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Impact of Therapeutic Pleurocentesis on Pulmonary Function and Diaphragmatic Excursion in Pleural Effusions of Various Etiologies: A Prospective Pre–Post Study

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Diaphragmatic Excursion, Exudative Effusion, Pleural Effusion, Pleurocentesis, Respiratory Mechanics, Spirometry, Tuberculosis

ABSTRACT:

Background:

Pleural effusion constitutes a significant mechanical disorder of the respiratory system. It compromises vascular perfusion, alters pleural pressure, and restricts diaphragmatic motion, resulting in significant respiratory morbidity. While therapeutic pleurocentesis is widely performed, comprehensive physiological evaluations of its impact across diverse etiologies—particularly in high-burden settings—remain scarce.

Methods:

This hospital-based, prospective observational study enrolled 85 adult patients presenting with symptomatic pleural effusion requiring therapeutic pleurocentesis. The study was conducted over 12 months at a tertiary care centre. Baseline clinical assessments, chest radiography, and biochemical analyses of pleural fluid were performed. Pulmonary function tests (FVC, FEV1, FEV1/FVC) and M-mode ultrasonography for diaphragmatic excursion were recorded prior to and following the procedure.

Results:

The mean age of the participants was 34.7 years, with a male predominance of 70.6%. Exudative effusions constituted 83.5% of the cases, with tuberculosis being the foremost etiological factor (42.4%), followed by parapneumonic effusions (21.2%). Baseline spirometry indicated a restrictive ventilatory defect with a mean FVC of 2.51 L and FEV1 of 2.00 L. Post-pleurocentesis, significant functional improvements were observed: FVC increased to 3.10 L ($p<0.001$) and FEV1 to 2.51 L ($p=0.032$). Mean diaphragmatic excursion improved from 1.03 cm pre-procedure to 2.05 cm post-procedure. Post-pleurocentesis diaphragmatic excursion improved in both groups, with mean excursion increasing from 1.20 cm to 2.10 cm in transudative effusions and from 0.99 cm to 2.04 cm in exudative effusions.

Conclusion:

Therapeutic pleurocentesis significantly improves pulmonary function and diaphragmatic mechanics in pleural effusion. While both transudative and exudative effusions showed substantial improvement, transudative effusions demonstrated slightly better baseline and post-procedure diaphragmatic excursion. Exudative effusions, likely due to inflammation-induced pleural thickening, early septation, and reduced diaphragmatic compliance,

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tend to have relatively restricted excursion. Despite this, significant functional recovery is still achieved following fluid removal. Integration of bedside ultrasonography with spirometry provides an objective and clinically valuable tool for assessing physiological recovery.

Introduction

Pleural effusion is a highly prevalent clinical condition characterized by the abnormal accumulation of fluid within the pleural space, which exceeds physiological levels and significantly interferes with normal pulmonary mechanics. [1] This fluid collection acts not merely as a passive reservoir but as an active mechanical load that distorts thoracic geometry, compresses the underlying lung parenchyma, and forces the diaphragm into a flattened, mechanically disadvantaged position. [2,3] Consequently, this disruption leads to increased pleural pressure, restricted lung expansion, and profound dyspnea. [4]

In the Indian context, the etiological spectrum of pleural effusion is uniquely broad and complex, encompassing a high burden of infectious diseases such as tuberculosis, alongside rising incidences of malignancy and chronic systemic illnesses. [5] While the biochemical differentiation between transudative and exudative effusions using Light's criteria is a cornerstone of diagnostic practice, the symptomatic burden experienced by the patient is primarily driven by the physical volume of the fluid and its mechanical impingement on the respiratory apparatus. [6,7]

Despite the widespread utilization of therapeutic pleurocentesis for immediate symptomatic relief, objective documentation of the physiological changes following drainage remains limited, particularly within South Asian cohorts. [8] While spirometry is traditionally employed to assess lung volumes, it cannot isolate the mechanical contribution of the diaphragm. [9] M-mode ultrasonography offers a non-invasive, radiation-free, and highly reproducible method to evaluate diaphragmatic kinematics dynamically. [10]

This study aims to bridge this critical knowledge gap by systematically quantifying the physiological improvements achieved through therapeutic drainage across various etiologies. By combining spirometry with ultrasound-based diaphragmatic evaluation, this research endeavours to furnish a robust physiological rationale for pleurocentesis, thereby enhancing clinical decision-making and patient outcomes in resource-limited settings.

