INDIAN HEART JOURNAL 65 (2013) 666–670

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Original Article

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

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ARTICLE INFO

Article history:
Received 19 December 2012
Accepted 9 October 2013
Available online 9 November 2013

Keywords:
Hemodynamics
Echocardiography
Tamponade
Pleural effusion

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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0019-4832/$ – see front matter
http://dx.doi.org/10.1016/j.ihj.2013.10.013

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

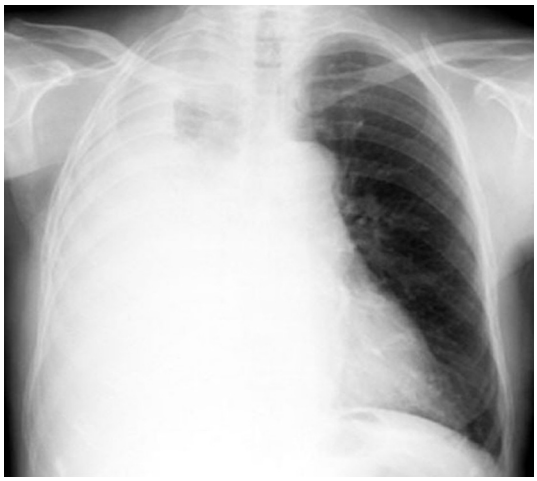

Fig. 1 – Chest X-ray PA view showing large right sided pleural effusion.

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.

Table 2 – Changes in cardiac dimensions before and after thoracocentesis.

Chamber size	Before thoracocentesis	After thoracocentesis
	Mean	Mean
Left atrium	3.5 cm	3.42 cm
Right atrium	3.4 cm	3.32 cm
Left ventricle systole	3.12 cm	3.26 cm
Left ventricle diastole	5.1 cm	5.2 cm
Right ventricle		
Basal	2.4 cm	2.45 cm
Mid	2.93 cm	2.98 cm
Base – Apex	7.37 cm	7.45 cm

3. Results

Results of the study are tabulated in Tables 1–3.

4. Discussion

There are few case reports regarding the impact of pleural effusion on cardiovascular hemodynamics. Previously Traylor et al[10] had studied a total of 27 patients dividing them into 2 groups, one with pleural effusion greater than ½ of hemi thorax and the other with pleural effusion less than half of a hemi thorax and all those patients had underlying minimal pericardial effusion. He concluded the impact of pleural effusion on the underlying pericardial effusion amounted to tamponade and the treatment for those should be thoracocentesis and not pericardiocentesis.

Since associated pericardial effusion was included in earlier studies it had a confounding effect. In our study we included patients with only pleural effusion and excluded patients with even minimal pericardial effusion in order to study the impact of pleural effusion per se on cardiovascular hemodynamics.

In this study, respiratory flow velocity variations typical of cardiac tamponade was detected in 85% patients with large

