Alcohol-Associated Bowel Disease (ABD) in a Young Patient Presenting with Mixed Gastrointestinal Bleeding: A Case Report

Agustien Bayu Ristanti[*,**], *Muhammad Erlangga Putra Harimurti*[*,**], *Mohammad Amin Rais Perfernandi Ilham*[*], *Agastjya Wisjnu Wardhana*[**]

[*]Faculty of Medicine Universitas Indonesia - Cipto Mangunkusumo Hospital, Jakarta, Indonesia
**Bunda Jakarta General Hospital, Jakarta, Indonesia

Corresponding Author:
Agustien Bayu Ristanti, Faculty of Medicine Universitas Indonesia - Cipto Mangunkusumo Hospital, Bunda Jakarta General Hospital, Jakarta, Indonesia, Email: agustienristantia@gmail.com Hp:+6285775414450

ABSTRACT

Gastrointestinal (GI) bleeding is a common condition that can be life-threatening and requires prompt diagnosis and treatment. In younger adults without significant comorbidities, GI bleeding presents a diagnostic challenge, as it is uncommon and requires thorough evaluation to identify the underlying cause. Chronic excessive alcohol consumption is a significant yet often underrecognized risk factor. In recent studies, the term alcohol-associated bowel disease (ABD) has been introduced to describe GI problems mainly related to alcohol consumption. We present a case of a 29-year-old male with a history of chronic alcohol use who presented with melena and hematochezia. Laboratory evaluation revealed anemia and hypoalbuminemia, while abdominal ultrasonography revealed moderate fatty liver disease. Esophagogastroduodenoscopy confirmed erosive gastroduodenitis with a mucosal break greater than 5 mm, accompanied by esophageal candidiasis and grade B esophagitis. Meanwhile, colonoscopy confirmed the diagnosis of ulcerative colitis with active erosive ulcer bleeding. This case highlights severe mixed GI bleeding associated with chronic alcohol use, which may be classified as ABD. This case report aimed to demonstrate the importance of considering ABD in a relatively healthy young adult presenting with mixed and severe gastrointestinal bleeding.

Keywords: *Alcohol-associated Bowel Disease, Hematochezia, Melena, Mixed Gastrointestinal Bleeding*

ABSTRAK

Perdarahan saluran cerna merupakan kondisi yang umum terjadi namun dapat mengancam jiwa, yang memerlukan diagnosis dan penanganan segera. Selain itu, perdarahan saluran cerna pada orang dewasa muda tanpa penyakit penyerta yang nyata menjadi sebuah tantangan dalam diagnostik, karena jarang terjadi dan memerlukan evaluasi menyeluruh untuk mengidentifikasi penyebab yang mendasarinya. Konsumsi alkohol berlebihan kronis merupakan faktor risiko yang signifikan namun seringkali diabaikan. Penelitian terkini memunculkan istilah penyakit usus terkait alkohol (alcohol-associated bowel disease atau ABD) untuk mengklasifikasikan masalah saluran cerna yang terutama terkait dengan konsumsi alkohol. Pada kasus ini, seorang pria berusia 29 tahun dengan riwayat konsumsi alkohol kronis yang datang dengan melena dan hematochezia. Hasil laboratorium menunjukkan anemia dan hipoalbuminemia, sedangkan USG menunjukkan perlemakan hati derajat sedang. Esofagogastroduodenoskopi mengkonfirmasi gastroduodenitis erosif dengan robekan mukosa >5 mm, dengan kandidiasis esofageal dan esophagitis grade B. Sementara itu, kolonoskopi mengkonfirmasi diagnosis kolitis ulseratif dengan perdarahan ulkus erosif yang jelas. Dalam kasus ini,

perdarahan saluran cerna campuran yang berat dikaitkan dengan penyalahgunaan alkohol, yang dapat diklasifikasikan sebagai ABD. Oleh karena itu, laporan kasus ini bertujuan untuk menunjukkan bagaimana perdarahan gastrointestinal campuran dan berat, yang ditandai dengan tinja yang sebagian besar berdarah, pada orang dewasa muda yang relatif sehat dapat dikaitkan dengan ABD.

