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Study of alcoholic myocardiopathy

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A STUDY OF ALCOHOLIC MYOCARDIOPATHY

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Introduction

This is a study of cases of alcoholic myocardiopathy as reported in recent literature and cases reviewed in the autopsy records of the Omaha Veterans Administration Hospital.

When taken in excess and habitually, alcohol is known to produce injury of the nervous system and involvement of the liver which progresses into an irremediable stage if abstinence is not immediately practiced to halt it. There is little reported of the effect of alcohol on the heart other than the beriberi syndrome from thiamine deficiency. Only in recent literature has there appeared the concept of damage to the heart muscle from alcohol.

Alcohol affects the heart in two ways: (1) heavy consumption of alcohol replaces customary meals and this discrepancy in the daily diet can lead to a deficiency in Vitamin B_1 within a short period of time and to the syndrome of beriberi; (2) excessive and habitual drinking over many years causes direct injury to the heart muscle to establish the condition which has become known as myocardiopathy.

Sometimes the two states appear side by side in the same patient, the mixed type, but usually they are met with separately and as distinct clinical states. (8).

The minimum daily requirement of Vitamin B_1 (aneurin or thiamine) for an adult is about one mg. Thiamine is used in the natural metabolism of carbohydrate, being concerned with the oxidation of pyruvic acid which is derived from lactate. Insufficient thiamine halts the breakdown of carbohydrate at this point, and this leads to an accumulation of pyruvic acid and the ensuing clinical state of beriberi(8).

Thiamine is present in small amounts in natural foods and in a higher concentration in rice, wheat and other grains, liver, and yeast. The deliberate exclusion of the vitamin in the experimental animal induces cardiac effects, either in form of heart failure (7) or myocardial changes which have included obvious fibrosis (11).

There are two ways to acquire the syndrome of beriberi. First, the amount of thiamine in the diet may be inadequate, as in the orient where persons subsist largely on a diet of polished rice (1,12,14), persons on certain special diets, patients with anorexia nervosa in whom vomiting affects the diet, and following resection of large portions of the digestive tract resulting in unsatisfactory absorption of food. The second cause of beriberi is due to a marked increase in carbohydrate metabolism which results from the consumption of large quantities of alcohol. Large amounts of thiamine are

utilized so that it becomes deficient. The above process is accentuated if food intake is decreased at the same time as often happens in alcoholism (3).

The patient with beriberi from excessive use of alcohol is usually an obese male who works in a position with ready access to alcohol, beer and wine. Common presenting symptoms usually include exertional dyspnea or paroxysmal and nocturnal dyspnea on some occasions. Swelling of the ankles is often present.

Other signs seen are associated with high output failure. The pulse is regular and rapid. There is frequently a gallop rhythm and venous distention of the neck veins. Crepitations may be heard over the lung bases. The liver is frequently distended (3).

Electrocardiogram changes in beriberi heart disease as reported in the literature are variable. The changes are probably transient and easily reversible with treatment. Wenckebach (17) and Keefer (13) report no electrocardiogram changes. Weiss and Wilkins (18) list inversion of the T waves as the commonest change. They state that this deformity decreased or disappeared after treatment with thiamine. Schrire and Grant (16) state that electrocardiogram readings may remain normal in alcoholic beriberi. Their study of 50 cases with this disease showed T wave inversion as a characteristic change in most cases with eight showing no or minimal electrocardiogram changes. The commonest changes were in chest leads

over the right ventricle. The changes in the leads over the left ventricle were also seen in the limb leads.

These variable findings are probably related to missing transient changes and not recording both chest and limb leads. The T wave changes are probable related to the concentration and mobilization of electrolytes within the myocardial cells (2).

Radiographic changes seen in the chest in beriberi include generalized enlargement of the heart shadow with prominent enlargement of the pulmonary artery and right ventricle. These changes are readily reversible following adequate treatment. Diagnosis can be made by excluding other causes of cardiac dilatation and response to specific treatment (2).

Alcoholic Myocardiopathy

The term "Alcoholic Myocardiopathy" is used to describe a type of cardiac disease apparently due to excessive use of alcohol (6). It is a type of heart disease which does not respond to thiamine and is apparently caused by the direct toxic effect of alcohol on the myocardium (7).

The clinical features of the patient with alcoholic cardiomyopathy may suggest the presence of valvular heart disease. This patient is often in relatively good physical condition with no overt evidence of malnutrition. He usually consumes alcohol in the form of beer rather than whiskey, gin, or brandy. He usually has a long

history of consistent use of 4 quarts of beer or a pint or more of alcohol per day (7). Brigden (4) in a series of 13 patients diagnosed as having alcoholic cardiomyopathy states that seven drank only beer, consuming an average of 10 pints a day from 10 to 40 years. Six patients consumed one or more bottles of strong liquor a day for 10 to 20 years. Evidence of signs and symptoms of heart disease appeared earlier and were more severe in the patients consuming hard liquor.

