CLINICAL VIGNETTE

Hematemesis after Binge Drinking: Acute Changes on Serial EGDs

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Introduction

Excessive alcohol consumption is a leading global cause of morbidity and mortality. We present a 47-year-old female with alcohol use disorder (AUD) and alcohol-associated liver disease with decompensated cirrhosis and a MELD score of 29 who had undergone outpatient esophagogastroduodenoscopy (EGD) earlier in the day followed by a binge drinking episode at home leading to hematemesis necessitating hospitalization and repeat EGD which showed significant acute changes. This natural experiment illustrated acute toxic effects of alcohol on the GI tract as seen EGDs and the importance of binge drinking as a modifiable risk factor contributing to acute GI bleeding.

Case

Six weeks prior to presentation, our patient underwent an EGD revealing grade II esophageal varices with red whale markings indicative of potential for and/or recent bleeding and mild portal hypertensive gastropathy. Her varies were banded, and she was counseled to abstain from alcohol. On the day prior to admission, follow-up EGD as an outpatient at 1 PM, revealed grade I esophageal varices and moderate portal hypertensive gastropathy. A few antral and duodenal erosions were also observed, but there was no evidence of recent bleeding (Figure 1). She returned home in the afternoon and proceeded to consume alcohol, and later in the evening, developed hematemesis and presented to our emergency department.

