Case Report

A Case Report of Covid-19 Associated Mitral Stenosis: A Causative Association?

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Abstract

Back Ground: *Coronavirus disease 2019 (COVID-19) has reached the pandemic level and is causing a numerous post viral effects including the cardiovascular system. The present case focuses on Covid -19 and its relation with the valvular heart disease.*

Case Summary: *A 36 year old female, presenting with complains of persistent breathlessness for 3 months post Covid-19 infection. She had no risk factors except for hypertension. Patient was evaluated for the same and was found to have Mild mitral stenosis. Rheumatic heart disease was ruled out by necessary examinations and laboratory tests. Patient was managed with diuretics and was advised regular follow-up.*

Discussion: *Elevated inflammatory markers like IL-6 and CRP might have been implicated in the pathogenesis of the Development of Mitral Stenosis.*

Previously, other viral infections have also been linked to the causation of various valvular lesions. However, the pathogenetic mechanism is still unclear and needs further studies.

Conclusion: *Covid -19 infection with higher inflammatory markers might be responsible for the development of the Mitral stenosis. However, further cases and studies are needed to support the finding.*

Keywords: *Covid-19, Mitral stenosis, Valvular heart disease, breathlessness.*

Introduction

Coronavirus is a single stranded RNA virus, which is prone for rapid mutations and recombinations, resulting in variations in its presentations and sequelae. The potential effects of covid-19 on the cardiovascular system has been published which include its effect in causing myocardial infarction, myocarditis, arrhythmias and heart failure. The present case discusses the relation of covid-19 disease in the pathogenesis of mitral stenosis in a young, non-smoker female.

Case Presentation

A 36 year old female, moderately built, non smoker, came to the OPD with history of unresolving dry cough and breathlessness NYHA Grade II. Apart from this, she had no other complaints. Previous history revealed that she had Covid-19 infection 3 months back. She was treated with anti – pyretics, anti tussives, low dose steroids and other supportive medications, after which she recovered except for persistent cough and breathlessness. Breathlessness, which was initially Grade III decreased to Grade II over 3 months. She had no history of chest pain, palpitations, pedal edema or syncopal attacks. She had no history of repeated URTI or LRTI or previous hospitalizations. She is a known hypertensive since 7 years on treatment with CCB. Her menstrual history was insignificant. She has 2 children with last child birth 7 years back, both full term normal vaginal deliveries with no complications. There was no history of any drug allergies or medications. No History of loss of weight or any arthritis or skin lesions was present. Family history is insignificant.

On examination, she was afebrile with PR 84/min, BP 126/82 mm Hg, SpO2 98% on Room air and RR 16/min. Chest expansion was equal on both sides. On Auscultation, S1 and S2 were heard. There were

no additional sounds. Breath sounds were clear and equal on both sides. There was no rash or erythema or any skin lesions noted. There were no lymphnodes palpable on general examination.

Patient was evaluated for persistent breathlessness with a number of required tests. Her Hb% was 12.5% with Normocytic and Normochromic RBC ruling out Anaemia. TLC was 7500 with normal distribution. Platelets were adequate. These CBP findings ruled out active bacterial infection.ASO and CRP levels were normal. ESR was within normal limit ruling out tubercular etiology. Chest X Ray PA view showed both lung fields clear with normal markings. Cardiac size appears to be normal. No pleural or pericardial effusion. No signs suggestive of Pulmonary Hypertension or Pulmonary edema. Pulmonary function test revealed good tidal volume with normal peak expiratory flow rate. ECG showed Normal sinus rhythm with no significant ST-T changes. Lipid profile shows mild elevation of LDL levels. Total cholesterol, HDL and VLDL levels were within normal limits. D- Dimer was negative. CK- MB within normal limits and Troponin I was negative.

2D Echo was done which showed mild concentric left ventricular hypertrophy with IVS and PW measuring 1.2cms. LV function was good with EF of 61%. There was no obvious RWMA noted. Mitral valve septal leaflet showed hockey stick like appearance with restricted mobility and thickening. The Mitral valve area measured with planimetry method was 1.84 cm2, which signifies that there was mild mitral stenosis. Thickening of the commisure was noted. The subvalvular apparatus was not severely thickened. The rest of the parameters on echocardiography were within the normal limits. There was no pericardial effusion. No clots or vegetations were noted during the study. The flow gradient across the other valves was normal without any significant regurgitations.

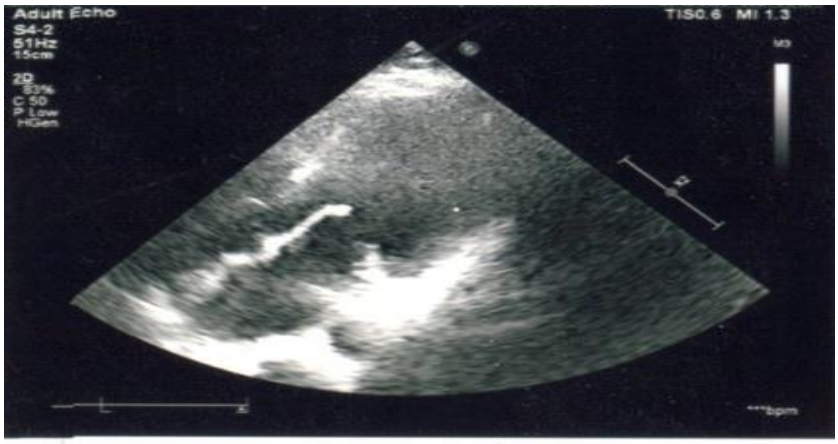

Fig 1: Mitral valve depicting typical hockey stick appearance suggestive of Mital stenosis in apical view.

