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Alcohol Binge Drinking–Induced ST-Segment-Elevation Acute Coronary Syndrome

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Abstract

We present the case of a 26-year-old man, without any apparent cardiovascular risk factors, who experienced an ST-segment-elevation acute coronary syndrome after binge drinking high-proof alcohol, which was successfully managed with primary percutaneous coronary intervention and comprehensive, guideline-directed medical therapy.

Keywords

alcohol, binge drinking, acute coronary syndromes, ST-segment-elevation, myocardial infarction

Key Clinical Message: The clinician should be aware that alcohol binge drinking can precipitate an acute coronary syndrome in the absence of conventional cardiovascular risk factors.

Introduction

The National Institute on Alcohol Abuse and Alcoholism (NIAAA) defines binge drinking as a pattern of drinking alcohol that leads to a blood alcohol concentration of 0.08% within 2 hours.[1] This practice is negatively associated with deleterious cardiovascular effects.[1] It has also been demonstrated that binge drinking has been associated with an increased risk of myocardial infarction (MI) in the geriatric subpopulation.[2,3]

We present the case of a 26-year-old man, without any apparent cardiovascular risk factors, who experienced an ST-segment-elevation acute coronary syndrome (STE-ACS) after binge drinking high-proof alcohol, which was successfully managed with primary percutaneous coronary intervention (PPCI) and comprehensive, guideline-directed medical therapy (CGDMT).

Case Report

A 26-year-old South Asian man with no significant medical history presented to the emergency room with typical chest pain that awakened him from sleep. His social history was significant for consuming 15 "drinks" of high-proof alcohol (75% volume) 2 hours prior during a 3-hour window. He did not report tobacco or any illicit, recreational drug use such as cannabis or cocaine.

His vital signs on presentation revealed a blood pressure of 158/89 mm Hg, heart rate of 111 beats per minute, and regular, with pulse oximetry 100% on ambient air, temperature of 36°C, and body mass index of 23.2 kg/m^2. On physical examination, he was alert and oriented without any neurological deficits. His heart sounds were normal, with vesicular breath sounds on auscultation of the lung fields. There was no peripheral edema.

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His laboratory investigations revealed a mild leukocytosis of 12.3 $\times$ 10^3 /μL (normal range: 4-10 $\times$ 10^3/μL) with normal hemoglobin and platelet count. His coronavirus 2019 polymerase chain reaction (COVID-19 PCR) test was negative. Renal function tests and coagulation studies were normal. His hepatic panel revealed an elevated aspartate aminotransferase of 54 U/L (normal range: 5-45 U/L) and alanine aminotransferase of 64 U/L (5-50 U/L). A 12-lead electrocardiogram indicated sinus rhythm and 5-millimeter ST-segment-elevation in leads V_2 to V_6, with reciprocal ST-segment-depression in II, III, and aVF (Image 1).

He subsequently underwent PPCI. Coronary angiography revealed thrombotic occlusion of the proximal left anterior descending (LAD) and first diagonal coronary arteries with Thrombolysis in Myocardial Infarction (TIMI) flow 0 and TIMI thrombus grade 6 (Image 2A and B). Initial aspiration thrombectomy was performed with "modified marination" (intracoronary cocktail of adenosine, nitroglycerin, nicardipine) prior to provisional drug-eluting stent (DES; Resolute Onyx, Medtronic plc, Minneapolis, Minnesota, United States) implantation with a good angiographic result (TIMI 3 antegrade flow) and no complications (Image 2C and D). The patient was then transferred to the cardiac care unit, where he was commenced on the cardiovascular medications of aspirin, ticagrelor, apixaban, valsartan-sacubitril, carvedilol, eplerenone, empagliflozin, rosuvastatin, ivabradine, and colchicine. His low-density lipoprotein level, glycosylated hemoglobin, and high-sensitivity C-reactive protein were normal. A urine drug screen was unrevealing. A 2-dimensional transthoracic echocardiogram revealed mild anterior hypokinesis with an estimated left ventricular ejection fraction of 40% to 45% without any mechanical complications, apical mural thrombus, or pericardial effusion. The remainder of his subsequent hospitalization was uneventful, and the patient was eventually discharged with outpatient follow-up in 1 week, and his medication regimen was de-escalated after 1 month.

