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A CASE OF NON-RHEUMATIC CARDITIS IN A YOUNG MALE LEADING TO DILATED CARDIOMYOPATHY

General Medicine

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ABSTRACT

Introduction:- Carditis is a group of inflammatory cardiac diseases with the affection of myocardium, i.e. "Myocarditis is inflammation of the myocardium". It is caused by an infection or toxins or autoimmunity or other causes. It is present in all age groups, but more frequent in children of the first years of life with predominance of boys. From data of autopsy, nonrheumatic carditis are found in 3-9% of children, who died from unknown causes. Its manifestations are age dependent. In early infancy- viral myocarditis often occurs as a acute, fulminant disease. In toddlers, young children - it occurs as an acute myopericarditis. In older children, adolescents it is often asymptomatic and comes to clinical attention primarily as a precursor to DCM. Here I present a case of a young boy who presented with dyspnoea of few years duration. He was found to have dilated cardiomyopathy secondary to chronic CMV infection. He responded to treatment.

KEYWORDS

Introduction:-

Myocarditis is characterized by myocardial inflammation, injury or necrosis, and ultimately fibrosis. Cardiac enlargement and diminished systolic function occur as a direct result of the myocardial damage. Viral myocarditis may also become a chronic process with persistence of viral nucleic acid in the myocardium, and the perpetuation of chronic inflammation secondary to altered host immune response including activated T lymphocytes and antibody-dependent cell mediated damage. Cytokines such as tumor necrosis factor-α and interleukin-1 are inhibitors of myocyte response to adrenergic stimuli and result in diminished cardiac function. The final result of viral-associated inflammation can be dilated cardiomyopathy[1]. Here I present a case of dilated cardiomyopathy caused by chronic CMV infection in a young boy.

Case Report:-

The patient was a male aged 14 years and is a student by occupation and is a right handed person. Patient is unmarried at present. The patient presented with chief complaints of

- Dyspnoea for the past 3 years,
- Palpitations for the past 2 years,
- Fever for the past 4 days

The patient complained of dyspnoea for the past 3 years which had been gradually progressive. It was mild to start with and the patient could do his routine activities but as the dyspnoea had progressed gradually over the next few years, the patient had been experiencing breathlessness even at rest. There was history of orthopnoea present. There was no history of PND. History of edema feet was present (in past few weeks). Patient complained of palpitations for the past 2 years which had been gradually increasing. It was more on exertion earlier but now was present even at rest. Patient gave history of fever for the past 4 days which was of mild to moderate degree. It was not associated with chills and was continuous in nature. There was no diurnal variation. There was history of nasal discharge and sore throat. There was no history of syncope, no history of giddiness, no history of cough, no history of joint pains, no history of chest pain, no history of rash/petechiae/bleeding, no history of altered sensorium, no history of any preferential weakness, no history of any painful nodules. Past history showed that the patient had an episode of high grade fever when he was about 11-12 years old. He also had sore throat at that time. There was no h/o any joint pains at that time. There is no previous history of DM, HTN, CAD, TB or any cardiac problem. Treatment history had nothing significant. Family history showed no history of similar complaints in the family. There was no history of any cardiac problem in parents or siblings, no history of sudden cardiac death in the family. Personal history of the patient showed that he was vegetarian by diet, no addictions, sleep was disturbed, bowel and bladder were normal. On GPE, the patient was conscious and cooperative and sitting comfortably in the bed. He was febrile (Temp 101*F), PR- 120/min, regular, pulsus dicroticus, low volume, no radiofemoral/radioradial delay, equal both sides, all PPWF, condition of vessel wall normal. RR- 22/min, abdominothoracic, BP- 100/70 mm Hg right arm supine, Pallor was present, no icterus,no cyanosis, no clubbing, no lymphadenopathy, minimal edema feet was present, JVP was raised (10 cm). There was no signs of active rheumatic activity/ infective endocarditis. On CVS examination,

Inspection-

- Precordial bulge was present
- Apex beat was lateral to the MCL
- Epigastric pulsations were present
- Pulsations were seen in the left 2nd ICS
- No visible scar mark was present.

Palpation-Apex beat was in the left 6th ICS in the anterior axillary line, 14 cms lateral to MSL

- Systolic thrill was present at the apex
- Diastolic shock was present in the pulmonary area
- Epigastric pulsations were felt on the tip of the finger
- Parasternal heave was present.

Percussion-

- Cardiac borders were delineated
- Left lateral border coincided with the apex
- Right heart border was retrosternal

Auscultation-

- Mitral Area-
- S1 was soft
- P2 was loud
- S3 was present

Pansystolic murmur was present radiating to the left axilla and back, Grade IV/VI, high pitched, better heard with diaphragm and in expiration, exazerbated by maneuvres like hand grip, leg elevation and squatting from standing position.

