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SEVERE ETHANOL-INDUCED CHEMICAL PROCTITIS WITH PROLONGED RECTAL BLEEDING: A CASE REPORT

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Abstract

Chemical proctitis is an uncommon colorectal injury caused by direct mucosal exposure to irritant or caustic substances. Rectal ethanol exposure is particularly rare and may cause severe inflammation, ulceration, necrosis, and hemorrhage. We present a 60-year-old woman with constipation who self-administered an unknown volume of approximately 50% homemade ethanol-based beverage (rakia) into the rectum using a syringe, believing that it would relieve constipation. Within several hours, she developed severe abdominal pain, tenesmus, and bright-red rectal bleeding and was admitted to a local hospital without endoscopic facilities. Her hemoglobin concentration at admission was 85 g/L, and she received two units of blood. She remained hospitalized for seven days without specific pharmacological treatment, while the bleeding gradually subsided and had ceased by day 7. After referral, a complete colonoscopy showed normal mucosa throughout the colon proximal to the rectum. The rectum demonstrated marked erythema and edema, loss of the normal vascular pattern, irregular superficial ulceration, and focal fibrinous or necrotic-appearing areas, without active bleeding. Histopathology showed lamina propria edema, lymphocytic and plasmacytic infiltration, macrophages, congested capillaries, and necrotic areas, supporting acute chemical mucosal injury. Follow-up sigmoidoscopy on day 14 showed normalization of the rectal mucosa. This case demonstrates that intrarectal exposure to high-concentration ethanol may produce severe, transfusion-requiring rectal bleeding despite subsequent rapid mucosal recovery. A directed history of rectal exposure

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and careful endoscopic evaluation are important for distinguishing chemical proctitis from ischemic, inflammatory, and infectious causes.

Keywords: *chemical proctitis; ethanol; rectal bleeding; colonoscopy; case report*

INTRODUCTION

Chemical proctitis and proctocolitis are uncommon inflammatory conditions resulting from direct mucosal exposure to irritant or caustic substances introduced into the rectum. The clinical presentation may include abdominal or rectal pain, tenesmus, diarrhea, and rectal bleeding, with severity ranging from mild, self-limited inflammation to extensive mucosal ulceration, necrosis, and significant hemorrhage (4-8). Because the clinical, endoscopic, and histopathological findings are often nonspecific, recognition of chemical exposure is essential for establishing the correct diagnosis.

Alcohol-induced colorectal injury is particularly rare, and the available literature consists predominantly of individual case reports and small case-based reviews (1–8). Reported presentations range from localized proctitis to pancolitis, and the endoscopic and histologic appearances may closely resemble those of ischemic colitis (4,5,8). A careful history regarding recent enemas, rectal cleansing procedures, accidental exposure, or self-administered substances is particularly important in patients presenting with unexplained acute proctitis.

The severity and distribution of injury appear to vary according to ethanol concentration, the administered volume, duration of mucosal contact, and proximal spread of the instilled solution. Published cases describe injury extending from the rectum to more proximal colonic segments (4,8). Most nonfatal cases have improved with conservative management, although severe systemic ethanol toxicity and fatal outcomes after rectal administration have also been reported (9).

We report a case of severe chemical proctitis following self-administration of approximately 50% homemade ethanol-based beverage (rakia) as an attempted home remedy for constipation. The case is notable for prolonged rectal bleeding with significant anemia requiring

blood transfusion. The injury was limited to the rectum, with complete endoscopic recovery within two weeks without specific treatment.

CASE PRESENTATION

A 60-year-old woman with constipation self-administered an unknown volume of approximately 50% homemade ethanol-based alcoholic beverage (rakia) into the rectum using a syringe. She used the alcohol as a home remedy because she believed that rectal administration would relieve her constipation.

Within several hours of exposure, she developed severe abdominal pain, tenesmus, and bright-red rectal bleeding. She presented to a local hospital where endoscopy was not available. Her hemoglobin concentration at admission was 85 g/L. Because of clinically significant bleeding and anemia, she received two units of blood.

She remained hospitalized for seven days. She was treated conservatively. The rectal bleeding stopped by day 7.

