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Original Research Article

An Echocardiographic Assessment of Cardiovascular Hemodynamics in Patients with Large Pleural Effusion at VAMCRH, Banthra, Shahjahanpur, U.P.

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Abstract

Background: Pleural effusion is a common medical problem existing all over the world, especially in developing countries. Pleural effusion of tuberculous origin is very common in India. The effect of pleural effusion depends on the cause and the amount of fluid in pleural space. This study aims to assess the tamponade physiology occurring in cases of large pleural effusion even in absence of any pericardial effusion and treatment of this condition should be thoracocentesis rather than pericardiocentesis.

Material and Methods: This cross-sectional study was done at VAMCRH, Banthra, Shahjahanpur, U.P. from February 2020 to October 2021. 73 patients were randomly selected from patients presented with large pleural effusion in medical emergency and medical ward. Detail history and clinical examination were done immediately. All patient with large pleural effusion whether unilateral or bilateral were enrolled in this study, while patients with pleural effusion of cardiac etiology, pericardial effusion and terminally ill were 11 cases excluded from this study. So total 62 cases included in this study.

Results: Sixty two cases of large pleural effusion were selected randomly irrespective of age, sex, etiology or sidedness of pleural effusion and were enrolled in this study group. General clinical parameters like pulse rate, respiratory rate, blood pressure etc. were measured. All sixty two patients with large pleural effusion were evaluated with echocardiography and echocardiographic parameters like chamber size of heart, Right ventricular diastolic collapse, right atrial diastolic cpllapse and respiratory flow variation across Mitral, Tricuspid, Pulmonary and Aortic valve were noted. Then 1000 to 1500 ml of pleural fluid were removed by thoracocentesis and a check x-ray was again repeated to make sure that pleural effusion remain below half of total lung field.A repeat echocardiography was done within 24 hour of thoracocentesis of large pleural effusion. Echocardiographic parameters like chamber size of heart, right ventricular diastolic collapse , right atrial diastolic collapse and respiratory flow variation across Mitral, Tricuspid, Pulmonary and Aortic valve were noted again. All the data were collected and kept for final study and were statistically analysed. The result of present study was to assess the effect of large pleural effusion on cardiovascular haemodynamics by means of echocardiography.

Conclusion: Patient's with tamponade physiology who has both large pleural effusion and pericardial effusion a pleurodesis is probably the safest initial procedure and might result in complete recovery from tamponade physiology.

Keyword: Tuberculous, Tamponade physiology, Large pleural effusion, Echocardiography

Background

Pleural effusion is excess amount of fluid that accumulates in the pleural cavity, the fluid-filled space that surrounds the lungs. Pleural effusion is a common medical problem existing all over the world, especially in developing countries. Pleural effusion of tuberculous origin is very common in India. The effect of pleural effusion depends on the cause and the amount of fluid in pleural space.

It is recognized that pleural effusion can compromise pulmonary function resulting in dyspnea and cough. Large pleural effusions are typically associated with dyspnea and potential respiratory compromise.

Experimental evidence suggests that with large pleural effusions, increased intrapleural pressure may be transmitted to the pericardial space, resulting in cardiac compromise similar to pericardial tamponade [1].

Pericardial effusion is collection of fluid in pericardial space. Small amount of pericardial effusion may remain asymptomatic for years while moderate to large pericardial effusion is one of the common medical emergencies.

When pericardial effusion accumulates rapidly or when it becomes excessive then it causes pressure on cardiac chambers causing tachycardia, and cardiovascular compromise (i.e. tamponade physiology).

Cardiac tamponade occurs when the pressure in the pericardium exceeds the pressure in cardiac chambers, resulting in impaired cardiac filling. As pericardial pressure increases, filling of each cardiac chamber are sequentially impaired leading to cardiac function compromise and decrease in cardiac output.

Rapid accumulation of even small volume of fluid can lead to a marked increase in pericardial pressure and can be a reason for cardiac tamponade. Cardiac tamponade is medical emergency and if it is not decompressed urgently it is invariably fatal.

Many of the medical condition which gives rise to pleural effusion can also causes pericardial effusion leading to cardiac tamponade.

There is very close relationship between pleural space and pericardial space and the dependence of their pressure kinetics are well known [2]. Many authors have studied the effects of pleural effusion on the pulmonary function and diaphragmatic contraction [3-5].

Literature is scarce about the effects of pleural effusion on cardiovascular haemodynamics.