Tricuspid Area-

S1 was soft
P2 was loud

Pansystolic murmur was present, no radiation, high pitched, better heard with diaphragm and in inspiration, grade III/VI.

Pulmonary Area-

P2 was loud, no murmur

Aortic Area-

A2 was normal, no murmur.

Other systems-

- RS- Trachea was central
- No kyphosis/ scoliosis
- B/l fine basal crepitations were present P/A-

- Liver was just palpable, soft, tender
- Spleen was not palpable
- Bowel sounds were presentCNS-

Patient was conscious, oriented, no FND.
Differential diagnosis was kept as -

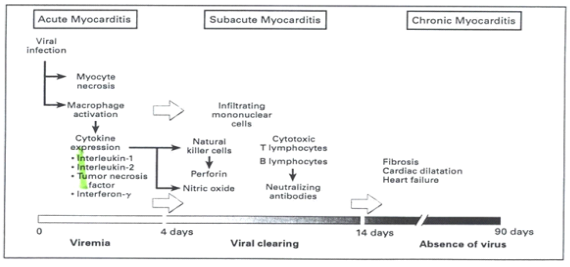
1.Rheumatic heart disease
- Severe MR
- Pulmonary hypertension
- Severe TR
- CCF
- Anemia
- Viral Fever

2. Chronic Viral Myocarditis with sequelae.
The investigations done showed-CBC
- Hb- 8.7 g%
- TLC- 11,400/cu.mm
- P71/L26/M2/E1
- MCV- 60 fl
- RFT
- Bl Urea- 33 mg%
- Sr Creat- 1 mg%
- Sr Uric Acid- 6 mg%
- Sr Calcium- 8.6 mg%
- Sr Phosphorus- 2.9 mg%
- Sr Sodium- 140 mEq/Lt
- Sr Potassium- 4.0 mEq/Lt
- RBS- 132 mg%
- ESR- 60 mm 1st hour
- Viral markers- negative
- LFT-
- Bilirubin (T)-1.8 mg%
- (D)-0.7 mg%
- Sr AST/ALT- 680/1475 IU/Lt
- Sr Alk P- 212 IU/Lt
- Sr GGT- 68 IU/Lt
- Urine R,M- normal
- Typhidot- negative
- USG Abdomen-
- ? Acute Hepatitis (?Congestive)
- Mild ascites
- B/l pleural effusions (Rt>Lt)
- CPK-MB 32 IU/Lt
- CXR(PA)- Cardiomegaly
- ECG- LVH, LAA, T-wave inversions leads V3-V6
- TFT- normal
- Sr Iron Studies-
- Sr Iron- 18 mcg% (65-175)
- TIBC- 302 mcg% (250-450)
- % Satn- 6% (13-45)
- Sr LDH- 339 IU/Lt (100-190)
- ASO titres- 124 IU/ml (0-200)
- ANA- positive 1+
- end point titre of 1:80
- ds-DNA negative
- Toxoplasma IgM, IgG- negative
- Rubella IgM, IgG- negative
- HSV 1&2IgM, IgG- negative
- LeptospiraIgM- borderline
- IgG- negative
- CMV IgM- 0.30 IU/Lt(normal <0.8)
- IgG- 20 IU/Lt (normal <0.8)
- Echocardiography-
- Dilated cardiomyopathy
- Severe MR, TR
- Severe pulmonary hypertension
- EF 20% .
- So, the final diagnosis was made as-
- Dilated Cardiomyopathy,
- Secondary to CMV induced chronic myocarditis,
- Severe MR,
- Pulmonary hypertension,
- Severe TR,
- CCF,
- Iron deficiency anemia.
- The treatment given included-
- Cardiac monitoring was done,
- Diuretics were given,
- Digoxin,
- Beta Blockers,
- ACE Inhibitors,
- Ganciclovir,
- Iron preparations.

The patient improved with the treatment and was discharged with an advice to consult the cardiology dept at PGI, Chandigarh for the need of ICD insertion and cardiac transplantation as it is not available at our centre.

Discussion:-

Manifestations of myocarditis range from asymptomatic or nonspecific illness to acute cardiogenic shock and sudden death. Infants and young children more often have a fulminant presentation with fever, respiratory distress, tachycardia, hypotension, gallop rhythm and cardiac murmur. Associated findings may include a rash or signs of hepatitis, aseptic meningitis. Patients with acute or chronic myocarditis may present with dyspnoea, chest discomfort, fever, palpitations, easy fatigability, or syncope. Hepatic enlargement, peripheral edema, and pulmonary findings such as wheezes or rales may be present in patients with decompensated heart failure.



Time Course of Experimental Viral Myocarditis in Mice.
Adapted from Kawai[11] with the permission of the publisher. The timeline is not drawn to scale.

