CASE REPORT

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Profuse haematochezia related to Crohn's disease: a rare case report

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ABSTRACT

Background: Lower gastrointestinal bleeding (LGIB) can be caused by irritable bowel disease. Haematochezia is one of the LGIB symptoms. Profuse haematochezia is a rare complication in Crohn's disease.

Case Presentation: We reported a 24-year-old patient with profuse haematochezia with severe anaemia and hypovolemic shock. Anamnesis, physical examination and colonoscopy showed that haematochezia was caused by Crohn's disease. Colonoscopy was done after the patient was hemodynamically stable with findings of external haemorrhoid and multiple ulcers with varying size (0.5–2 cm) on ascending colon, caecum and terminal ileum. The tissue histopathology of ileocecal junction indicated intestinal epithelia with partially eroded, crypts infiltrated by inflammatory cells, swollen lamina propria with proliferation and dilation of blood vessels indicting the Crohn's disease. The patient then treated with Crohn's disease medical therapy including low dose oral steroid, sulfasalazine and antibiotics. Clinical improvement was found on one week follow-up after discharge from hospital.

Conclusion: Profuse haematochezia could be associated with Crohn's disease and the comprehensive approaches should be taken to manage the Crohn's disease with such presentation.

Keywords: haematochezia, lower gastrointestinal bleeding, irritable bowel disease, Crohn's disease.
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INTRODUCTION

Lower gastrointestinal bleeding (LGIB), any bleeding distal to the ligament of Treitz, can present as chronic or acute. Acute LGIB can be presented as melena with hemodynamic instability and require resuscitation that might require transfusion. Anaemia and haematochezia are usually presented as chronic LGIB. Haematochezia, passing of fresh blood from the rectum or in the stool, is one of the symptoms of LGIB.[1] LGIB incidence is approximately 33/100,000 population per year in the United Kingdom.[2] The bleeding originates mostly from the distal ileocecal.[3] The most common causes of LGIB are diverticulosis, ischemic colitis, colorectal polyps and haemorrhoids.[4,5] Acute severe LGIB caused by Crohn's disease is a rare case and its therapeutic is challenging.[6,7]

LGIB in Crohn's disease (CD) is not as much as in ulcerative colitis, but haematochezia is one of the most common manifestations (45%) of CD.[8] Profuse bleeding is a rare complication of CD, approximately 0.9-2.5% (9). The incidence and prevalence of inflammatory bowel disease (IBD) in developed countries is higher than in developing countries (10). Therefore, CD in developing country such as Indonesia is relatively rare in particular with haematochezia. Here we present a 24-year-old male with acute profuse haematochezia caused by CD.

CASE PRESENTATION

A 24-year old male referred to Emergency Room of Dr. Soetomo General Hospital with chief complaint of profuse bloody stool for about 1500 ml. The patient had a history of bloody stool since four days before admission, approximately 200 ml with frequency 2–3 times a day. The patient also felt nausea, vomiting, dizziness, fever and feeling weakness for four days before admission. The patient complained about pain in all quadrants of the abdominal areas when defecating and a mass from rectum that intermittently came out six months ago. The body weight loss was about 3 kg for last month. No history of diabetes mellitus or hypertension. The patient's dietary routine rarely consumed fruit and vegetables and usually ate fried foods. The patient has been an active smoker with 3–5 cigarettes daily since five year ago.

On admission, vital signs showed blood pressure 90/60 mmHg, heart rate 140 beats per min, respiration rate 22 breaths per minute, temperature 37°C, capillary refill time >2 seconds,

oxygen saturation 98% with nasal cannula 3 litters per minute (lpm) and alert consciousness. Body mass index (BMI) status 24.2 kg/m^2. Physical examination showed conjunctiva anaemic, wet, cold and oedema on lower extremities. Digital rectal examination found a mass suspected as haemorrhoids and faeces with blood. Laboratory findings suggested anaemia (4.1 g/dl), leucocytosis (17.310 µl), neutrophilia (72.5%), hypoalbuminemia (2.03 g/dl) and elevated liver enzymes (ALT 210 IU/l and AST) 390 IU/l). Human immunodeficiency virus (HIV), hepatitis B surface antigen (HBsAg), hepatitis C virus (HCV) tests showed negative results. Abdominal ultrasound (USG) assessment showed slight hepatomegaly. The patient was first diagnosed with hypovolemic shock with profuse haematochezia caused by colitis as differential diagnose with grade III internal haemorrhoid.