The mediastinal pleura and the pericardium are closely related entities [6]. It has been shown in a canine model that large bilateral pleural effusions can lead to an increase in intrapleural pressure, which causes a linear increase in intrapericardial pressure, finally leading to right ventricular diastolic collapse which is sign of tamponade physiology [7].

Material and Methods

This cross-sectional study was conducted in different units of Department of Medicine,

at VAMCRH, Banthra, Shahjahanpur, U.P. from February 2020 to October 2021. Patients of pleural effusion of different etiology attending medical emergency ward of Varunarjun Medical College and Rohilkhand Hospital, Banthra, Shahjahanpur (UP), were randomly selected for study.

Methodology

All patients were evaluated irrespective of sidedness of pleural effusion. The chest x-ray of patients was done immediately for assessing the size of pleural effusion. On the basis of x-ray chest and clinical evaluation, patients were classified as having large, moderate or small pleural effusion. Pleural effusion involving more than 3/4th area of lung field were considered as large while those involving less than 3/4th area of lung field were considerd moderate to small. All patients with large pleural effusion were taken into study.

Inclusion criteria for patients

1. Patients with unilateral pleural effusion involving more than ¾th area on chest x ray.
2. Patients with bilateral pleural effusion involving more than ¾th area on chest x ray.

Exclusion criteria

1. All patients with pleural effusion involving less than 3/4th area on chest x-ray.
2. All patients with pericardial effusion of cardiac etiology.
3. All patients with pleural effusion of cardiac etiology.
4. All patients who were seriously ill.

In this cross-sectional study 73 patients were randomly selected from patients presented with large pleural effusion in VAMCRH, Banthra, Shahjahanpur, U.P., medical emergency and medical ward. Detail history and clinical examination were done immediately.

All patients with large pleural effusion whether unilateral or bilateral were enrolled in this study, while patients with pleural effusion of cardiac etiology, pericardial effusion and terminally ill were excluded from this study.

Out of 73 patients 6 patients were having pleural effusion of cardiac etiology and 3 patients were seriously ill. All these 9 patients were excluded from this study so as to prevent direct effect of cardiac disease on study finding and fear of loss to follow up study respectively. After excluding 9 patients 64 patients were left and all were enrolled in this study.

All patients included were transferred from medical ward for echocardiography.

On echocardiography 2 patients were found to have pericardial effusion along with pleural effusion. Patient with pericardial effusion (2) as seen on echocardiography were also excluded from this study.

After exclusion of all these patients, 62 patients were left and all were enrolled in this cross-sectional study for further evaluation.

Echocardiography was performed in all patients to measure chamber dimensions and pulse wave Doppler across mitral, tricuspid, pulmonary and aortic valve during quite respiration.

The data's observed in this study was tabulated. The mean value, standard deviation (SD) and p values were analyzed statistically. $P < 0.05$ is significant.

Results

In this present study 62 cases of large pleural effusion from both sexes presented to the indoor and outdoor department of medicine were studied.

The following tables show the results of the present study.

Table 1: Sex Distribution among Study group

Sex	Number of Patients (n)	Percentage of Patients
Male	44	70.96%
Female	18	29.04%
Total	62	100%

Table 2: Diagnosis of patients in Study group

Diagnosis	Total number of patients	Percentage
Pulmonary Tuberculosis	48	77.42%
Chronic liver disease	02	03.23%
Pulmonary malignancy	08	12.90%
Nephrotic syndrome	04	06.45%
0thers	00	0%

Table 3: Distribution of patients by sidedness of Pleural Effusion

Side	Total No. of Patients	Percentage
Left sided pleural effusion	09	14.52%
Right sided pleural effusion	47	75.80%
Bilateral pleural effusion	06	09.68%

Table 4: Abnormal transvulvular respiratory flow velocity change among study group

Patients showing	Number of patients before thoracocentesis	Percentage	Number of patients After thoracocentesis	Percentage
>40 % transvulvular flow velocity change across tricuspid valve	52	83.87 %	04	6.45%
>25 % transvulvular flow velocity change across mitral valve	49	79.03 %	01	1.61%
>15 % transvulvular flow velocity change across pulmonary valve	51	82.26 %	11	17.74%
>10 % transvulvular flow velocity change across aortic valve	53	85.48 %	12	19.35%

Table 5: Abnormal transvulvular Respiratory flow variation with reference to sidedness of pleural effusion (Before thoracocentesis)

