Challenges in Clinical Electrocardiography

# Recurrent Angina After Alcohol Consumption

Caifeng Fan, MD; Hao Wang, MD; Yunfei Gu, MD

## Case Presentation

A patient in their 50s was admitted with recurrent abdominal pain and syncope after drinking alcohol. They had this problem for 2 years but experienced worsening on admission. The patient was a smoker and had alcohol overconsumption for about 20 years. The average alcohol intake was 250 g/wk. They had hypertension diagnosed 3 months prior to admission. On admission, high-sensitivity troponin I was 0.01 ng/mL (to convert to µg/L, multiply by 1) and serum potassium was 4.6 mEq/L (to convert to mmol/L, multiply by 1), both of which were in normal range. The patient's Holter was shown in the **Figure**.

**Questions:** What are the key findings in the electrocardiograms (ECGs)? What would you do next?

## Interpretation

The Figure, A, shows a high-degree atrioventricular block and premature ventricular contractions. The ST segments in leads II, III and aVF are 4 to 6 mm elevated, while the ST segments in leads $V_2$ through $V_6$ are substantially depressed.

The ECG in the Figure, B, shows a sinus rhythm with short bursts of ventricular tachycardia. It shows also widespread ST-segment elevations (4-15 mm) in leads II, III, aVF, and $V_2$ through $V_6$.

## Clinical Course

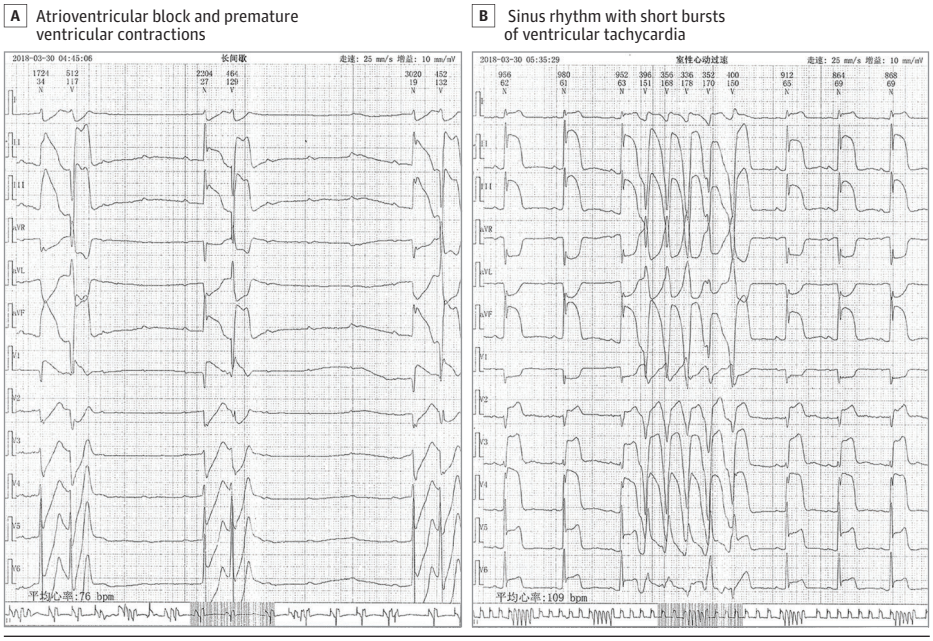
The patient was originally admitted to the department of gastroenterology due to upper abdominal pain, dizziness, and syncope. The symptoms usually occurred 6 to 20 hours after alcohol ingestion and lasted 5 to 10 minutes. Syncope occurred only after a large amount of alcohol intake. The ECGs (Figure) were recorded during 1 of the episodes. Because of these ECG findings, the patient was transferred to the cardiology department. He underwent coronary angiography that showed only 10% stenosis in the proximal left anterior descending artery (LAD) and right coronary artery (RCA). No stenosis was shown in the left main and circumflex coronary artery. Given the clinical course and ECG changes, the result of coronary angiography raised the suspicion of alcohol-induced Prinzmetal variant angina.[1,2] The patient was treated with diltiazem, a calcium antagonist, which could relieve and shorten the duration of abdominal pain during hospitalization.

In addition to diltiazem, we added atorvastatin, aspirin, and clopidogrel. Importantly, the patient stopped smoking and consuming alcohol. Since discharge, the patient had no more abdominal pain and syncope during the follow-up of 2 years.

## Discussion

Prinzmetal angina is a type of variant angina with transient ST-segment elevation caused by the spasm of coronary arteries.[1] The variant angina, different from the classic one which is often provoked by exertion, occurs mostly at rest and can be precipitated by cold, coffee, emotional agitation, alcohol, and other factors. Takizawa et al[3] found that 26.8% of patients with variant angina pectoris have alcohol ingestion. A recent large-scale trial[4] has also shown that alcohol consumption is a risk factor for coronary artery spasm. Sato et al[5] reported a case of such variant angina, where chest pain oc-