Paroxysmal dyspnea and palpitation are common in alcoholic myocardiopathy and are often the first symptoms. Sweating is commonly seen. Chest pain may be present but is not common. Syncope is rarely ever present. Nutrition is universally good at the onset of symptoms. A systolic murmur and gallop rhythm may develop. Tachycardia is usual. The liver is often enlarged and ankle edema may be present (6).

The fact that pathologic changes of the myocardium may be caused by alcohol is not generally appreciated. Chronic alcoholism affects the heart in two different ways. In alcoholic beriberi, the effect on the heart --- edema of the walls and enlargement of its cavities--- is reversible and the result of thiamine deficiency (13). In alcoholic myocardiopathy, irreversible enlargement of the left ventricle follows fibrosis of the myocardium which is the result of the toxic action of long-standing and excessive use of alcohol (8).

The changes of alcoholic myocardiopathy present as two types. In one type the heart may show enlargement from compensating hypertrophy of its muscle fibers. The cut surface may appear normal, but microscopic examination of tissue sections will reveal insular areas of fibrosis, accompanied by a variable degree of cellular reaction. The other type demonstrates more gross changes with large and more confluent areas of fibrosis. Cardiac enlargement in cases of alcoholic myocardiopathy may be impressive (8).

Evans (8) describes electrocardiogram changes of four designs which concern T wave changes in association with alcoholic myocardiopathy. These changes appear as spinous, dimple, cloven, or narrow inversions.

The spinous T wave is more sharply pointed and is differentiated from a peaked T wave occasionally found in electrocardiograms of healthy persons, in patients with early cardiac infarction and in patients with beriberi heart disease as reported by Benchimol and Schlesinger (2). The spinous T wave is drawn out into a needle-like spike (Figure 1). This spinous deformity is the earliest electrocardiogram sign of alcoholic myocardiopathy.

The dimple T wave is a shallow and narrow notch in an isoelectric S-U period (Figure 2). This same deformity has been reported on rare occasions in healthy youths under the age of 20 years after ingestion of a meal (15). This deformity when seen in a postprandial electrocardiogram is a temporary episode.

The cloven T wave appears as a cleft at the summit of a T wave whose amplitude is subdued (9) (Figure 3). This deformity may be seen in healthy children, but is associated with inversion of the T wave in leads over the right ventricle. It may also signify a limited cardiac infarction. It becomes significant if it appears in an electrocardiogram of an adult who has a history of habitual consumption of alcohol.

The inversion of narrow T waves appears with gradual developing myocardial fibrosis (Figure 4). Accompanied by other T wave changes as dimpling and and cloven T waves and a history of habitual alcoholic use and absence of cardiac pain this deformity may be distinguished from changes following cardiac infarction from coronary artery disease.

Other electrocardiogram changes that may be seen are paroxysmal tachycardia, atrial fibrillation, faulty conduction, and depression of the S-T segment (8).

Whereas beriberi heart disease demonstrates radiographic changes predominantly involving right heart failure, alcoholic mycardiopathy changes indicate predominantly left ventricular involvement. These changes are also less reversible with treatment than are the changes seen in beriberi heart involvement. As the condition progresses, cardiac enlargement becomes considerable and hilar clouding is added as evidence of heart failure (5).