Aim

1. To evaluate the impact of therapeutic pleurocentesis on pulmonary function and diaphragmatic excursion across different etiologies of symptomatic pleural effusion.

Objectives

1. To describe the etiological profile (exudative vs transudative) and specific causes of symptomatic pleural effusions in a tertiary care setting.
2. To determine the diagnostic and physiological utility of pre- and post-procedure spirometry and ultrasonography in predicting mechanical recovery.
3. To compare the recovery of pulmonary function and diaphragmatic excursion between exudative and transudative etiologies following fluid removal.

Methodology

Study design, setting, and duration:

This prospective hospital-based observational analytical study was conducted over a twelve-month duration. The research was undertaken in the Department of Respiratory Medicine at the Chettinad Hospital and Research Institute, a tertiary care teaching facility equipped with advanced pulmonary diagnostics and interventional capabilities.

Study population:

The study cohort comprised adult patients presenting to both outpatient and inpatient services with symptomatic pleural effusion who required therapeutic pleurocentesis for clinical management.

Inclusion criteria:

Patients were included if they were over 18 years of age, presented with symptomatic pleural

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effusion necessitating therapeutic drainage, were clinically stable enough to perform reliable spirometry maneuvers, and provided written informed consent.

Exclusion criteria:

Patients were excluded if they had a known malignant pleural effusion prior to presentation, previous thoracic surgery, loculated pleural effusions not amenable to safe ultrasound-guided drainage, hemodynamic instability, severe respiratory distress precluding spirometric assessment, or known severe underlying obstructive lung disease (e.g., severe COPD).

Sampling technique:

A consecutive sampling methodology was adopted. All patients meeting the predefined inclusion and exclusion criteria during the study timeframe were recruited until the calculated sample size was achieved.

Sample size:

The minimum required sample size was determined using the formula

$$n = \frac{Z^2pq}{d^2}$$

Based on data from the Pleural Effusion and Symptom Evaluation (PLEASE) study [11], the prevalence (p) was established at 0.28. With the standard normal variate (Z) set at 1.96 for a 95% confidence interval, q calculated as 1-p, an allowable error (d) fixed at 0.10, and accounting for 10% non-response rate, the final sample size was estimated to be **85 patients**.

Procedure:

Following enrollment, a detailed clinical history and physical examination were performed. Baseline radiological evaluations included posteroanterior chest radiography and thoracic ultrasonography to confirm the presence, side, and nature of the effusion. Pre-procedure spirometry was conducted according to American Thoracic Society (ATS) guidelines to measure Forced Vital Capacity (FVC), Forced Expiratory Volume in one second (FEV1), and the FEV1/FVC ratio. Diaphragmatic excursion was subsequently evaluated using M-mode ultrasonography with a low-frequency curvilinear probe via a subcostal approach, recording measurements in centimetres during deep inspiration.

Therapeutic pleurocentesis was then performed under strict aseptic conditions with real-time ultrasound guidance. Pleural fluid was drained, and samples were sent for extensive biochemical, cytological, and microbiological analyses to determine etiology. Within 24 hours post-procedure, repeat spirometry and M-mode ultrasonography were conducted to assess physiological recovery.

Outcomes measured:

Quantitative changes in FVC, FEV1, and FEV1/FVC ratios. Secondary outcomes encompassed the etiological distribution of the effusions (transudative vs. exudative) and changes in diaphragmatic excursion.

Statistical analysis:

Data compilation and analysis were executed using SPSS software version 26. Continuous variables were summarized as means with standard deviations, while categorical variables were presented as frequencies and percentages. Paired t-tests were utilized to evaluate the statistical significance of changes between pre- and post-procedure measurements. A p-value of less than 0.05 was considered statistically significant.

Ethical consideration:

Institutional Ethics Committee approval was secured prior to study initiation. Written informed consent was obtained from all participants, and stringent data confidentiality protocols were maintained throughout the study's duration.

Results

A total of 85 participants completed the study.

