin B_{12} producing microorganism. That is the reason why human must obtain vitamin B_{12} solely from the diet. Dhopeshwarkar et al observed that lactovegetarians had distinctly lower serum vitamin B_{12} concentrations than did nonvegetarians.[8] Measurement of vitamin B_{12} levels can be helpful for diagnosing vitamin B_{12} deficiency. Generally, normal serum B_{12} levels range from 200 to 900 pg/mL.[10] Clinical features of vitamin B_{12} deficiency involve the hematopoetic, the gastrointestinal tract, and the nervous system. Clinical manifestation may occur without abnormality in other systems.[5,11,12]

Gastrointestinal manifestations reflect the effect of cobalamin deficiency on rapidly proliferating gastrointestinal epithelium. Anorexia with moderate weight loss may also be evident, possibly accompanied by diarrhea and other gastrointestinal symptoms. These latter manifestations may be caused in part by megaloblastosis of the small intestinal epithelium, which results in malabsorbtion.[12]

On physical examination, the patient with cobalamin deficiency is pale with mild jaundice. Elevated serum bilirubin levels are related to high erithroid cell turnover in the marrow.[12] Management of cobalamin deficiency is related to the underlying disorder. If the deficiency is caused by malabsorbtion, initial parenteral treatment of 1,000 μg cobalamin per week for 8 weeks, followed by 1,000 μg cyanocobalamin injected intramuscularly every month. However, cobalamin deficiency can also be managed very effectively by oral replacement therapy of 2 mg crystalline B_{12} per day.[12]

CASE ILLUSTRATION

Male, 18 years old, was admitted to Cipto Mangunkusumo hospital in March 7, 2004 with chief complaint of nausea and vomiting since 3 days prior to

admission. Since one month prior to admission, the patient also complained of cough, sometimes with blood streak and shortness of breath, weight loss, loss of appetite and fluctuating fever, but he did not have night sweat. The patient went to Medika Lestari Hospital and was given some medicine from the doctor which was not known by the patient. According to the patient, the doctor said that he was suffering from typhoid fever and was suspected to have lung tuberculosis. After he completed the medications, he found no improvement at al. Since 3 days prior to admission he suffered from nausea and vomiting. He also complained about abdominal discomfort and constipation for 3 days. There was no history of diarrhea. He went back to Medika Lestari hospital, and underwent abdominal X-ray and some others laboratory examination. He had been hospitalized in Medika Lestari for 3 days and got some medications (ceftriaxone 2 g daily, ranitidin and metoclopramide injection 3 times daily). Nasogastric tube (NGT) was inserted also but there was not any improvement of his condition at all. The doctor suggested that he should be referred to Cipto Mangukusumo hospital for exploration. So he went to Cipto Mangunkusumo hospital, and the doctor in emergency room said that he was suffering from gastrointestinal disorder and lung infection, but because the wards were occupied, he went to Usada Insani hospital. According to the patient, the doctor at the Usada Insani hospital suggested that he undergo operation because of intestinal perforation. The patient and his family rejected the procedure decided to bring him back to Cipto Mangunkusumo hospital.

From the past medical history, the patient denied suffering from hepatitis, malaria, and he has no history of traveling out of town. He also denied using neither intravenous drugs nor free sex. He was a senior high school student, and had low social economic background.

On physical examination, the patient was moderately ill, alert and fully oriented but looked underweight. Blood pressure was 100/60 mmHg, pulse rate was regular at 110 beats per minute, temperature was 37.7°C and respiratory rate was 24 per minute. His height was 165 cm and the body weight was about 40 kg. Conjunctiva was not pale and his sclera was icteric. JVP was 5-2 cmH_2O, and there were no palpable lymph nodes. Lips and tongue was dry. Nasogastric tube (NGT) had already been inserted for gastric content drainage. NGT drainage was 200 cc greenish fluid. On chest examination, the heart sounds were normal without any murmur or gallop, the breath sound was vesicular with rales at both of the lungs, and no wheezing. The abdomen was distended, liver and spleen were not palpable, and the bowel sound decreased. Epigastric pain was felt on abdominal palpation. The extremities were warm and there was no edema or clubbing finger digital rectal examination found weakness of anal sphincter muscle, ampula of rectum did not collapsed, no tenderness, no mass, smooth inner surface of rectum and no feces or blood on hand gloves.