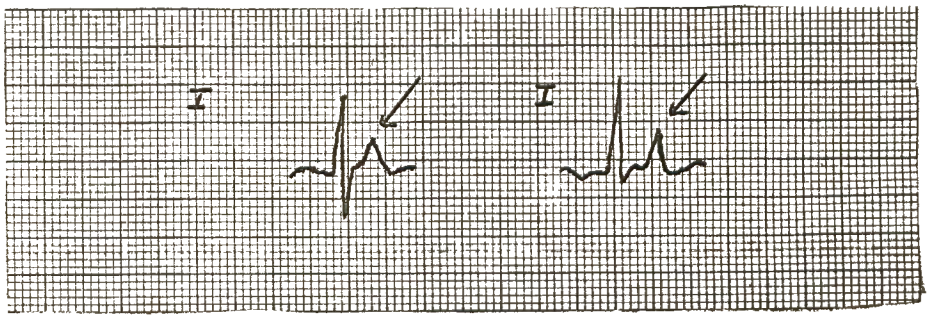

Fig. 1. Spinous T waves

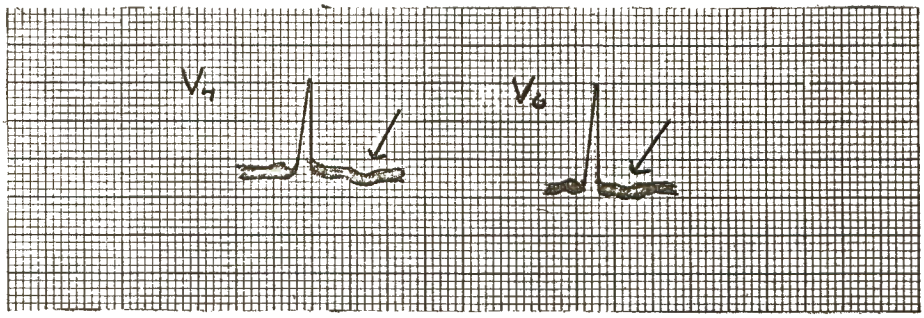

Fig. 2. Dimple T waves

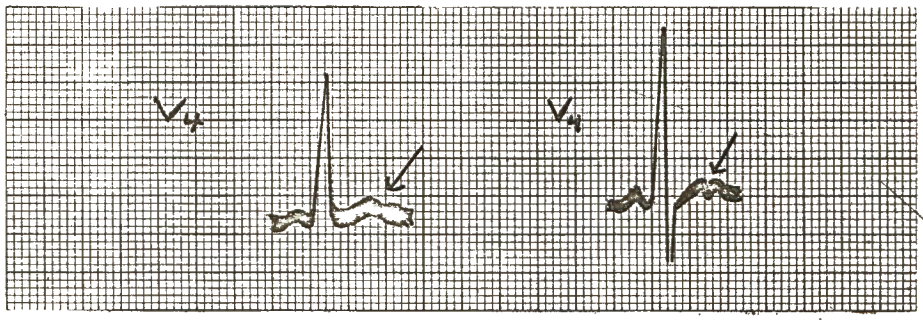

Fig. 3. Cloven T waves

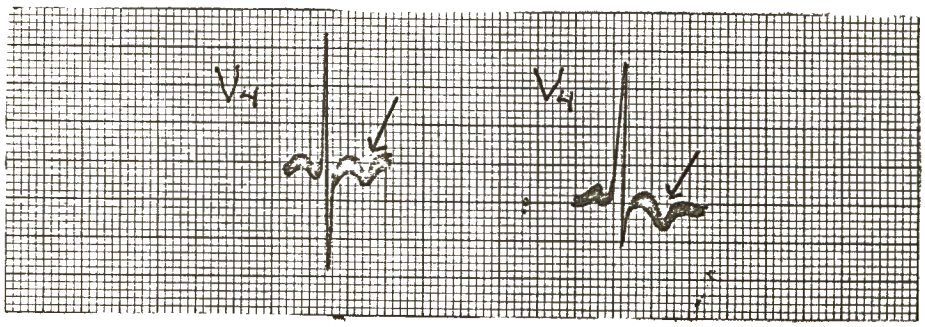

Fig. 4. Inverted T waves

(9)

In a mixture of alcoholic and beriberi myocardiopathy the features may be difficult to interpret. Following treatment with thiamine, part of the cardiac enlargement will disappear. The left ventricular enlargement will remain along with the pulmonary congestion (8). Bridgen (4), Dines (6), and Eliaser and Giansiracusa (7) reported studies of patients with alcoholic myocardiopathy with results similar to Ferrans, Hibbs, and others (10) who reported a study consisting of eight patients in whom the diagnosis of alcoholic myocardiopathy was made clinically. These patients were subjects of extensive clinical and laboratory studies and presented several features in common: (1) an exceedingly heavy consumption of alcohol preceded the onset of cardiac symptoms; (2) all but two patients had a prolonged history of inadequate diet; (3) no other etiological factor could be found to account for the heart disease; (4) most of them responded fairly well initially to a therapeutic regimen that consisted of the usual measures for the treatment of congestive heart failure, prolonged bed rest and discontinuance of alcohol; (5) the clinical course was marked by relapses related to the resumption of alcoholic intake, inadequate nutrition, and excessive physical activity whenever these patients were able to resume outpatient status.

Review of Veterans Administration Autopsies

The review of 1,185 autopsy cases at the Omaha Veterans Administration Hospital from 1960 to 1965 revealed numerous patients

with congestive heart failure which were not included in this study because there was evidence of significant valvular heart disease, hypertensive heart disease, or coronary artery disease.

A group of eight patients (VA-65-136, VA-65-103, VA-65-70, VA-65-65, VA-65-17, VA-63-47, VA-62-163, VA-61-64) including five with the clinical diagnosis of alcoholic myocardiopathy and three with the diagnosis of toxic myocarditis and congestive heart failure who did not respond to the usual therapeutic regimen and who demonstrated no other cause of heart failure were selected for this study. These patients presented with the same several features as those reported by Ferrans, Hibbs, and others (10). The age distribution of these eight patients was age 26 years to 53 years with most patients in the range of 40 to 50 years old. The predominant history was repeated episodes of congestive heart failure. The known duration of the disease varied from 2 to 15 years with death occurring within 2 to 5 years in most cases.

