Section: Miscellaneous

Original Research Article

A MULTIDISCIPLINARY DIAGNOSTIC APPROACH TO PLEURAL EFFUSION- A ONEYEAR RETROSPECTIVE STUDY

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ABSTRACT

Background: Pleural fluid evaluation is an important minimally invasive diagnostic approach for identifying the possible etiology of pleural effusion and guiding further clinical workup and management. Integrated assessment of cytological, biochemical and microbiological parameters provides valuable supportive information in differentiating transudative and exudative effusions and in identifying infective, tuberculous, malignant and inflammatory patterns. The objective is to evaluate the diagnostic significance of pleural fluid total cell count, differential cell predominance, adenosine deaminase, glucose, protein, lactate dehydrogenase and culture-sensitivity findings in patients with pleural effusion.

Materials and Methods: This retrospective observational study was conducted at a tertiary care centre on 111 pleural fluid samples received over a one-year period. Pleural fluid samples were analysed using a multidisciplinary laboratory approach involving cytological, biochemical and microbiological evaluation. The parameters assessed included total cell count, differential cell type, adenosine deaminase, glucose, protein, lactate dehydrogenase and culture-sensitivity testing.

Results: Exudative effusions were more common than transudative effusions, accounting for 60% and 40% of samples, respectively. Total cell count was below 500 cells/cu mm in 65% of samples. Lymphocytic predominance was observed in 74.7% of samples, while neutrophilic predominance was observed in 25.3%. Adenosine deaminase levels were below 40 IU/L in 73.5% of samples and above 40 IU/L in 26.5%. Biochemical analysis showed mean pleural fluid glucose, protein and lactate dehydrogenase levels of 118.6 mg/dL, 4.28 g/dL and 1401.6 U/L, respectively. Pleural fluid culture was positive in 3.6% of samples.

Conclusion: Multidisciplinary pleural fluid evaluation combining cytological, biochemical and microbiological parameters provides useful diagnostic support in the assessment of pleural effusion. Since no single parameter is independently diagnostic in all cases, integrated interpretation of cell count, differential pattern, adenosine deaminase, glucose, protein, lactate dehydrogenase and culture findings improves etiological assessment and helps guide further clinical management.

Keywords: Pleural effusion; Pleural fluid analysis; Adenosine deaminase; Lactate dehydrogenase; Culture sensitivity; Multidisciplinary diagnosis.

INTRODUCTION

Pleural effusion is a common and clinically significant manifestation of several pulmonary, pleural and systemic disorders. It occurs due to abnormal accumulation of fluid within the pleural space, usually resulting from an imbalance between pleural fluid formation and absorption. The underlying causes are diverse and include cardiac failure, renal disease, liver disease, infection, tuberculosis, malignancy and inflammatory disorders. Therefore, pleural effusion should be approached as a clinical sign of an underlying disease process rather than as an isolated diagnosis. A systematic diagnostic evaluation is essential because appropriate management depends on accurate identification of the underlying etiology.[1,2]

Diagnostic thoracocentesis and pleural fluid analysis remain central to the initial evaluation of pleural effusion. The first diagnostic step is usually classification of pleural fluid into transudative and exudative categories, as this helps narrow the differential diagnosis and directs further clinical workup. Transudative effusions are commonly related to systemic disturbances in hydrostatic or oncotic pressure, whereas exudative effusions are more often associated with local pleural or pulmonary pathology, including infection, tuberculosis, malignancy and inflammatory disease.[2-13]

Among exudative effusions, infective pleural effusions require particular attention because they may be associated with increased morbidity, prolonged antimicrobial therapy, need for pleural drainage and adverse clinical outcomes. Infective effusions commonly occur as parapneumonic effusions secondary to pneumonia and may progress to complicated parapneumonic effusion or empyema when the inflammatory process extends into the pleural space. Early recognition is important because delayed diagnosis and delayed pleural intervention may worsen clinical course and increase the need for further therapeutic procedures.[12-16]