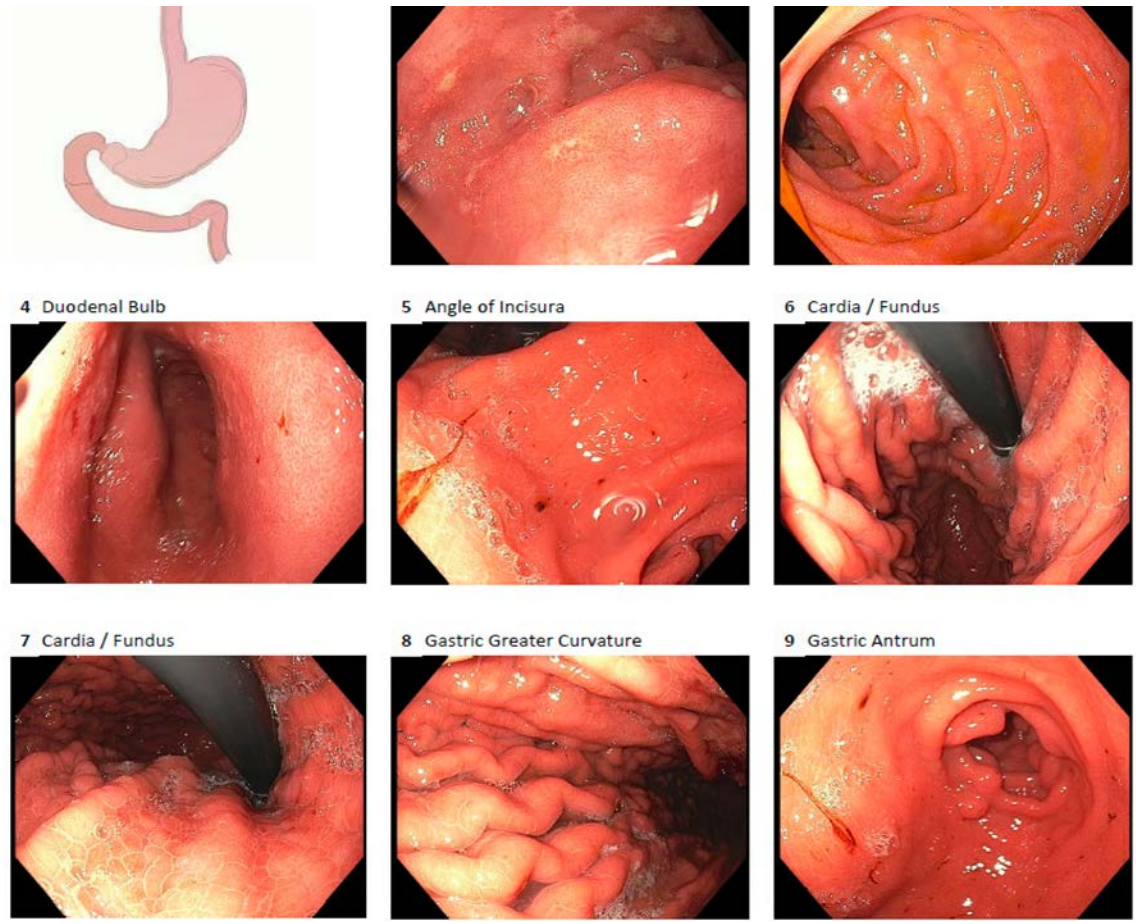

Figure 1: Initial EGD at 1 PM, the day prior to admission

She arrived at emergency room around 2:30 AM reporting an hour of gross hematemesis. Initial vital signs were T36.5°C, HR of 91 beats per minute, BP of 117/71 mmHg, RR of 20/min, and room air O2 sat of 94%. Labs included hemoglobin of 11.7 g/dL, platelet count of 60,000/µL, blood alcohol concentration (BAC) of 290 mg/dL, and INR of 1.8. She was admitted to the general medicine service, started on proton pump inhibitor (PPI) and octreotide continuous IV infusions, and the gastroenterology service was consulted for endoscopic evaluation. Her hemodynamics were stable, and EGD was performed at 10 AM revealing a mix of fresh and old blood within the GI tract, non-bleeding residual esophageal varices, moderate portal hypertensive gastropathy, and two small oozing ulcers in the duodenal bulb and antrum. There was also fundic ulcer with an actively oozing vessel for which hemoclips were applied to achieve hemostasis (see Figure 2).

With pharmacologic and mechanical treatment as above, gross hematemesis stopped, and her hgb stabilized around 8-9 g/dL. Over the next three days, she was continued on PPI and octreotide infusions for 72 hours, followed by a maintenance regimen of BID PPI. Alcohol cessation counseling was again emphasized, and alcohol cessation resources were provided by social work and addiction medicine, with close outpatient follow-up scheduled.

