Case Report

Dilated Alcoholic Cardiomyopathy and Incidental Lymphoma Occuring in A 56 Year Old Man who was being Managed for Hypertensive Heart Disease in Enugu Nigeria - A Rare Finding

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ABSTRACT

We present a case of dilated alcoholic cardiomyopathy occurring in a 56-year-old Nigerian male. He admitted to taking alcoholic beverage, approximately 2-3 bottles of different brands of beer per day for about 30 years, but stopped three years ago on medical advise. He had a history of progressively worsening dyspnoea and encephalopathy, from decreasing ejection fraction. This resulted in a poor blood supply to the vital centers of the brain. Autopsy confirmed a dilated cardiomyopathy with an incidental fairly advanced Bcell lyphoma involving the liver and spleen .The latter was thought to be a coincidental finding.

KEY WORDS Dilated Cardiomyopathy, male, alcohol, lymphoma.

INTRODUCTION

The most recent WHO/ISFC classification of cardiomyopathies (1995) describes as cardiomyopathies all heart muscle diseases, which demonstrate a disturbance of cardiac function. It distinguishes primarily according to hemodynamic criteria the following 5 forms: 1. dilated (DCM), 2. hypertrophic (HCM), 3. restrictive (RCM) from 4. arrhythmogenic right ventricular (ARVCM) and assembles in 5. non-classified cardiomyopathies (NKCM) the non-classifiable forms[1]. Dilated cardiomyopathy is therefore a type of cardiomyopathy and, depending on the diagnostic criteria used, the reported annual incidence varies between 5 and 8 cases per 100,000 population[2,3,].

However, the true incidence of DC is probably underestimated by those figures, since many asymptomatic cases remain unrecognized. In our environment few studies have been done and no figures are available although DC remains a rare disease. The age-adjusted prevalence of DC in the United States averages 36 cases per 100,000 population,[4] and it accounts for 10,000 deaths annually[2].

Blacks and males have a 2.5-fold increase in risk, as compared with whites and females, that is unexplained by socioeconomic factors, alcohol intake, or other variables[2,5]. Convincing associations have been reported between DC and hypertension, the use of beta-adrenergic agonists, and moderate alcohol consumption[5,6]. Excess alcohol consumption has been reported in up to 40 percent of patients with DC. Obtaining a quantitative history of alcohol consumption is of paramount importance, since abstinence may result in a dramatic increase in the ejection fraction[2,7].

CASE REPORT

We present a case of a 56 year civil servant and a known hypertensive for 3-years with poor drug adherance. He was also being managed for congestive cardiac failure on an outpatient basis. He was seen at the emergency unit of University of Nigeria Teaching Hospital on 02/02/06 with a two-week history of dyspnoea and bilateral leg swelling. Dyspnoea was progressive, initially on mild exertion and later at rest. There was associated cough with white frothy sputum, dizziness but no vertigo. Paroxymal nocturnal dyspnoea and orthopnoea were also present.

Pedal oedema was bilateral, progressive and got worse as the day progressed. Past Medical history was significant with previous admission for the same illness. There is no family history of hypertension or diabetes mellitus. He took alcoholic beverages heavily and also smoked heavily for about 30 years but stopped three years ago on medical advice.

On examination he was found to be in mild respiratory distress, moderately icteric, with bilateral pitting pedal oedema up to the knee.

CARDIOVASCULAR SYSTEM

He had a wrist pulse of 80/min, full volume, regular. Blood pressure was 120/80mmHg.(supine).Jugular venous pressure was raised to the angle of the jaw. Heart sounds 1,2,3 were heard, with grade 3 apical murmurs and a cooing component**. Chest-**20 breathes /min.with bilateral fine crepitations hard on both lung bases. **Abdomen** full and soft, with tender hepatomegaly 8cm below the right costal margin, with a span of 16cm.**Central Nervous System** was intact.

Results of ordered investigations were-Full blood count Packed cell volume 44%, White blood cells 7,100/mm^3, neutrophils 53%, Eosinophils 4%, Lyphocytes 42%, Platelets 160,000/mm3. Erythrocyte sedimentation rate 6mm/1st hour. Serum electrolytes were normal. **Chest-x-ray**-showed gross cardiomegaly, mildly unfolded aorta, prominent hilar vessels. **Electrocardiogram** showed

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Figure 1 shows the gross features of the patient's heart. It weighed 850g.