Pleural fluid cytology and biochemical analysis provide important supportive information in identifying infective and inflammatory patterns. Neutrophilic predominance in pleural fluid is commonly associated with acute inflammatory conditions such as parapneumonic effusion and empyema, while lymphocytic predominance is more frequently seen in tuberculosis, malignancy and chronic inflammatory conditions. However, differential cell count alone is not diagnostic and must be interpreted along with biochemical parameters, microbiological results, imaging findings and clinical presentation.[5,6,10]

Biochemical markers such as glucose, protein and lactate dehydrogenase are useful in characterizing pleural effusions and assessing inflammatory activity within the pleural space. Low pleural fluid glucose and elevated LDH may be seen in pleural infection, tuberculosis, malignancy and inflammatory pleural diseases. LDH is a sensitive marker of cellular injury and inflammation but is not disease-specific; therefore, it should be interpreted as part of a combined diagnostic panel rather than in isolation.[9,10,13]

Adenosine deaminase is an important adjunctive marker in the evaluation of exudative pleural effusions, especially in regions where tuberculosis is common. Raised pleural fluid ADA is frequently associated with tuberculous pleuritis, but elevated ADA levels may also be observed in empyema, parapneumonic effusion, lymphoma, malignant pleural effusion and selected inflammatory disorders. Hence, ADA estimation improves diagnostic interpretation when used along with cell count, biochemical findings and clinical correlation, but it should not be considered an independent confirmatory test in all cases.[7,8,11]

Microbiological culture and sensitivity testing are essential in suspected infective pleural effusion because organism identification allows targeted antimicrobial therapy. However, culture yield may be limited in routine practice due to prior antibiotic exposure, delayed thoracocentesis, low organism burden, fastidious organisms or sampling-related limitations. Meyer et al. reported that patients with microbiology-negative pleural infection had outcomes comparable to those with known bacterial etiology, emphasizing that culture negativity should not be interpreted as absence of clinically significant pleural infection.[12] Recent literature also supports that microbiological diagnosis in pleural infection may be limited by conventional culture methods and should be interpreted within the broader clinical and biochemical context.[14,16,17]

In this context, multidisciplinary pleural fluid evaluation has practical diagnostic value. A combined assessment of total cell count, differential cell pattern, glucose, protein, LDH, ADA and microbiological culture provides a broader framework for differentiating transudative and exudative effusions and for identifying infective, tuberculous, malignant and inflammatory patterns. Such an approach is particularly relevant in tertiary care settings, where patients may have received empirical treatment before pleural fluid sampling. Therefore, integrated pleural fluid analysis remains an important component of etiological assessment and clinical decision-making.[2,9,13]

The present study was undertaken to evaluate the diagnostic significance of pleural fluid cytological, biochemical and microbiological parameters, including total cell count, differential cell predominance, ADA, glucose, protein, LDH and culture-sensitivity findings, in patients with pleural effusion. This integrated laboratory approach was used to assess the role of multidisciplinary pleural fluid evaluation in supporting etiological interpretation and guiding further clinical workup.

Aim

To evaluate the diagnostic utility of a multidisciplinary pleural fluid analysis approach by integrating cytological, biochemical and microbiological parameters in patients with pleural effusion.

Objectives

1. To assess the distribution of pleural effusions based on cytological and biochemical characteristics, including total cell count, differential cell predominance, adenosine deaminase, glucose, protein and lactate dehydrogenase levels.
2. To evaluate the microbiological culture and sensitivity findings in pleural fluid samples and correlate them with the overall laboratory pattern to support etiological interpretation of pleural effusion.

MATERIALS AND METHODS

This retrospective observational study was conducted at a tertiary care centre over a one-year period. Pleural fluid samples received for laboratory evaluation from January 2025 to December 2025 were included in the study. The study followed a multidisciplinary diagnostic approach involving the Departments of Pathology, Biochemistry and Microbiology.

A total of 111 pleural fluid samples received during the study period were included. Samples were evaluated using cytological, biochemical and microbiological parameters. Cytological evaluation included total white blood cell count and differential cell type analysis. Based on the predominant cell population, the pleural fluid samples were categorized as lymphocyte-predominant or neutrophil-predominant. This distinction was used as supportive information for interpreting chronic inflammatory, infective and other pleural disease processes.

