



RECURRENCE OF SYMPTOMS OF CONGESTIVE CARDIAC FAILURE: A CASE REPORT

PATIL S.*, SARMA V., YADAV K.S., PENDSE M., GHOSHAL J., GOENKA P. AND MENON S.

Padmashree Dr. D.Y. Patil Medical College, Hospital & Research Center, Navi Mumbai- 400706, MS, India.

*Corresponding Author: Email- sppsmitta9@gmail.com

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Abstract- The morbidity and mortality caused by the dilated cardiomyopathy arise from fatal complications like ventricular tachy and bradyarrhythmias and congestive cardiac failure. A 36 year's old male residing at Navi Mumbai was admitted with the chief complaints of breathlessness on exertion since last 4 months, swelling over the body with distension of abdomen since last 3 months. There is no history of chest pain, syncope, and weight / appetite loss, Hypertension (HT), Ischemic Heart Disease (IHD), Valvular Heart Disease (VHD), Tuberculosis (TB) or any drugs in the past.

On examination it was found that 2 D Echo-Dilated LA, LV, RA, RV; Mild MR,TR; LVEF <25%, normal blood reports, chest X-ray shows a marked cardiomegaly, ECG-Sinus tachycardia, LBBB, intra-ventricular conduction defects, poor progression of r waves. Other investigation shows TB PCR, Thyroid profile, Anemia profile, RFT and Urine (r/m) has not been detected any abnormality. USG (abdomen) detected minimal ascites with hepatomegaly. A liver enzyme, cardiac enzymes and serum potassium shows marginal elevation.

Keywords- cardiomyopathy; ventricular tachy; bradyarrhythmias; congestive cardiac failure.

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Introduction

The cardiomyopathies are a relatively common group of serious diseases characterized by damage to the heart muscle. They form the most frequent indication for cardiac transplantation.

Three main types of cardiomyopathy are; hypertrophic (HCM) dilated (DCM) and restrictive cardiomyopathies. Duchenne and Becker muscular dystrophies often produce some very resembling cardiac symptoms. A recent work by Towbin suggested that contractile protein dysfunction predominantly gives rise to HCM, whereas defects in cytoskeletal proteins may produce dilated cardiomyopathy [1].

According to the classification of the 1995 World Health Organization/International Society and Federation of Cardiology (WHO/ISFC) Task Force on the Definition and Classification of Cardiomyopathies [2], there are five types of cardiomyopathy but three are major that can be appreciated and differentiated by echocardiography. These conditions can affect either ventricle but are most often recognized when they involve the left ventricle. American Heart Association (AHA) year 2006 classification and European Society of Cardiology (ESC) year 2008 classification systems include these five types but differ from the earlier WHO/ISFC classification in emphasizing the distinction between familial/genetic and non-familial/non-genetic causes of cardiomyopathy and excluding heart disease secondary to coronary artery disease, valvular or congenital heart disorders [3,4].

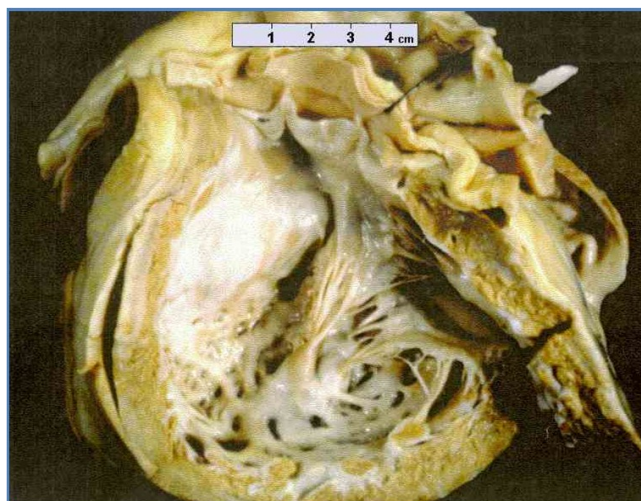


Fig. 1- Pathological changes seen in dilated cardiomyopathy

Dilated cardiomyopathy is a biochemical abnormality of cardiac muscle in which the heart chambers become grossly enlarged, with a greatly reduced contractility. It is a relatively common disease with an incidence of about 37 per 100,000. At least 25% and possibly as many as 50% of the cases may be familial associated with particular HLA phenotypes. The disease is heterogeneous and no

specific genetic defect has yet been clearly identified. The post mortem specimen below illustrates some of the pathological changes seen in dilated cardiomyopathy [Fig-1]. The left ventricle is grossly enlarged and the muscular walls are thin, in relation to the size of the ventricle.