The patient was then transferred to the intensive care unit (ICU) until hemodynamically stable. The tamponade dressing still detected bloody stool until day 3 after admission but the volume was reduced. The patient received two bags of whole blood transfusion, and five bags of packed red cell transfusions (two transfusions each day) to gain haemoglobin at level 11.6 g/dl on the day 7 of admission. Intravenous antibiotics (levofloxacin and metronidazole), antifibrinolytic, vitamin K, antiemetic and proton pump inhibitors were given until day of 6 of admission. Tamponade dressing was placed in the patient's anal canal to control and monitoring the bleeding.

Patient was planned for faecal calprotein test and colonoscopy. On the day 3 of admission, faecal calprotein showed 232 µg/g. Colonoscopy was conducted on the day 7 of admission (Figure 1). Colonoscopy findings showed intact external haemorrhoid (size 2 x 1 x 1 cm), erythema spots on rectosigmoid mucosa, multiple ulcers with varying size (0.5–2 cm) on ascending colon, caecum and terminal ileum, and the edges of appendix was found hyperaemic and oedematous. The patient was then diagnosed with Crohn's disease and external haemorrhoids. Low dose oral steroid (4 mg methylprednisolone each 8 h), sulfasalazine (500 mg each 8 h), and proton pump inhibitor (omeprazole 20 mg once a day). The patient was discharged after nine day hospitalization. The histopathology of the tissues from ileocecal junction showed mucosal tissue covered with intestinal epithelium partially eroded, crypts infiltrated by neutrophils and lymphocytes, the lamina propria was slightly swollen with the distribution of neutrophil cells, histiocytes, lymphocytes, plasma cells, proliferation and dilation of blood vessels (Figure 2). The conclusions of histopathology finding was chronically active colitis.

DISCUSSION

Haematochezia is a common clinical presentation of LGIB with incidence of hospitalization rate approximately 35.7/100,000 in the US.[11,12] The differential diagnosis for haematochezia is broad and needs careful medical evaluation. Management approach for haematochezia should be started by evaluating the hemodynamic instability, obtain the source of bleeding, assess any abdominal and pelvic pain and perform digital rectal examination.[13] Haematochezia can be presented on some diseases such as haemorrhoids, neoplasia, IBD, radiation enteritis/colitis and infectious colitis.[11] The differential diagnosis of haematochezia can be dependent on the patient age. CD is a potential cause of haematochezia in younger patients (<20 years old).[13] IBD contributes to 9.5% of LGBI in adults in China.[14] A rare but life-threatening complication of IBD is an acute profuse haematochezia. In patients with CD the prevalence of profuse haematochezia is very low ranges from 0.6 to 6%.[15,16] This indicates that the case of profuse haematochezia in CD patient is a rare patient.

CD lesions can be from mouth to anus and mostly found on ileum and colon or both.[17] During the disease course, the CD could have period of remissions and flares. The pathogenesis of CD involves the interactions of environmental factors, innate immunity, susceptibility genes and the host gut microbiota. These factors lead to intestinal mucosa disruption.[18] The incidence and prevalence of CD have been found increasing over the last three decades and the most common is reported in Europe and north America, but is probably increasing in Asia and Africa. The incidence of CD in Asia has peaked in age 20–24 years and 40–44 years. One of the risk of developing CD is associated with smoking.[19,20] Smoking could be one of the risk factor for this patient who was active smoker for almost five years with 3–5 cigarettes daily.