Biochemical analysis included estimation of pleural fluid adenosine deaminase, glucose, total protein and lactate dehydrogenase. These parameters were assessed to support classification and etiological interpretation of pleural effusions. Protein and LDH were used as important biochemical indicators in differentiating exudative and transudative patterns, while glucose and LDH were interpreted as supportive markers of inflammatory or infective pleural activity. ADA was assessed as an additional marker relevant to the evaluation of tuberculous and chronic inflammatory effusions.

Microbiological evaluation was performed by culture and sensitivity testing of pleural fluid samples. Culture-positive isolates were identified, and antimicrobial susceptibility patterns were recorded to provide clinically relevant information for targeted antimicrobial therapy.

Data were collected retrospectively from laboratory records. The parameters analysed included total cell count, differential cell predominance, ADA, glucose, protein, LDH and culture-sensitivity findings. Data were entered, tabulated and analysed using descriptive statistics. Categorical variables were expressed as frequencies and percentages, while continuous variables were expressed as mean, standard deviation and range, wherever applicable. The study was designed to assess the diagnostic utility of integrated pleural fluid analysis in the evaluation of pleural effusion.

RESULTS

In this study, a total of 111 cases of pleural effusion were studied and the following observations were made. Out of total 111 samples 40% were transudative, about 60% were exudative.

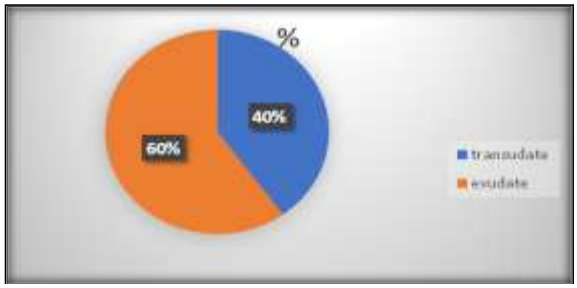

Figure 1: Pleural Fluid Classification

Total cell countupto 500 cells/ cu mm seen in 65% of cases, >500cells/cu mm seen in 35% of cases, out of which lymphocytic predominance noted in 83 cases constituting (74.7%), neutrophils predominance noted in 28 cases constituting (25.3%).

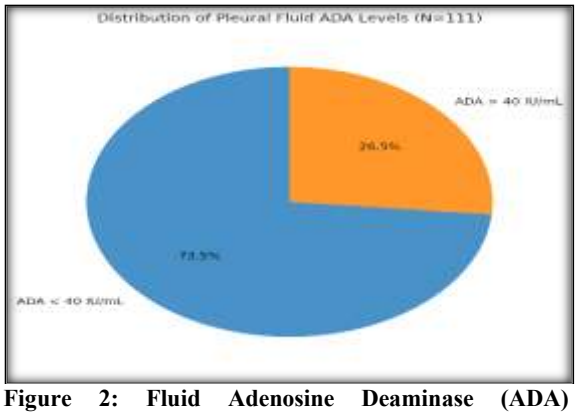

Figure 2: Fluid Adenosine Deaminase (ADA) Distribution

Table 1: Pleural Fluid Cellular Characteristics(N=111)

Cellular Parameter	Case count (n)	Percentage (%)
Total cell count		
<500 cells/mm3	72	65
>500 cells/mm3	39	35

Differential Predominance		
Lymphocytic	83	74.7
Neutrophilic	28	25.3

Out of total 111cases ADA levels <40IU/ml seen in 73.5% of cases, >40IU/ml seen in 26.5% of cases.
In present study biochemical analysis showed fluid glucose displayed mean 118.6±16.27mg/dl, fluid protein mean was 4.28±0.98g/dL, fluid LDH mean 1401.6±385.8U/L.

Table 2: Quantitative Biochemical Parameter Summary (N=111)

Biochemical parameter	Mean + SD	Range
Fluid glucose (mg/dL)	118.6 ± 16.27	2 - 336
Fluid protein (g/dL)	4.28 ± 0.98	1.2 - 42
Fluid LDH (U/L)	1401.6 ± 385.8	54 - 20,300

Fluid culture and sensitivity revealed positive culture in 3.6 % of the cases.