Cardiac enlargement visible on chest X-rays creates a suspicion of dilated cardiomyopathy, but it is important to systematically eliminate all the other possible explanations for the symptoms and to treat any intercurrent diseases. Laboratory tests, even when prove negative, can play a decisive role by excluding other possible etiology, and thereby confirm that the preliminary diagnosis was correct.

Case Report

A 36 year old man, residing at Navi Mumbai, was admitted in D. Y. Patil Hospital Nerul, with the chief complaints of breathlessness on exertion since last 4 months, swelling over the body with distension of abdomen since last 3 months.

There is no history of chest pain, syncope, and weight / appetite loss. There is no history of Hypertension (HT), Ischemic Heart Disease (IHD), Valvular Heart Disease (VHD), Tuberculosis (TB) or any drugs (Anti-neoplastic, Tri-cyclic Antidepressant (TCA), phenothiazine's, lithium, cocaine abuse) in the past. There is no history of any major surgery in the past. Patient is a chronic smoker with 4-5 sticks per day and an occasional alcoholic.

On physical examination patient has a moderate built with a Heart Rate (HR)-108/min (low volume regular), Blood Pressure (BP)-100/70 mm Hg, Respiratory Rate (RR)-24/min, the jugular venous pressure (JVP)-engorged and pulsatile. No pallor, icterus, clubbing, cyanosis and lymphadenopathy were reported.

Cardiovascular (CVS) Examination

Cardiac impulse lies in 6th Intercostal space (ICS) 2 cms lateral to mid clavicular line (MCL), heaving in character. On auscultation, 1st heart sound is muffled, 2nd heart sound is soft. S3 gallop is present in mitral area. Soft systolic murmur present in mitral and tricuspid areas, with no radiation. Left sided murmur increases on handgrip and decreases by Valsalva maneuver and standing.

Other System Examination

On examination of per-abdomen, a 5 cm soft tender palpable liver is recorded. Chest has bilateral basal rales present. Investigations showed a normal blood profile except Liver enzymes, cardiac enzymes and serum potassium levels. Chest X-ray shows a marked cardiomegaly. Electrocardiography, chest X-ray and echocardiographic findings were consistent with dilated cardiomyopathy [10].

Before 1968 there were numerous reports of electrocardiographic findings in patients with hypertrophic cardiomyopathy [5-9]. Electrocardiogram (ECG) shown in [Fig-2b] shows sinus tachycardia, Left bundle branch block (LBBB), intra-ventricular conduction defects and poor progression of r waves. Electrocardiography (ECG) may detect abnormalities in the electrical activity of the heart. However, these abnormalities are usually not sufficient evidence for a diagnosis. Echocardiography uses ultrasound waves to produce an image of the heart is the most useful procedure because it can show the size and pumping action of the heart. Magnetic resonance imaging (MRI), produces very detailed images of the heart, may be used to confirm the diagnosis.

Fig-2a & Fig-2b showing Chest X-ray [Fig-2a] and ECG [Fig-2b] taken during admission in medicine ward.



Fig. 2a- Chest X-ray taken during admission in medicine ward

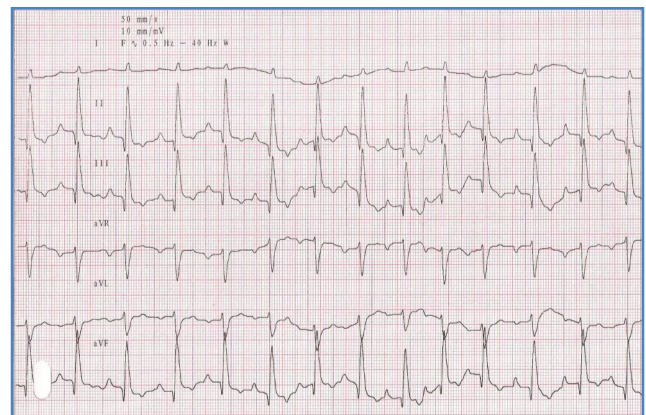


Fig. 2b- ECG taken during admission in medicine ward.