On day 7 after the ethanol exposure, she was referred for endoscopic evaluation. A complete colonoscopy was performed. The mucosa throughout the colon proximal to the rectum was normal. In the rectum, there was mucosal erythema and edema, loss of the normal vascular pattern, irregular superficial ulceration, focal fibrinous necrotic-appearing areas. No active bleeding was present at the time of colonoscopy (Figure 1).

Histopathologic examination of rectal biopsy specimens demonstrated edema of the lamina propria, lymphocytic and plasmacytic inflammatory infiltration, macrophages, congested capillaries, and necrotic areas, supporting acute chemical mucosal injury.

A follow-up sigmoidoscopy performed on day 14 after exposure showed normalization of the rectal mucosa. No further rectal bleeding was reported during this interval.

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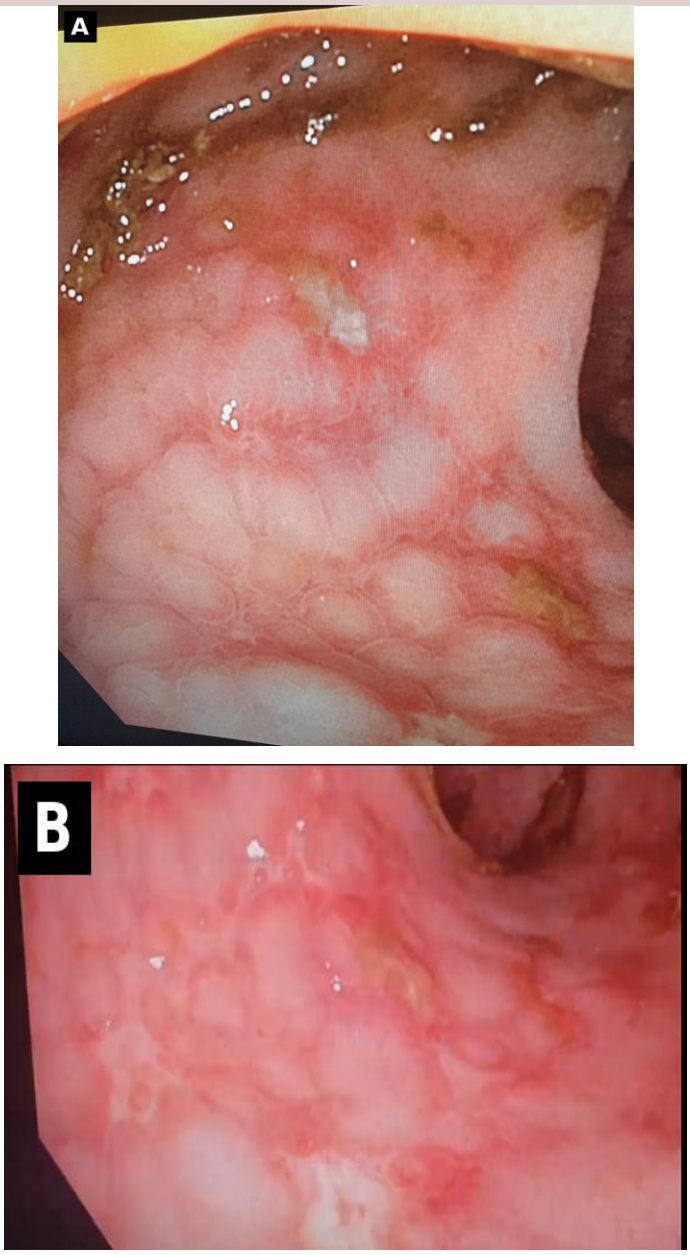


Figure 1. Colonoscopic appearance of the rectum on day 7 after intrarectal ethanol exposure. (A) Marked mucosal erythema, edema, and loss of the normal vascular pattern. (B) Irregular superficial ulceration with focal fibrinous/necrotic-appearing areas. No active bleeding was observed.

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DISCUSSION

Rectal administration of ethanol is an unusual but recognized cause of chemical proctitis or proctocolitis. Published reports have remained uncommon over several decades (1–10). This rarity can create a diagnostic challenge, particularly when patients do not have a history of rectal exposure.

