Reversibility of alcoholic cardiomyopathy

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Summary

A patient with alcoholic cardiomyopathy is described in whom cardiovascular function capacity and radiological heart size fluctuated widely with periods of heavy drinking and abstinence. On two occasions, cessation of drinking resulted in clinical recovery from severe degrees of congestive failure and in complete reversal of cardiac enlargement. Echocardiographic follow-up showed, however, that the true rapidity of myocardial recovery was much slower than estimated clinically and radiologically and than suggested by the previous reports.

KEY WORDS: cardiac failure, echocardiography.

Introduction

The natural course of congestive alcoholic cardiomyopathy is poor: over 40% of such patients died within an average time of 3 years in a prospective study (Demakis *et al.*, 1974). By abstention from alcohol, the patients seemed to benefit so that 61% of abstaining patients were clinically improved in contrast with only 10% of those who continued heavy drinking. Moreover, three previous case reports (Schwartz, Sample and Wigle, 1975; Baudet *et al.*, 1979; Hung *et al.*, 1979) have objectively shown that even severe degrees of congestive failure due to alcoholic heart muscle disease are potentially totally reversible. The recovery from overt failure to normal cardiac function in these patients took from 8-18 months.

The patient reported in this paper serves to emphasize the necessity of total and permanent cessation of drinking for successful treatment of alcoholic heart disease. This case also demonstrates the usefulness of serial echocardiographic studies in following the restoration of myocardial performance.

Case report

A 32-year-old seaman was admitted to hospital in December 1978 because of advancing heart failure. He had been well until 6 weeks earlier when shortness of breath and fatigue on exertion first appeared after a 3-week period of exceptionally heavy alcohol abuse. He admitted to excessive drinking since 1970 and his approximate alcohol consumption had amounted to 150-200 grams/day during the last 2 years before admission. He gave no history of hypertension, chest pain or recent viral illness and was a non-smoker.

On examination, the patient appeared well nourished and without signs of chronic liver disease. Heart rate was 102 beats/min and blood pressure was 110/80 mmHg. There was a third heart sound, marked jugular venous distension and the liver was palpable 3 cm below the right costal margin. The electrocardiogram showed sinus tachycardia, P-wave changes indicative of left atrial strain and ST segment depression as well as T-wave inversion suggestive of left ventricular strain. The chest roentgenogram disclosed cardiac enlargement; the cardiothoracic ratio was 0.55 (Fig. 1a). Apart from macrocytosis of the red cells, the routine blood tests were non-revealing. Tests for antinuclear antibodies were negative as were the serologic reactions for syphilis and streptococcal, viral, and rickettsial diseases. A tentative diagnosis of alcoholic cardiomyopathy was made and the patient was discharged on digoxin and hydrochlorthiazide and advised to refrain from drinking.

The condition of the patient improved rapidly during abstinence and he resumed his professional activities 4 months after the discharge from hospital. All medications were discontinued in June 1979. In December 1979, 1 year after admission, the patient was symptom-free and the clinical cardiac examination was interpreted normal. The chest X-rays showed a return of heart size to normal; the

FIG. 1. X-ray studies of the chest: on admission in December 1978 (A), after 1 year of abstinence in December 1979 (B), on readmission in March 1981 (C), and after a 6-month abstinence in September 1981 (D).

cardiothoracic ratio was 0.38 (Fig. 1b). Though much diminished, T-wave inversion still persisted in the leads V_4 – V_6 of the electrocardiogram. The patient was considered almost fully recovered and was discharged from follow-up.

In March 1981, the patient was readmitted because of congestive heart failure (Fig. 1c). He had been well and maintained abstinence until 2 weeks earlier when he relapsed into alcohol abuse imbibing approximately 3/4 litres of whisky daily till the onset of

breathlessness 4 days before admission. On examination, there was a summation gallop as well as murmur of mitral insufficiency. The electrocardiogram showed left atrial and left ventricular strain and occasional ventricular premature beats in addition to sinus tachycardia. The chest roentgenogram revealed a recurrence of cardiac enlargement as well as pulmonary venous engorgement and interstitial extravasation. The routine blood tests were normal and the antiviral antibodies showed no acute reactions.

TABLE 1. Non-invasive follow-up of left ventricular performance during abstinence after the second admission

Variable	Months of abstinence				Reference range*
	0	6	12	18	
Heart rate (beats/min)	94	81	72	66	
Blood pressure (mmHg)	100/70	114/70	116/70	116/70	
LVEDD (mm)	74	62	67	63	41-57
LVESD (mm)	67	55	58	52	26-41
PWT (mm)	11	13	11	11	7-12
LVM (g)	242	239	216	200	90-192
Fractional shortening (%)	10	12	14	18	24-42
PEP/LVET	0.54	0.57	0.48	0.40	0.26-0.40

LVEDD and LVESD = left ventricular end-diastolic and end-systolic diameters, respectively, measured by the European standardization (Roelandt and Gibson, 1980); PWT = posterior wall end-diastolic thickness; LVM = left ventricular mass (Troy, Pombo and Rackley, 1972); PEP/LVET—pre-ejection period/left ventricular ejection time ratio, derived from simultaneous recordings of electrocardiogram, phonocardiogram, and carotid arterial pulse tracing (Lewis *et al.*, 1977).

*The reference ranges are the 95% confidence intervals in twenty healthy male subjects aged 23–62 years (mean age, 36 years).

FIG. 2. M-mode echocardiographic studies of the left ventricle on readmission in March 1981 (A) and after a 6-month abstinence from alcohol (B). Note the diminution of left ventricular transverse diameters; the depth scales are nearly identical.

haemochromatosis was also excluded. A left ventricular echocardiographic study and measurement of the systolic time intervals gave findings typical of congestive cardiomyopathy (Fig. 2a, Table 1). The patient was started on digoxin and prazosin and a small dose of metoprolol was later added to the regimen.

After discharge from hospital, the patient main-

tained total abstinence and again rapidly improved. All medications were discontinued in June 1981 and the patient resumed his working activities. In September 1981, 6 months after the readmission, he was symptom-free apart from slight dyspnoea on effort; neither murmurs nor gallop sounds were heard on auscultation. The chest X-rays revealed again a normal-sized heart (Fig. 1d). Nevertheless, left ven-

tricular dilatation and hypokinesia, although diminished, still persisted on the echocardiographic study (Fig. 2b, Table 1) and the electrocardiogram showed negative T-waves in the leads V_4 – V_6 . Until September 1982, the patient has maintained abstinence, and repeated echocardiographic studies 12 and 18 months after the 2nd admission have shown a steady but slow improvement in left ventricular performance (Table 1).

Discussion

The case presented herein supports the few earlier reports on the reversibility of alcoholic cardiomyopathy. On the other hand, this case shows also that, although the initial clinical improvement can be rapid, the restoration of cardiac performance during abstinence either may remain incomplete or can take a much longer time than suggested by the previous case histories (Schwartz *et al.*, 1975; Baudet *et al.*, 1979; Hung *et al.*, 1979). On clinical grounds alone, without the use of echocardiography, the rapidity of myocardial recovery would have been greatly overestimated in the present case. The slowness of this process helps to explain the surprisingly rapid recurrence of congestive failure on resumed heavy drinking before the 2nd admission. So, although symptom-free and having normal function capacity, there were still subclinical manifestations of cardiomyopathy

which were acutely exacerbated by the toxic insult from heavy alcohol intake. Abstinence from drinking must be total and permanent to be curative in alcoholic heart disease.

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(Accepted 16 March 1983)