Kata Kunci: *Alcohol-Associated Bowel Disease, Hematochezia, Melena, Perdarahan Saluran Cerna Campuran*

INTRODUCTION

Gastrointestinal (GI) bleeding is a common condition that can be life-threatening and poses a significant global health problem. GI bleeding is generally classified into upper and lower GI bleeding. The distinction between the two categories depends on the location of the source of bleeding, with the ligament of Treitz, also known as the suspensory ligament of the duodenum, serving as the anatomical boundary between upper and lower GI tract. Upper gastrointestinal bleeding (UGIB) originates from the esophagus, stomach, or duodenum, while lower gastrointestinal bleeding (LGIB) originates from the small intestine, colon, or anorectal region.[1,2] The incidence of UGIB is reported to range from 15.0−172.0 cases per 100,000 person-years, while that of LGIB varies from 20.5−87.0 cases per 100,000 person-years. Mortality rates associated with GI bleeding ranges from 0.9−9.8 cases per 100,000 person-years and 0.8−3.5 cases per 100,000 person-years for UGIB and LGIB, respectively.[1] Clinically, UGIB commonly presents with hematemesis, coffee-ground vomiting, or melena. In contrast, LGIB is characterized by bright red rectal bleeding, blood clots, or blood mixed with stool. However, rapid UGIB may also manifest as fresh blood or blood clots from the rectum.[1,2]

Alcohol consumption is a well-recognized risk factor for major GI bleeding.[3] Excessive alcohol consumption is estimated to have caused 3 million deaths globally in 2016, representing 5.3% of total global mortality. Among the 230 alcohol-related diseases, the leading causes of alcohol-related deaths include gastrointestinal disorders (21.3%), unintentional injuries (20.9%), and cardiovascular conditions (19.0%).[4] Excessive alcohol consumption, defined as more than 42 drinks per week, is associated with a four-fold increased risk of peptic ulcer bleeding. However, even moderate alcohol consumption (1–2 drinks per day) is significantly associated with an increased risk of peptic ulcer bleeding.[3-5] Furthermore, alcohol consumption is believed to induce pathological changes in both the small and large intestines, potentially contributing to alcohol-related bowel disease (ABD) and further exacerbating GI complications.[6,7] Severe GI bleeding in otherwise healthy young adults is uncommon, as the condition is generally associated with pre-existing comorbidities that predispose individuals to GI bleeding. Therefore, a thorough understanding of initial management strategies, treatment approaches, and the mechanisms by which chronic alcohol consumption may contribute to GI bleeding is essential, particularly in emergency situations requiring immediate intervention.

In this report, we present a case of a 29-year-old healthy patient presented with severe GI bleeding which resulted in a syncopal episode. The patient was successfully treated with medication, and the source of bleeding was identified through esophagogastroduodenoscopy (EGD)-Colonoscopy. This case highlights the significant role of alcohol in GI bleeding, even in young adults without comorbidities or other risk factors.

CASE ILLUSTRATION

A 29-year-old male presented to the emergency department with complaint of bloody stool since the day prior to admission. The patient had experienced bloody stool twice and had six episodes on the day prior to presentation. The symptoms were accompanied by generalized weakness, light-headedness, and a syncopal episode before hospital admission. He reported persistent epigastric pain and cramping sensations associated with the urge to defecate, and the passage of bloody stool. He had experienced similar symptoms one year prior, although the severity at that time was less than in the current episode. An upper endoscopy performed during the previous visit revealed gastroesophageal reflux disease (GERD). The patient also reported having recurrent epigastric discomfort for approximately five years, but no prior endoscopic evaluation had been conducted until the procedure performed one year ago.

The patient had been a recreational alcohol user for the past five years and had developed an addiction, with the most recent consumption was prior to presentation, averaging 8-10 drinks per week. He also had a history of

coffee consumption, with an average of one cup per day. He was a former smoker and denied any intravenous (IV) drug use. The patient denied experiencing unintentional weight loss, hematemesis, or changes in bowel habits. There was no history of non-steroidal anti-inflammatory drug, anticoagulant medications, antiplatelet agents, or iron supplementation. There was a history of GERD, while hypertension, diabetes mellitus, and hepatitis were denied.

