Research Article

Evaluation of Pediatric Pleural Effusion: Insights from a Tertiary Care Center in North India

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Article Info

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Keywords

Children, Empyema, Inter-costal drainage, Pleural Effusion, Tuberculosis

Abstract

Background: Pediatric pleural effusion, characterized by abnormal fluid accumulation in the pleural space, arises from various etiologies, including infections and systemic diseases. This study evaluated the clinical, radiological, and bacteriological profiles of pleural effusion and related outcomes in children in North India.

Methodology: A prospective observational study was conducted on 133 children aged 0–19 years with confirmed pleural effusion at a tertiary care centre. Comprehensive clinical history, socioeconomic data, physical examinations, imaging, and pleural fluid (PF) analysis were performed. Microbiological testing, including CBNAAT for Mycobacterium tuberculosis, was utilized when tuberculosis was suspected. Follow-ups were conducted until 6 weeks post-discharge.

Results: The mean age was 8.67 ± 4.93 years, with a male predominance (54.1%). Common symptoms included fever (85.7%) and cough (67.7%), and right-sided effusion (49.6%) was most frequent. Diagnoses included tuberculosis (21.1%), bacterial empyema (20.3%), and sepsis (16.5%). PF cultures detected Staphylococcus aureus in 3.8% and Streptococcus pneumoniae in 2.3%. CBNAAT was positive in only 7.1% of PF samples but in 57.1% of respiratory specimens (sputum or gastric aspirate). Most cases (62.4%) were managed conservatively, 20.3% required intercostal tube drainage, and 10.5% underwent surgery. The median ICT duration was 239 hours, and median hospital stay was 342 hours. Mortality was 6.8%, mainly from sepsis.

Conclusion: This study underscores diverse etiologies of pediatric pleural effusion, emphasizing tuberculosis and empyema. Timely diagnosis and management, including surgical intervention when needed, are essential to enhance outcomes.

Introduction

Pleural effusion results from an abnormal accumulation of fluid in the pleural space, often due to an imbalance between fluid production and drainage [1]. In children, the leading causes include infections, congestive heart failure, and nephrotic syndrome. Pneumonia often serves as a common precursor, accounting for up to 70% of cases [1]. Some cases of pneumonia can progress to empyema, which occurs in approximately 0.6-2% of children with bacterial pneumonia [2]. The most frequently isolated organisms in such cases include Staphylococcus aureus, followed by Streptococcus pneumoniae and Klebsiella [3,4]. The prognosis of pleural effusion in children largely depends on the underlying disorder and the treatment administered [5]. In India, particularly within the pediatric population, factors such as delayed medical consultation and the indiscriminate use of antibiotics contribute to poor diagnostic yields, leading to increased morbidity from complications like empyema [6].

Tuberculous effusions are generally observed in adolescents, whereas empyema is more prevalent among children aged 1 to 5 years, with a higher incidence in males and those from lower socio-economic backgrounds [5,7,8]. Predisposing factors such as cerebral palsy, congenital heart disease, and immunosuppression are commonly associated with pleural effusion; although it can also occur without any underlying conditions [9]. Differentiating between exudative and transudative effusions using Light's criteria can aid in identifying the underlying pathology [10,11]. Imaging modalities, including chest radiographs, ultrasound, and, in specific cases, contrast-enhanced computed tomography (CECT), play a crucial role in diagnosis and management [12,13,14].

Given the diverse etiologies and outcomes associated with pleural effusion, understanding the clinical, radiological, and bacteriological profiles is essential for optimizing management strategies. The high incidence of complications such as empyema, combined with regional variations in presentation and outcomes, highlights the need for localized data to inform treatment approaches. Therefore, this study was designed to elucidate the clinical, radiological, and bacteriological profiles of pleural effusion in hospitalized children in North India. Additionally, the study aimed to evaluate the outcomes related to the type and etiology of pleural effusion in the pediatric age group.

