HOLIDAY HEART SYNDROME

(A Case Report)

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Introduction

Alcohol has direct depressant effects on the myocardium both after acute ingestion and chronic intake and produces demonstrable cardiac dysfunction even when ingested by normal individuals in quantities consumed in social drinking [1]. Alcoholics without other evidence of heart disease are often seen developing an acute cardiac rhythm disturbance or conduction disturbance associated with heavy ethanol consumption and disappearing with abstinence. This is dubbed as the 'holiday heart syndrome' [2].

CASE REPORTS

Case 1

A 38-year-old married male, a known case of alcohol dependence syndrome, was hospitalized on 26 Dec 92 by his colleagues with the history that he had become abusive after consuming alcohol continuously since the previous afternoon. The patient complained of episodic palpitations and giddiness. Clinical examination revealed an averagely built and poorly nourished patient smelling strongly of alcohol. His pulse rate was 122 per minute irregularly irregular, blood pressure 130/85 mmHg, temperature 99.2°F and respiratory rate 24 per minute. He looked pale, was tremulous and was sweating profusely. The examination of the cardiovascular system revealed normal heart size and sounds. No cardiac murmur or adventitious sounds were discerned. Examination of the central nervous system revealed slurring of speech, digital and lingual tremors, and ataxia. Examination of abdomen revealed a firm, mildly tender liver palpable 4cm below the costal margin. Psychiatric examination showed a dishevelled individual wearing dirty clothes and smelling strongly of alcohol. He was garrulous and exhibited disinhibited behaviour. Affect was euphoric. There were no features of psychosis. Investigations showed blood hemoglobin 10.2 g%, serum bilirubin 1.7 mg%, SGOT 45 IU/L, SGPT 55 IU/L, blood glucose: fasting 105 mg%, post-prandial 160 mg%. Blood urea, serum creatinine, serum electrolytes, serum uric acid and urinalysis were within normal limits. Electrocardiogram showed atrial fibrillation with a ventricular response of 130 per minute. There were no changes in ST segment and T

wave morphology. Echocardiography (M mode and 2D) showed normal heart valves and functions. Ultrasonography of abdomen showed altered echogenicity of liver suggestive of fatty liver.

Since the patient had no hemodynamic abnormality he was managed conservatively with vitamins including 100 mg of thiamine parenterally, tab chlorthalidone 10 mg 8 hourly and other supportive measures. Repeat ECG after 18 hours showed reversion to normal sinus rhythm. Treadmill test (Bruce protocol) done 2 weeks later was negative for inducible ischemia.

Case 2

This 38-year-old married male, was initially hospitalized on 28 Feb 94 (Monday) with the complaints of pain chest and palpitations following an alcoholic binge since Saturday. On examination the patient looked anxious and was smelling of alcohol. Pulse rate 106/min regularly irregular. Blood pressure was 140/90 mm of Hg. Central nervous system examination revealed the presence of digital tremors. Psychiatric examination showed a dishevelled appearance. He was garrulous, worried, and anxious. There were no features of psychosis.

Investigations showed blood hemoglobin 13.5 g%, serum bilirubin 0.6 mg%, SGOT 14 IU/L, SGPT 12 IU/L, blood glucose fasting 72 mg% and post-prandial 86 mg%. Blood urea, serum creatinine, serum cholesterol, serum electrolytes, serum proteins, serum uric acid, urinalysis and chest radiography were normal. Electrocardiography showed the presence of monomorphic ventricular ectopy with fixed coupling interval (Lown's grade III). Echocardiography revealed normal valves and function.

He was treated with supplemental vitamins, anxiolytics and other supportive measures. Repeat ECG done after 24 hours was normal. Treadmill testing (Bruce protocol) done thereafter was negative for provokable ischemia. He was counselled and discharged with the advice that he should abstain from alcohol. Patient did not touch alcohol for 2 months. On 22 Jun 94 he again went on an alcoholic binge and consumed about 1 bottle of rum. He was hospitalized again on 23 Jun 94 with recurrence of pain chest, palpitations, and dizziness. Physical examination showed tremulousness, pulse rate of 112/min regularly irregular and blood pressure of 160/100 mm of Hg. ECG showed recurrence of ventricular ectopy which abated spontaneously after six hours.

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Discussion

An association between alcohol use and cardiac arrhythmias, particularly atrial fibrillation has long been suspected. The "holiday heart syndrome" in which alcoholics without overt cardiomyopathy developed cardiac arrhythmias after heavy alcohol intake was first described by Ettinger et al [2]. They observed 32 separate episodes requiring hospitalization in 24 alcoholics with history of at least 10 years of heavy ethanol consumption, and especially heavy ingestion prior to the arrhythmia. Atrial fibrillation was most common, followed by atrial premature beats, atrial tachycardia and ventricular premature beats. Junctional tachycardia, ventricular tachycardia, conduction blocks, and AV nodal blocks were observed in a few cases. Treatment given included digoxin, DC cardioversion, quinidine, procainamide, and carotid sinus massage. Eight episodes went untreated and resolved spontaneously. None of the patients had evidence of overt heart disease after treatment of arrhythmia [2]. These arrhythmias were often labelled idiopathic because once the rhythm disturbance resolved no clinical evidence of underlying heart disease was found, and the provocative nature of previous alcohol ingestion was not appreciated. In a given individual the same arrhythmia tends to recur on subsequent occasions with alcohol abuse, as seen in Case 2. Other reports have also highlighted an association between alcohol consumption and cardiac arrhythmias [3-5]. These arrhythmias are usually self-limiting (as was seen in our patients) and require treatment only if asso-

ciated with underlying heart disease or hemodynamic compromise.

The mechanisms by which ethanol promotes the induction of ventricular arrhythmias are complex. Chronic alcohol intake induced myocardial injury promotes an arrhythmogenic substrate characterized by prolonged action potentials, whereas acute alcohol intake provides the trigger "of increased adrenergic stimulation by encouraging early after-depolarizations". An additional arrhythmogenic factor might be the low serum magnesium concentrations frequently found in alcoholics which can also encourage early after-depolarizations. These early after-depolarizations lead to increased automaticity and also favour the development of micro-reentry circuits [3,6].

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