On physical examination, the patient appeared pale and dehydrated but was euvolemic and hemodynamically stable at presentation. Vital signs were within normal limits. The conjunctivae appears anemic. There was no jaundice, spider nevi, or stigmata of chronic liver disease. Cardiopulmonary examination findings were normal. Abdominal tenderness was noted, particularly in the left upper quadrant. Murphy's sign was negative. No palpable masses, organomegaly, or ascites were detected. Increased bowel sounds were observed, and melena mixed with hematochezia was seen from the patient's rectum, measuring approximately 200–250 mL, along with a lump in the anus.

Laboratory results showed a hemoglobin level of 7.5 g/dL (reference range: 13.5–17.5), indicating anemia. The white blood cell count was 6870/µL (reference range: 5000–10,000), platelet count was 255,000/µL (reference range: 150,000–400,000), prothrombin time was 11.2 seconds (reference range: 10.4–12.9), and the activated partial thromboplastin time was 28.6 seconds (control: 32.6), all within normal limits. Albumin was low at 3.41 g/dL (reference range: 3.97–4.94). Cholinesterase levels were normal at 4770 (range: 4620–11,500). A whole abdomen ultrasound showed moderate fatty liver, and no abnormalities were detected on the chest X-ray.

Following initial stabilization in the emergency department, the patient was monitored in the High Care Unit. Therapeutic interventions included the administration of Ringer's lactate and B-fluid for fluid resuscitation, carbazochrome diluted in normal saline for hemostasis, intravenous tranexamic acid for bleeding control, vitamin K for coagulation support, a continuous intravenous infusion of a proton pump inhibitor (PPI) to reduce gastric acid secretion, and intravenous ondansetron for antiemetic management. A nasogastric tube (NGT) was inserted for decompression and drainage, and the patient was kept fasting. Furthermore, packed red blood cells (PRBC) were transfused to address anemia and blood loss. Once hemodynamic stability was achieved after a total of four packs (886 mL) of PRBC transfusion, with hemoglobin level above 9 g/dL, the patient was planned for esophagogastroduodenoscopy (EGD) and colonoscopy for further evaluation.

EGD and colonoscopy were performed under general sedation after bowel preparation. The EGD (**Figure 1**) revealed mucosal break exceeding 5 mm in two folds of the lower esophageal sphincter (LES), along with visible fungal infection and esophageal edema. The gastric fundus was swollen, while the cardia and corpus were swollen, hyperemic, and showed erosions exceeding 30%. The antrum was also swollen and hyperemic, with bleeding spot erosions. The duodenum was swollen and hyperemic. The EGD findings were suggestive of Esophageal Candidiasis, confirmed Esophagitis Grade B, and Erosive Gastroduodenitis. Biopsy samples were taken from the cardia, antrum, and duodenum.

Colonoscopy (**Figure 2**) revealed edema and hyperemia in multiple areas of the colon and rectum, with mucus, bleeding spot erosions, and traces of blood. The anus showed edema but no evidence of hemorrhoids, and the appendix and terminal ileum appeared swollen and hyperemic, with traces of blood. Biopsy samples were taken from the ascending colon, transverse colon, and descending colon. These sites were selected to provide representative sampling of right, mid, and left colon involvement, as the inflammatory changes were observed in multiple and generally comparable areas across the examined segments. Additional biopsies were not performed because further sampling would likely not provide additional diagnostic value and could increase procedural risks.

