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Original Research Article

# CLINICAL SIGNIFICANCE OF PLEURAL FLUID C-REACTIVE PROTEIN IN THE DIAGNOSIS OF EXUDATIVE PLEURAL EFFUSION

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## ABSTRACT

**Background:** Exudative pleural effusion is a common clinical presentation with diverse etiologies, including tuberculosis, parapneumonic infection, and malignancy. Accurate etiological differentiation is essential for appropriate management but remains challenging due to the limitations of existing diagnostic tests. C-reactive protein (CRP), an acute-phase reactant, may serve as a simple and cost-effective biomarker to distinguish inflammatory from non-inflammatory pleural effusions. This study aimed to evaluate the diagnostic role of pleural fluid CRP in differentiating the etiology of exudative pleural effusions. **Materials and Methods:** A prospective observational study was conducted on 264 patients with exudative pleural effusion at a tertiary care center in North India. Etiology was established based on clinical, biochemical, cytological, and microbiological findings. Pleural fluid CRP was estimated by high-sensitivity turbidimetric immunoassay and compared among tubercular (n = 142), parapneumonic (n = 67), and malignant (n = 55) groups. Statistical analysis included ANOVA and receiver operating characteristic (ROC) curve analysis to determine diagnostic accuracy. **Result:** The mean pleural CRP levels were significantly higher in parapneumonic effusions (93.7 ± 34.1 mg/L) than in tubercular (49.8 ± 22.6 mg/L) or malignant effusions (21.4 ± 12.7 mg/L) ($p < 0.001$). At a cut-off value of 35 mg/L, CRP differentiated infectious (tubercular + parapneumonic) from malignant effusions with an AUC of 0.912 (95% CI: 0.875–0.949), sensitivity 88.7%, and specificity 83.6%. CRP correlated positively with LDH ($r = 0.71$, $p < 0.001$) and ADA ($r = 0.46$, $p < 0.001$), and negatively with glucose ($r = –0.54$, $p < 0.001$). **Conclusion:** Pleural fluid CRP is a valuable adjunct biomarker in the etiological diagnosis of exudative pleural effusion. A threshold of 35 mg/L effectively distinguishes infectious from malignant effusions, supporting its use as a rapid, inexpensive, and reliable diagnostic tool in resource-limited settings.

## INTRODUCTION

Pleural effusion, defined as the accumulation of fluid within the pleural space, is a frequent manifestation of diverse pulmonary and systemic diseases. Globally, it is estimated that over 1.5 million new cases of pleural effusion occur annually, accounting for a substantial proportion of hospital admissions for respiratory illness.[1] The etiologies vary widely, with congestive heart failure, pneumonia, malignancy, and tuberculosis (TB) being the leading causes.[2] In India, tubercular pleural effusion remains the most prevalent form of exudative effusion, contributing to nearly 50–60% of cases in tertiary care settings.[3]

The initial and most crucial step in evaluating pleural effusion is to distinguish transudates from exudates, as it directs further diagnostic workup and management. Light's criteria, based on pleural fluid and serum total protein and lactate dehydrogenase (LDH) ratios, remain the gold standard owing to their high sensitivity (≈98%) but have limited specificity (≈80%).[4,5] Moreover, their accuracy diminishes in patients on diuretic therapy or in borderline biochemical profiles.[6] Once an effusion is categorized as exudative, identifying the underlying etiology—most commonly tubercular, parapneumonic, or malignant—is often challenging because traditional diagnostic tests are either invasive, time-consuming, or have suboptimal yield.


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Conventional markers such as adenosine deaminase (ADA), interferon-γ, and pleural fluid cytology are widely used, but each has limitations. While ADA is sensitive for tubercular effusion, its specificity drops in parapneumonic or empyematous effusions.[7,8] Pleural fluid cytology has a diagnostic yield of only 40–60% in malignant effusions,[9] and microbiological confirmation in tubercular or parapneumonic cases is achieved in less than 30% of cases due to the paucibacillary nature of the fluid.[10] Consequently, there is an unmet need for an additional biochemical marker that is simple, rapid, inexpensive, and capable of distinguishing inflammatory from non-inflammatory causes of exudative pleural effusion.

C-reactive protein (CRP), an acute-phase reactant synthesized by hepatocytes in response to pro-inflammatory cytokines (especially IL-6), has been extensively studied as a systemic marker of inflammation.[11] Elevated CRP levels in pleural fluid are indicative of local inflammatory activity within the pleural cavity. Previous studies have shown that pleural fluid CRP concentrations are significantly higher in parapneumonic and tubercular effusions compared to malignant or transudative ones.[12,13] For instance, it is reported mean pleural CRP levels of 80–120 mg/L in parapneumonic, 40–70 mg/L in tubercular, and <20 mg/L in malignant effusions, suggesting its discriminative potential.[14] Furthermore, pleural CRP estimation is inexpensive and can be performed using standard laboratory assays, making it feasible for use in resource-limited settings.