USG examination of abdomen detected minimal ascites with hepatomegaly. 2-D Echo in [Fig-3b] shows dilated LA, LV, RA, RV; Mild MR, TR; LVEF <25%. Instead of measuring the surface area ratio directly, with some experience the intensivist will simply compare the RV-to-LV size and subjectively classify the RV as normal, moderately enlarged or severely enlarged. The accuracy of this simplified technique was demonstrated by Viellard-Baron et al. Dilated cardiomyopathy arises as a primary myocardial disorder of unknown etiology or disorders xenos or of infective origin. In clinical practice, ischemic cardiomyopathy is frequently viewed as a type of dilated cardiomyopathy, although current major society classification systems i.e. American Heart Association (AHA) year 2006 and European Society of Cardiology (ESC) year 2008 exclude it [3,4]. [Fig-3a] and [Fig-3b] showing USG 2D-Echo taken during admission in medicine ward.

Other Special Investigations

TB PCR; Thyroid profile; Sr. Iron/Ferritin/TIBC; RFT and Urine (r/m) shows no abnormality. Ultra-sonography (USG) of abdomen shows minimal ascites with hepatomegaly. Investigations showed a normal blood profile except Liver enzymes, cardiac enzymes and serum potassium levels which is shown in [Table-1] and [Table-2].

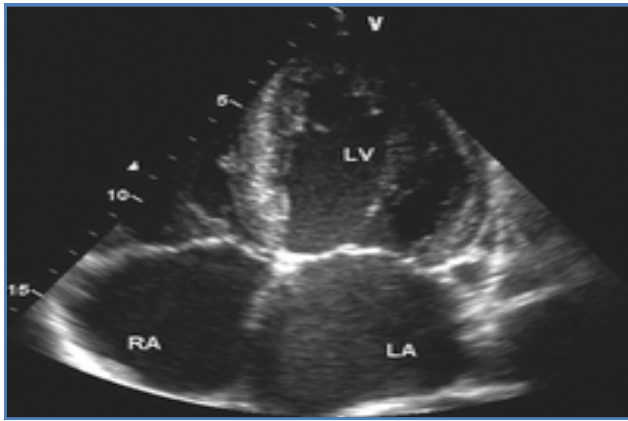


Fig. 3a-

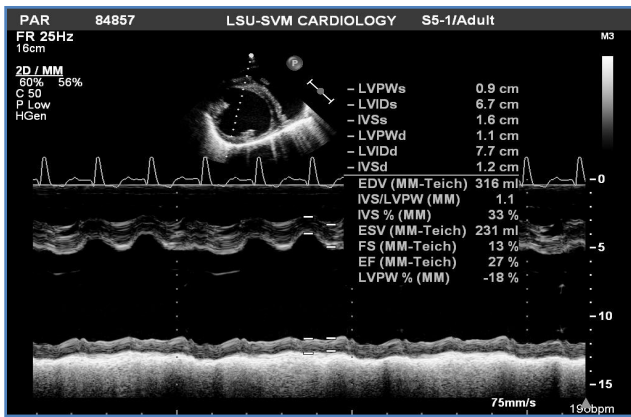


Fig. 3b-

Table 1- Enzymes measured for six times with fix days interval followed with treatment.

Investigation	1st Visit	1st FU	2nd FU	3rd FU	4th FU	5th FU
Alanine transaminase	189	224	131	89	56	29
Aspartate transaminase	22	29	47	38	24	17
Creatinine Kinase	404	421	376	222	109	88
Lactate dehydrogenase	222	346	301	295	267	221
Alkaline phosphatase	59	66	82	76	61	53

[Fig-4a] and [Fig-4b] 2 D Line diagram of Enzymes measured for six times with fix days interval followed with treatment.