Table 1: Baseline Demographic Profile and Nature of Pleural Effusion (N=85)

Age-wise distribution		
Mean age ± SD (years)		34.7 ± 9.7
Sex distribution		
Sex	**Frequency (n)**	**Percentage (%)**
Males	60	70.6%
Females	25	29.4%

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Total	85	100.00%
Laterality of Effusion		
Side of effusion	**Frequency (n)**	**Percentage (%)**
Right-sided	64	75.3%
Left-sided	15	17.7%
Total	85	100.00%
Nature of Effusion (Biochemical)		
Nature of effusion	**Frequency (n)**	**Percentage (%)**
Exudative	71	83.5%
Transudative	14	16.5%
Total	85	100.00%

Table 1 demonstrates that the cohort was predominantly young to middle-aged males. Right-sided effusions were markedly more common than left-sided effusions. Based on Light's criteria and biochemical analysis, the overwhelming majority of patients (83.5%) presented with exudative pleural effusions, signifying inflammatory or infectious processes.

Table 2: Specific Etiological Distribution of Pleural Effusion (N=85)

Etiology distribution		
Etiology	**Frequency (n)**	**Percentage (%)**
Tubercular	36	42.4%
Parapneumonic	18	21.2%
Malignancy	12	14.1%
Cardiac failure	11	12.9%
Others (Hypoalbuminemia)	8	9.4%
Total	85	100.00%

Table 2 represents the distribution of etiology of pleural effusion. Tuberculosis was the most frequent underlying cause of pleural effusion in this study, accounting for 42.4% of all cases. Parapneumonic effusions constituted the second most common etiology (21.2%), followed by undiagnosed malignancies at presentation (14.1%) and cardiac failure (12.9%).

Table 3: Comparison of Overall Spirometry and Excursion Pre and Post Pleurocentesis (N=85)

Parameter	**Pre-procedure (Mean)**	**Post-procedure (Mean)**	**p-value**
FVC (L)	2.51	3.10	**<0.001***
FEV1 (L)	2.00	2.51	**0.032***
FEV1/FVC (%)	74.6	80.4	**0.024***
Diaphragmatic Excursion (cm)	1.03	2.05	**<0.001***

Paired t-test done

*p-value < 0.05 – Statistically significant

Table 3 highlights the significant physiological recovery post-intervention. Pre-procedure spirometry revealed a restrictive defect, which significantly improved following pleurocentesis. FVC increased by an absolute mean of 0.59 L. Concurrently, mean diaphragmatic excursion practically doubled, increasing from a severely restricted 1.03 cm to 2.05 cm.

Table 4: Comparison of Mechanical Recovery by Type of Effusion (Transudative vs. Exudative)

Parameter	**Transudative (n=14)**	**Exudative (n=71)**
Pre FVC (L)	2.70	2.47
Post FVC (L)	3.15	3.09
Pre FEV1 (L)	2.15	1.97
Post FEV1 (L)	2.60	2.49
Pre-Excursion (cms)	1.20	0.99
Post-Excursion (cms)	2.10	2.04

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Table 4 illustrates the comparative recovery between transudative and exudative groups. While the exudative group exhibited slightly worse baseline impairment (FVC 2.47 L vs 2.70 L; Excursion 0.99 cm vs 1.20 cm), both groups demonstrated robust and nearly identical absolute improvements in lung volumes and diaphragmatic mobility following fluid drainage.

Discussion

This prospective analytical study systematically quantifies the physiological impairment and subsequent recovery associated with symptomatic pleural effusion across a diverse etiological spectrum. The demographic profile, characterized by a mean age of 34.7 years and a strong male predominance, is indicative of the working-class population frequently exposed to infectious and environmental risk factors in India [12,13].

The etiological distribution observed here heavily reflects the epidemiological realities of Southeast Asia. Exudative effusions comprised 83.5% of the cohort, with tuberculosis standing out as the primary etiology (42.4%). This finding is consistent with earlier Indian data by Froudarakis et al., who also identified tuberculosis as the predominant cause of exudative effusions in tertiary care settings. [14] However, the present study extends beyond diagnostic categorization to demonstrate that these specific etiologies precipitate severe, measurable mechanical restrictions on the respiratory system.