Patients with	Total no (n)	Patients showing abnormal transvalvular respiratory flow variation	Patients showing abnormal transvalvular respiratory flow variation percentage	Patients showing normal transvalvular respiratory flow variation	Patients showing normal transvalvular respiratory flow variation percentage
Right sided pleural effusion	47	39	82.98%	08	17.02%
Left sided pleural effusion	09	07	77.78%	02	22.22%
Bilateral pleural effusion	06	06	100%	00	00%

Table 6: Abnormal transvulvular Respiratory flow variation with reference to sidedness of pleural effusion (After thoracocentesis)

Patients with	Total no (n)	Patients showing abnormal transvulvular respiratory flow variation	Patients showing abnormal transvulvular respiratory flow variation percentage	Patients showing normal transvulvular respiratory flow variation	Patients showing normal transvulvular respiratory flow variation percentage
Right sided pleural effusion	47	00	00.00%	47	100%
Left sided pleural effusion	9	01	11.11%	08	88.89%
Bilateral pleural effusion	6	03	50.00%	03	100%

Table 7: Transvulvular mean respiratory flow velocity variation in left sided pleural effusion

	Before thoracocentesis mean respiratory variation	After thoracocentesis mean respiratory variation
Tricuspid E	42.22	27.00
Tricuspid A	45.66	29.66
Mitral E	30.66	13.88
Mitral A	24.66	15.77
Pulmonary artery	43.44	15.00

Aorta	16.44	09.44

Table 8: Transvulvular mean respiratory flow velocity variation in bilateral effusion

	Before thoracocentesis mean respiratory variation	After thoracocentesis mean respiratory variation
Tricuspid E	58.83	39.00
Tricuspid A	77.16	40.66
Mitral E	41.67	20.66
Mitral A	39.17	20.66
Pulmonary artery	67.33	23.66
Aorta	29.16	14.83

Table 9: Mean Chamber Size before and after thoracocentesis

Cardiac Chamber	Mean size before thoracocentesis	Mean size after thoracocentesis
Left atrium	3.6 cm	3.42cm
Right atrium	3.5cm	3.34cm
Left ventricle systole	3.14cm	3.30cm
Left ventricle diastole	5.2cm	5.3cm
Right ventricle basal	2.4cm	2.45cm
Right ventricle mid	2.91cm	2.96cm
Right ventricle Base-apex	7.36cm	7.44cm

Table 10: Mean transvulvular flow velocity change across different valve before and after thoracocentesis

	Before thoracocentesis mean respiratory variation	After thoracocentesis mean respiratory variation	'p' value
Tricuspid E	45.13±07.79	27.53±6.08	<0.001
Tricuspid A	52.56±13.03	29.98±5.56	<0.001
Mitral E	31.50±08.02	14.95±4.06	<0.001
Mitral A	25.77±06.44	16.97±4.25	<0.001
Pulmonary artery	40.06±17.30	15.21±3.43	<0.001
Aorta	20.23±07.06	9.66±2.56	<0.001

Discussion

Out of 62 patients 18(29.04%) patients were female and 44(70.96%) patients were male (TABLE 1). In this study group, male patients outnumbered female patients.

Patients were between the age group of 14 years to 72 years; with mean age of presentation was 38.4 years. Out of 62 patients 46(75.80%) patients were having right sided pleural effusion, 9(14.52%) patients were having left sided pleural effusion while 6(09.68%) patients were having bilateral pleural effusion. (Table 2)

The prevalence of right sided pleural effusion was very high (74.19%) in this study group as compared to left sided (14.51%) and bilateral pleural effusion (11.29%).In Sundar Chidambaram *et al* [8] (2013) study group too there was higher

prevalence of right sided pleural effusion as compared to left sided and bilateral effusion. This probably reflects higher prevalence of right sided pleural effusion compared to left sided pleural effusion and bilateral pleural effusion in community.

Out of 62 patients, 48(77.42%) patients were having pulmonary tuberculosis, 8(12.90%) patients were having malignancy, 4(6.45%) patients were having nephrotic syndrome and 2(3.23%) patients were having chronic liver disease (TABLE 3) In Sundar Chidambaram *et al* [8] study there were 7 diabetics, 25 tuberculosis, 14 malignancies and 1 hepatic failure patient.

Echocardiography of all 62 patients was obtained immediately before thoracocentesis. Echocardiographic findings of the study group were corroborative with clinical findings such as elevated jugular venous pressure, pulses paradoxus etc. These clinical findings such as elevated jugular venous pressure, pulsus paradoxus show the impact of large pleural effusion on the right side of heart.