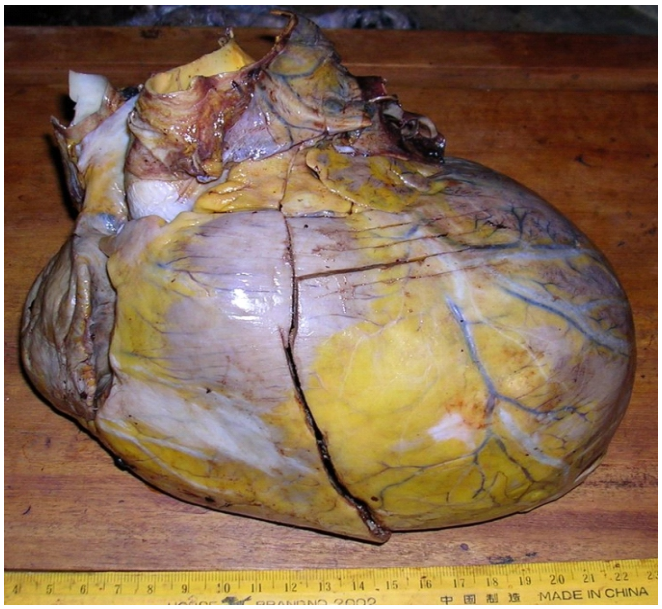
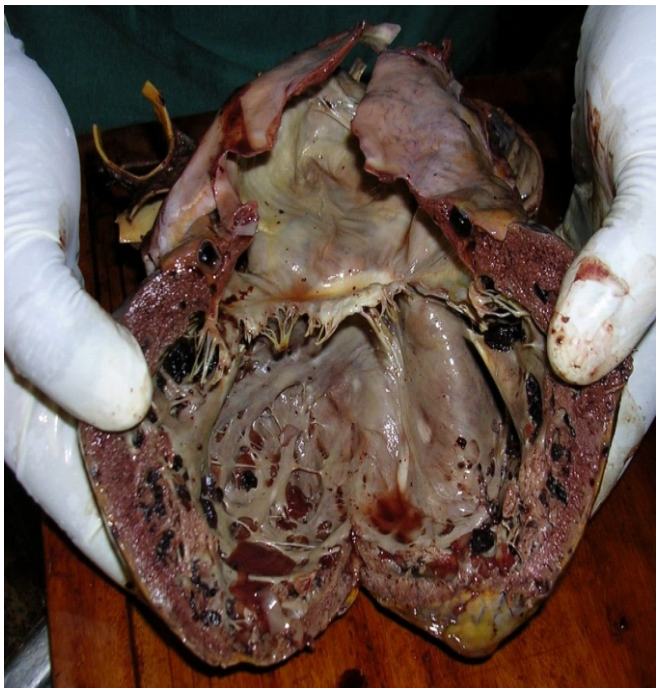
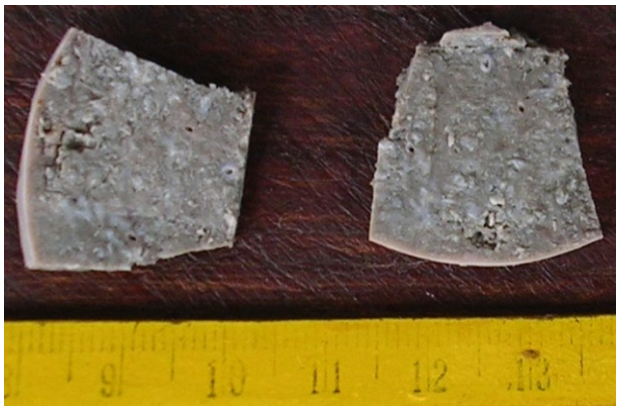