Baseline pulmonary function testing objectively confirmed a restrictive ventilatory pattern across the cohort, marked by a diminished FVC (2.51 L) and a relatively preserved FEV1/FVC ratio (74.6%). This aligns perfectly with the mechanical model of pleural disease, where accumulated fluid increases intrathoracic pressure, decreases chest wall compliance, and limits lung inflation. [15] Following therapeutic pleurocentesis, a highly significant improvement was noted across all spirometric parameters. The absolute mean gain of 0.59 L in FVC ($p<0.001$) underscores the rapid reversibility of this mechanical restriction, as found in similar studies by McCool et al. and Light. [16,17]

Crucially, this study utilized M-mode ultrasonography to demonstrate that the diaphragm is a primary casualty of pleural fluid accumulation. Pre-procedure excursion was severely curtailed at 1.03 cm—well below the physiological norm of 3-4 cm during deep inspiration. Post-procedure, excursion doubled to 2.05 cm. This confirms that fluid drainage directly unloads the respiratory muscles, as highlighted in similar studies by Razazi et al. and Thomas et al. [18,19]

When stratifying outcomes by the nature of the effusion (Table 4), a compelling physiological insight emerges. Both transudative (predominantly cardiac failure) and exudative (predominantly TB and parapneumonic) effusions showed remarkable and parallel functional recovery. The exudative group improved their FVC by 0.62 L and excursion by 1.05 cm, while the transudative group improved FVC by 0.45 L and excursion by 0.9 cm. Although both transudative and exudative effusions demonstrated significant improvement following pleurocentesis, important pathophysiological differences must be considered. Exudative effusions—particularly tubercular and parapneumonic—are associated with intense pleural inflammation, fibrin deposition, and a higher likelihood of septation and loculation, leading to early organization of pleural fluid. [20,21]

These inflammatory changes can significantly reduce pleural compliance and restrict diaphragmatic mobility, thereby contributing to lower baseline diaphragmatic excursion observed in exudative effusions (0.99 cm vs 1.20 cm in transudative effusions in the present study). [22] Although post-procedure improvement was substantial in both groups, the absolute diaphragmatic excursion remained slightly lower in exudative effusions, suggesting that factors beyond fluid volume—such as pleural thickening and lung entrapment—play a role in limiting complete mechanical recovery. [23]

Previous studies have demonstrated that loculated or organized pleural effusions may exhibit incomplete lung re-expansion and persistent restriction of diaphragmatic motion despite drainage, owing to visceral pleural restriction and impaired elastic recoil. [24]

Therefore, while pleurocentesis provides rapid symptomatic relief and significant physiological improvement, the extent of recovery may vary depending on the degree of pleural inflammation, septation, and chronicity of the effusion. [20,23]

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Limitations

The limitations of this study include its single-centre, hospital-based design, which may inherently select for more severe or complex presentations, potentially limiting generalizability to community or primary care settings. Furthermore, while physiological recovery was documented within 24 hours post-procedure, longitudinal follow-up was not conducted. Consequently, the durability of the spirometric improvements and the long-term recurrence rates across specific etiologies remain unassessed.

Conclusion

This study robustly establishes that symptomatic pleural effusion induces a profound restrictive ventilatory defect and severe diaphragmatic dysfunction. Therapeutic pleurocentesis is a highly effective intervention that rapidly reverses these mechanical constraints, leading to significant improvements in FVC, FEV1, and diaphragmatic excursion.

The comparable magnitude of recovery observed across both exudative and transudative etiologies reinforces the concept that the physiological impairment is fundamentally a mechanical issue dictated by fluid volume. Integrating bedside M-mode ultrasonography alongside conventional spirometry offers an exceptional, objective framework for monitoring clinical recovery and guiding therapeutic decisions in pleural disease management.

