Leukocytoclastic Vasculitis with Manifestations of Hematemesis and Melena

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ABSTRACT

Vasculitis is an inflammation of the blood vessel accompanied by the presence of leukocytes on the vascular wall, followed by structural damage. It often involves the liver and gastrointestinal system, with manifestations of hematemesis and melena. We report a case of hematesis and melena due to leukocytoclastic vasculitis due to the use of antibiotics.

Key words: Leukocytoclastic vasculitis, Hematemesis and melena

INTRODUCTION

Vasculitis is an inflammation of the blood vessel. It is characterized by the presence of leukocytes on the walls of the blood vessels and resulting structural damage, causing tissue ischemia and necrosis. Vasculitis may cause primary or secondary process as a result of a primary illness.[1,2]

Vasculitis often involves systemic organs, such as the liver and the gastrointestinal system. Gastrointestinal system disturbances may be the initial manifestation of vasculitis, particularly in certain types of vasculitis, such as Henoch-Schonlein purpura, systemic lupus erythematosus, and leukoclastic vasculitis.[2,3]

The level of vessel and organ damage varies from being life-threatening, such as ischemia of the mesenteric vessel or acute pancreatitis to non-specific esophageal dysmotility.[3]

In determining gastrointestinal manifestations due to vasculitis, it is very important to determine the size of the vessel involved. The size of the vessel sometimes determines the site of ischemia and the level of organ damage. Involvement of small vessels such as the intramural artery and the vasa recta of the intestinal wall creates a "patchy" look, focal ischemia and mucous ulceration. Involvement of moderate-size to large vessels can cause intestinal infarct, gangrene, and perforation. Aneurysm dilatation and rupture may occur on moderate-size vessels, causing bleeding of the intestinal wall and lumen.[3,4]

In the year 1990, the American College of Rheumatology formulated 5 criteria for leukocytoclastic or drug-induced vasculitis, as follows:[1,5]

- Age > 16 years
- Drug use during the time of the development of symptoms
- Palpable purpura
- Maculopapular rash
- Lesion biopsy on the skin demonstrating neutrophil deposit on arterioles and venules.

The presence of 3 or more criteria above has a sensitivity rate of 71% and a specificity rate of 84% for the diagnosis of hypersensitive/leukocytoclastic vasculitis.

PATHOGENESIS

Leukocytoclastic vasculitis is a complex immune process. It is characterized by leukocytoclasis, which is debris of cell nucleus originating from neutrophils infiltrating into and around the blood vessel during the acute phase. The sub-acute and chronic phases are dominated by mononuclear cells, while in certain groups, there is deposit of eosinophylic cells. There is often extravasation of erythrocytes from the blood vessel involved, causing the development palpable purpura.[6]

CLINICAL MANIFESTATIONS

Aside from skin lesions, palpable purpura and petechiae, other clinical manifestations include fever, urticaria, arthralgia, lymphadenopathy, low serum complement, and increased blood sedimentation rate.[1,5,7] Most

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skin lesions are located on locations influenced by gravity, such as the lower extremities of ambulatory patients, and the sacrum of bed rest patients.[8] In most patients, these symptoms develop from 7 to 10 days after antigen exposure, since adequate time is needed for the development of antigen-antibody complex.[1,5]

DIAGNOSIS

The diagnosis of leukocytoclastic vasculitis is established based on clinical findings and exposure to drugs or infection. Skin biopsy demonstrates findings of leukocytoclastic vasculitis, while the presence of IgA deposit indicates the possibility of Henoch-Schonlein purpura.[1,5] If IgA deposit is still unknown, the following clinical criteria can assist in differentiating leukocytoclastic vasculitis and Henoch-Schonlein purpura. If there are no more than the following 2 criteria, the diagnosis is most probably (74%) hypersensitive vasculitis. The criteria are as follows:

- Palpable purpura
- Bowel angina
- Gastrointestinal bleeding
- Hematuria
- Onset equal to or below the age of 20 years

TREATMENT

Termination of the use of the drug believed to cause allergy can reduce symptoms and signs within days to several weeks.