Table 3: Bacterial Isolates and Antibiotic Susceptibility Testing (AST) Profiles (N=111)

Culture Status / Isolate	Case Count (n)	Proportion (%)	Antibiotics Sensitive	Antibiotics Intermediate / Resistant
Culture Negative	107	96.4%	—	—-
Culture Positive	4	3.6%	—	—-
Klebsiella pneumoniae	2	1.8%	AMK, SAM, ATM, FEP, CTX, CHL, CIP, IPM, MEM, NET, TZP, SXT	Resistant: AMP
Citrobacter koseri	1	0.9%	AMK, ATM, FEP, CTX, CAZ, CIP, DOX, GEN, IPM, MEM, NET, OFX, TZP, TOB, SXT	Pan-sensitive (None)
Enterococcus faecium	1	0.9%	PCN, CIP, GEN (High-Level Synergy), LEV, NIT, TEC, TGC, VAN	Intermediate: ERY, LZD Resistant: TET

Abbreviations: AMK: Amikacin; AMP: Ampicillin; SAM: Ampicillin/Sulbactam; ATM: Aztreonam; CAZ: Ceftazidime; CTX: Cefotaxime; FEP: Cefepime; CHL: Chloramphenicol; CIP: Ciprofloxacin; DOX: Doxycycline; ERY: Erythromycin; GEN: Gentamicin; IPM: Imipenem; LEV: Levofloxacin; LZD: Linezolid; MEM: Meropenem; NET: Netilmicin; NIT: Nitrofurantoin; OFX: Ofloxacin; PCN: Benzylpenicillin; TEC: Teicoplanin; TET: Tetracycline; TGC: Tigecycline; TOB: Tobramycin; TZP: Piperacillin/Tazobactam; SXT: Trimethoprim/Sulfamethoxazole; VAN: Vancomycin.

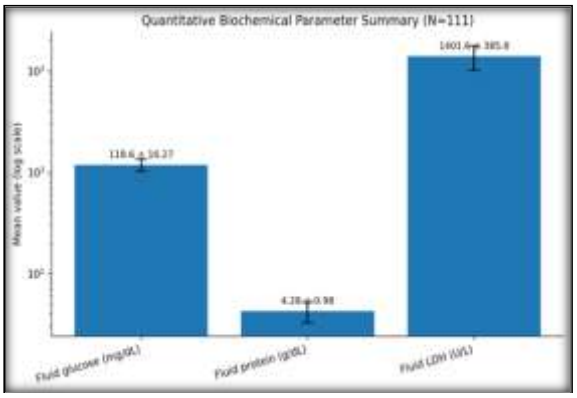

Figure 3: Quantitative Biochemical Parameter Summary

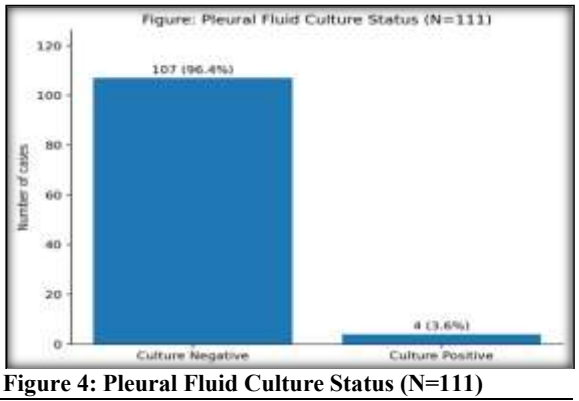

Figure 4: Pleural Fluid Culture Status (N=111)

