

**WCC 2016-118 : A RARE CASE REPORT OF CARDIAC SARCOIDOSIS-** Raghu Kishore Galla .

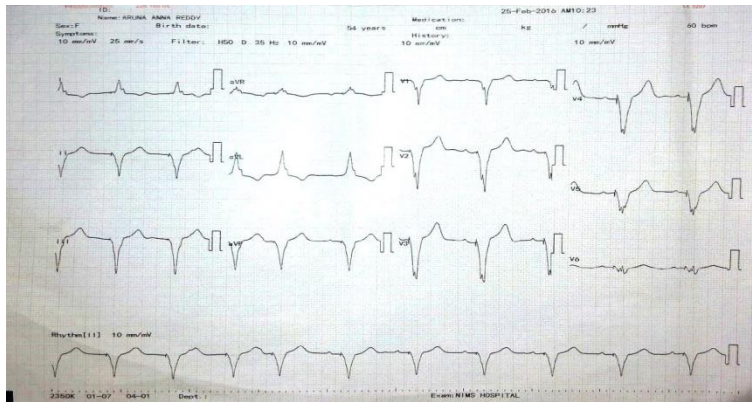
**BACKGROUND**

Sarcoidosis is a multisystem disease characterized histologically by the formation of granulomas in many tissues. Most patients present with pulmonary involvement of whom approximately half have asymptomatic pulmonary involvement, characterized by hilar lymphadenopathy on chest x-ray. Cardiac involvement in patients with sarcoidosis is being increasingly recognized, involving 10-25% of the patients of sarcoidosis and is associated with poor prognosis. Although environmental and genetic factors have been implicated in its pathogenesis, the aetiology of cardiac sarcoidosis remains obscure. Clinical manifestations include advanced heart block, arrhythmias, and congestive heart failure. To date, cardiac sarcoidosis has been extremely difficult to diagnose clinically because the clinical manifestations are non-specific, and the sensitivity and specificity of diagnostic modalities are limited. The optimal management of cardiac sarcoidosis has not been well defined. Although corticosteroids remain the mainstay of treatment, there is little evidence for the optimal initiation, dosage, or duration of therapy. Here we are reporting a case of cardiac sarcoidosis presented with advanced heart block, congestive heart failure, CAD and other manifestations.

Case report - A 56 yrs/ female, came with chief complaints of shortness of breath on exertion for 2 months , Orthopnea and PND for the last 1 week. Pedal edema and facial puffiness for the last 3 days. SOB is insidious in onset, started as NYHA class II, progressed to NYHA class IV, associated with dry cough, orthopnea and frequent PND episodes during the night times. H/o bilateral pitting edema of feet and facial puffiness for the last week and H/o fatigue present. No h/o chest pain, palpitations or syncope or No h/o decreased urine output or fever.

Past history - Known HTN and known DM for 20 years on insulin. Known Hypothyroid for the last 15 years. k/c/o CHB S/P PPI (VVI) in 2007 (Medtronic). S/P Change of PG of pacemaker and upgradation to DDDR mode 2012. H/O CAD and PTCA to RCA in 2007 and PTCA to proximal and distal RCA in 2015. H/O Acute Left common femoral artery occlusion s/p successful thrombolysis by urokinase 2002. H/O ureteric stenting in 2002. H/O ? ILD (NSIP type) in 2014 treated with short course of steroids. H/O recurrent admissions for heart failure 3 times in the last year. H/O AKI recovered in 2014 after PTCA. No H/O drug non compliance.

ECG showed



2D Echo showed global hypokinesia of LV, severe LV dysfunction with EF 28%, moderate MR and grade 1 diastolic dysfunction. No TR/AR/PAH, No PE/Vegetation/Clot. Holter recording – uneventful. HRCT chest showed Extensive ground glass opacities of both the lung fields with mosaic attenuation, Cardiomegaly and mediastinal lymphadenopathy. Investigations showed Hb 12.8 gr%, TLC 11,800 PC 2.0 L, RBS 132 Urea 28, S.Creatinine 0.7, Na 132, K 3.8, CL 104, TSB 0.8, DB 0.2, SGOT 31, SGPT 28, ALP 85 TP 6.8, Albumin 3.8. PT 14 sec. INR 1.1, APTT 28 sec, CPK 142, LDH 256, NT Pro BNP 24800. F 18 FDG PET scan showed hypermetabolic mediastinal and right axillary lymphadenopathy. Diffuse ground glass opacities involving bilateral lung fields with minimal heterogeneous metabolic activity. Abnormal multifocal patchy metabolic activity in the LV myocardium- ? Sarcoidosis. Patient started on steroids, improved symptomatically including functional class. On regular follow up with optimal medical therapy for the CCF