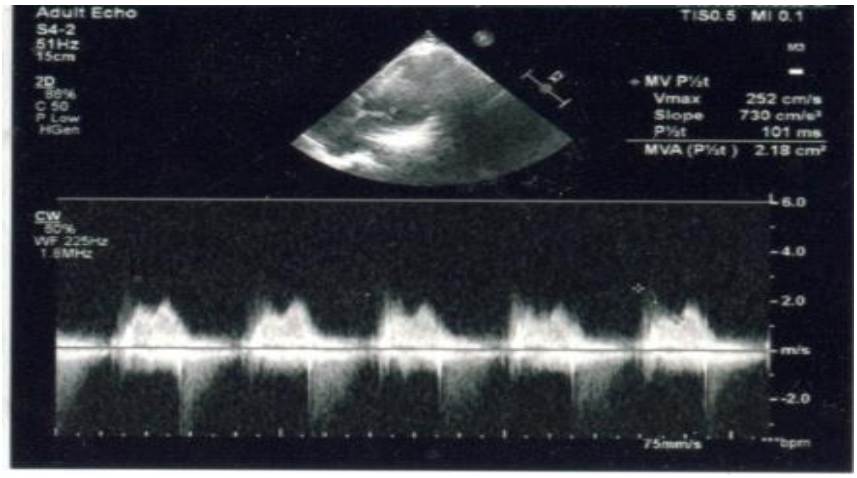

Fig 2: Mitral valve area by PHT method showing mild mitral stenosis.

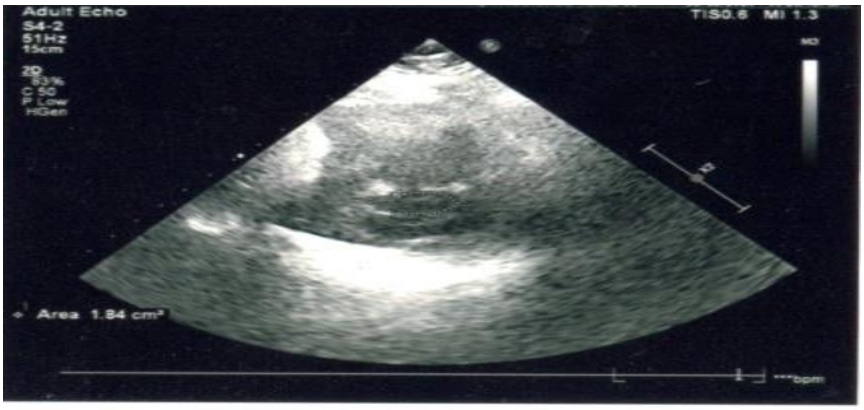

Fig 3: Mitral valve area by Planimetry method showing mild mitral stenosis.

Patient was diagnosed to have mild Mitral Stenosis, possibly post viral. The patient was managed with cardioselective beta blockers and diuretics. Patients improved symptomatically. She was advised for medical management and regular periodic follow up with ECG and 2D Echo and follow up in case of exacerbations.

Discussion

A number of studies has recently been published that link Covid-19 infection to various cardiac complications like heart failure1, arrythmias2 and myocardial infarction3. However, till date, there is rarely any case that clearly relates Covid-19 disease manifesting as a valvular heart lesion. Rheumatic heart disease still remains one of the leading causes of the valvular heart disease in developing nation. In this case, a young female had no history or symptoms suggestive of Rheumatic Heart Disease. Only significant history was of Hypertension and recent Covid-19 infection.

Viral infection, specifically HIV4 and Coksackie virus5 has previously been linked to the causation of valvular heart disease. Studies has shown that even H1N1 has been associated with valvular heart disease especially Mitral Stenosis.6 Based on the data available linking the viruses and the valvular disease, it is possible that Covid-19 disease causing Corona virus may play a role in the pathogenesis of development of denovo Mitral Stenosis in this particular case.

Covid-19 infection is associated with rise in inflammatory markers.7 C reactive protein is a type of protein that is produced in the liver and Serves as a marker of infection and inflammation.8 Elevated levels of CRP were observed in upto 86% in severe Covid-19 patients.9 Many previous studies have shown that inflammatory markers like C-reactive protein is associated with active phase and progression of the valvular heart disease. Persistant elevation of the CRP is also associated with chronicity of the valvular disease.10 Elevated CRP levels might have been implicated in the development of Mitral Stenosis in this case.

Interleukin 6 is a cytokine that controls the immune response in addition to cell proliferation and differentiation. The triggering of the IL-6 after a viral infection causes a fatal immune reaction due to the hyperactivation of the T cells.11 IL-6 levels correlated with the severity of the COVID-19 disease including pulmonary inflammation and extensive lung damage.12 IL-6 play a significant role in the pathogenesis of Aortic stenosis. Pi mediated activation of NF-kB regulates positively IL-6 which entrain mineralisation of the valvular interstitial cells.13 This increase in the IL-6 levels might have played a role in the development of Mitral stenosis.

Covid-19 disease was associated with significantly higher levels of serum ferritin, correlating directly with the severity of the disease.14 Studies have shown that H-ferritin inhibits mineralization and osteoblastic differation of human valvular interstitial cells. It also decreases the expression of inflammatory markers like TNF α and IL-1β in valvular interstitial cells15. This may act as a protective factor in preventing the mineralisation of the heart valves.

Conclusion

Pre existing cardiovascular disease is a documented risk factor for severe Covid-19 infection. But the literature of the cases and studies analysing the effect of Covid-19 disease on the heart valves is limited and in preliminary phase. The present case represents the valvular effect of covid-19 virus, resulting in development of denovo Mitral Stenosis. However, further studies and trials are needed to confirm whether this is just a coincidental finding or a causal relationship exists.

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