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Uncommon manifestation of common disease:"Cardio Vocal Syndrome"

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Abstract
We are presenting a case of 19 year female who was symptomatic since 4-year's forprogressive shortness of breath and 3 months for hoarseness of voice. Physical examination revealed a mid-diastolic murmur. Echocardiography confirmed severe mitral stenosis with severe pulmonary hypertension. Indirect laryngoscopy revealed left vocal cord palsy.She underwent successful percutaneous balloon mitral valvotomy.Improvement in voice quality was noted in subsequent visits.

Keywords:
Mitral stenosis (MS), New York Heart association(NYHA), Jugular Venous Pressure(JYP), Percutaneous Ballon Mitral Valvotomy(PBMV),Midclavicular line (MCL), Intercostal space (ICS),Left Recurrent Laryngeal Nerve Palsy (LRLN),Echocardiography(ECHO)

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I. Introduction

Rheumatic mitral stenosis is commonly seen cardiac disease in our country. Hoarseness of voice due to left recurrent laryngeal nerve palsy is an uncommon manifestation of this common disease.Ortner syndrome or cardiovocal syndrome is a rare condition characterized by hoarseness of voice associated with cardiovascular pathology. Mitral stenosis is a common cause, but several cardiac and non-cardiac conditions may be responsible as well. This case illustrates an uncommon manifestation of common disease.

Uncommon manifestation of common disease: "CardioVocal Syndrome"

19 year female was admitted withprogressive shortness of breathsince 4 years. She had paroxysmal nocturnal dyspnea since 4 months and hoarseness of voice since 3 months.She was admitted thrice in last 4 months for heart failure.No history suggesting of infective endocarditis or stroke in past.No history suggestive of rheumatic fever in childhood. On examination she had mitral facies.Her pulse was 84/min, regular with low volume.Her blood pressure was 92/70 mmHg.JVP was raised. There was no peripheral oedema, cyanosis or clubbing. Cardiovascular examination revealedtapping apex beat in 5th ICS medial to MCL.Left parasternal heave with diastolic shock was present. On auscultation, the first heart sound was loud and the pulmonary component of the second heart sound was prominent at the apex and pulmonary area respectively. A grade 3/6 mid diastolic rumbling murmur with presystolic accentuation with opening snap was heard at the apex in left lateral position with bell of stethoscope.A grade 3/6 pansystolic murmur was heard at left lower parasternal region which increases in intensity with inspiration. Breath sounds were normal with no added sounds. Abdominal and neurological examination was normal.Blood investigations including complete hemogram,ESR,Kidney function test and liver function test were normal. ECG showed normal sinus rhythm with right axis deviation and left atrial enlargement and right ventricular pressure overload pattern.Chest Xray PA view revealed straightening of the left heart border and enlarged main pulmonary artery (trunk) suggesting significant pulmonary hypertension (Fig 1). M mode showed enlarged left atrium{Left atrial diameter =40mm}(Fig 2).2D ECHO showed mean pressure gradient across mitral valve of 14 mmHg(Fig 3).Planimetry showed mitral valve area of 0.4 cm2(Fig 4). Patient also had severe tricuspid regurgitation (Fig 5) with severe pulmonary hypertension (Fig 6).Wilkin's score was 8/16.Other valves were normal.No clot was seen in left atrial appendage on transesophageal echocardiographyprior to PBMV.In view of hoarseness of voice patient was referred to ENT where indirect laryngoscopyshowed left vocal cord palsy. As patient was symptomatic with severe mitral stenosis with pliable valve sheunderwent successfulPBMV. Post procedure, the mid-diastolic murmur was no longer audible. On subsequent visits improvement in voice quality was noted.

II. Conclusion

Cardiovocal syndrome is rarely seen nowadays due to greater awareness of the disease and earlier intervention.In our case, the left recurrent laryngeal nerve was most probably compressed by the dilated pulmonary artery, not by the left atrium because the latter was only mildly dilated.Laryngeal nerve reassessment should be done yearly. Many times the hoarseness of voice gets reversed as iseen in our case but if doesn't recover then thyroplastyshould be strongly considered if patient develops aspiration pneumonia or wishes to improve voice quality.