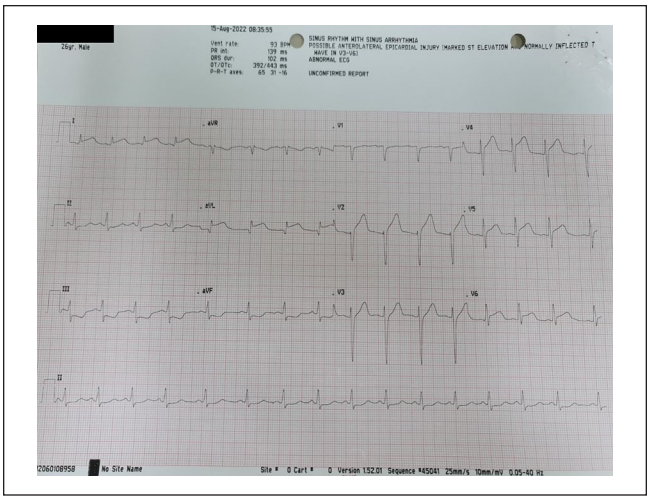

Image 1. The patient's 12-lead electrocardiogram indicated sinus rhythm and 5-millimeter ST-segment-elevation in leads V_2 to V_6, with reciprocal ST-segment-depression in II, III, and aVF.

Discussion

In 1996, the American Heart Association recommended that moderate consumption of alcohol, defined as 1 to 2 drinks per day, may confer cardioprotective effects. Generally, a standard drink is defined as (1) a 12-ounce bottle of beer, (2) a 4-ounce glass of wine, or (3) a 1½-ounce shot of spirits. In middle-aged and elderly adults, mortality rates were lower in those who consumed 1 to 2 drinks per day than those without consumption.[4] Further large-scale epidemiological studies have reinforced that moderate alcohol consumption is associated with a decreased risk of major adverse cardiovascular events (MACEs).[5-8] However, some studies demonstrate increased mortality for those with ≥ 3 drinks per day which was predominantly attributed to cerebrovascular events (CVEs) and heart failure but also non-cardiovascular diseases such as pancreatitis, cirrhosis, cancers, and suicide.[4] While moderate consumption of alcohol is cardioprotective, the pattern of drinking is impactful. Binge drinking has been associated with hypertension, atrial fibrillation (AF), CVE, and increased mortality.[9,10]

The INTERHEART study demonstrated an increased risk of MI with heavy episodic drinking in elderly adults.[3] Although this association is commonly seen in this subpopulation, our case is exceedingly unusual, as we present a young South Asian male patient (26-year-old) presenting with STE-ACS with a high thrombus burden. To the authors' knowledge, there has only been one other documented report of a similar case in a young male patient.[11] Additional studies show the incidence of acute coronary syndrome (ACS) is increased within 1 hour of alcohol consumption among people who are not daily drinkers, and this signal is more robust in those who consume liquor.[12] In Trinidad and Tobago, there are alcohol users in 64% of households, with 57% reporting heavy episodic drinking. Our patient reported binge drinking 15 units of "white" rum (75% alcohol by volume) during a 3-hour window 2 hours prior to presentation.[13]

In addition to increasing the risk of MACE, binge drinking has been associated with a higher mortality post-ACS.[14] A Finnish study reported that the risk of death from fatal ACS was > 6 times greater in men who consumed ≥ 6 drinks per session compared with those who consumed ≤ 3.[10] In another study, there was an odds ratio of approximately 2.6 for MACE in men who consumed ≥ 9 drinks daily in 1 to 2 days compared with men who consumed the same quantity of alcohol during an entire week.[15] Jabbari demonstrated that patients with STE-ACS who consumed ≥ 8 drinks per week were at an increased risk of developing ventricular fibrillation (VF) compared with non-drinkers.[16] Our patient experienced peri-procedural accelerated idioventricular rhythm during reperfusion, with no AF or VF observed on telemetry during his hospitalization.