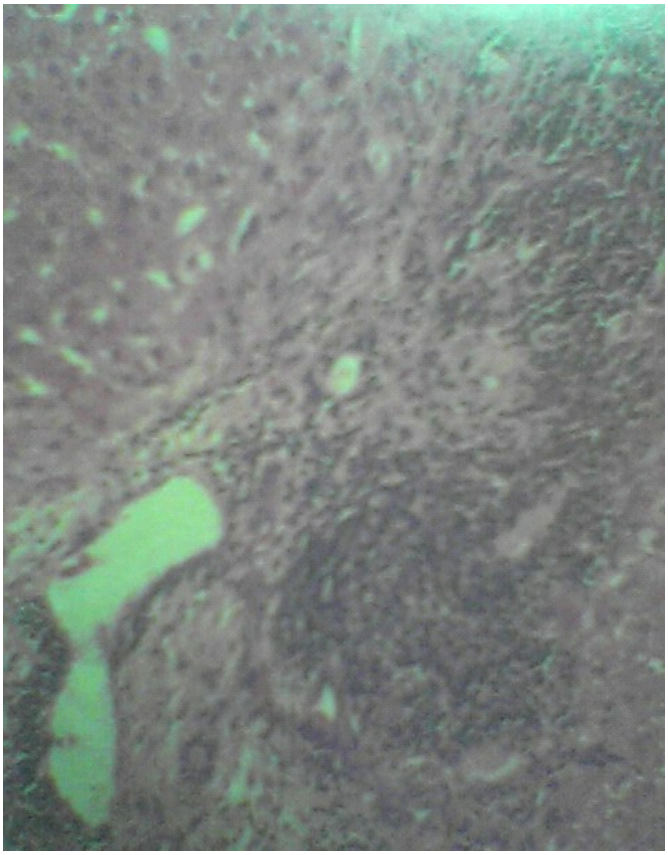
Fig 2 cut surface showing a markedly dilated right side of the heart. With some residual hypertrophy and a degree of intimal fibrosis.



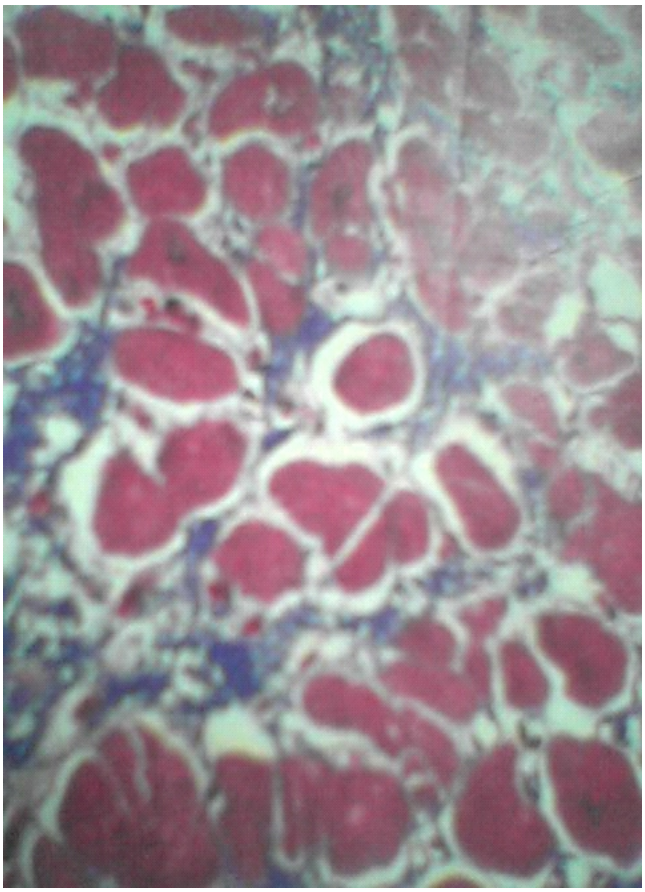
Cut surface of the spleen showing prominent B-cell areas or white pulp.



Photomicrograph showing dense accumulation of Small lymphocytes invading the portal tracts.



Photomicrograph of hypertrophied myocytes with dense Collagen fibres inbetween trichrome stain.



features consistent with left ventricular hypertrophy with strain. Dilatation of all four-heart chambers and grade 4 aortic regurgitation were demonstrated at echocardiography. **An impression of Congestive cardiac failure secondary to a hypertensive heart disease was made** and appropriate treatment was commenced. Patient was then discharged home 10days later on account of clinical improvement. But was readmitted on 18/03/2006 for restlessness, confusion and irrational talk. Blood pressure then was lower at100/80mmHg. Wrist pulse was 96/min small volume. Patient's condition continued to deteriorate and died 12days later after repeated seizures.