The colonoscopy result confirmed ulcerative colitis. Histopathological examination of the gastric biopsies from the antrum and corpus revealed chronic non-active gastritis with preserved glandular architecture, no epithelial dysplasia, and no evidence of *Helicobacter pylori*. Duodenal biopsies demonstrated chronic duodenitis, characterized by blunt, rounded villi with a mild to moderate mononuclear inflammatory infiltrate and the presence of Brunner's glands, without features of active inflammation or dysplasia. Colon biopsies from the cecum to the descending colon demonstrated non-specific colitis, consisting of mild chronic inflammatory infiltrate without crypt distortion, basal plasmacytosis, or neutrophilic activity. Together, these findings indicate chronic but non-active inflammatory changes in the stomach, duodenum, and colon without evidence of *H. pylori* infection or inflammatory bowel disease-defining features.

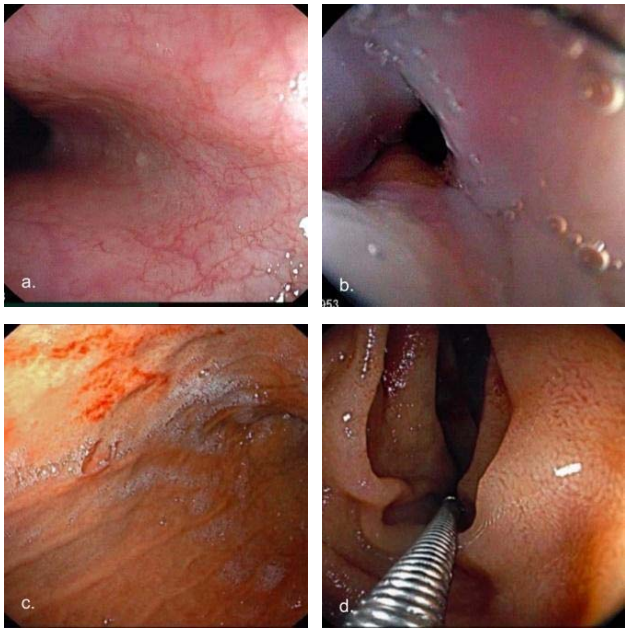

Figure 1. EGD findings: a. candidiasis esophagitis b. mucosal break greater than 5 mm at the LES c. erosions exceeding 30% of the gastric cardia, corpus, and antrum d. edematous and hyperemic duodenum

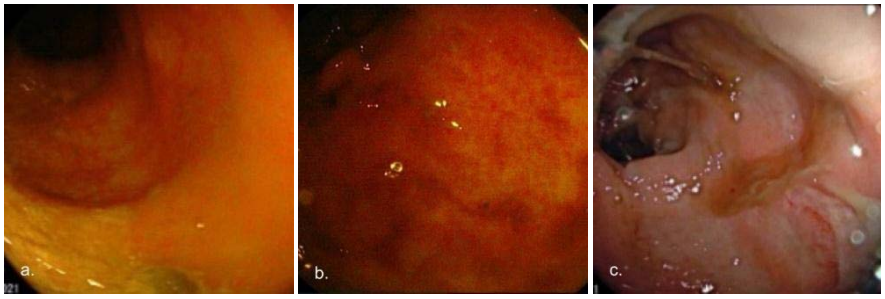

Figure 2. Colonoscopy findings: a & b. edema, hyperemia, and erosions observed in the ascending colon c. presence of mucus, traces of blood, and erosive ulcer bleeding in the descending colon

After undergoing the procedure, the patient was treated with 3 x 15 cc sucralfate orally, 3 x 100 mg rebamipide orally, 4 x 1 cc nystatin orally, 3 x 500 mg Mesalazine orally, 1 x 1 sachet probiotics (*Lactobacillus acidophilus* Rossel-52 dan *Lactococcus rhamnosus* Rossel-11) orally, 2 x 40 mg intravenous esomeprazole, 3 x 8 mg intravenous ondansetron, and 3 x 500 mg intravenous tranexamic acid. During treatment, there were no episodes of bloody stools. The abdominal pain subsequently subsided and the patient's general clinical condition improved. After two days of inpatient care, the patient was discharged and continued with outpatient treatment.