The heart weight varied from 420 grams to 760 grams with the majority near 500 grams. The coronary arteries demonstrated minimal or no atherosclerotic changes.

The lungs and liver were observed to show primarily chronic passive congestion. The patients in Ferrans (10) study demonstrated no cirrhosis and only two patients had areas of liver fibrosis and one had central necrosis. Only one patient in the Omaha Veterans Administration Hospital study demonstrated diffuse

nodular cirrhosis. Three patients were found to have central necrosis and two patients had fatty change of the liver. The patients in this study appear to have had more extensive liver involvement than those in Ferrans (10) discussion.

Other findings demonstrated at autopsy in the Veterans Administration Hospital study indicate five patients had kidney involvement ranging from pyelonephritis to renal infarcts. Three patients in Ferrans (10) study had similar renal findings.

The significant finding in both groups of patients was the predominance of congestive heart failure, usually right sided in nature, with minimal to moderate damage to the liver.

The radiologic changes observed in the eight patients in the Veterans Administration Hospital study demonstrated "cardiac enlargement" in four patients. Two other patients had chest x-rays revealing predominantly left ventricular enlargement. The chest x-rays in the two remaining patients were described as "normal." Evans (8) describes generalized enlargement of the heart shadow after the disease has progressed and left ventricular enlargement during the earlier phases of the disease.

The predominent electrocardiogram changes noted were sinus tachycardia and low voltage. However, two patients demonstrated cloven T wave changes, one on two separate electrocardiograms. One patient's electrocardiogram revealed spinous T waves. Two other patients demonstrated narrow inverted T waves, one with associated early dimple T wave changes. A fifth patient had a tracing showing faulty conduction with prolonged Q-T intervals.

Treatment

In the treatment of the alcoholic beriberi syndrome, total abstinence and a return to a normal diet should be reinforced with the giving of thiamine for two weeks or more. Thiamine chloride (100 mg.) should be given intravenously daily for five days and thereafter given orally 50 mg. daily until abstinence from alcohol and a return to an adequate diet have insured a sufficient supply of natural Vitamin B_1 (2).

Patients in which alcoholic myocardiopathy has progressed to considerable fibrosis and hypertrophy with heart failure, abstinence from alcohol does little or no good because irreversible changes have occurred. Measures to treat heart failure is the only help that can be given; reduction in fluid and salt intake, digitalization, and oral diuretics if needed. At this stage, any form of therapy may be fruitless and every effort should be made to establish early diagnosis in order to begin therapy before hope of recovery is gone. The early treatment

of alcoholic myocardiopathy consists primarily of abstinence from alcohol. This should be reinforced with an adequate diet, adequate rest, and avoidance of excessive physical exercise (8).

Summary

The review of autopsy cases at the Omaha Veterans Administration Hospital reveal that the histories, physical findings, radiological and electrocardiogram changes, pathologic alterations, and clinical courses of these patients are similar to those reported in the literature.

Alcoholic myocardiopathy has only recently been recognized as a distinct disease entity. There are several reasons for this, the most significant being the stealthy onset of the condition, its similarity to beriberi heart disease, and the clinicians emphasis on the effects of alcohol on the liver and the nervous system. The electrocardiographic signs of alcoholic myocardiopathy have gone unrecognized for they have been attributed to other forms of heart disease.

The patient with alcoholic myocardiopathy generally is in late middle age and presents with gradually progressing congestive heart failure with a rather sudden worsening of the failure leading to hospitalization. He usually appears to be well-nourished and has a relatively short cardiac history with a long history of

overuse of large amounts of alcohol. He usually dies of right heart failure.

The electrocardiograms in patients with alcoholic myocardiopathy often show characteristic changes. These changes include distinctive deformities of the T wave which may be spinous, cloven, dimple, or peculiarly inverted. When these changes are present, it is not unusual to find some left ventricular enlargement.

The clinical picture and course of alcoholic patients with nonspecific heart failure in the absence of clinical malnutrition and significant lack of response to thiamine therapy gives rise to consideration of the possibility that alcohol per se may have a direct toxic effect on the heart. A combination of consistent histories, signs, symptoms, physical findings, x-ray, and electrocardiogram changes are gradually forming diagnostic boundaries as criteria of alcoholic myocardiopathy.

Early recognition of alcoholic myocardiopathy through the use of electrocardiographic signs in patients who have a history of excessive use of alcohol and with symptoms of breathlessness, palpitations, tachycardia, or atrial fibrillation without its usual causes is urgent since only treatment in the early stages of this condition gives any hope of cure.

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