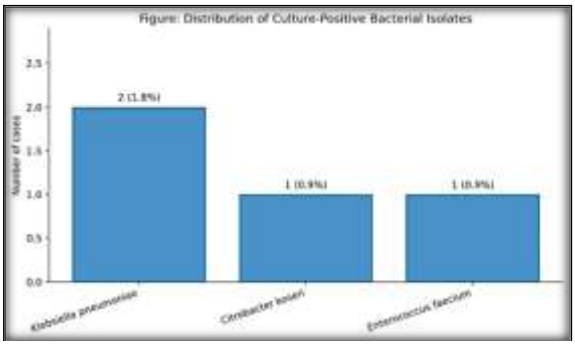

Figure 5: Distribution Of Culture Positive Bacterial Isolates

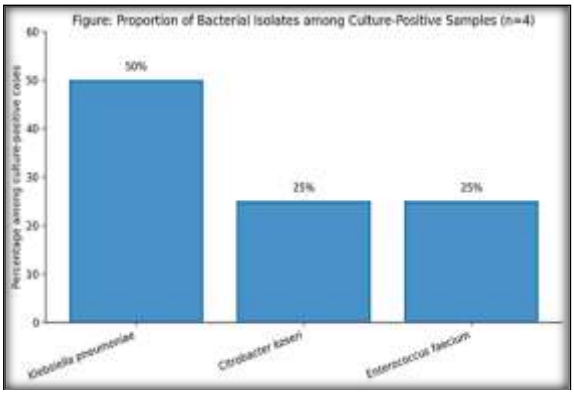

Figure 6: Distribution of Culture-Positive Bacterial Isolates in Pleural Fluid Samples

DISCUSSION

Pleural effusion is defined as the abnormal accumulation of fluid within the pleural space and reflects an imbalance between pleural fluid formation and removal. It is not a disease by itself, but an important clinical manifestation of underlying pulmonary, pleural or systemic pathology. Accurate etiological identification is essential because management varies according to the underlying cause. Evaluation of pleural fluid cytology, biochemical parameters, microbiological findings and clinical context provides valuable diagnostic information and helps guide appropriate treatment decisions.[1,2]

Light's criteria remain the most widely used method for differentiating transudative and exudative pleural effusions. Transudative effusions usually occur due to systemic alterations in hydrostatic or oncotic pressure, such as congestive cardiac failure, nephrotic syndrome, liver cirrhosis, hypoalbuminemia and peritoneal dialysis. In contrast, exudative effusions are commonly associated with local pleural or pulmonary pathology, including pulmonary infections such as pneumonia and tuberculosis, malignancy, inflammatory disorders, pancreatitis, lupus, rheumatoid arthritis, post-cardiac injury syndrome, chylothorax, hemothorax and post-coronary artery bypass grafting.[2,4,9,13]

In the present study, 40% of pleural effusions were transudative and 60% were exudative, indicating a predominance of exudative effusions in the study population. This finding is comparable with observations reported by Jaison et al. and Kushwaha et al.[4,5] The higher proportion of exudative effusions highlights the importance of detailed laboratory evaluation, as exudates are associated with a wide range of infective, tuberculous, malignant, inflammatory and systemic etiologies requiring further clinical correlation.

Differential cell count provides useful supportive information in pleural fluid interpretation. In the present study, lymphocytic predominance was observed in 74.7% of samples, while neutrophilic predominance was noted in 25.3% of samples. Lymphocyte-predominant effusions are commonly associated with tuberculosis, malignancy and chronic inflammatory conditions. Neutrophil-predominant effusions are more commonly seen in acute inflammatory conditions, particularly parapneumonic effusion, empyema, pulmonary embolism and acute pancreatitis.[6.10] However, cellular predominance alone is not diagnostic and should always be interpreted along with biochemical findings, microbiological results, radiological features and clinical presentation.

Adenosine deaminase is a useful adjunctive marker in the evaluation of exudative pleural effusions, particularly in settings where tuberculous pleuritis is common. In the present study, ADA levels were below 40 IU/L in 73.5% of samples and above 40 IU/L in 26.5% of samples. ADA values above 40 IU/L are commonly associated with tuberculous pleurisy, but elevated ADA may also be observed in empyema, parapneumonic effusion, malignant pleural effusion, lymphoma and selected inflammatory conditions.[7,11] Therefore, ADA should not be interpreted as an isolated diagnostic marker. Low ADA levels make tuberculous pleuritis less likely, but do not exclude other non-tuberculous infective, inflammatory or malignant causes.[8]

Biochemical analysis in the present study showed a mean pleural fluid glucose of 118.6 mg/dL, mean protein of 4.28 g/dL and mean LDH of 1401.6 U/L. Pleural fluid glucose and LDH are particularly useful in identifying inflammatory or infective activity within the pleural space. Low glucose and elevated LDH are commonly associated with pleural infection, malignancy and inflammatory pleural diseases. LDH is a sensitive marker of cellular injury and inflammation, but it is not disease-specific. Pleural fluid LDH values greater than 1000 U/L may be seen in pleural infection, rheumatoid pleurisy, tuberculous pleurisy and malignancy.[9,13] Therefore, LDH must be interpreted together with cell count, glucose, protein, ADA, culture results and clinical findings.