Despite encouraging data, considerable variation exists in the reported cut-off values and diagnostic performance of pleural CRP across studies. Sensitivity and specificity have ranged from 75–95% and 70–90%, respectively, depending on study population and assay method.[15] Indian data remain limited, and few studies have systematically compared CRP levels across the three predominant exudative etiologies—tubercular, parapneumonic, and malignant effusions—in the same cohort.

Hence, this study was aimed to evaluate the role of pleural fluid C-reactive protein in the etiological diagnosis of exudative pleural effusion, and to determine its diagnostic accuracy in differentiating between tubercular, parapneumonic, and malignant causes. Establishing reliable CRP thresholds may help clinicians in early, cost-effective differentiation of exudative effusions, thereby improving patient outcomes, especially in high TB-burden regions like India.

## MATERIALS AND METHODS

**Study Design and Setting:** This hospital-based prospective observational study was conducted in the Department of General Medicine at a tertiary care teaching hospital in North India, over a period of 24 months from June 2023 to May 2025. The study aimed to evaluate the diagnostic utility of pleural fluid C-reactive protein (CRP) in differentiating among the major etiologies of exudative pleural effusion—tubercular, parapneumonic, and malignant. Institutional Ethics Committee approval was obtained prior to initiation of the study, and written informed consent was obtained from all participants.

**Study Population :** Adult patients aged 18 years and above presenting with clinical and radiological evidence of pleural effusion and subsequently confirmed to have exudative pleural effusion based on Light's criteria were consecutively enrolled. Exudative effusion was defined by the presence of one or more of the following: pleural fluid to serum protein ratio >0.5, pleural fluid to serum lactate dehydrogenase (LDH) ratio >0.6, or pleural fluid LDH > two-thirds of the upper limit of normal serum LDH. Patients were excluded if they had transudative effusions due to cardiac, hepatic, or renal disease; those with mixed effusions; individuals on immunosuppressive therapy; and those with co-existing systemic inflammatory conditions such as rheumatoid arthritis, systemic lupus erythematosus, or recent major surgery or trauma. Patients who had received prior anti-tubercular or antibiotic therapy for more than two weeks before presentation were also excluded to avoid confounding effects on CRP levels.

**Clinical Evaluation and Diagnostic Work-up:** All patients underwent a detailed history and clinical examination, including evaluation of symptoms such as cough, fever, chest pain, breathlessness, and weight loss. Chest radiography and ultrasonography were performed to confirm the presence and site of pleural effusion. Diagnostic thoracentesis was performed under aseptic precautions, and pleural fluid was analyzed for routine biochemical, cytological, and microbiological parameters.

Pleural fluid analysis included appearance, total and differential cell count, protein, glucose, LDH, adenosine deaminase (ADA), and C-reactive protein (CRP) levels. Gram stain, Ziehl–Neelsen (ZN) staining for acid-fast bacilli, bacterial culture, and cytological examination for malignant cells were also performed. Simultaneous blood samples were collected for serum protein and LDH estimation to apply Light's criteria.

**Etiological Classification:** The etiology of pleural effusion was determined based on a combination of clinical features, biochemical parameters, and diagnostic investigations: Tubercular effusion was diagnosed when pleural fluid ADA ≥40 U/L, lymphocytic predominance (>70%), and exclusion of other causes, or when Mycobacterium tuberculosis was demonstrated in pleural fluid or pleural biopsy; Parapneumonic effusion was diagnosed in patients with pneumonia or lung abscess evident on imaging, with neutrophilic pleural fluid, positive bacterial culture or Gram stain, and/or clinical response to antibiotic therapy; and Malignant effusion was diagnosed when malignant cells were detected in pleural fluid cytology or confirmed by pleural biopsy


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or histopathology of a primary tumor with secondary pleural involvement.

**Estimation of Pleural Fluid C-Reactive Protein:** Pleural fluid CRP concentration was estimated using a high-sensitivity turbidimetric immunoassay method on an automated clinical chemistry analyzer (e.g., Beckman Coulter AU680 or equivalent). The assay was based on the principle of antigen–antibody agglutination reaction, where the increase in turbidity is directly proportional to CRP concentration. Results were expressed in milligrams per liter (mg/L). All samples were analyzed within two hours of collection to prevent protein degradation. Internal quality controls were maintained for each batch of testing, and samples with hemolysis or contamination were discarded.