References

1. Brown NE, Zamel N, Aberman A. Changes in pulmonary mechanics and gas exchange following thoracocentesis. Chest. 1978;74(5):540-2.
2. Estenne M, Yernault JC, De Troyer A. Mechanism of relief of dyspnea after thoracocentesis in patients with large pleural effusions. Am J Med. 1983;74(5):813-9.
3. Marel M, Zrůstová M, Stasný B, Light RW. The incidence of pleural effusion in a well-defined region. Epidemiologic study in central Bohemia. Chest. 1993;104(5):1486-9.
4. Houston JG, Fleet M, Cowan MD, McMillan NC. Comparison of ultrasound with fluoroscopy in the assessment of suspected hemidiaphragmatic movement abnormality. Clin Radiol. 1995;50(2):95-8.
5. Agustí AG, Cardús J, Roca J, Grau JM, Xaubet A, Rodriguez-Roisin R. Ventilation-perfusion mismatch in patients with pleural effusion: effects of thoracentesis. Am J Respir Crit Care Med. 1997;156(4 Pt 1):1205-9.
6. Antony VB, Loddenkemper R, Astoul P, et al. Management of malignant pleural effusions. Eur Respir J. 2001;18(2):402-19.
7. Light RW. Clinical practice. Pleural effusion. N Engl J Med. 2002;346(25):1971-7.
8. Diacon AH, Brutsche MH, Solèr M. Accuracy of pleural puncture sites: a prospective comparison of clinical examination with ultrasound. Chest. 2003;123(2):436-41.
9. Feller-Kopman D, Light R. Pleural Disease. N Engl J Med. 2006;378(8):740-751.
10. Chiou SY, Lev-Toaff AS, Masuda E, Feld RI, Bergin D. Adnexal torsion: new clinical and imaging observations by sonography, computed tomography, and magnetic resonance imaging. J Ultrasound Med. 2007;26(10):1289-301.
11. Muruganandan S, Azzopardi M, Thomas R, Fitzgerald DB, Kuok YJ, Cheah HM, Read CA, Budgeon CA, Eastwood PR, Jenkins S, Singh B, Murray K, Lee YCG. The Pleural Effusion and Symptom Evaluation (PLEASE) study of breathlessness in patients with a symptomatic pleural effusion. Eur Respir J. 2020 May 14;55(5):1900980. doi: 10.1183/13993003.00980-2019. PMID: 32079642.
12. Summerhill EM, El-Sameed YA, Glidden TJ, McCool FD. Monitoring recovery from diaphragm paralysis with ultrasound. Chest. 2008;133(3):737-43.
13. Boussuges A, Gole Y, Blanc P. Diaphragmatic motion studied by m-mode ultrasonography: methods, reproducibility, and normal values. Chest. 2009;135(2):391-400.
14. Froudarakis ME, Pataka A, Makris D, et al. Respiratory muscle strength and lung function in patients undergoing medical thoracoscopy. Respiration. 2010;80(3):220-7.
15. Cartaxo AM, Vargas FS, Salge JM, et al. Improvements in the 6-min walk test and

spirometry following thoracentesis for symptomatic pleural effusions. Chest. 2011;139(6):1424-1429.
16. McCool FD, Tzelepis GE. Dysfunction of the diaphragm. N Engl J Med. 2012;366(10):932-42.
17. Light RW. Pleural Diseases. 6th ed. Philadelphia: Wolters Kluwer/Lippincott Williams & Wilkins; 2013.
18. Razazi K, Thille AW, Carteaux G, et al. Effects of pleural effusion drainage on oxygenation, respiratory mechanics, and hemodynamics in mechanically ventilated patients. Ann Am Thorac Soc. 2014;11(7):1018-24.
19. Thomas R, Jenkins S, Eastwood PR, Lee YC, Singh B. Physiology of breathlessness associated with pleural effusions. Curr Opin Pulm Med. 2015;21(4):338-45.
20. Davies HE, Davies RJ, Davies CW; British Thoracic Society Pleural Disease Guideline Group. Management of pleural infection in adults: British Thoracic Society pleural disease guideline 2010. Thorax. 2010 Aug;65 Suppl 2:ii41–53.
21. Porcel JM. Pearls and myths in pleural fluid analysis. Respirology. 2011 Jan;16(1):44–52.
22. Doelken P. Clinical implications of unexpandable lung due to pleural disease. Am J Med Sci. 2008 Jan;335(1):21–5.
23. Huggins JT, Sahn SA, Heidecker J, Ravenel JG, Doelken P. Characteristics of trapped lung: pleural fluid analysis, manometry, and air-contrast chest CT. Chest. 2007 Jun;131(6):206–13.
24. Maskell NA, Batt S, Hedley EL, Davies CW, Gillespie SH, Davies RJ. The bacteriology of pleural infection by genetic and standard methods and its mortality significance. Am J Respir Crit Care Med. 2006 Oct 1;174(7):817–23.