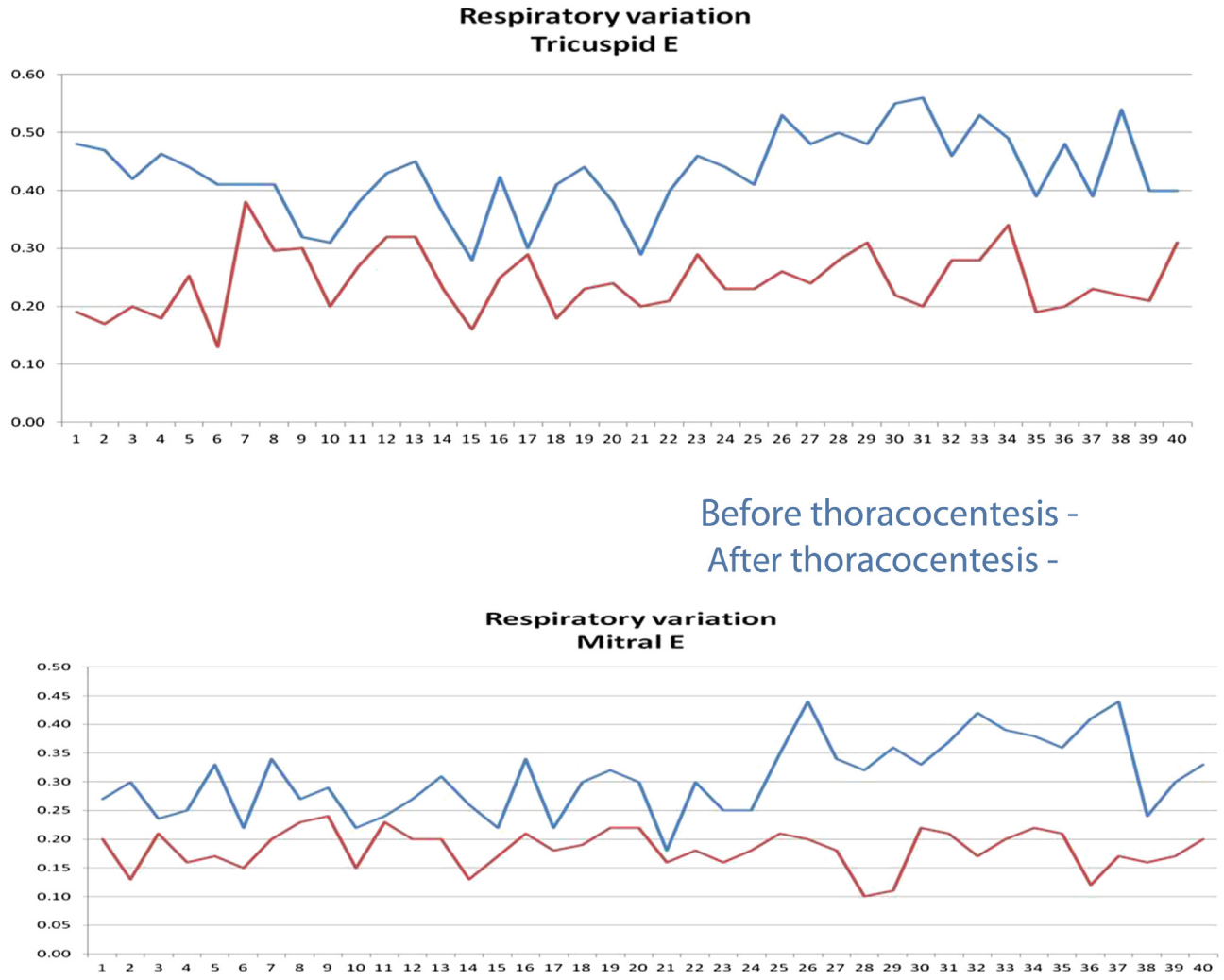

Fig. 2 – Figure shows the pattern of respiratory variation across tricuspid and mitral valve before and after thoracocentesis.

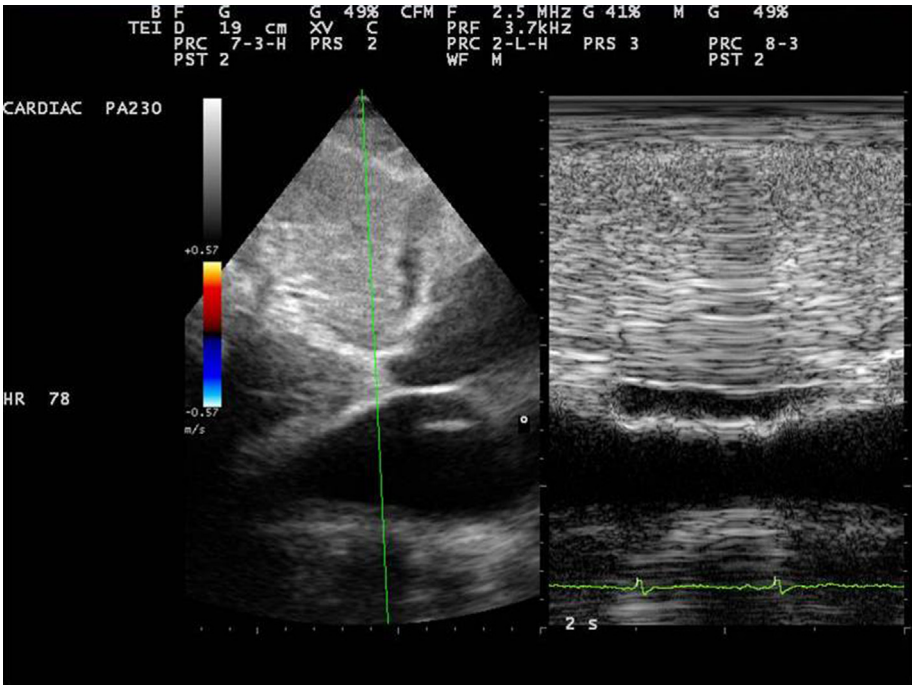

Fig. 3 – Non-compressible IVC during a sniff.