Out of 62 patients 18 (29%) patients were having right atrial diastolic collapse on echocardiography. This finding was in accordance with the finding of Sadaniantz A *et al* [9] study, in which they reported that, the frequency of chamber collapse was 18% in patients with pleural effusion in the absence of pericardial effusion, thus cardiac chamber collapse occurs in patients with pleural effusion. Anim J Am *et al* [10] had similarly reported a case of marked pleural effusion causing right atrial collapse simulating cardiac tamponade in dog.

Respiratory flow velocity variation across mitral, tricuspid, pulmonary and aortic valve was obtained. Respiratory flow velocity change across valve is reliable echocardiographic sign of tamponade physiology.

Materazzo C *et al* [11] in 2003, studied respiratory changes in transvalvular flow velocities versus two-dimensional echocardiographic findings in the diagnosis of cardiac tamponade, they concluded that in the diagnosis of cardiac tamponade: right atrial collapse is the most sensitive sign but lacks any specificity; except for the tricuspid valve, the respiratory variations in the transvalvular flow velocities have a reliability and a predictive value comparable with those of right ventricular collapse.

There was inspiratory increase and expiratory decrease in the transvulvular flow across tricuspid and pulmonary valve which was exaggerated. The percentage of change in transvulvular velocity were obtained .The change in transvulvular velocity of more than 40% across tricuspid valve and more than 15% across pulmonary valve were considered significant and termed as abnormal flow velocity or flow velocity paradox.

Similarly there was inspiratory decrease and expiratory increase in transvulvular flow velocity across mitral and aortic valve. The changes in transvulvular velocity of more than 25 % across mitral valve and more than 10% across aortic valve were considered significant.

Out of 62 patients, 52(83.87%) patients showed abnormal respiratory flow velocity variation typical of cardiac tamponade. 52(83.87%) patients showed more than 40% transvulvular flow velocity change across tricuspid E, 49(79.03%) patients showed more than 25% transvulvular flow velocity change across mitral valve, 51(82.26%) patients showed more than 15% transvulvular flow velocity change across pulmonary valve, 53(85.48%) patients showed more than 10% transvulvular velocity change across aortic valve which was typical of cardiac tamponade physiology. The parameters reverted to

normal values after thoracocentesis which were statistically significant. This was direct effect of tamponade physiology. (Table-4)

Sundar Chidambaram *et al* [8] had also reported reported similar prevalence of respiratory variations typical of cardiac tamponade in 85% patients with large pleural effusion which resolved after thorecocentasis.

In out of 46 patients with right sided pleural effusion 39 (82.98%) patients were showing abnormal transvulvular flow velocity changes typical of tamponade physiology. (Table-5)

In out of 9 patients with left sided effusion 7 (77.78%) patients were showing abnormal transvulvular flow velocity change typical of tamponade physiology. (Table-5)

There have been some speculations that left sided pleural effusions were more likely to cause tamponade since major cardiac mass is on the left side. This study observed no difference in the occurrence of tamponade physiology with reference to the sidedness of pleural effusion. In this study 82.98% of patients with right sided pleural effusion were showing abnormal transvulvular flow velocity changes typical of tamponade physiology as against 78.78 % of patients with left sided pleural effusion. (Table-5). After thoracocentesis none of the patients with right sided pleural effusion was showing transvulvular respiratory flow velocity change typical of tamponade physiology, while 11.11% of patients with left sided pleural effusion and 50% of patients with bilateral pleural effusion were showing abnormal transvulvular flow velocity changes typical of tamponade physiology.

This finding is in accordance with the finding of Sundar Chidambram *et al* study in which they similarly did not found any differences in occurrence of tamponade physiology with refrence to sidedness of pleural effusion.

In out of 6 patients with bilateral effusion all (100%) were showing abnormal transvulvular flow velocity changes typical of tamponade physiology. (TABLE-6)

This study did found severe form of tamponade physiology associated with bilateral pleural effusion (Table-8). In bilateral pleural effusion ,before thoracocentesis mean respiratory flow variation across Tricuspid E, Tricuspid A Mitral E, Mitral A, Pulmonary and Aortic was 58.83%,77.16%,41.67%,39.17%,67.3% and 29.16% respectively, which was variation of extreme degree and was clearly a sign of more severe tamponade physiology. After thoracocentesis these parameters reverted into normal range. Parameters after thoracocentesis across Tricuspid E, Tricuspid A, Mitral E, Mitral A, Pulmonary and Aortic valve was 39.00%, 40.66%, 20.6%, 20.66%, 23.66% and 14.83% respectively.