III. Discussion

Ortner syndrome was first described by NobertOrtner, a Viennese physician in 1897, in a case of mitral stenosis with dilated left atrium.(1)Subsequently it was reported with mitral stenosis, mitral regurgitation, atrial myxoma, primary pulmonary hypertension,thoracic aortic aneurysm,aortic dissection,pulmonary embolism, defibrillation,trans catheter ablation of atrial fibrillation, cardiothoracic surgery and heart-lung transplantation. The basic abnormality is paralysis of the left recurrent laryngeal nerve.Though an initially enlarged left atrium was thought to be the main culprit, the current understanding favours pressure in the pulmonary artery playing the most important role in causing the nerve compression in a majority of the cases.(2)

The incidence of cardiovocal syndrome in mitral stenosis ranges from 0.6% to 5%.(3)LRLN cause the vocal cord to be in paramedian position;on laryngoscopy the position can be variable.Symptoms include hoarseness,dysphagia and shortness of breathduring speech because of loss of air,which is secondary to glottis incompetence.Effective cough cannot be mounted.LRLN palsy can be significant risk for aspiration because the paralysed cord cannot protect from aspiration especially liquid.The degree of symptoms depends on the extent of paresis and compensation by other vocal cord.The most common manifestation of Ortner syndrome is hoarseness of voice. Unilateral vocal cord paralysis increases the risk of aspiration which may be present in up to 40% of patients.The recurrent laryngeal nerve paralysis is mainly due to the compression by an enlarged pulmonary artery as initially thought.(4)

Fetterroff and Norris showed distance between aorta and pulmonary artery at aortic window is only 4mm,and compression betweentwo structures is responsible for palsy.This hypothesis was further strengthened when compression of LRLN was observed by Ari et al.between aorta and pulmonary artery near the ligamentunmarteriosum in patients with mitral stenosis undergoing mitral commisurotomy.(5)

The prognosis of recurrent laryngeal nerve paralysis depends on the degree and duration of nerve compression. The treatment of unilateral vocal cord palsy consists of early rehabilitation, treatment of the primary etiology and endoscopic insertion of a prosthesis or injection of fat or collagen.There are two absolute indications for surgery: aspiration pneumonia and the patient's desire to improve the voice-related quality of life. (4)

If the symptoms are well tolerated without any evidence of aspiration, reassessment of laryngeal nerve can be made within a year.Those who remain symptomatic after observation should be strongly considered for surgery. Information regarding the reversibility of hoarseness in cardiovocal syndrome after correction of the underlying cardiovascular disease is limited. Of those reported 12 of 14 cases resolved within 1 week to 3 years, with duration of preexisting hoarseness ranging from 1 month to 10 years. It appears that the recurrent nerve palsy in cardiovocal syndrome can be reversed. The chance of recovery, however, might depend on the degree and duration of RLN injury (6).

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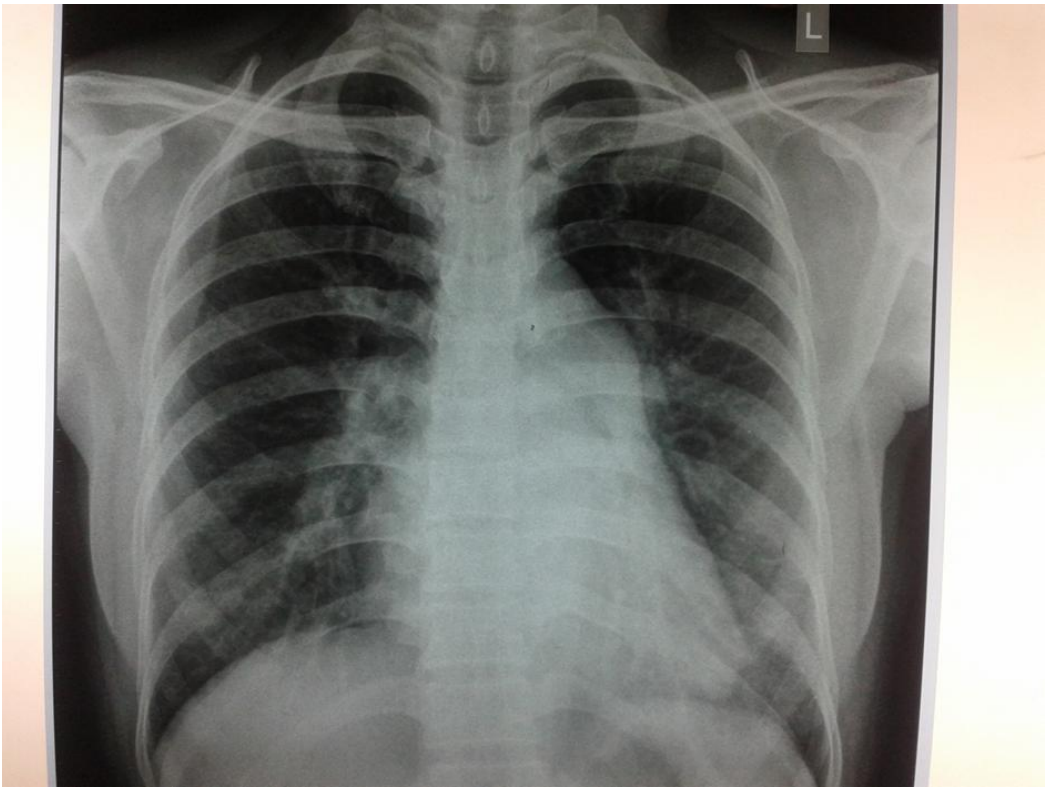