Figure. Electrocardiogram (ECG) Findings From Holter Recording




A, The ECG showed high-degree atrioventricular block and premature ventricular contractions. The ST segments in leads II, III and aVF are 4 to 6 mm elevated, while the ST segments in leads $V_2$ through $V_6$ are substantially depressed. B, The ECG has a sinus rhythm with short bursts of ventricular tachycardia. It shows also widespread ST-segment elevations (4-15 mm) in leads II, III, aVF, and $V_2$ through $V_6$.

curred each time 10 to 11 hours after alcohol ingestion, when blood concentration of alcohol had already decreased to 0, indicating a withdrawal from an acute exposure of alcohol as a possible trigger to coronary spasm. Although the mechanism of such variant angina is unclear, alcohol-induced vascular endothelial dysfunction is associated with coronary spasm.[3-5] This includes alcohol-induced elevation in plasma endothelin-1 and decrease in plasma prostaglandin F1 alpha and cyclic guanosine monophosphate.[3-5] The present patient might have experienced endothelial dysfunction due to long-term smoking and alcohol use. Moreover, the high prevalence of aldehyde dehydrogenase 2 deficiency in East Asian populations may predispose patients to alcohol-induced vasospasm.[6]

The ECG changes of variant angina depend on the duration of vessel spasm and which coronary arteries are affected. Longer spasms of the RCA can lead to inferior myocardial infarction and atrioventricular block while vessel spasm in the LAD may introduce anterior myocardial infarction. However, spasm of any coronary artery can lead to malignant ventricular arrhythmias.[7] The present patient experienced 1 episode with transient ST-segment elevation in the inferior leads and concomitant high-degree atrioventricular block, suggesting the involvement of RCA at that moment. The other episode with widespread ST-segment elevation in the anterior and inferior leads and simultaneously one burst of nonsustained ventricular tachycardia perhaps involved LAD arteries.

The central treatment of alcohol-induced Prinzmetal variant angina is alcohol cessation. If Prinzmetal variant angina has introduced severe outcomes of cardiovascular events, eg, ventricular tachycardia and complete atrioventricular block as in the present case, anti-ischemia and anticoronary spasm therapy can be considered. We have therefore added clopidogrel and aspirin as known dual antiplatelet therapy in the present treatment.

## Take-home Points

- Prinzmetal variant angina should be considered a differential diagnosis if the chest pain begins within 24 hours after alcohol consumption.
- Vasospasm can involve one or more coronary arteries, causing variant ECG changes and cardiac arrhythmias.
- Most patients with alcohol-induced variant angina have good prognoses. The central treatment is alcohol cessation.


## ARTICLE INFORMATION

**Author Affiliations:** Department of Cardiology, Luoyang Central Hospital Affiliated to Zhengzhou University, Luoyang, Henan Province, China.



**Corresponding Author:** Yunfei Gu, MD, Department of Cardiology, Luoyang Central Hospital Affiliated to Zhengzhou University, 288 Zhongzhouzhong Rd, Henan Province, Luoyang 471000, China (yunfeigu@126.com).



**Published Online:** January 3, 2023. doi:10.1001/jamainternmed.2022.5411

**Conflict of Interest Disclosures:** None reported.

**Additional Contributions:** The authors thank all the staff members in our institution. The authors thank Tong Liu, MD, Second Hospital of Tianjin Medical University. Guo-Liang Li, MD, The First Affiliated Hospital of Xi'an Jiaotong University and Yang-Zhen Shao, MD, Sahlgrenska University Hospital, Gothenburg, Sweden. for their helpful comments. These individuals were not compensated for their contributions.


## REFERENCES


1. Prinzmetal M, Kennamer R, Merliss R, Wada T, Bor N. Angina pectoris I: a variant form of angina pectoris; preliminary report. *Am J Med*. 1959;27:375-388. doi:10.1016/0002-9343(59)90003-8

2. JCS Joint Working Group. Guidelines for diagnosis and treatment of patients with vasospastic angina (coronary spastic angina) (JCS 2013). *Circ J*. 2014;78(11):2779-2801. doi:10.1253/circj.CJ-66-0098

3. Takizawa A, Yasue H, Omote S, et al. Variant angina induced by alcohol ingestion. *Am Heart J*. 1984;107(1):25-27. doi:10.1016/0002-8703(84)90128-5

4. Sohn SM, Choi BG, Choi SY, et al. Impact of alcohol drinking on acetylcholine-induced coronary artery spasm in Korean populations. *Atherosclerosis*. 2018;268:163-169. doi:10.1016/j.atherosclerosis.2017.11.032

5. Sato A, Taneichi Y, Sekine I, et al. Prinzmetal's variant angina induced only by alcohol ingestion. *Clin Cardiol*. 1981;4(4):193-195. doi:10.1002/clc.4960040408

6. Seki T, Okayama H, Isoyama S, et al. The role of alcohol dehydrogenase 2 and aldehyde dehydrogenase 2 genotypes in alcohol-induced vasospastic angina. *Tohoku J Exp Med*. 1999;187(4):311-322. doi:10.1620/tjem.187.311

7. Nishizaki M, Arita M, Sakurada H, et al. Polymorphic ventricular tachycardia in patients with vasospastic angina–clinical and electrocardiographic characteristics and long-term outcome. *Jpn Circ J*. 2001;65(6):519-525. doi:10.1253/jcj.65.519