Patients with systemic complications and involvement of gastrointestinal tract should receive the following treatment (administered for 3 months, usually up to 6 months following the establishment of the diagnosis):[6,9]

- 2 mg/kg bodyweight/day of cyclophosphamid (up to a maximum of 200 mg/day). For those over 60 years of age, the dose is reduced 25%.
- 1 mg/kg bodyweight/day of prednison (up to a maximum of 80 mg/day), reduced to 25 mg/day for 8 weeks, and then to 10 mg/day for 6 months.

CASE REPORT

Mr. H, 16 years, a high-school student residing in Matraman, was hospitalized with chief complaint of abdominal pain since 12 days prior to admission.

Abdominal pain was continuous and localized, particularly in the epigastric region. The patient vomited after every meal or drink. The vomit contained food remains. Three days prior to hospitalization, the patient vomited approximately 5 tablespoons of reddish brown substance. He found it difficult to defecate, with little, solid, black, pungent stool, amounting to approximately 2 tablespoons. He had no problems urinating.

Since one year prior to admission, red spots similar to mosquito bites appeared on the skin of his hands, legs, and low back. The patient visited a dermatologist, and received 4 x 500 mg of erythromycin. The spots remained.

One week prior to admission, the patient complained of nausea and vomiting. The vomit contained food remains. The patient defecated black stool. He suffered from pain of the whole abdomen, particularly of the epigastrium, which remained even with antacids. The patient's conditions deteriorated, and he was finally escorted to Dr. Cipto Mangunkusumo General Central National Hospital.

Based on physical examination during admission, the patient was moderately ill and fully conscious. His blood pressure was 120/80 mmHg. His pulse rate was 88 times/minute, regular, with adequate volume. His respiratory rate was 16 times per minute, his temperature was 37.1° Celcius. His conjunctiva was not pale, his sclera demonstrated no signs of jaundice. His jugular venous pressure (JVP) was 5-2 cmH_2O. Cardiac evaluation demonstrated pure first and second heart sounds, without murmur or gallop. The lungs demonstrated vesicular breath sounds, without ronchi and wheezing. During abdominal examination, the patient's abdomen was not distended, there was tenderness of the whole abdomen, there were no signs of defans muskulaire. The liver and spleen were not palpable. Bowel sound increased. Skin turgor was satisfactory. The patient's extremities were warm, there was no edema, but there were petechie distributed unevenly on both hands and legs.

Laboratory data during admission were as follows: Hemoglobin level 16 g/dl, Hematocryte 46 vol%, leukocyte 10.400/ul, platelet count 309,000/ul, ureum 36 mg/dl, creatinine 0.5 mg/dl, and 102 mg/dl.

Based on history, physical and laboratory examination, the patient was diagnosed with melena accompanied with abdominal pain, petechie suspected to be due to vasculitis, and hyponatremia.

The problem of melena accompanied with abdominal pain was thought to be due to pancreatitis, with a differential diagnosis of erosive gastritis, established based on continuous, localized, intensifying abdominal pain for 12 days, particularly in the epigastric region, accompanied by nausea and greenish vomit. The patient was scheduled for peripheral blood evaluation, ureum, creatinine, electrolyte, and cito blood sugar, amylase and li-

pase laboratory evaluations, as well as endoscopy. For treatment, a nasogastric tube was inserted, the patient was asked to fast, and parenteral treatment combined with aminoleban:triofusin E-1000 1:1/12 hours and 0.9% NaCl/12 hours were administered. The patient also received 2 x vials of ranitidine and 3 x 1 teaspoon of sucralphate. Results of laboratory evaluation of amylase and lipase were 63 S. Somogyi and 34 S. Cherry Crandall respectively.

Results of laboratory evaluation of amylase and lipase of 63 S. Somogyi and 34 S. Cherry Crandall respectively, eliminated the possibility of pancreatitis. Peripheral blood evaluation of the patient demonstrated a hemoglobin level of 16 g/dl, hematocryte of 46 vol%, leukocyte count of 10,400/ul, and platelet count of 509,000/ul. The ureum and creatinine levels were 36 mg% and 0.5 mg%, respectively. His albumin level was 3.6 g/dl, and his globulin level 35 g/dl.