Fig 1: Chest X Ray PA view showing straightening of left heart border with dilatation of pulmonary trunk and prominence of right pulmonary artery.

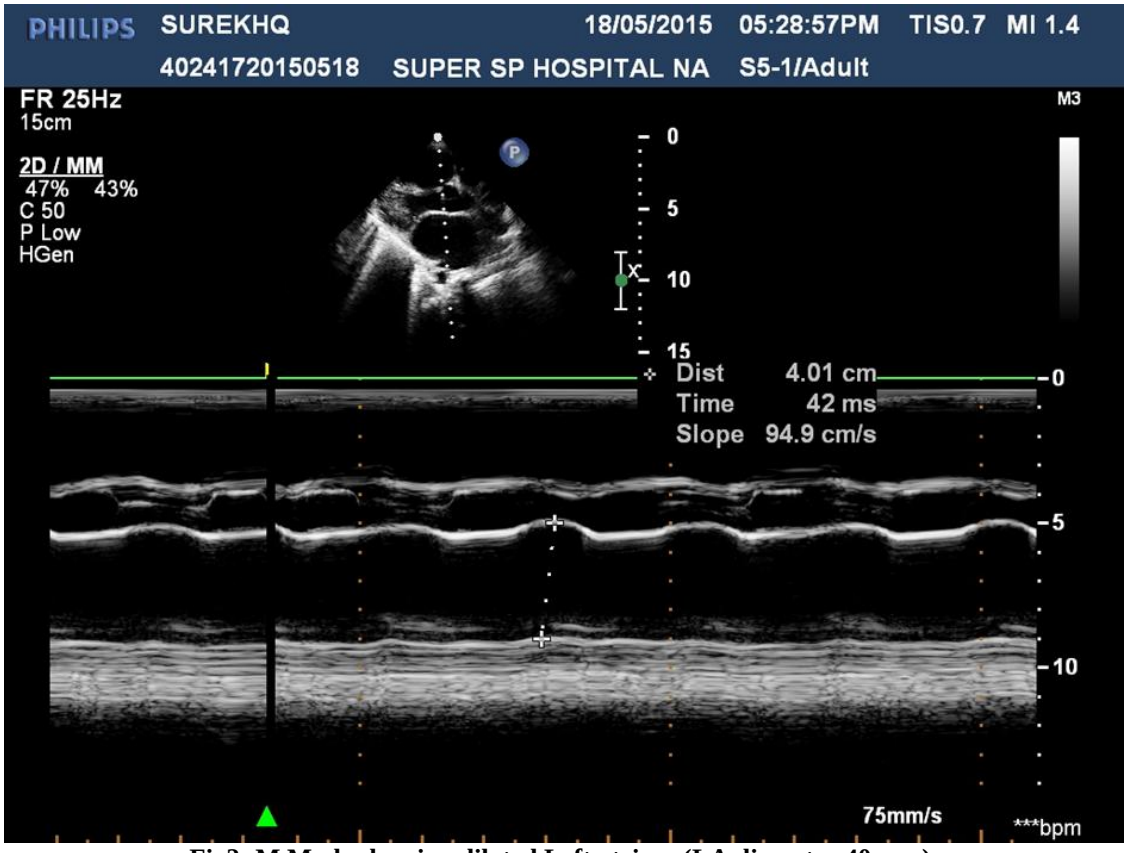

Fig2: M Mode showing dilated Left atrium (LA diameter 40 mm)

