A 24 year female had normal echocardiographic examination for unexplained chest pain. There was no evidence of mitral or tricuspid valve prolapse or pericarditis. One month later she presented with fever, cough and chest pain. Mild cardiomegaly was seen on skiagram of chest. Echocardiography revealed moderate pericardial effusion. She did not agree for pericardial aspiration. ESR was 68 mm at 1 hr and Mantoux test was strongly positive (30 mm). She was advised antitubercular therapy along with prednisolone. She gradually became asymptomatic. After 1½ months she presented with pain in epigastrium and breathlessness. Jugular venous pressure was raised and liver was enlarged. Echocardiography confirmed clinical impression of mild mitral and tricuspid regurgitation secondary to pericardial constriction.
of pericardial constriction. Thickened pericardium (Figure 1A), dilated inferior vena cava (Figure 1B). Hepatic vein flow revealed increased diastolic flow, exaggerated respiratory variation and increased expiratory reversal (Figure 1C). Tissue Doppler imaging was normal (Figure 1D). However, colour Doppler imaging revealed mild mitral regurgitation (Figure 2A). Mitral valve structure (Figure 2B) and pulsed Doppler flow of mitral valve (Figure 2C) were normal. She was continued on antitubercular treatment, tapering dose of Prednisolone and small dose of Frusemide + Spironolactone. She recovered clinically. Repeat echocardiographic examination after another two months revealed persistence of mild MR and new appearance of minimal TR (Figure 2D) with normal right ventricular pressure.

Tricuspid regurgitation has been reported as a consequence of constrictive pericarditis. However, pathogenesis of such TR has not been explained. We could not find any reference of mitral regurgitation as a consequence of pericardial constriction. Sequential echocardiographic evaluation of this patient suggests that mild mitral and tricuspid regurgitation could be secondary to pericardial constriction. Earliest and maximal collection of pericardial fluid occurs in the posterior atrioventricular groove which is the most dependent portion in supine position. It is possible that during the process of healing, maximal fibrous tissue is also laid down along posterior atrioventricular groove. This could affect the functioning of posterior mitral annulus in a manner similar to that produced by fibrosis and calcification seen in “calcification of mitral annulus”. This can explain mild mitral and tricuspid incompetence secondary to pericardial constriction. Relatively thinner wall of right ventricle and right atrium could result in greater proximity of tricuspid annulus to pericardium and reported occurrence of tricuspid regurgitation in pericardial constriction. Our patient probably had greater and earlier involvement of mitral annulus resulting in earlier appearance of MR. Constriction predominantly localised to either inflow or outflow regions of one or more cardiac chambers are known. Myocardial fibrosis and atrophy accompanying long standing severe calcific constriction may also contribute to atrioventricular incompetence. However, our patient did not have any evidence of such pathology.

Pericardial constriction can produce mild mitral and tricuspid regurgitation in absence of any other structural disease of valve leaflets, chordae and papillary muscles. Fibrosis along posterior atrioventricular groove could hamper annular function.

References