AUTOPSY REPORT
On the autopsy table is the embalmed body of middle-aged man of African descent with a healed thyroidectomy scar on the neck, pitting pedal oedema up to the tibia.

CHEST
Mediastinal lymph nodes: Markedly enlarged.
Lungs: Weight 1,300g (right), 850g (left). The right is oozing of whitish frothy fluid
HEART: Weight 850g; **Gross Cardiomegaly** (boot-shaped); dilatation of all the hambers, Tricuspid valve diameter: 12.5cm (normal 9.5-11.5cm), Mitral valve diameter: 14cm(normal 9.5-12cm), Pulmonary valve: 7cm,Right ventricular thickness: 0.3cm,Left ventricular wall thickness: 1.5cm,Aorta and great vessels: yellow plaques
Pulmonary artery: yellowish plaques on the major branches. Histology done on cardiac muscle and liver are shown in figs 5&4.

RETICULO-ENDOTHELIAL SYSTEM
Spleen: Weight 300g; mosaic whitish lesions are seen on the cut surface.
Liver: Weight 2,100g, congested

URINARY SYSTEM: Pale and mildly enlarged.200gRight. 180gLeft.

CENTRAL NERVOUS SYSTEM
Brain weight: 1,400g cerebral oedema
Cerebrum: Oedematous

FUNDAMENTAL DISEASES
1. Dilated cardiomyopathy

CONCOMITANT ALTERATIONS
Heart: Four-chamber enlargement; cardiomegaly (850g); interstitial fibrosis; myocytes necrosis and hypertrophy; mild inflammatory cell infiltrate.
Kidneys: Diffuse cortical necrosis due to acute ischemic tubular necrosis
Lungs: Severe pulmonary oedema; alveolar capillary congestion.
Brain: Cytotoxic cerebral oedema.

INDEPENDENT ALTERATIONS
Mediastinal lymph nodes: lymphoma , with secondary and diffuse involvement of spleen, kidney, and liver.
Arterial system: Atherosclerosis Ib (mild). Pulmonary hypertension with atherosclerosis.

CAUSE OF DEATH
1. Cardiopulmonary failure

DISCUSSION
The chief morphologic feature of DC is dilatation of both ventricles[7]. Mural thrombi are frequently present in the left ventricle and not infrequently in the atria, which are also usually dilated[7]. The heart is markedly increased in weight, indicating hypertrophy, but the maximal thicknesses of the left ventricular free wall and septum are typically normal because of the abnormally dilated chambers as in this case. Mild focal scarring of the mitral and tricuspid leaflets and secondary dilatation of their annuli are frequently present. The histological findings were consistent with dilated cardiomyopathy (Figure 4)[8].

The mechanism of the cardiac damage produced by alcohol remains unclear. Over many years, several theories have arisen based on clinical and scientific data obtained in both human and animal studies[9,10]. Original theories regarding the mechanism focused on nutritional deficiencies (eg, thiamine deficiency), secondary exposures (eg, tobacco, cobalt, arsenic), and other co morbidities (eg, hypertension). However, although these mechanisms may continue to play a role in selected patients, most evidence in the literature indicates that the effects of alcohol on the myocardium are independent of these factors and that the effect is a direct toxic result of ethanol or its metabolites[10]. In this patient there was a clear history of long-term heavy ingestion of alcohol, hypertension, and cigarette smoking. The progressively worsening dyspnoea and encephalopathy we believe ensued as a result of worsening ejection systolic fraction which is a major determinant of cardiac out put and indirectly of total peripheral resistance. This is as a result of poor blood supply to the brain stem where the autonomic centers controlling the peripheral resistance is located. Incidentally this patient also had a fairly advanced B-cell lymphoma with clear liver and splenic involvement were noted although we do believe this is merely coincidental and may not have a causal relationship. This case highlights the existence of this disease in our environment where hypertension is almost labeled the sole cause of cardiac failure.

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NEWS AND NOTICES

Erratum: Nigerian Journal of Medicine Vol. 20 No. 2, April - June, 2011. Correct name should read I. U. Takai and not I. U. Takie.