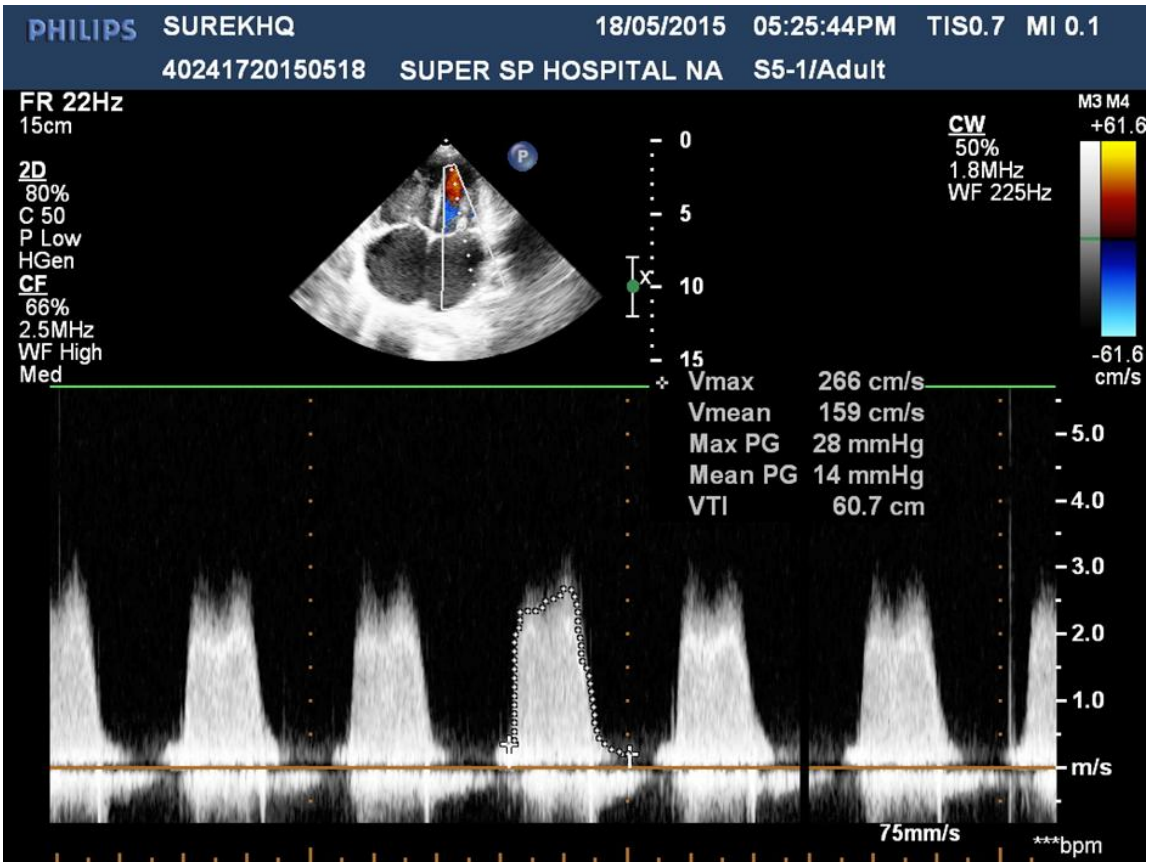

Fig3:2DECHO showing mean pressure gradient of 14 mmHg suggesting severe MS

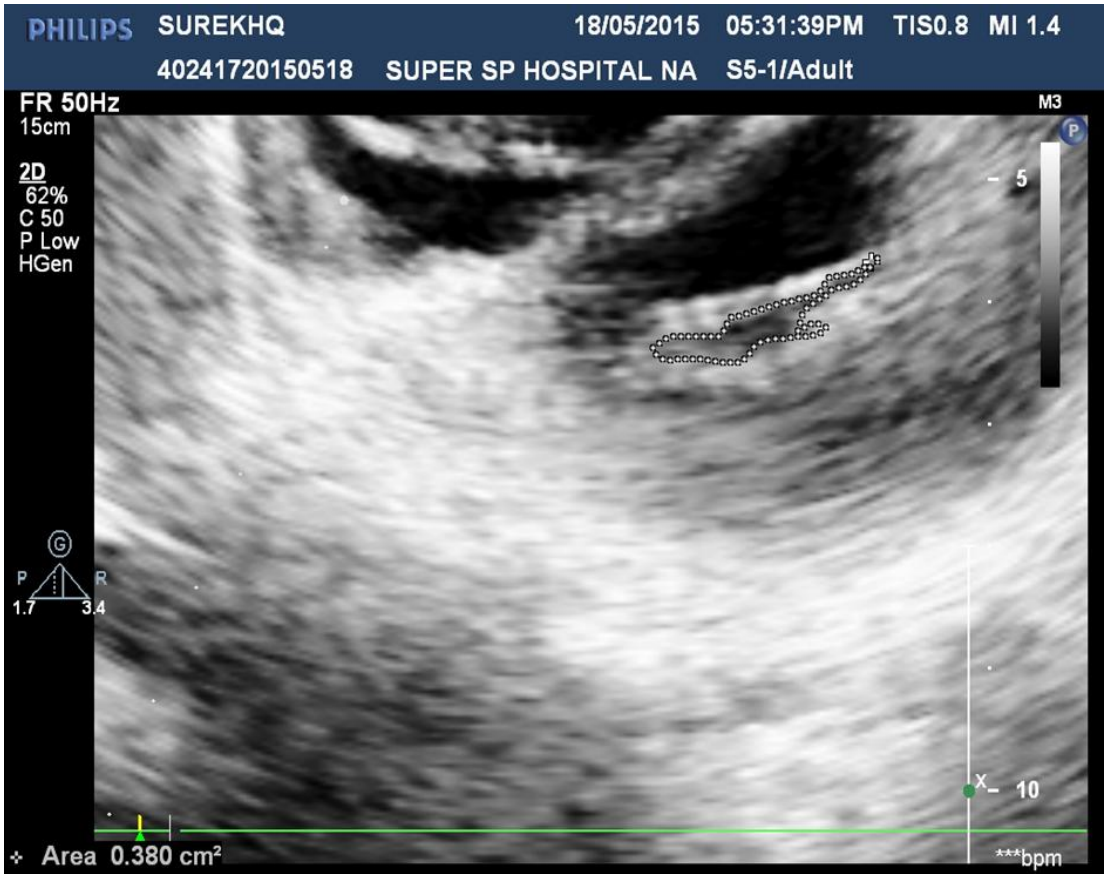

Fig4:2DECHO planimetry showing Mitral Valve area of 0.4cm2 suggesting severe mitral stenosis.