The causes include-
- Bacterial
- Staph aureus
- Diphtheria
- Lyme disease
- Salmonella
- Tuberculosis
- Beta haemolytic streptococci
- Meningococci
- Leptospirosis
- Viral
- Coxsackie B virus
- HIV
- Influenza
- Poliomyelitis
- HCV
- CMV
- EBV
- Fungal
- CandidaBlastomyces
- Cryptococci Aspergillus
- Rickettsia
- R typhii
- R tsutsugamushi
- Chlamydia
- Protozoa
- Trypanosomiasis
- Toxoplasmosis
- Acute Rheumatic Fever
- Physical Agents
- Radiation
- Heat stroke
- Chemicals
- CobaltArsenic
- Antimony
- Drugs
- Phenothiazines
- TCAD
- Emetine
- Penecillin
- Methyldopa

- Fiedler's Giant Cell Myocarditis
-

Criteria for diagnosis according to the New York Cardiology Association is-

- Presence of proofs of the clinical or laboratory infection
- Cardiomegaly
- Changes of rate - tachycardia
- Pathological changes in ECG
- Congestive cardiac failure
- Echo evidence

Presence of at least 3 signs testifies to the credible diagnosis of carditis[2].

DCM in children is characterized by varying degrees of dilatation of ventricles, most prominently the left. LV dilatation and systolic dysfunction cause increased heart size and weight, ventricular dilatation, normal wall thickness, heart dysfunction out of portion to fibrosis. An enlarged LV with decreased systolic function as measured by LVEF characterizes the DCM. Incidence in children < 18 years is approx 0.57cases/100,000 persons/year. African-American children have a higher incidence and worse outcome. It is more common in HIV-1 infected persons and childhood cancer survivors. Approximately 40% undergo cardiac transplantation or die within 5 years of diagnosis. Cardiac transplantation carries a lifelong risk of immunosuppression**.** Older age, worse LV function and more advanced heart failure are associated with worse outcomes. Cardiac transplantation remains the only and definitive treatment for children progressing to end stage heart failure. Non-white race, presence of myocarditis are asso with a higher risk of death after transplantation. The cause - a genetic basis or are the sequelae of viral myocarditis. In 20-50 % of cases, the disease is recognized as familial. Autosomal dominant inheritance is most commonly encountered and mutations in several cardiac structural or metabolic genes have been identified. Failure of the LV causes an increase in end-diastolic volume, which results in increase in LA, pulmonary venous and pulmonary capillary pressure. Mitral valve regur gitation may result from papillary muscle dysfunction or severe dilatation of the valve annulus. All age group may be affected. The onset is insidious, but sometimes symptoms of heart failure occur suddenly.Irritability, anorexia, abdominal pain, cough from pulmonary congestion and dyspnoea with exertion are common.Infants and younger children have respiratory symptoms and failure to thrive. The diagnosis is based on-

- Clinical signs
- Chest x-ray
- CT Scan
- Echocardiogrphy
- 12-lead ECG
- Cardiac biopsy
- MRI (magnetic resonance imaging)
- Cardiac catheterization

Echocardiography usually shows, dilatation of the LA and LV, poor contractility

Doppler studies usually show, decreased flow velocity through the aortic valve and mitral regurgitation. In long-standing cases, evidence of Pulmonary hypertension may exist. Serious complications_include ventricular arrhythmias leading to syncope and sudden death, pulmonary or systemic emboli from intracardiac thrombi and development of pulmonary vascular disease from chronically elevated left atrial pressure. The course of disease is progressively downhill, although some patients may remain stable for years. Treatment of HF may result in temporary remission, but relapses are common and in time – patients become resistant to therapy. Prognosis for survival beyond a year is poor. Patients with severely depressed myocardial function should be monitored for arrhythmias and, if present, treated aggressively with antiarrhythmic agents or an implantable cardioverter-defibrillator (ICD). In the management of DCM, limit activity based on functional status, salt restriction of a 2-g Na^+ (5g NaCl) diet, fluid restriction for significant low Na+, initiate medical therapy- ACE inhibitors, diuretics, digoxin, metoprolol, carvedilol. Consider adding ß-blocking agents if symptoms persists. Anticoagulation (history of thromboemboli, presence of mural thrombi), intravenous dopamine / dobutamine, cardiac transplantation[3].

Conclusion:-

This was a case of non-rheumatic carditis in a young male. Patient had presented with dyspnoea of gradual progression. On investigation it was found that the cause was chronic CMV infection. Treatment was started and the patient responded to Ganciclovir. This case shows the importance of early recognition of the cause of DCM before dire complications occur so that the cause specific treatment can be given early. It also shows that the cause of DCM is not only ischaemic but there are many other rarer causes as well. It also alerts us to the fact that DCM should be looked for when suspected, in the young also.

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