The previous laboratory results from Medika Lestari hospital on 4th March 2004 revealed hemoglobin level 11.1 g/dL, hematocryte 33%, leukocyte 17,600/µL and platelet count 325,000/µL, blood glucose 125 mg/dL, AST 114 µ/L and titer of serologic widal test were paratyphi BO 1/160, AH 1/160, BH 1/80.

On laboratory examination at the emergency room revealed hemoglobin 11.3 g/dL, hematocryte 33%, leukocyte 14,600/µL and platelet count 149,000/µL, blood glucose 99 mg/dL, liver function tests showed elevated ALT 135 µ/L and AST 183µ/L, BUN 51 g/dL, creatinine 0.5 g/dL, serum electrolyte were normal with sodium 144 mmol/L, potassium 4.1 mmol/L and chloride 105 mmol/L. There were slightly elevated levels of amylase 110 U/L and lipase 97 U/L. Three positional abdominal X-ray showed dilated gaster and duodenum with minimal air fluid level at intralumen, no herring bone appearance, minimal air distribute to distal, and absent of free intraperitoneal air. Chest X-ray showed infiltrates in both of the lung, especially at the right lung.

Based on all data above, the problems of this patient were: (1) Paralytic ileus; (2) Pneumonia; (3) Jaundice. Paralytic ileus was based on symptoms of nausea, vomiting, abdominal discomfort, sign of decreased bowel sound, leukocytosis and slightly elevated levels of amylase-lipase enzymes, and on three positional abdominal X-ray showed dilated gaster and duodenum with minimal air fluid level at intralumen, no herring bone appearance, minimal air distribution to distal, and absent of free intraperitoneal air. Acute pancreatitis was suspected of the etiology of paralytic ileus, with differential diagnosis of obstructive ileus and peritonitis. Pneumonia was based on cough for one month, with bloody streak, weight loss, loss of appetite, fluctuating fever, rales at both of the lung, leukocytosis, and infiltrate at both of the lung on chest X-ray. Differential diagnosis was lung tuberculosis. Jaundice was detected on the sclera accompanied by leukocytosis and elevated levels of ALT and AST suggested possible causes like cholecystitis and cholelythiasis with differential diagnosis of leptospira infection and acute hepatitis.

Our management included decompression of gastrointestinal, fasting with adequate parenteral nutrition by giving triofusin E 1,000 each 12 hours, normal saline and dextrose in saline 1,500 cc per day, 2 liters oxygen, 1 gram of cefotaxime 3 times daily, 1 ampoule pantoprazole once daily and fluid balance for 24 hours and consulted to Surgery Department. The result of the consultation, they suggested that the patient be rehydrated, decompressed, were given total parenteral nutrition and at that time no surgical intervention was indicated but need further evaluation. We planned to perform abdominal ultrasonography, repeat amylase-lipase test, liver function test, serologic marker of hepatitis such as HbSAg and Anti HCV, acid fast bacilli stained and sputum culture with microorganism resistance test.

On the third day of hospitalization, the patient's condition was moderately ill, blood pressure 120/80 mmHg, pulse rate 100 times per minute, respiratory rate 20 times per minute, no fever, but icteric sclera and decreased bowel sound. We continued to decompress with fasting and nasogastric tube, total parenteral nutrition, 1 g cefotaxime 3 times daily, 1 ampoule pantoprazole once daily, and twice daily alinamin F. Production of gastrointestinal secretion observed from NGT was 500 cc greenish fluid per 24 hours. We consulted to surgical department for further evaluation.