The diagnosis of CD can be concluded from clinical finding, imaging data with histologic finding and laboratory test. The most common symptom is chronic diarrhoea followed by abdominal pain in the right lower quadrant, weight loss, haematochezia, fatigue (due to inflammation, anaemia, deficiency of vitamin and mineral deficiencies), fever and extraintestinal manifestations (arthropathy, erythema nodosum, uveitis, hepatobiliary diseases).[21] Faecal calprotectin is a useful test to rule out CD in adult with 93% and 94% sensitivity and specificity, respectively at 50 µg/g

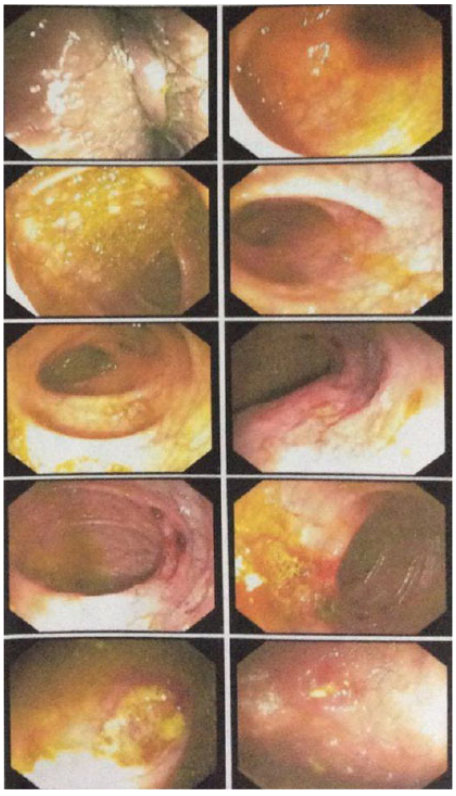

Figure 1. Colonoscopy finding showed external haemorrhoid and multiple ulcers with varying size (0.5–2 cm) on ascending colon, caecum and terminal ileum.

