

When Celebration Becomes Arrhythmia: Recurrence of Alcohol-Induced Paroxysmal Atrial Fibrillation in a Young Adult

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Abstract: Atrial fibrillation is the most common sustained arrhythmia and may occur in young individuals without structural heart disease when triggered by reversible factors. Alcohol consumption is a well-established precipitant, particularly in the context of binge drinking, known as Holiday Heart Syndrome. We report the case of a 43-year-old man without cardiovascular risk factors who presented to the emergency department with palpitations lasting approximately 48 hours following acute alcohol binge intake during New Year celebrations. Electrocardiography confirmed atrial fibrillation with controlled ventricular response. Transthoracic echocardiography demonstrated normal cardiac chambers and preserved biventricular systolic function. Laboratory evaluation, including thyroid function and serum electrolytes, was unremarkable. Rate control with bisoprolol was initiated, followed by spontaneous conversion to sinus rhythm within 48 hours. Given a CHA₂DS₂-VA score of 0, no anticoagulation was indicated. This case highlights alcohol-induced atrial fibrillation as a reversible arrhythmogenic condition and reinforces the importance of lifestyle modification in preventing recurrence.

Keywords: Atrial fibrillation; Alcohol Consumption; Holiday Heart Syndrome.

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1. Introduction

Atrial fibrillation (AF) is the most prevalent sustained arrhythmia in clinical practice and constitutes a major public health challenge due to its high prevalence, prognostic impact, and increasing socioeconomic burden. Although the incidence increases with age and the presence of cardiovascular risk factors, AF can also occur in young individuals without structural heart disease, frequently triggered by reversible factors [1, 2]. Among these factors, alcohol consumption is one of the environmental triggers most consistently associated with AF, with a well-established dose-dependent relationship [2, 3]. However, episodic excessive consumption presents greater arrhythmogenic potential, which can trigger AF episodes in individuals without previously known structural heart disease [3, 4].

The term Holiday Heart Syndrome (HHS) was introduced by Ettinger et al., in 1978, to describe cardiac arrhythmias, predominantly AF, triggered after excessive alcohol intake during festive periods in individuals without apparent heart disease [5]. In recent decades, the understanding of the pathophysiology of HHS has evolved significantly. Experimental and clinical evidence suggests that alcohol-induced arrhythmogenesis results from the interaction between autonomic imbalance, alterations in intracellular calcium handling, oxidative stress, transient myocardial inflammation, and electrophysiological effects mediated by acetaldehyde, the main metabolite of ethanol [6]. Furthermore, recent studies indicate that many AF episodes occur during the hangover phase or acute alcohol withdrawal, characterized by rebound sympathetic hyperactivity, and not necessarily during the peak of alcohol intoxication [7,8].

The diagnosis is primarily based on the temporal association with alcohol exposure, the electrocardiographic documentation of arrhythmia, and the exclusion of secondary causes and clinically relevant structural heart disease. Contemporary guidelines for the management of AF emphasize the identification and correction of modifiable risk factors, including the reduction or abstinence of alcohol consumption, as a fundamental component of recurrence prevention and the integrated approach to the patient with AF [9,10].

Despite HHS being a recognized clinical entity for several decades, the growing understanding of the mechanisms involved in alcohol-induced arrhythmogenesis and the evolution of contemporary recommendations for the management of AF justify the reassessment of this syndrome from a current perspective. In this context, we present the case of a young adult who developed paroxysmal AF after episodic excessive alcohol consumption, discussing the most recent pathophysiological mechanisms, the diagnostic approach, and the therapeutic implications considering contemporary evidence.

2. Case Report

A 43-year-old black man, without cardiovascular risk factors, relevant personal history, history of alcohol use disorder or illicit drug use, presented to the emergency department with persistent palpitations for approximately 48 hours. The patient reported a history of a similar episode during the New Year's Eve celebrations from 2023 to 2024, triggered by excessive alcohol consumption. On that occasion, he was seen at the Emergency department, where a diagnosis of atrial fibrillation was confirmed. The arrhythmia was self-limiting, with spontaneous return to sinus rhythm after approximately 48 hours.