Table-9 is showing changes in cardiac dimensions before and after thoracocentesis. It shows that there were increase in ventriclle size of left and right and decrease in atrial size of left and right after thoracocentesis. Mean chamber size before and after thoracocentesis of left atrium was 3.6 and 3.42, right atrium 3.5 and 3.34, left ventricle systole 3.14 and 3.30, left ventricle diastole 5.2 and 5.3, right ventricle basal 2.4 and 2.45, right ventricle mid-2.91 and 2.96, right ventricle base-apex 7.36 and 7.44 respectively. This finding was similar to the Sundar Chidambaram *et al* [8] finding of Mean chamber size before and after thoracocentesis of left atrium 3.5 and 3.42, right atrium 3.4 and 3.22, left ventricle systole 3.12 and 3.26, left ventricle diastole 5.1 and 5.2, right ventricle basal 2.4 and 2.45, right ventricle mid-2.93 and 2.98, right

ventricle base-apex 7.37 and 7.45 respectively.

Table-10 shows mean respiratory variation across different valve before and after thoracocentesis. Before thoracocentesis mean respiratory variation across Tricuspid E, Tricuspid A Mitral E, Mitral A, Pulmonary and Aortic was 45.13±7.79%, 52.56±13.03%, 31.50±8.02%, 25.77±6.94%, 40.06±17.30% and 20.23±7.06% respectively.

This transvulvular respiratory flow velocity variation was well above the cut of value of transvulvular respiratory variation for tamponade physiology across tricuspid, mitral, pulmonary and aorta as 40%, 25%, 15% and 10% respectively. This was direct evidence of tamponade physiology. This means that tamponade physiology was present in these patients. Sundar Chidambaram *et al* [8] had shown almost similar data in cases of large pleural effusion in their study group. These shows that patient with large pleural effusion can mimic cardiac tamponade.

Once approximately 1000ml to 1500ml of the pleura fluid was removed by thoracocentesis the respiratory variation across Tricuspid E, Tricuspid A Mitral E, Mitral A, Pulmonary and Aortic changed to 27.53±6.08%, 29.98±5.6%, 14.95±9.06%, 16.97±4.25%, 15.21±4.3% and 9.66±2.56% respectively and patients respiratory and cardiac distress improved dramatically. This changed in transvulvular mean respiratory flow velocity was highly significant statistically. This respiratory variation was well below the cut of value of respiratory variation for tamponade physiology across tricuspid, mitral, pulmonary and aorta as 40%, 25%, 15 % and 10 % respectively. This was direct evidence of tamponade physiology. This shows that large pleural effusion has a potential to cause adverse outcome on cardiovascular haemodynamics which could manifest as tamponade physiology even in absence of pericardial effusion. So when assessing a case of large pleural effusion presenting as tamponade physiology an echocardiography should be done and thoracocentesis should preceeds pericardiocentesis.

Monia Werlang *et al* [12] in 2015 had similarly reported a case of large pleural effusion due to myxedema, which was showing tamponade physiology. In their study they concluded that large pleural effusion can be associated with clinical and echocardiographic signs of cardiac tamponade and its evacuation leading to complete recovery of patient's haemodynamic status.

Conclusion

Cardiac tamponade physiology can occur in situations other than pericardial effusion. Tamponade physiology can occur in cases of large pleural effusion. Pleural effusion can compromise cardiovascular haemodynamics mimicking pericardial effusion which can manifest as cardiac tamponade physiology. Bilateral large pleural effusion is generally associated with more severe cardiovascular haemodynamic compromise as compared to unilateral large pleural effusion. The impact of pleural effusion on cardiovascular haemodynamics is not related to sidedness of pleural effusion and effect is almost similar between right sided and left sided pleural effusion but bilateral pleural effusion are associated with severe form of tamponade physiology. In addition to fluid in lung parenchyma and pleural space, altered cardiovascular haemodynamics could be an important contributor in mechanism of dyspnea in large pleural effusion. Thoracocentesis of large pleural effusion improves cardiac tamponade physiology in pleural effusion so treatment of this condition is thoracocentesis rather than pericardiocentesis. Echocardiography is

valuable investigation in case of cardiac tamponade physiology in presence of large pleural effusion. Patient presenting with signs of cardiac tamponade with large pleural effusion should undergo echocardiography before pericardiocentesis. Large pleural effusion should be drained by thoracocentesis as early as possible to prevent cardiovascular compromise and tamponade physiology.

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