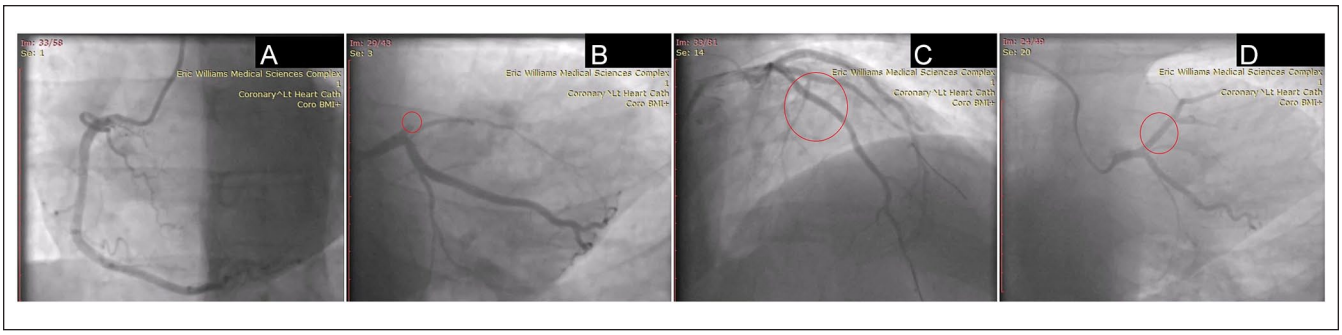

Image 2. The patient's primary percutaneous coronary intervention (PPCI). (A) Left anterior oblique cineangiography of the right coronary artery was angiographically normal. (B) Left anterior oblique caudal cineangiography revealing a thrombotic occlusion of the proximal left anterior descending (LAD) and first diagonal coronary arteries with Thrombolysis in Myocardial Infarction (TIMI) flow 0 and TIMI thrombus grade 6 (encircled in red). (C) Left anterior oblique cranial cineangiography with initial aspiration thrombectomy was performed with "modified marination" (intracoronary cocktail of adenosine, nitroglycerin, nicardipine; encircled in red). (D) Left anterior oblique caudal cineangiography demonstrating provisional drug-eluting stent (DES; Resolute Onyx, Medtronic plc, Minneapolis, Minnesota, United States) implantation with a good angiographic result (TIMI 3 antegrade flow) and no complications (encircled in red).

Acute coronary syndrome may occur after binge drinking due to maladaptive mechanistic effects in fibrinolysis, platelet hyperreactivity, endothelial dysfunction, and oxidative stress, which may persist for up to 24 hours. There are increased endogenous levels of plasminogen activator inhibitor-1 (PAI-1) antigen, PAI-1 activity, and decreased tissue plasminogen activator (tPA) activity.[17] Alcohol also accentuates platelet aggregation via thromboxane or indirectly via hypertension, tachycardia, and resultant shear stress forces.[18,19] Repeated chronic binge drinking is associated with an accentuated oxidative response, which correlates to increased blood pressure and angiotensin II levels.[19]

Our patient presented with STE-ACS 2 hours after an episode of alcohol binge drinking. This alludes to a robust temporal relationship without any incipient cardiovascular risk factors.

Conclusion

We present the case of a 26-year-old man, without any apparent cardiovascular risk factors, who experienced an STE-ACS after binge drinking high-proof alcohol, which was successfully managed with PPCI and CGDMT. The clinician should be aware that alcohol binge drinking can precipitate ACS in the absence of conventional cardiovascular risk factors.

Availability of Data and Materials

All available data can be obtained by contacting the corresponding author.

Author Contributions

All authors contributed equally to writing the manuscript, and all authors read and approved the final manuscript.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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Ethics Approval

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed Consent

The patient has provided informed consent to have the details and images of his case published, and institutional approval was not required for publication.

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