Between December 31, 2025 and January 3, 2026, he ingested approximately 1.5 L of gin/vodka and an unspecified amount of beer as part of the New Year celebrations. He stated it was an isolated episode of excessive alcohol consumption, denying habitual intake or alcohol dependence. The symptoms began on January 4, initially attributed by the patient to a hangover, but persisted continuously, prompting him to seek medical care on January 6. He denied chest pain, dyspnea, syncope, fever, symptoms of recent infection, or prior use of illicit drugs.

On admission, found hemodynamically stable, with blood pressure of 129/82 mmHg, heart rate of 90 bpm, weight of 74 kg, height of 1.75 m and body mass index of 24.2 kg/m². The cardiovascular examination revealed an irregular rhythm, without murmurs or other pathological findings, with the remainder of the physical exam being normal. The 12-lead electrocardiogram demonstrated AF with controlled ventricular response (95 bpm) and an early repolarization pattern (Figure 1). The transthoracic echocardiogram showed cardiac cavities of normal dimensions, including a left atrial volume index of 24 mL/m² and a right atrial volume index of 21 mL/m², preserved biventricular systolic function, left ventricular ejection fraction of 68% and absence of structurally significant valvular heart disease.

Laboratory investigation revealed no relevant changes, including hemoglobin of 12.4 g/dL, creatinine of 0.9 mg/dL, serum potassium of 4.1 mEq/L, serum magnesium of 1.9 mg/dL, TSH of 1.8 mUI/L and free thyroxine (free T4) of 1.2 ng/dL. Toxicological screening was not performed due to the unavailability of this test at the institution. Thromboembolic

risk was stratified using the CHA₂DS₂-VA score, according to contemporary recommendations, resulting in 0, which is why there was no indication for anticoagulation. An initial strategy of ventricular rate control with Bisoprolol 2,5 mg/day was chosen. The patient presented spontaneous conversion to sinus rhythm after approximately 48 hours (Figure 2), allowing for the suspension of the beta-blocker before hospital discharge.

Figure 1. Electrocardiogram shows atrial fibrillation, HR=95bpm.

