

The Silent Danger: Examining the Escalating Issue of Alcohol-Related Premature End Organ Damage in Young Adults - A Case Study

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ABSTRACT/INTRODUCTION

Alcohol abuse has long been acknowledged as a pressing public health issue. Recent data points to a worrying trend of spike in excessive alcohol consumption amongst young adults who as a result are prone to suffer its harmful effects more frequently.^[1] Severe alcohol addiction is accounted to around 4% of fatalities globally by disrupting physiology and affecting multiple organ systems.^[2] Here we present one such case with a trifecta of pancreatitis, hepatic cirrhosis, and hypertriglyceridemia, in a male who is only 21 years old with no previous medical history. This case report intends to draw attention to the notably increase in growth of end-organ failures among people in this age range. Therefore, recognizing the gravity of this issue, it is crucial to prioritize prevention.

Keywords: Alcohol; Young Adults

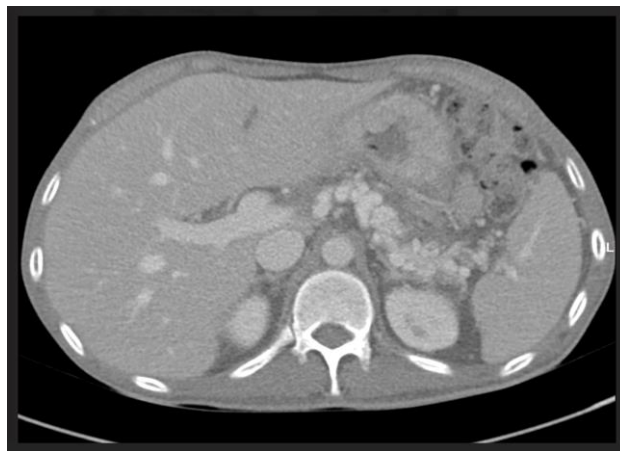
DESCRIPTION

A 21-year-old male with no past medical history and care gaps, presented with complaints of worsening epigastric pain, radiating to his back for a duration of two days. He admitted to drinking 10-12 cans of beer, 3-4 days a week but denied tobacco or illicit drug use. He had no history of recent travel or OTC medication consumption. His family history was also grossly insignificant.

In the ED, his vitals were significant for a HR- 97, BP-116/82, RR- 20, afebrile and saturating 98% on room air. The patient looked malnourished, and the abdominal examination was significant for tenderness in the epigastric area, with no rigidity, rebound tenderness, or distention. No discoloration or erythema was noticed on inspection

and bowel sounds were heard normal in all four quadrants. His labs were significant for a WBC count of 2.4 K/mcl, Hb- 11.2mg/dl, AST-124 U/L, ALT- 221 U/L, ALP-208 U/L, Creatinine- 1.0, Na- 140 and K-5.4 mmol/L, AG- 21mEq/L, Blood glucose-310mg/dl. VBG showed a pH of 7.1, pCO₂- 29 mmHg, pO₂-171 mmHg, HCO₃- 10.3 mmol/L, and lactate-0.8 mmol/L. Triglycerides were elevated to 512 mg/dL, Lipase 10 unit/L, HbA1c- 12.1%, UA positive for ketones and Elevated BHB. Hence, he was admitted to the ICU for further evaluation and management of DKA and was started on an insulin drip as per DKA protocol and symptomatically managed. He was also placed on CIWA protocol.

An extensive workup for DKA and elevated liver enzymes were pursued. Serology was negative for viral hepatitis A, B, and C. CT abdomen was performed which showed chronic calcific pancreatitis and features of cirrhosis with portal hypertension.(Figure 1)



Ferritin level was checked to rule out hemochromatosis and he was also worked up for autoimmune disorders which were all unremarkable. Anti GAD ab was also negative.

Patient's DKA and pain abdomen resolved, and condition improved. He was started on pancreatic enzymes and insulin and was discharged to alcohol Rehabilitation along with referral to Hepatologist and Endocrinologist.

DISCUSSION

Alcohol misuse has been linked to an increased risk of pancreatitis and liver cirrhosis. For young people, early-onset pancreatitis can have serious repercussions. Repeated pancreatitis attacks can develop into chronic pancreatitis, which is marked by ongoing inflammation and permanent pancreatic damage. Impairing symptoms, such as persistent pain, malabsorption, diabetes, and a higher risk of pancreatic cancer, can be brought on by chronic pancreatitis.

Cirrhosis, on the other hand, can lead to insulin resistance and impaired glucose utilization, which can also contribute to hyperglycemia and DKA. Cirrhosis of the liver in young adults is frequently brought on by the hepatitis B and C viruses (HBV, HCV), non-alcoholic fatty liver disease (NAFLD), autoimmune hepatitis, primary biliary cholangitis, and primary sclerosing cholangitis (PSC).^[3] However, we try to enhance that with growing alcohol consumption among young adults and adolescents, alcoholic cirrhosis should not be missed.

This case also signifies one such young patient with only medical history of alcohol abuse was found to have chronic calcific pancreatitis and Liver Cirrhosis indicating that he most likely developed pancreatogenic DM which is also known as Type 3c DM.^[4] and alcoholic cirrhosis leading to DKA.

CONCLUSION

Underage Drinking is a significant public health problem in the US. Excessive drinking is responsible for more than 3900 deaths and 225000 years of potential life lost among people under the age of 21 each year.^[5] A comprehensive strategy is thus required to address the surge in alcohol consumption among young adults and the related health effects. Included in this should be education and prevention.^[6] Regular alcohol screening in adolescent pediatric and primary care visits enables early detection of those who are at risk and prompt intervention through counseling, treatment, and rehabilitation programs.^[7,8] Also by improving accessibility to specialized medical care, such as addiction treatment, mental health counseling, and rehabilitation programs, to offer all-encompassing care to people impacted by alcohol abuse. Implementing stricter policies and regulations on the sale and marketing of alcohol to minimize accessibility to underage individuals can also help reduce alcohol-related harm.^[9]

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