On the fourth day we reexamined the levels of amylase-lipase enzymes because production of NGT was 300 cc per 8 hours contained greenish fluid. We examined the liver function test and scheduled for abdominal ultrasonography. The results of the level of amylase-lipase enzymes were slightly increased with amylase: 129 U/L and lipase 128 U/L.

On the fifth day, bowel sound started to improve, and we planned to administer liquid diet started from 50 cc, if there were no complain of abdominal pain we planned to increase the volume of liquid diet on the following day. Anamnesis on diet lifestyle revealed that he had been vegetarian since he was 6 years old. Paralytic ileus might be correlated with his vegetarian lifestyle. We also suspected diagnosis of vitamin B_{12} deficiency in this patient.

On the 9th day lung condition were improved, with minimal rales at both of the lung, and we changed some medications to oral preparation (lanzoprazole once daily and oral alinamin F two times daily and hepatoprotector). On the 10th day the patient underwent abdominal ultrasound and the conclusion were chronic hepatitis appearance, no obstruction, and was suggested to check serologic marker of viral hepatitis. He was suggested to examine of vitamin B_{12} serum at the laboratory because of his life style. Unfortunately, the patient's family refused to do the examination because it was considered too expensive.

On the 12th day the patient had defecation, jaundice disappeared, minimal rales at the right lung, and he started to have soft diet.

On the 15th day the patient was discharged from hospital, given some medications (cefixime 100 mg 2 times daily, lanzoprazole once daily, and hepatoprotector). We still motivated him to check his vitamin B_{12} serum level to confirm diagnosis vitamin B_{12} deficiency. We also educated the patient to change his vegetarian lifestyle.

Two weeks after being discharged, he underwent the examination of vitamin B_{12} serum and chest X-ray for evaluation. The concentration of vitamin B_{12} serum was 175 pg/mL (normal value: 179-1132 pg/mL) and appearance of chest x-ray was improved. We suggested the patient to undergo bronchoscopic examination to help establish the diagnosis of pulmonary infection.

March 7, 2004: Three positional abdominal X-ray showed dilated gaster and duodenum with minimal air fluid level at intralumen, no herring bone appearance, minimal air distribute to distal, absent of free intraperitoneal air. On March 9, 2004, leptospirosis was negative. March 11, 2004. Culture of sputum was positive Pseudomonas sp.

DISCUSSION

Diagnosis of paralytic ileus is based on the complaint of nausea, vomiting, abdomen discomfort, with decreased bowel sound, there were leukocytosis and slightly elevated amylase-lipase enzymes, and on three positions abdominal X-rays dilated gaster and duodenum were found with minimal air fluid level at intralumen, no herring bone appearance, minimal air distribute to distal, and absent of free intraperitoneal air.

Before all the data were available, we suspected the etiology of paralytic ileus in this patient was acute pancreatitis with differential diagnosis tuberculosis peritonitis or leptopsirosis because of normal concentration of electrolyte serum. But after we repeated amylase-lipase enzymes, the result did not support the diagnosis of acute pancreatitis because the enzymes increased less than 2 times than normal value. Leptospirosis was also negative. One of the clinical conditions that may cause increased risk of paralytic ileus in this patient was pneumonia infection. Sepsis and all infections of the body could induce paralytic ileus.[2,3]

But clinical status of pneumonia infection was not severe in this patient, so we tried to explore other conditions that may aggravate paralytic ileus. After we asked the patient about his diet, we found that he was lactovegetarian. From the literature we know that lactovegetarian has cobalamin deficiency compared to non vegetarian.

The patient was lactovegetarian. He still consumed milk for his diet, but did not consume egg, meat and fish. That indicated he could be suffering from vitamin B_{12} deficiency. Clinical manifestation of vitamin B_{12} deficiency are various, one of them gastrointestinal disorder. Unfortunately, vitamin B_{12} serum could not be examined at that time when the symptoms and sign of paralytic ileus appeared.

Pneumonia infection was suspected of the etiology of paralytic ileus, aggravated by vitamin B_{12} deficiency, because all the data we collected did not support other causes like acute pancreatitis or electrolyte abnormalities. He was suggested to check vitamin B_{12} serum level to confirm the diagnosis.