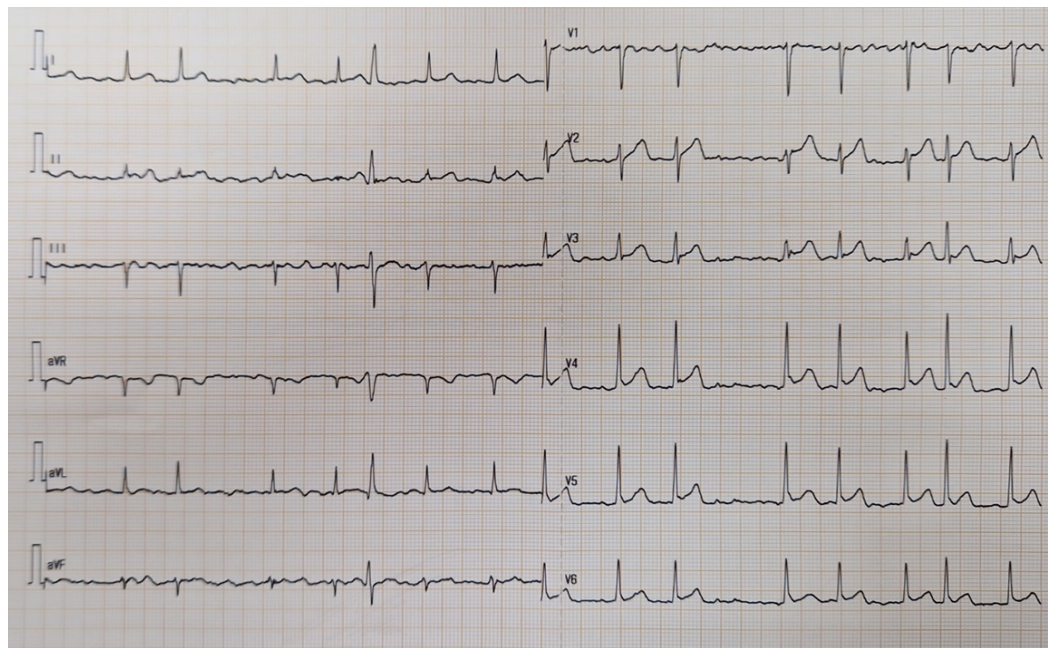
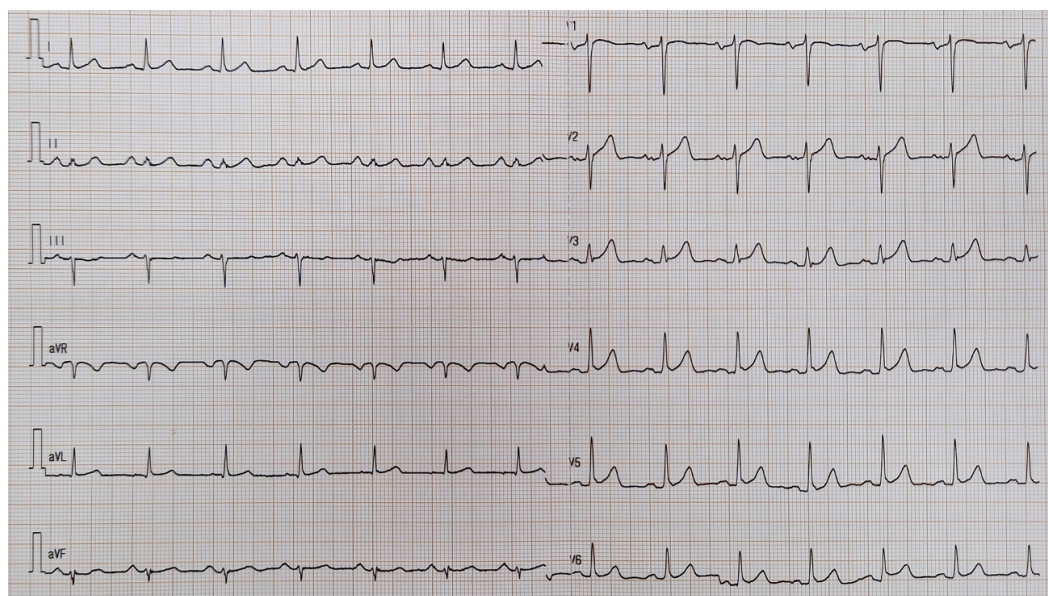


Figure 2. Electrocardiogram shows Sinus rhythm, HR=75bpm.



At discharge, lifestyle modification measures were reinforced, with an express recommendation for abstinence from excessive alcohol consumption. Outpatient follow-up was scheduled after one month, with a request for a follow-up transthoracic echocardiogram, 48-hour Holter outpatient electrocardiographic monitoring and laboratory reassessment. Considering the close temporal relationship between episodic excessive alcohol consumption and the onset of arrhythmia, the recurrence under similar circumstances, the

absence of structural heart disease and the exclusion of more frequent secondary causes of AF, the diagnosis of alcohol-induced paroxysmal AF was established.

3. Discussion

The present case illustrates a classic presentation of HHS, characterized by the development of paroxysmal AF after episodic excessive alcohol consumption in an individual without known structural heart disease. Although originally described by Ettinger et al. in 1978, HHS remains a frequently underdiagnosed entity, especially in young adults, in whom the absence of cardiovascular risk factors may delay the recognition of the association between excessive alcohol consumption and the onset of arrhythmia [5]. Epidemiological studies demonstrate a consistent and dose-dependent relationship between alcohol intake and the risk of AF, with binge drinking being one of the most important triggers for acute episodes in previously healthy individuals [3,4].

In this patient, several elements reinforce the clinical diagnosis of HHS: the close temporal relationship between excessive alcohol consumption and the onset of AF, the recurrence of a similar episode under virtually identical circumstances in the previous year, the absence of structural heart disease on echocardiogram, normal levels of electrolytes and thyroid hormones, and spontaneous reversion to sinus rhythm after conservative treatment. Taken together, these findings make other secondary causes of AF unlikely and support the diagnosis of alcohol-induced AF.