During treatment, the patient suffered from melena and hematoschaezia for 7 times. At first, the patient was suspected to have suffered from erosive gastritis due to injection dexamethasone. The patient thus received 40 mg of omeprazole injection (replaced with 2 x 20 mg of oral OMZ). During consultation, the gastroenterology division recommended to continue the administration of dexamethasone, with additional protection of PPI and sucralphate. Three days following administration of PPI and sucralphate, complaints subsided. The patient underwent endoscopy of the gastrointestinal tract to determine the etiology of hematemesis melena in the patient. Endoscopic evaluation found hyperemic patches with a diameter ranging from 3 to 8 mm. The biopsy of the gastric mucosa demonstrated non-active, non-atrophic chronic gastritis. There was no *H. pylori* or signs of malignancy. There was no melena and hematoschaezia. The nasogastric tube was removed, and the patient received gastric diet and was gradually given solid foods.

The problem of vasculitis was established based on a history of red spots on the hands, legs, and parts of the body, accompanied by malaise, and prior history of antibiotics administration. The patient was suspected to have suffered from drug-induced vasculitis, with a differential diagnosis of: 1. Wegener's granulomatosis, 2. Polyartheritis nodosa. The patient was scheduled for evaluation of ANA, anti-DS-DNA, ACA, and ANCA. The patient was scheduled for treatment with 5 mg x 2 vials of Dexamethasone (Prednisolone dose 1 ½ mg/kg bodyweight/day). Since the patient weighs 40 kg, the patient received 60 mg of prednisolone (4-4-4). ANCA titer and anti-DS-DNA evaluation turned out negative (anti-DS-DNA 97.9 iu/ul-Normal 0-200; ANCA titer 1:20 negative), thus eliminating the diagnosis of Wegener's granulomatosis. Skin biopsy by the department of Dermatovenereology found pathologic findings to be in accordance with leukocytoclastic vasculitis.

The problem of hyponatremia in this patient is based on a sodium level of 128 and a low osmolarity, probably due to excessive vomiting prior to hospitalization Post-correction evaluation demonstrated a sodium level of 135 and a potassium level of 4.5. The patient received 500 cc infusion of NaCl 0.9% every 12 hours, combined with parenteral nutrition. The patient was scheduled for re-evaluation of electrolyte balance following correctional treatment.

DISCUSSION

This case was deliberately brought up as a demonstration of vasculitis with manifestations of hematemesis and melena.

Leukocytoclastic vasculitis is a disorder of the blood vessel due to allergy to various substances including antibiotics. In this case, the patient is most probably allergic to clindamycin. However, previous history of petechiae needs to be further explored to acquire additional information on other substances causing the vasculitis in question.

Hematemesis and melena that developed in this patient is most probably due to vasculitis of the mesenteric vein, since signs of erosive gastritis was not found during endoscopic examination of the gastric mucosa. However, such information should be supported by arteriography and venography of the mesenteric vein and artery. However, these examinations were not performed, due to the possibility of more severe systemic side effects due to administration of contrast agents.

Treatment of hematemesis and melena in this patient was the same as in patients with general erosive gastritis. The patient was required to fast; a nasogastric tube was inserted; and antacids, proton-pump inhibitor (PPI), and mucoprotector agents were administered. Then, if the nasogastric tube turned out to be free of blood during rinsing, treatment is continued in the form of administration of gastric diet.

During gastroendoscopy, the examining physician found an uncommon finding in the form of aggregating red patches. It was still unclear whether this finding was due to vasculitis or other, since histopathological evaluation demonstrated no abnormality.

To establish a definite diagnosis in this patient, aside from lesion biopsy, immunofluorescence microscopic

evaluation should be performed to find IgA deposits. This is performed to differentiate leukocytoclastic vasculitis and Henoch-Schonlein purpura.[1,5] Since this last evaluation was not performed, we based the diagnosis on a tally of clinical criteria, where if no more than 2 of the following criteria is found, the diagnosis most probably (74%) points toward hypersensitive vasculitis: palpable purpura, bowel angina, gastrointestinal bleeding, and an onset of 20 years or less. Thus, the patient most probably suffers from Henoch-Schonlein purpura.

Treatment of vasculitis in this patient is in the form of steroid prednisolone 1½ mg/kg bodyweight/day for 3 months (prednisolone 5 mg 4-4-4). Then the patient was then tapered off. Based on the symptoms, the prognosis in this patient was quite satisfactory, bearing in mind that the etiology is drug allergy.

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