The extent of injury is variable. Seki and Fukushima summarized cases in which damage ranged from the rectum alone to the ascending colon. In their review, larger instilled volumes were associated with more proximal involvement (8). Their patient had administered 1.8 L of 35% alcohol and developed extensive proctocolitis. In contrast, our patient had injury in the rectum despite exposure to an approximately 50% homemade alcoholic beverage. The administered volume in our patient is unknown, limiting direct comparison with previous reports. The present case was clinically severe because bleeding persisted for approximately one week, hemoglobin was 85 g/L at hospital admission, and two units of blood were required. No active bleeding was seen at colonoscopy on day 7, and the mucosa had normalized by day 14 without specific pharmacological treatment (4,5,8).

An important feature of this case is the reason for exposure. The patient used homemade rakia rectally as an attempted treatment for constipation rather than for intoxication. Because endoscopy was unavailable during the initial hospitalization, the colonoscopic images obtained on day 7 probably represent a healing phase rather than the peak mucosal injury.

The main limitations are the unknown volume of ethanol administered and absence of endoscopic documentation during the acute first days. The documented exposure, rectal bleeding requiring transfusion, findings limited to the rectum, and rapid recovery support the diagnosis.

Chemical proctitis is not specific to ethanol. Intrarectally administered corrosive or irritant compounds can produce toxic colitis, and the clinical presentation may include abdominal or rectal pain, tenesmus, diarrhea, and hematochezia. Endoscopic and histologic findings may be nonspecific (11).

Alcohol-related injury may cause erythema, edema, erosions, ulceration, and bleeding, with findings similar to ischemic colitis (8). Histology may show edema, hemorrhage, and inflammatory changes. In our patient, biopsy showed lamina propria edema, inflammatory cell infiltration, congested capillaries, and areas of necrosis.

The severity of mucosal injury may depend on ethanol concentration and volume. Published cases include high-concentration alcohol exposures with recovery under conservative management, while the proximal extent of injury appears to be influenced by the volume administered (5,8). Seki and Fukushima reviewed previous cases were 200 mL had been associated with injury reaching the descending colon, whereas larger volumes of approximately 1,000 mL or more were associated with more proximal disease (8). In our patient, the unknown volume is an important limitation, but confinement of disease to the rectum suggests limited proximal distribution of the instilled liquid.

The timing of endoscopy is another clinically relevant point. Our first endoscopic examination was performed seven days after exposure, when overt bleeding had already stopped. Therefore, the observed erythema, edema, superficial ulceration, and fibrinous/necrotic-appearing areas should be interpreted as residual injury during mucosal healing rather than necessarily representing the maximum acute damage. Complete normalization on day 14 documents rapid endoscopic recovery and supports the self-limited course reported in many nonfatal alcohol-enema cases (5,6,8).

There is no established specific pharmacological treatment for ethanol-induced chemical proctitis. Published cases have generally been managed supportively, with bowel rest, intravenous fluids, observation, and treatment of complications. Individual reports have used anti-inflammatory therapy, but the available case-based literature does not establish a standard drug regimen (6,8). In our patient, no specific therapy for proctitis was given. The clinically important intervention was transfusion of two units of blood for significant blood-loss anemia, followed by spontaneous cessation of bleeding and complete endoscopic recovery.

The differential diagnosis at presentation includes ischemic proctocolitis, inflammatory bowel disease, infectious proctitis, and other forms of chemical injury. In this case, the immediate association with intrarectal rakia, localization of abnormalities exclusively to the

rectum on complete colonoscopy, compatible nonspecific histopathologic injury, and rapid resolution without disease-specific therapy strongly support an ethanol-induced chemical mechanism.

CONCLUSION

Intrarectal ethanol can cause severe chemical proctitis with mucosal injury and prolonged rectal bleeding. In patients with unexplained acute proctitis, it is important to ask about enemas, home remedies, and other rectally administered substances. In our patient, conservative treatment was followed by rapid and complete mucosal recovery.

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