**Statistical Analysis:** Data were entered in Microsoft Excel and analyzed using IBM SPSS Statistics version 25.0 (IBM Corp., Armonk, NY, USA). Quantitative variables such as CRP, protein, and LDH were expressed as mean ± standard deviation (SD) or median (interquartile range) depending on data distribution. Qualitative variables were summarized as frequencies and percentages. Comparison of mean CRP levels among different etiological groups (tubercular, parapneumonic, and malignant) was performed using one-way analysis of variance (ANOVA) followed by post hoc Tukey's test. The diagnostic accuracy of pleural fluid CRP was assessed using receiver operating characteristic (ROC) curve analysis to determine the area under the curve (AUC), optimal cut-off value, sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) for differentiating infectious (tubercular and parapneumonic) from non-infectious (malignant) effusions. A p-value <0.05 was considered statistically significant.

**Ethical Considerations:** The study adhered to the principles of the Declaration of Helsinki (2013 revision). Participation was voluntary, and informed written consent was obtained from all patients prior to enrollment. Patient confidentiality was strictly maintained throughout the study, and data were anonymized before statistical analysis.

## RESULTS

A total of 264 patients with exudative pleural effusion were enrolled. The mean age of the study population was 48.6 ± 15.2 years, with a male-to-female ratio of 1.7:1. The tubercular group constituted the majority (n = 142; 53.8%), followed by parapneumonic effusion (n = 67; 25.4%) and malignant effusion (n = 55; 20.8%). Common presenting symptoms were fever (68.9%), cough (63.3%), chest pain (58.7%), and weight loss (46.2%). The mean duration of symptoms was longest in the malignant group [Table 1].

**Table 1: Baseline Demographic and Clinical Characteristics of Patients (N = 264).**

| Parameter | Tubercular (n = 142) | Parapneumonic (n = 67) | Malignant (n = 55) | p-value |
|---|---|---|---|---|
| | Frequency (%)/ mean ± SD | | | |
| Age (years) | 43.1 ± 14.8 | 49.2 ± 13.5 | 59.8 ± 11.2 | <0.001 |
| Gender | | | | |
| Male | 93 (65.5%) | 47 (70.1%) | 29 (52.7%) | 0.028 |
| Female | 49 (34.5%) | 20 (29.9%) | 26 (47.3%) | |
| Male:Female ratio | 1.9:1 | 2.3:1 | 1.1:1 | 0.028 |
| Symptoms | | | | |
| Fever | 115 (81.0%) | 58 (86.6%) | 9 (16.4%) | <0.001 |
| Cough | 91 (64.1%) | 53 (79.1%) | 23 (41.8%) | <0.001 |
| Chest pain | 73 (51.4%) | 51 (76.1%) | 31 (56.4%) | 0.008 |
| Weight loss | 69 (48.6%) | 21 (31.3%) | 32 (58.2%) | 0.014 |
| Duration of symptoms (weeks) | 5.6 ± 2.4 | 3.9 ± 2.1 | 7.2 ± 2.9 | <0.001 |

Pleural fluid CRP levels were markedly elevated in parapneumonic effusions (mean ≈ 94 mg/L), moderate in tubercular effusions (≈ 50 mg/L), and lowest in malignant effusions (≈ 21 mg/L), demonstrating a significant gradient with inflammatory etiology. Pleural fluid analysis revealed that mean protein and LDH levels were significantly higher in parapneumonic effusions, while ADA was markedly elevated in tubercular cases. Pleural fluid CRP levels varied widely across groups, with highest values in parapneumonic effusions, followed by tubercular and malignant effusions ($p < 0.001$) [Table 2].

**Table 2: Pleural Fluid Biochemical Characteristics Across Etiological Groups.**

| Parameter | Tubercular (n = 142) | Parapneumonic (n = 67) | Malignant (n = 55) | p-value |
|---|---|---|---|---|
| | Frequency (%)/ mean ± SD | | | |
| Total protein (g/dL) | 5.3 ± 0.7 | 5.8 ± 0.8 | 4.9 ± 0.6 | <0.001 |
| LDH (U/L) | 472 ± 135 | 682 ± 158 | 403 ± 121 | <0.001 |
| Glucose (mg/dL) | 69.5 ± 21.6 | 48.2 ± 19.4 | 77.1 ± 24.3 | <0.001 |
| ADA (U/L) | 68.4 ± 22.5 | 33.1 ± 14.2 | 17.6 ± 8.9 | <0.001 |
| CRP (mg/L) | 49.8 ± 22.6 | 93.7 ± 34.1 | 21.4 ± 12.7 | <0.001 |


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Pleural fluid CRP at a cut-off of 35 mg/L reliably differentiated infectious from malignant effusions with an AUC = 0.91, high sensitivity (89%) and specificity (84%). A higher threshold (70 mg/L) improved specificity for identifying parapneumonic effusions. Receiver Operating Characteristic (ROC) analysis was performed to evaluate the discriminative power of pleural fluid CRP in differentiating infectious (tubercular + parapneumonic) from non-infectious (malignant) effusions [Table 3].

