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Original Article

An echocardiographic assessment of cardiovascular hemodynamics in patients with large pleural effusion

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ABSTRACT

Background: The close relationship between pleural space and pericardial space and the dependence of their pressure kinetics are well known. This study evaluates the effects of increased intra pleural pressure due to pleural effusion on cardiovascular system.

Methods: Forty patients above the age of 12 who had massive unilateral/bilateral pleural effusion due to non-cardiac etiology were included in the study. Therapeutic thoracocentesis was done for massive pleural effusion. The echocardiographic parameters measured before and after thoracocentesis were compared.

Results: Mean age of the patients 46.6 years. Out of 40 patients 8 were females (20%). 7 patients had right atrial collapse on echo. 85% of patients had significant flow velocity changes across both tricuspid valve and mitral valve during phases of respiration.11 patients (47.82%) had IVC compressibility of <50% during inspiration. Mean flow velocity respiratory variations across tricuspid valve before thoracocentesis and after thoracocentesis E 45.04 ± 10.3,32 ± 11.3% (p value <0.001), A 53.71 ± 28%, 32.08 ± 12.5% (p < 0.001) across mitral valve E 32.30 ± 12%, 19.78 ± 7.8% (p < 0.001), A 26 ± 11.2%, 21 ± 9.3% (p 0.006) across pulmonary artery 42.63 ± 31.3%, 17.70 ± 6.2% (p < 0.001), across aorta 21.57 ± 11.4%, 14.08 ± 7.6% (p < 0.001).

Conclusion: Large pleural effusion has a potential to cause adverse impact on the cardiovascular hemodynamics, which could manifest as tamponade physiology. Altered cardiac hemodynamics could be an important contributor in the mechanism of dyspnea in patients with large pleural effusion.

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1. Introduction

Pleural effusion is one of the common medical emergencies. It is well recognized that pleural effusion can compromise pulmonary function resulting in dyspnea. The close relationship between pleural space and pericardial space and the dependence of their pressure kinetics are well known. Many authors have studied the effects of pleural effusion on the pulmonary function and diaphragmatic contraction.[1–4] Literature is scarce in this specific issue about the effects of pleural effusion on cardiovascular hemodynamics[5,6] The mediastinal pleura and the pericardium are closely related entities.[7,8] It has been shown in a canine model that large bilateral pleural effusions lead to an increase in intrapleural pressure, which causes a linear increase in intrapericardial pressure, finally leading to right ventricular diastolic collapse.[5,9] This suggests that cardiac tamponade physiology can occur in situations other than pericardial effusion. In this context this, study was undertaken to analyse the impact of pleural effusion on cardiovascular hemodynamics.

2. Patients and methods

The study was conducted in Rajiv Gandhi Government General Hospital, Chennai which is a tertiary care center between February to December 2011. All patients with large pleural effusion who were admitted to our medical ward were enrolled in the study. The clinical data like pulse rate, respiratory rate, blood pressure, saturation and systemic examination were noted (Table 1). The size of the pleural effusion was graded as mild, moderate and large based on the chest X-ray (Fig 1). Effusion involving more than $^{3}/_{4}$ th of the lung field on the chest X-ray was considered a large effusion.[10] Computed tomography scan of the chest was done in all the patients to assess the thickness of pericardium to rule out any constriction.

Table 1 – Baseline characteristics of patients.

Age	46.6 years (14–56 years)
Male	32
Female	8
Unilateral (right sided effusion)	30
Unilateral (left sided effusion)	7
Bilateral	3
Clinical parameters	
Pulse rate	101.75 ± 4.8
Respiratory rate	20 ± 4
Spo2	97% ± 1.3%
Systolic blood pressure	110.2 ± 9.2 mm Hg
Diastolic blood pressure	75 ± 7.3 mm Hg
Co morbid conditions	
Diabetes	7
Systemic hypertension	0
Renal failure	0
Tuberculosis	25
Malignancies	14
HIV	0
Heart failure	0
Constrictive pericarditis	0
Pericardial effusion	0
Hepatic failure	1

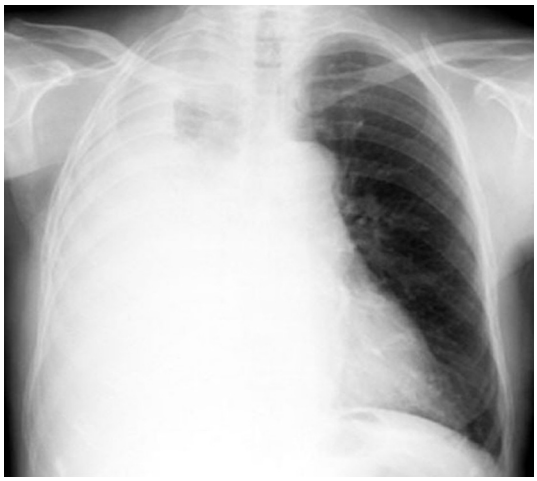

Fig. 1 – Chest X-ray PA view showing large right sided pleural effusion.

Out of 45 patients who had unilateral or bilateral pleural effusions of non-cardiac etiology, 40 patients who had large pleural effusions were included in the study. 5 patients were excluded from the study because of their moribund status and inability to obtain consent. Patients with smaller pleural effusions and pericardial effusion of any degree were excluded. In bilateral pleural effusion, the patients were included if at least one side of the effusion was more than moderate. The patients were transferred from medical ward for echocardiographic analysis.

Echocardiography was performed in all patients with reference to 2-D echocardiography to measure chamber dimensions and functions and pulse wave Doppler across mitral, tricuspid, pulmonary, aortic valves during quiet respiration (Table 2). Flow velocity across tricuspid, mitral, pulmonary, aortic valves and superior vena cava were measured during inspiration and expiration. IVC diameter and its collapsibility during inspiration and pulmonary artery pressures were measured (Fig. 3). Then the patients were transferred to medical ward for thoracocentesis.

All the patients underwent therapeutic thoracocentesis for massive pleural effusion if clinically indicated. About 2000–3500 ml of fluid was drained. A check chest X-ray was taken to assure that the pleural effusion remained less than ½ of a hemi thorax and all the clinical and echo parameters repeated within 24 h of thoracocentesis. Pre- and post-thoracocentesis, clinical and Echo parameters were compared. The mitral and tricuspid E velocities during inspiration and expiration were measured and the percentage of change in velocity was obtained. A change of 25% across mitral and 40% across tricuspid valve was considered significant and termed abnormal respiratory variation or flow velocity paradoxus (Fig. 2) (Table 3). Similarly respiratory flow velocity change across pulmonary and aortic valve during inspiration and expiration were measured and considered significant if the percent flow velocity change is more than 15% and 10%, respectively.