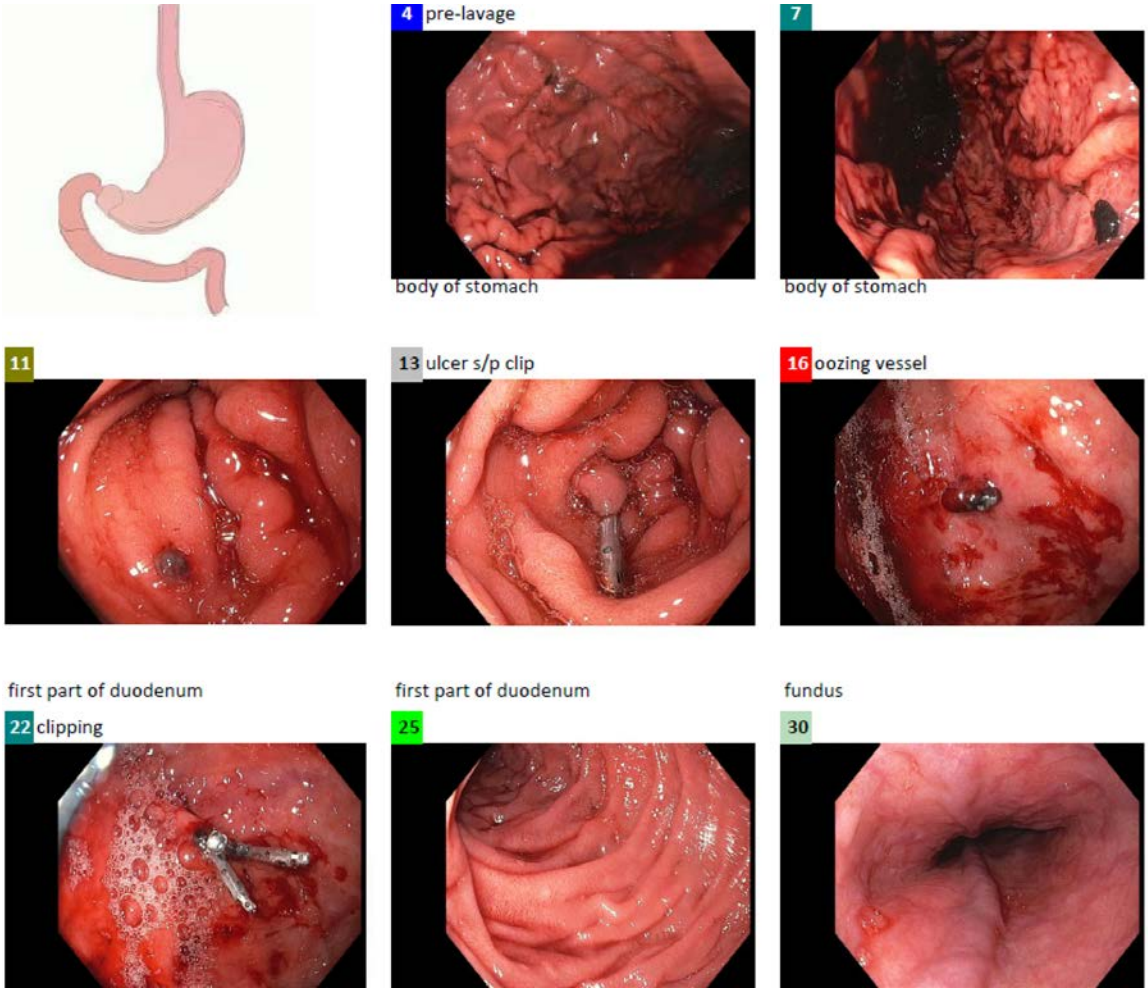

Figure 2: Repeat EGD on day of admission at 10 AM (approximately 21 hours after EGD in Figure 1, with binge-drinking episode in interim) showing acute ulcerations with bleeding.

Discussion

Excessive alcohol consumption is a leading global cause of preventable morbidity and mortality. Binge drinking in particular is responsible for a substantial portion of alcohol-attributable disease and deaths.[1] Binge drinking is defined as the consumption of five or more drinks during one occasion. It is responsible for over half of the estimated 80,000 average annual deaths and three quarters of the $223.5 billion in yearly economic costs due to excessive alcohol consumption in the U.S.[2,3] Although this patient had a history of AUD and cirrhosis, she initially denied recent alcohol use, and only with further prompting, revealed that while she had maintained sobriety for around ten years, she had relapsed in the two weeks

leading to admission and had consumed an unknown 'large amount' of alcohol the evening after her outpatient EGD. Her BAC at presentation was 290 mg/dL, well above the legal driving limit of 80 mg/dL consistent with binge drinking.[4] Long term and population based alcohol consumption elevates risk for major upper gastrointestinal bleeds in non-predisposed individuals with a clear dose-dependent effect.[5] In the short term and at the individual level, alcohol has a direct, dose-dependent toxic effect on the gastric and duodenal mucosa, disrupting the tight junctions between epithelial cells, making the mucosa more permeable and vulnerable to injury, and ultimately leading to inflammation, edema, and possible hemorrhage as in our case.[6-8] Our patient highlights why binge drinking in particular is dangerous and should be strongly discouraged.

Conclusion

Alcohol consumption may lead to significant detrimental health effects. An exemplified here, binge drinking has high risk for gastric bleeding due to acute gastric epithelial injury. This case served as a natural experiment with serial EGDs done just prior to and just after an acute binge drinking episode in a patient with AUD. EGD demonstrates the acute, toxic effects of alcohol on the stomach mucosa due to binge drinking. Inquiring and counseling about alcohol use habits should continue to prevent future episodes.

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