DISCUSSION

This case study of a 29-year-old male with a history of alcohol addiction who presented with melena, hematochezia, anemia, and significant gastrointestinal involvement highlights the complex interplay between chronic alcohol use and bowel disease. Alcohol contributes to GI bleeding through multiple mechanisms. Ethanol metabolism produces acetaldehyde and acetate, which disrupt the intestinal barrier, increasing permeability and systemic inflammation. This disruption facilitates the translocation of endotoxins, exacerbating mucosal damage and triggering inflammatory responses.[7] Alcohol-induced dysbiosis also alters the gut microbiome, reducing beneficial microbial populations, and allowing overgrowth of pathogenic bacteria. This microbial imbalance further disrupts the mucosal barrier and perpetuates inflammation. These changes play a crucial role in the established connection between alcohol-related oxidative stress, increased gut permeability, and the eventual development of alcoholic liver disease and other related conditions. Additionally, ethanol exposure in the small intestine

causes malabsorption due to reduced active transport of nutrients such as vitamins, monosaccharides, and several L-amino acids—across epithelium, which could also lead to stool abnormality.[7,8]

The presence of erosive gastroduodenitis and grade B esophagitis in this patient aligns with prior findings that alcohol-induced gastric mucosal damage can lead to significant UGIB. A prospective study by Strate *et al*. highlighted the role of alcohol in GI bleeding, demonstrating that men consuming more than 30 grams of alcohol daily had a 1.43-fold increased risk of major GI bleeding compared to non-drinkers, with the strongest association observed for upper GI bleeding associated with heavy drinking.[9] Alcohol-related GI bleeding often leads to iron deficiency anemia, as observed in this patient with a hemoglobin level of 7.5 g/dL. Furthermore, the patient presented with hypoalbuminemia (3.41 g/dL) and moderate fatty liver on ultrasound, suggesting early hepatic dysfunction associated with chronic alcohol consumption. The hepatotoxic effect of alcohol can lead to liver dysfunction, portal hypertension, and the formation of esophageal or gastric varices, significantly increasing the risk of bleeding. In acute conditions, alcohol can impair clotting mechanisms and platelet function, exacerbating bleeding tendency.[5-7] However, in this case, we were able to rule out esophageal varices as a possible cause of melena, as the EGD result showed erosive gastroduodenitis instead. As previously mentioned, gastritis is known to cause melena in alcoholism, even in the absence of peptic ulcer.

One interesting finding in this patient was the presence of Candida Esophagitis (CE), as revealed in the EGD. Alcohol might impair intestinal permeability and disrupt the gut microbiota, leading to systemic inflammation and increased susceptibility to infections such as esophageal candidiasis.[8] Interestingly, alcoholism is known to be a major risk factor for CE. Mushi *et al*. in their study stated that alcohol consumption is an independent risk factor for CE (OR 17.1; 4.94-58.87).[10] In non-HIV patients, approximately 70% of patients with CE consume alcohol. A study by Chen *et al*. also found that alcohol consumption is an independent risk factor for CE.[11] A case reported by Ali *et al*. in 2020 also identified CE in a patient with a history of heavy alcohol consumption, presenting with melena as a chief complaint.[12] In CE, the most common chief complaint is dysphagia, but other studies have shown that melena along with nausea, abdominal pain, and heartburn are also common complaints in patients.[13,14] However, in the present case, the UGIB was most likely due to erosive gastritis rather than CE, as erosive gastritis is a far more common and well-established cause of UGIB in immunocompetent patients.[15] Nevertheless, in immunocompromised patients, CE with ulceration should be considered as a potential source of GI bleeding.[16] Antifungal therapy administered in this case was appropriate according to existing guidelines.[17]