effusion .The parameters had reverted to normal values after thoracocentesis, which were statistically significant. This is a direct evidence for the occurrence of tamponade physiology.[11–17]

Echocardiographic findings were corroborative with clinical findings such as elevated jugular venous pressure, pulsus paradoxus. These clinical findings such as elevated jugular venous pressure, pulsus paradoxus show the impact of pleural effusion on the right side of the heart. We acknowledge respiratory flow velocity variations can occur in cases of chronic obstructive pulmonary disease and constrictive pericarditis also. So mean SVC flow was measured which was within normal range, which ruled out chronic obstructive pulmonary disease as a reason for respiratory variation of flow velocities. CT scan chest was taken and pericardial thickness was measured in all the cases and ruled out constrictive pericarditis.[18]

Table 3 – Confirmation of tamponade physiology.

	Before thoracocentesis	After thoracocentesis	*p* value
	Mean respiratory variations	Mean respiratory variations	
Tricuspid E	45.04 ± 10.4%	32 ± 11.3%	<0.001
Tricuspid A	53.71 ± 28%	32.08 ± 12.5%	<0.001
Mitral E	32.30 ± 12%,	19.78 ± 7.8%	<0.001
Mitral A	26 ± 11.2%	21 ± 9.3%	0.006
Pulmonary artery	42.63 ± 31.3%	17.70 ± 6.2%	<0.001
Aorta	21.57 ± 11.4%	14.08 ± 7.6%	<0.001

It is also suggested severely dyspneic patients can show fluctuations in the Doppler AV inflow pattern. We have considered this possibility hence the cut off value for significant variation is kept at 40% for tricuspid and 25% for mitral valve.

There have been some speculations that left sided effusions were likely to cause tamponade since major cardiac mass is on the left side. We have observed no difference in the occurrence of tamponade physiology with reference to the sidedness of effusion in this study.

As expected bilateral effusion showed severe forms of tamponade. In contrast to the observation in this study 15% of patients with large effusion did not show tamponade physiology. This can be probably be explained by the fact that there are significant inter individual variation in volume of various anatomical compartments of heart, lungs, pleural cavities within a fixed mediastinal volume.

In some of these patients, a good mediastinal compliance might have prevented the occurrence tamponade physiology.

Pulmonary arterial pressure was not significantly elevated in any of these patients This is contrary to our expectation, since an increase in pulmonary artery pressure due to the collapse of the lung parenchyma is expected. One of the explanations offered for absence of increase in PAP is underfiling of pulmonary circulation due the reduced RV stroke volume due to tamponade physiology.

5. Limitations of the study

1. Lack of catheterization data may reduce the accuracy of the study.
2. Poor echo windows.

6. Conclusion

Large pleural effusion has a potential to cause adverse impact on the cardiovascular hemodynamics, which could manifest as tamponade physiology. The impact of pleural effusion on cardiac hemodynamics does not seem to be related to the sidedness of effusion. Surprisingly, pulmonary arterial pressure remained normal in spite of mechanical effect of pleural effusion over the lung vasculature. Altered cardiac hemodynamics could be an important contributor in the mechanism of dyspnea in patients with large pleural effusion.

Contributorship

Venkatesan Sangareddi – study design.
Gnanavelu Ganesan, DM – study design.
Dhandapani V.E. DM – protocols.
Ravi M.S. DM – protocols.
Ravishankar. DM – manuscript preparation.
Meenakshi. K. DM – manuscript preparation.
Muthukumar. DM – final corrections.
Swaminathan. N. DM;- final corrections.

Conflicts of interest

All authors have none to declare.

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