Determining the underlying disease in paralytic ileus is not easy. Thus, conservative management must be given if clinical symptoms suggests diagnosis of paralytic ileus, while identify the etiologic diagnosis. Causal therapy can immediately be initiated when the underlying disease/condition has been identified.[2]

Decisions about management and selecting supporting examinations such as colonoscopy should be based on the patient's clinical status.[3] From the abdominal X-ray, dilated gaster and duodenum were found with minimal air fluid level at intralumen, no herring bone appearance, minimal air distribute to distal, and absent of free intraperitoneal air. No sign of total obstruction were found. But intestinal dilatation, had to be suspicion of intestinal perforation. If dilatation more than 12 cm or no resolution of colonic distention after six days, surgical exploration like caecostomy may be indicated. Risk of colonic perforation was reported higher if the caecal diameter exceeds 12 cm and if distention is present for more than six days.[2]Surgical intervention is required if there is clinical suspicion of mechanical obstruction[3]

Gastric decompression would help to eliminate upper GI secretions and decreases vomiting and gastric distention. The resolution of distention will be reached within 3 days. Ponec RJ recommended the use of neostigmine if conservative treatment shows no response.[4] Supportive management like fluid replacement, temporary or total parenteral nutrition, is an important thing for successful conservative management. Pneumonia infection may cause paralytic ileus, thus antibiotics should be administered according to the type of infection and the results of the antibiotic sensitivity test to achieve satisfactory outcome.

The levels of bilirubin, ALT, AST, phosphatase alkaline and gamma GT increased in this patient might be caused by cholestasis syndrome. We suspected that he was infected by hepatitis A virus, but unfortunately due to financial limitation the diagnosis could not be established. This condition could be attributed to the parenteral feeding that might mimicked cholestasis syndrome. The prognosis is usually good if the patient responds to conservative management, although sometimes the etiology remains undetermined.[2,3] This case was presented as demonstration case of paralytic ileus in vegetarian due to pneumonia infection with vitamin B_{12} deficiency.

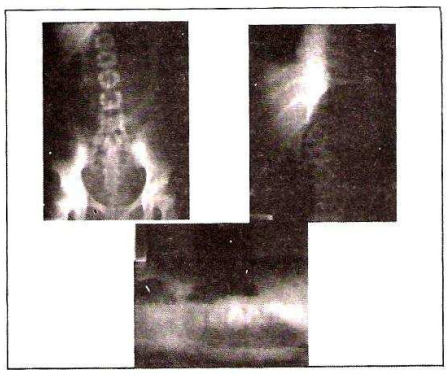

Figure 1. Three positional abdominal radiographic from Medika Lestari hospital

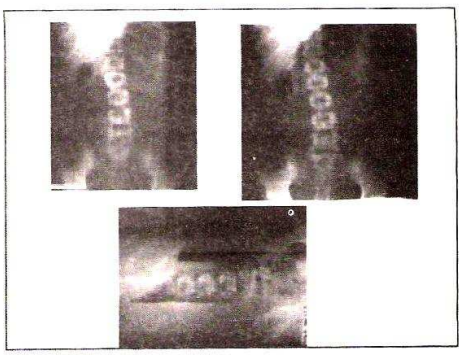

Figure 2. Three positional abdominal radiographic from Cipto Mangunkusumo hospital

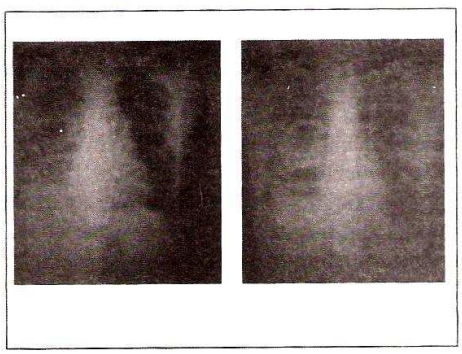

Figure 3. Comparison before and after treatment course of chest X-ray

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