A particularly relevant aspect of this case is the chronology of symptoms. Although excessive alcohol consumption occurred between December 31 and January 3, palpitations began only on January 4, during the hangover phase, and not during the period of maximum alcohol intoxication. This temporal sequence suggests that the arrhythmia was predominantly precipitated by the pathophysiological mechanisms associated with acute alcohol withdrawal, rather than by the immediate electrophysiological effects of ethanol. This observation is consistent with recent studies demonstrating an increased incidence of AF in the hours following excessive alcohol consumption, reinforcing the importance of the hangover phase as a period of greater atrial electrical vulnerability.

The mechanisms responsible for HHS are multifactorial and likely act synergistically. During the acute alcohol withdrawal phase, rebound sympathetic hyperactivity occurs, with an increase in circulating catecholamine levels, accompanied by significant autonomic imbalance and a reduction in the atrial effective refractory period, favoring triggered activity and reentry circuits. In parallel, ethanol metabolism leads to the accumulation of acetaldehyde, which promotes oxidative stress, mitochondrial dysfunction, and transient myocardial inflammation. Experimental models have also demonstrated that acute exposure to alcohol activates the protein kinase C (PKC)/glycogen synthase kinase-3 β (GSK-3 β)/nuclear factor of activated T-cells (NFAT) pathway, increasing the expression of T-type calcium channels, promoting intracellular calcium instability, and increasing susceptibility to AF [6,8]. These mechanisms provide a plausible pathophysiological explanation for the development of transient episodes of AF even in the absence of evident structural heart disease.

However, only a small proportion of individuals exposed to excessive alcohol consumption develop AF, suggesting that individual susceptibility factors also play an important role. Subclinical electrophysiological changes, genetic predisposition, or early atrial remodeling may explain this variability. In this context, advanced imaging methods, such as cardiac magnetic resonance imaging with late enhancement and atrial strain assessment by echocardiography, can identify occult atrial remodeling before the appearance of conventional structural changes. However, these tests are not routinely recommended by current guidelines for patients with a clearly reversible precipitating factor, a transthoracic echocardiogram without relevant structural changes, and favorable clinical evolution, and can be reserved for recurrent cases or when there is clinical suspicion of underlying cardiomyopathy [9,10].

The management of this patient was in accordance with contemporary recommendations for AF. The initial choice was ventricular rate control with Bisoprolol, without the need for pharmacological or electrical cardioversion, since spontaneous conversion to sinus rhythm occurred in approximately 48 hours. The stratification of thromboembolic risk using the CHA₂DS₂-VA score was zero, with no indication for anticoagulation. In addition, outpatient follow-up was scheduled with 48-hour Holter electrocardiographic monitoring and a new transthoracic echocardiogram, aiming to confirm the maintenance of sinus rhythm and re-evaluate possible structural changes should recurrences arise.

The modification of risk factors currently constitutes one of the pillars of AF treatment. Among them, the reduction or abstinence from alcohol consumption has a significant impact on the prevention of new episodes, especially in patients with HHS. Studies demonstrate that reducing alcohol intake reduces the recurrence of AF, improves rhythm control, and decreases the arrhythmic burden, which is why guidance for alcohol abstinence should be considered an integral part of treatment, and not just a behavioral recommendation [3,4,9].

This report reinforces the importance of recognizing episodic excessive alcohol consumption as a potentially reversible trigger for AF. Although HHS is a known clinical entity, its pathophysiology continues to be progressively clarified, highlighting the interaction between autonomic, metabolic, and inflammatory changes in the genesis of arrhythmia. Early recognition of the syndrome, associated with the careful exclusion of secondary causes, proper thromboembolic risk stratification, and intervention on modifiable factors, remains fundamental to reducing recurrences and avoiding unnecessary treatments.

4. Conclusion

This case clearly illustrates the causal relationship between excessive alcohol consumption and atrial fibrillation, reinforcing the importance of identifying Holiday Heart Syndrome as a relevant clinical diagnosis. Early recognition and appropriate intervention are essential to reduce recurrences and long-term complications.

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