**Table 3: Diagnostic Accuracy of Pleural Fluid CRP in Differentiating Exudative Effusion Etiologies.**

| Comparison | Optimal CRP Cut-off (mg/L) | AUC (95% CI) | Sensitivity (%) | Specificity (%) | PPV (%) | NPV (%) | p-value |
|---|---|---|---|---|---|---|---|
| Infectious (TB + Parapneumonic) vs Malignant | 35 | 0.912 (0.875–0.949) | 88.7 | 83.6 | 93.5 | 72.3 | <0.001 |
| Parapneumonic vs Tubercular | 70 | 0.816 (0.752–0.879) | 79.1 | 74.6 | 76.3 | 77.5 | <0.001 |
| Tubercular vs Malignant | 30 | 0.889 (0.842–0.935) | 85.9 | 80 | 90.6 | 71.1 | <0.001 |

Pleural fluid CRP levels showed a strong positive correlation with pleural fluid LDH (r = 0.71, p < 0.001) and a negative correlation with glucose (r = –0.54, p < 0.001). A moderate positive correlation was observed with ADA (r = 0.46, p < 0.001) in the tubercular subgroup [Table 4 and Figure 1].

**Table 4: Correlation Between Pleural Fluid CRP and Other Parameters.**

| Parameter | Spearman's r | p-value | Interpretation |
|---|---|---|---|
| LDH (U/L) | 0.71 | <0.001* | Strong positive correlation |
| ADA (U/L) | 0.46 | <0.001* | Moderate positive correlation |
| Glucose (mg/dL) | –0.54 | <0.001* | Strong negative correlation |
| Protein (g/dL) | 0.33 | 0.012* | Mild positive correlation |

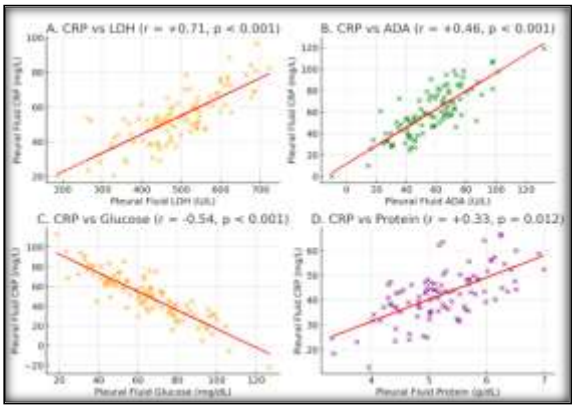


**Figure 1: Correlation between pleural fluid C-reactive protein (CRP) and other biochemical parameters (LDH, ADA, glucose, and protein).**

## DISCUSSION

In this prospective study involving 264 patients with exudative pleural effusion, we observed that tuberculosis (53.8%) remained the leading etiology, followed by parapneumonic (25.4%) and malignant effusions (20.8%), consistent with the prevailing pattern in Indian tertiary centers in studies by Reddy et al., and Bagal et al., where tuberculosis continues to dominate the exudative spectrum.[16,17]

Our findings align with Indian studies by Mandal et al., Bansal et al., and Raghavan et al., evaluating the diagnostic potential of pleural fluid CRP.[18-20] Mandal et al., reported significantly higher CRP levels in parapneumonic effusions (median 110 mg/L) than in tubercular (50 mg/L) or malignant effusions (18 mg/L), achieving an AUC of 0.90 for discriminating infectious from malignant etiologies.[18] Similarly, Bansal et al., and Raghavan et al., observed CRP levels >45 mg/L to indicate infectious etiology with sensitivity ranging from 84–92% and specificity 80–88%.[19,20] The present study corroborates these results, demonstrating that CRP values ≥35 mg/L reliably distinguished infectious effusions (tubercular and parapneumonic) from malignant ones (AUC = 0.91), highlighting comparable diagnostic accuracy in our Indian cohort. When analyzed separately, parapneumonic effusions in our study showed markedly elevated CRP (mean 93.7 mg/L) compared to tubercular effusions (mean 49.8 mg/L), consistent with observations by Barabde et al., and Sharma et al., who reported mean CRP values of 87–95 mg/L in bacterial effusions and 40–60 mg/L in tubercular cases.[21,22] These differences likely reflect the more intense neutrophil-mediated inflammatory response and cytokine surge—particularly interleukin-6 (IL-6) and tumor necrosis factor-α (TNF-α)—in bacterial infections compared to the lymphocyte-predominant immune response seen in tuberculosis.[23] In contrast, malignant effusions exhibited low CRP levels, mirroring the lower-grade, often non-purulent inflammatory milieu characteristic of tumor-related pleural involvement.[24]