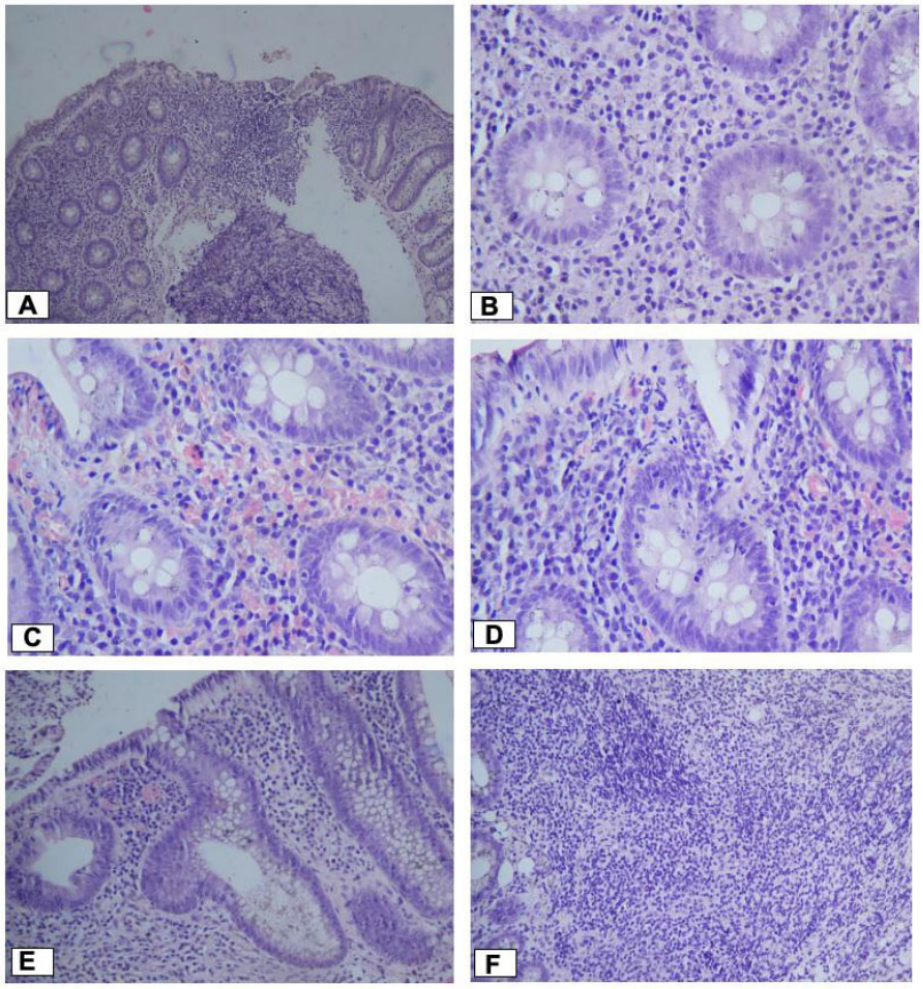

Figure 2. The histopathology of ileocecal junction tissue from the patient. The histopathology shows eroded epithelium (A), swollen lamina propria with the distribution of neutrophil cells (B), proliferation and dilation of blood vessels (C), infiltrated crypts with neutrophils and lymphocytes (D), distorted crypts (E) and aggregation of the lymphocytes (F). A: 100 magnifications, B-D: 400 magnifications, E and F: 200 magnifications.

cut-off.[22] Typical endoscopic findings in CD include: (1) cobblestone appearance; (2) focal distribution of longitudinal ulcers; and (3) small aphthous ulcerations arranged in a longitudinal fashion.[23] Histologic characteristics of CD include focal crypt architectural distortion, focal chronic inflammation, and granulomas not related to crypt injury.[24] In this case, the histologic showed intestinal epithelium that partially eroded, crypts infiltrated by inflammatory cells and swollen lamina propria suggesting the CD.

The management CD are established based on location, severity, complications, and future disease prognosis. The severity of disease can be calculated with Crohn's Disease Activity Index (CDAI).[25] Based on CDAI, the criteria as follow: (a) remission: CDAI score <150 points; (b) mild-to-moderate: 150–220 points; (c) moderate-to-severe disease: 220–450 points; and (d) severe: >450 points.[25] The CDAI total score in this patient was 443 indicates a moderate-severely active disease and therefore medical therapy was chosen. The start of medical therapy with a combination of biological and immunosuppressant, so called 'top-down' or start treatment with topical steroids then step up to systemic steroids, immunosuppression and biologicals ('step-up') treatment were still debatable. We treated this patient with a step-up method. Based on American College of Gastroenterology (ACG) guidelines, for induction phase, prednisone 40-60 mg/day or equivalent (for 1–2 weeks then reduced by 5 mg/week for 3 months), anti-tumour necrosis factor (TNF) (infliximab, adalimumab, certolizumab pegol), anti-integrin with or without immunomodulators can be given. The treatment then followed by maintenance phase with azathioprine (1.5–2.5 mg/kg/day) and 6-mercaptopurine (0.75–1.5 mg/kg/day), methotrexate (MTX) (15–25 mg/week), anti-TNF, anti-integrin, anti-p40 inhibiting IL-12 and IL-23 (for failed steroids, thiopurine, MTX, and anti-TNF).[26] Our patient achieved remission after steroid and 5-aminosalicylic acid (5-ASA) oral treatment. Clinical improvement was achieved on one week follow-up after hospital discharge.

CONCLUSION

We found acute life-threatening complications of Crohn's disease case. Profuse haematochezia was an indication of LGIB. Epidemiologic and historical features of current disease should be considered for LGIB diagnosis. Identification of the bleeding site is the most important initial step in treatment. The use of elective colonoscopy and laboratory studies such as faecal calprotein found to be useful in our case. The medical therapy using steroids and sulfasalazine showed a good result on our moderate-to-severe disease patients.

PATIENT CONSENT

Verbal informed consent was obtained from the patient's next of kin for inclusion in this report. Research approval is not required for case reports.

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DISCLOSURE OF CONFLICTS OF INTEREST

I would like to declare that there is no conflict of interest during completion of this case report.

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AUTHOR CONTRIBUTION

All authors contributed significantly to the study from the conceptual, data acquisition, data analysis and during manuscript preparation.

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