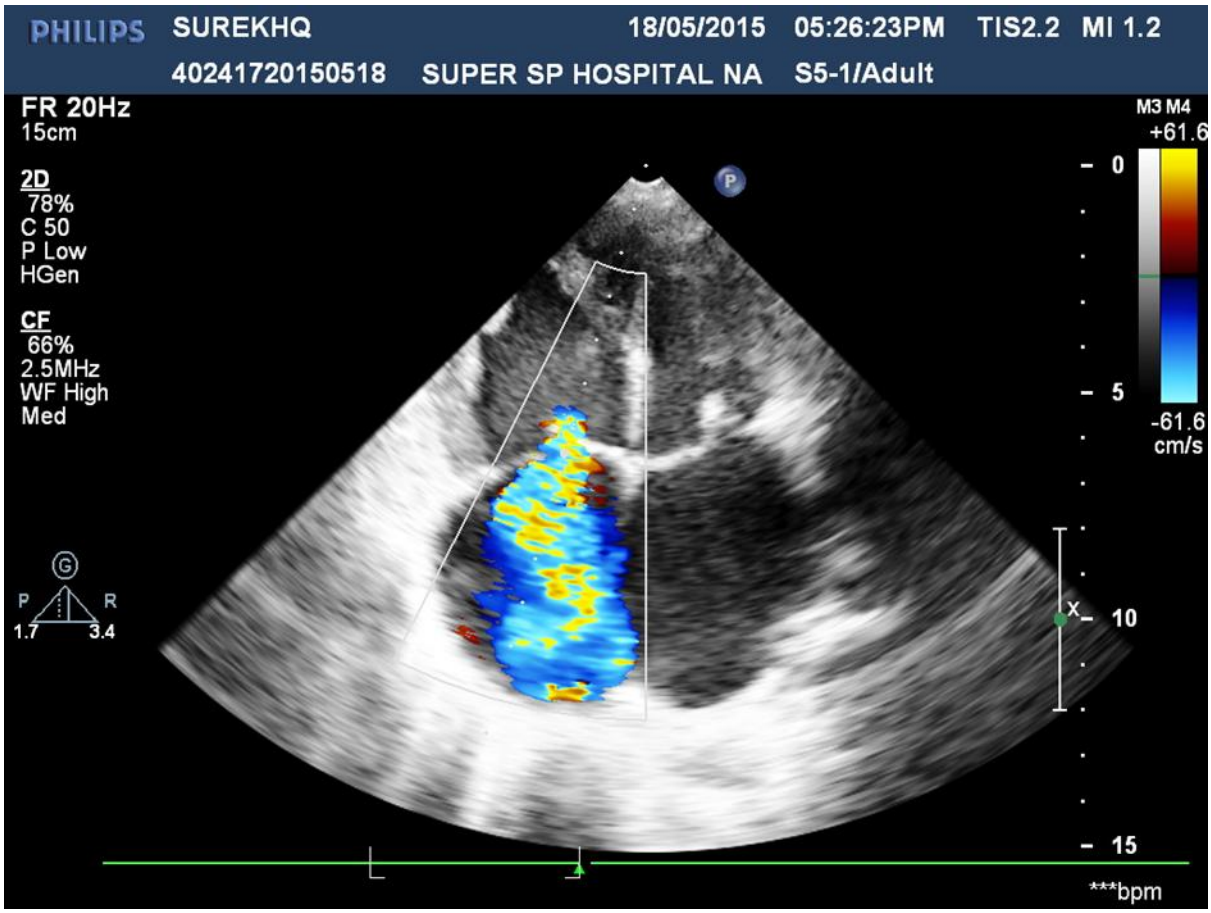

Fig5:2DECHO showing dilated Right atrium and right ventricle and severe Tricuspid Regurgitation