The observed strong positive correlation between pleural CRP and LDH (r = 0.71, p < 0.001) and negative correlation with glucose (r = –0.54, p < 0.001) further supports CRP's association with the biochemical hallmarks of inflammation and cell turnover in the pleural space. Similar correlations have been reported by Hussein et al., and Porcel et al., both noting that CRP parallels LDH levels as both derive from neutrophil degranulation and tissue injury.[25,26] Elevated ADA levels in tubercular


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effusions were associated with moderately raised CRP (r = 0.46), suggesting that while both reflect inflammation, ADA is more specific to T-cell activation, whereas CRP indicates generalized acute-phase response.[27]

Receiver operating characteristic (ROC) curve analysis identified a CRP cut-off of 35 mg/L as optimal for differentiating infectious (tubercular/parapneumonic) from non-infectious (malignant) effusions, with an area under curve (AUC) of 0.912, sensitivity of 88.7%, and specificity of 83.6%. The biological plausibility of elevated pleural CRP lies in its hepatic synthesis under IL-6 stimulation, followed by transudation and local production within the pleural space during active inflammation. In parapneumonic effusions, bacterial infection of the pleural cavity triggers intense cytokine-mediated exudation of CRP-rich plasma and possibly mesothelial secretion, explaining the higher concentrations observed. In tubercular pleuritis, chronic granulomatous inflammation results in moderately elevated CRP levels, while malignant effusions, characterized by minimal inflammatory response and obstruction-related fluid accumulation, exhibit comparatively lower CRP concentrations.[28,29] Thus, pleural CRP effectively mirrors the intensity and nature of the local inflammatory process.

**Clinical Implications:** The findings of this study highlight pleural fluid CRP as a simple, rapid, and cost-effective biomarker that can aid in the etiological differentiation of exudative pleural effusions, particularly in resource-limited settings where access to advanced molecular diagnostics may be constrained. In combination with ADA and cytology, CRP measurement enhances diagnostic confidence and helps prioritize treatment decisions. A cut-off of 35 mg/L demonstrated robust sensitivity and specificity for identifying infectious causes, suggesting its potential as a screening tool before resorting to invasive procedures like pleural biopsy or thoracoscopy. Additionally, since CRP measurement is routinely available in most clinical laboratories, it offers practical utility in day-to-day pulmonary practice.

While ADA remains the standard biomarker for tubercular effusions, its specificity is limited in empyema or parapneumonic effusions.[30-32] CRP, in contrast, provides complementary information—high in both tubercular and bacterial effusions but low in malignant ones. Thus, integrating ADA and CRP improves differential diagnosis: high ADA with moderate CRP suggests tuberculosis, while very high CRP with low ADA favors parapneumonic etiology. This synergistic approach has been advocated by Behera et al., and Mohapatra et al., as a practical diagnostic algorithm for exudative pleural effusion in high-burden regions.[33,34]

**Strengths and Limitations:** The major strength of our study lies in its adequate sample size (N = 264), robust biochemical comparison across major etiological categories, and use of ROC analysis to establish optimal diagnostic cut-offs. The findings are clinically generalizable to similar high-TB-burden settings. However, the study was conducted in a single center, and microbiological confirmation of tuberculosis and bacterial pathogens was limited by low yield, a common constraint in pleural fluid studies. Future multicentric studies incorporating molecular assays (GeneXpert, PCR-based CRP isoforms) may further refine the diagnostic accuracy of CRP.

## CONCLUSION

In summary, pleural fluid C-reactive protein serves as a reliable adjunct biomarker for distinguishing the etiology of exudative pleural effusions. Significantly higher CRP levels in parapneumonic and tubercular effusions compared to malignant effusions, coupled with strong correlation with other inflammatory markers, reinforce its pathophysiological relevance. A threshold of 35 mg/L provides optimal diagnostic discrimination between infectious and non-infectious effusions, making pleural CRP a valuable, cost-effective addition to routine pleural fluid analysis in clinical practice.

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