A unique finding in this patient was the presence of ulcerative colitis (UC) on colonoscopy with visible erosive ulcer bleeding, and chronic colitis confirmed by biopsy. Although UC is primarily an immune-mediated inflammatory condition, alcohol may act as a precipitating or exacerbating factor through dysbiosis, increased intestinal permeability, and direct mucosal toxicity. Alcohol-induced mucosal damage and immune modulation can intensify colonic inflammation, leading to more severe disease manifestations. Although alcohol does not directly cause UC, its contribution to symptom exacerbation, disease progression, and relapse is well recognized.[17,18] In recent years, this phenomenon has been classified as alcohol-associated bowel disease (ABD), as the diagnosis could be established from the history of alcohol consumption.[7,19] The aforementioned pathophysiological changes in the intestine due to alcohol consumption can trigger ABD and its development. However, it is quite tricky to diagnose ABD without considering the possibility of other confounding conditions that could cause GI bleeding. However, we were able to exclude several important diseases in this young and relatively healthy patient with the help of clinical and supporting examinations (including EGD and colonoscopy). The diseases ruled out included alcoholic liver disease (with only moderate fatty liver in ultrasound presentation), diverticulitis, and hemorrhoid. These exclusions led to the diagnosis of ABD, but other possibilities such as Inflammatory Bowel Disease (IBD), still need to be considered due to the difficulty of diagnosing ABD.

A major challenge in diagnosing ABD lies in the absence of clear histological criteria, particularly in patients with Alcohol Use Disorder (AUD). Studies have shown that AUD patients who are still actively drinking have shorter duodenal villi than healthy individuals without signs of epithelial damage, as well as increased intestinal permeability in some patients, particularly in the proximal small intestine, the primary site of alcohol absorption. Ethanol and its metabolites, such as acetaldehyde and acetate, are thought to influence malabsorption and intestinal barrier function, but their exact effects *in vivo* remain incompletely understood.[7]

Severe bleeding can be associated with hypoalbuminemia, either as a cause or as a consequence of the bleeding process itself. In our case, hypoalbuminemia most likely occurred as a result of gastrointestinal bleeding, in which gut loss mechanisms play a key role. In this condition, protein loss, including albumin, occurs through the inflamed or damaged gastrointestinal mucosa, resulting in albumin excretion exceeding the liver's production capacity. This ultimately contributes to decreased serum albumin levels during the bleeding episode.[20-22]

In terms of management, the patient required acute management focusing on hemodynamic stabilization, bleeding control, identification of the bleeding source, and management of potential infections due to the severe mixed GI bleeding. Pharmacological interventions, including proton pump inhibitors, antifungal agents, and mesalamine, were crucial for managing the identified GI pathologies. The drug selection was guided by the results of EGD and colonoscopy. Both examinations were thus crucial not only in the diagnostic process, but also in managing the pathologies identified. Long-term management includes complete cessation of alcohol consumption to prevent further mucosal damage and systemic complications, as further excessive ethanol consumption could worsen the symptoms in the future based on known pathophysiology. Additionally, nutritional support and monitoring for potential deficiencies are also important, considering the malabsorptive potential of alcohol-induced mucosal damage.

CONCLUSION

This case highlights the profound impact of chronic alcohol consumption on the GI tract, contributing to conditions such as erosive gastroduodenitis, esophagitis, and ulcerative colitis, even in young, otherwise healthy individuals. Alcohol-associated bowel disease manifests across a broad spectrum, ranging from upper GI bleeding to colon inflammation, requiring a comprehensive, multidisciplinary management approach. Understanding the role of alcohol in exacerbating pre-existing GI conditions, along with patient education about the importance of alcohol cessation, is essential to prevent recurrence and improve long-term outcomes.

CONFLICT OF INTEREST

The author(s) declare no conflicts of interest related to this manuscript.

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AUTHOR CONTRIBUTIONS

- Conceptualization: Agustien Bayu Ristanti, Muhammad Erlangga Putra Harimurti
- Methodology: Agustien Bayu Ristanti, Mohammad Amin Rais Perfernandi Ilham
- Data Collection: Agustien Bayu Ristanti, Agastjya Wisjnu Wardhana
- Data Analysis and Interpretation: Agastjya Wisjnu Wardhana, Agustien Bayu Ristanti
- Manuscript Drafting: Muhammad Erlangga Putra Harimurti, Mohammad Amin Rais Perfernandi Ilham
- Critical Revision of the Manuscript: All authors
- Final Approval of the Version to Be Published: All authors
- Accountability for All Aspects of the Work: All authors

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