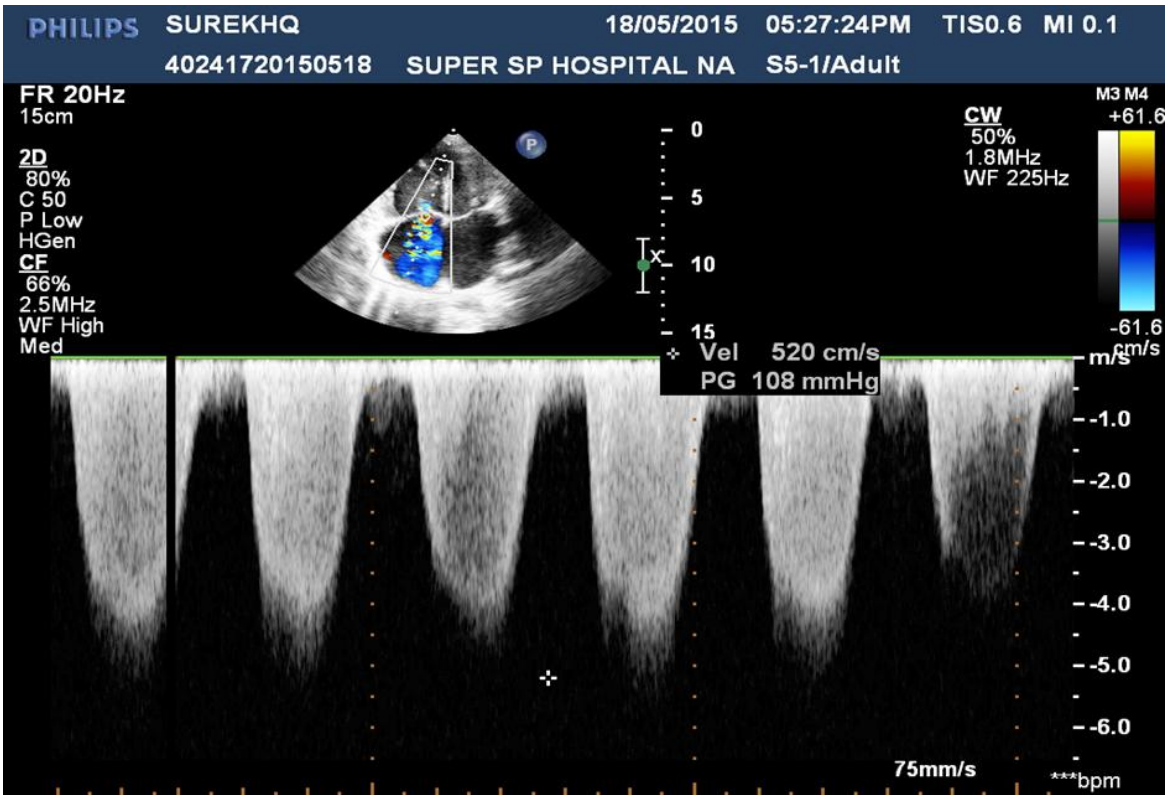

Fig6: 2DECHO in apical 4 chamber view showing severe pulmonary hypertension. (PASP by TR Jet 108 mmHg)