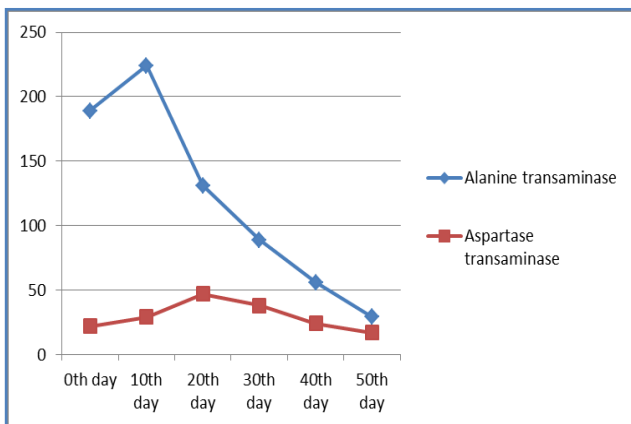


Fig. 4a-

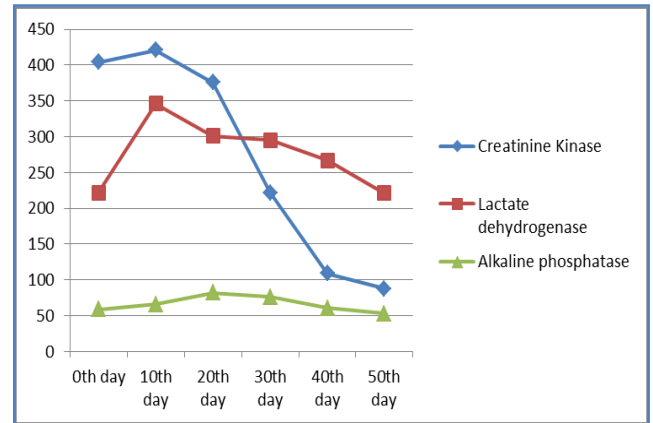


Fig. 4b-

Table 2- Blood Electrolytes (Sodium & Potassium) measured for six times with fix days interval followed with treatment.

Investigation	1st Visit	1st FU	2nd FU	3rd FU	4th FU	5th FU
Blood sodium	136	148	145	139	143	142
Blood Potassium	5.2	4.7	4.9	4.6	4.4	4.3
Blood Chloride	94	103	102	96	98	98

[Fig-5] shows 2 D line diagram of enzymes measured for six times with fix days interval followed with treatment.

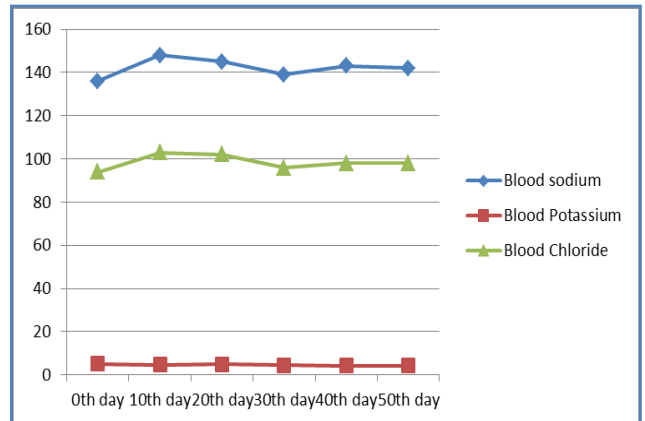


Fig. 5- 2 D Line diagram of enzymes measured for six times with fix days interval followed with treatment

Examination and investigations confirmed that this is case of dilated cardiomyopathy. Patient improved on standard therapy of salt restriction, ACE inhibitors, diuretics, digitalis but he used to have recurrences of symptoms with lots of morbidity.

Discussion

Dilated cardiomyopathy or DCM is a condition in which the heart becomes weakened and enlarged and cannot pump blood efficiently. The poor heart function can affect the lungs, liver, and other body systems.

Dilated Cardiomyopathy (DCM) is one of the cardiomyopathies, a group of diseases that primarily affect the myocardium (the muscle of the heart). Different cardiomyopathies have irrespective causes and affect the heart in different ways. In DCM a portion of the myocardium is dilated sometime without any obvious cause. Left or

right ventricular systolic pump function of the heart is impaired, leading to progressive cardiac enlargement and hypertrophy, the said progression of disease is called remodeling [2].

Dilated cardiomyopathy is the most common form of non-ischemic cardiomyopathy. It occurs more frequently in men than in women, and is most common between the ages of 20 and 60 years [3]. About one in three cases of congestive heart failure (CHF) is due to dilated cardiomyopathy [2].

Conclusion

This young case of dilated cardiomyopathy had repeated signs and symptoms of congestive cardiac failure. More research and trial is needed to improve long term prognosis like 'myocardial biopsy' proving inflammation to support immunosuppressive treatment and implantation of cardioverter and defibrillator (ICD) in patients who are at risk of sudden cardiac death due to ventricular fibrillation and ventricular tachycardia.

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