Microbiological evaluation in the present study revealed culture positivity in 3.6% of samples. Although the culture yield was low, culture-positive results remain clinically significant because they provide direct microbiological evidence and allow organism-specific antimicrobial guidance. The bacterial isolates included Klebsiella pneumoniae, Citrobacter koseri and Enterococcus faecium. The antimicrobial susceptibility patterns helped identify potentially useful therapeutic options and resistance patterns. However, because the number of culture-positive samples was small, these findings should be interpreted descriptively and should not be generalized as representative of the broader microbiological profile of pleural infections in the population.

Low culture positivity does not exclude infective pleural disease. Pleural fluid cultures may be negative due to prior antibiotic administration, delayed thoracocentesis, low bacterial load, inadequate sample volume, fastidious organisms or limitations of conventional culture methods. Meyer et al., in a large retrospective cohort of pleural infection, observed that patients with microbiology-negative pleural infection had outcomes comparable to those with known bacterial etiology. Their study emphasized that microbiology-negative pleural infection is common and that absence of culture positivity should not be interpreted as absence of clinically significant infection.[12] Recent literature on pleural infection also supports that microbiological confirmation may be limited in routine practice and that culture results should be interpreted within the broader clinical, biochemical and radiological context.[14,16,17]

The findings of Meyer et al. are relevant to the present study because they support the need for integrated interpretation of pleural fluid results. In suspected infective effusions, culture and sensitivity testing remain essential when positive, but culture negativity should be interpreted in the context of cytological and biochemical indicators such as neutrophilic predominance, elevated LDH, altered glucose and clinical-radiological evidence of infection. This is particularly important in routine clinical practice, where empirical antibiotics may be initiated before pleural fluid sampling.[12,17]

The present study supports the value of a multidisciplinary approach involving pathology, biochemistry and microbiology in the evaluation of pleural effusion. Cytological examination helps identify the predominant inflammatory cell pattern, biochemical analysis assists in classifying the effusion and assessing inflammatory activity, ADA contributes to the evaluation of tuberculous and chronic inflammatory effusions, and microbiological culture provides organism-specific guidance when positive. Since no single parameter is completely diagnostic, combined interpretation improves the diagnostic utility of pleural fluid analysis and helps clinicians plan further investigations and management.

CONCLUSION

Pleural fluid analysis is an essential diagnostic tool in the evaluation of pleural effusion. In the present study, an integrated multidisciplinary approach combining cytological, biochemical and microbiological parameters provided useful diagnostic support for differentiating transudative and exudative effusions and for assisting etiological interpretation.

Biochemical markers such as protein, glucose and lactate dehydrogenase help characterize the nature of the effusion and assess the degree of pleural inflammatory activity. Adenosine deaminase provides additional supportive information in the evaluation of tuberculous and chronic inflammatory effusions. Differential cell count further helps identify lymphocytic or neutrophilic patterns, which may guide clinical suspicion toward tuberculous, malignant, chronic inflammatory or infective etiologies.

Culture and sensitivity testing showed a low positivity rate, but remained clinically relevant in culture-positive cases by identifying bacterial isolates and their antimicrobial susceptibility patterns. However, culture-negative results should not exclude infective pleural disease, particularly when cytological, biochemical, clinical or radiological findings suggest infection. Evidence from pleural infection studies supports that microbiology-negative pleural infection is common and requires careful clinical correlation.

Overall, the study emphasizes that pleural fluid should be interpreted as a composite diagnostic specimen rather than through isolated parameters. A coordinated laboratory approach involving pathology, biochemistry and microbiology improves diagnostic clarity, supports timely clinical decision-making and helps guide appropriate management of patients with pleural effusion.

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