Material and Methods

This prospective observational study was conducted at a tertiary care center in North India after obtaining approval from the Institutional Ethics Committee(IEC/GMC/2019/787). The study included all the children aged 0 to 19 years admitted with clinical and radiological evidence of pleural effusion during the study period. The upper age limit of 19 years was adopted because, at our institution, patients up to 19 years of age are routinely managed under the Department of Pediatrics, in accordance with institutional admission policy Exclusion criteria included those who either did not provide informed consent or were lost to follow-up during the course of the study.

Data Collection

For each enrolled patient, a detailed clinical history was taken, which included information on symptoms, their duration, and any relevant medical history, such as prior infections, tuberculosis contact, or comorbidities like congenital heart disease, immunosuppressive conditions or any underlying chronic disease. Socio-economic status as per modified Kuppuswamy scale, was recorded for all participants to examine its association with disease outcomes. A thorough physical examination was performed for all patients.

Diagnostic Investigations

Both chest X-rays and ultrasound imaging were performed for all patients to confirm the diagnosis of pleural effusion and assess fluid characteristics such as size, location, and any associated conditions like lung consolidation or collapse. Ultrasound also provided guidance for pleural fluid (PF) aspiration when required. In cases of minimal effusion without respiratory compromise, PF aspiration was avoided to minimize unnecessary invasive procedures.

Computed tomography (CT) scans were reserved for cases where detailed anatomical evaluation was necessary, especially when surgical intervention was planned or if complications like loculated effusions, lung abscesses, or other associated anomalies were suspected, based on the clinician's discretion. PF aspiration was performed under aseptic conditions, and the aspirated fluid underwent biochemical (protein, glucose, lactate dehydrogenase), cytological (cell count and differential), and bacteriological testing. Light's criteria were used to classify the pleural effusion as either exudative or transudative. PF pH was measured using a blood gas analyzer. Samples were also sent for Gram stain, bacterial cultures, and polymerase chain reaction (PCR) for detecting Mycobacterium tuberculosis (M.tb) through CBNAAT (Cartridge Based Nucleic Acid Amplification Test) whenever indicated. Additional diagnostic workup was done depending on suspected etiology.

Pleural fluid samples were collected under aseptic precautions and transported promptly to the central clinical laboratory for analysis. Biochemical analysis included estimation of total protein, glucose, and lactate dehydrogenase (LDH), which were performed using standard automated spectrophotometric methods in accordance with the manufacturer's instructions and the institutional laboratory's internal quality control procedures. Simultaneous blood samples were obtained, wherever indicated, to facilitate the application of Light's criteria.. Pleural fluid samples for pH estimation were collected in a heparinized blood gas syringe, avoiding air bubbles, and analyzed immediately after collection to reduce pre-analytical variation. Samples intended for microbiological evaluation were processed without delay and subjected to Gram staining, bacterial culture, and

CBNAAT (Cartridge Based Nucleic Acid Amplification Test) for Mycobacterium tuberculosis (M. tb.), whenever clinically indicated, following standard laboratory protocols.
The final diagnosis was established based on the clinical presentation, radiological findings, pleural fluid analysis, microbiological investigations, and additional diagnostic tests, as appropriate. Children with pleural effusion secondary to pneumonia were classified as having either simple parapneumonic effusion or empyema. This distinction was based on thoracic ultrasonography and pleural fluid analysis. Empyema/ Complicated parapneumonic effusion was diagnosed by the presence of grossly purulent pleural fluid, positive Gram stain or bacterial culture of pleural fluid, or ultrasonographic features suggestive of a complicated pleural collection, including echogenic debris, septations, loculations, or pleural thickening (pleural peel). Pleural effusions associated with pneumonia that did not demonstrate these features were classified as simple parapneumonic effusions.

Follow-Up and Outcome Assessment

After the initial management during the hospital stay, all the patients were followed up on OPD basis four weeks and six weeks after discharge. The final outcome was recorded at six weeks with repeat chest x-ray and ultrasound thorax to assess any residual effusion or re-accumulation of PF. Any patient requiring surgical intervention was referred to surgical department and the final outcome was then recorded at 6 weeks post-discharge.

Statistical Analysis

Data were systematically entered into Microsoft Excel for organization and later analyzed using SPSS version 20.
The baseline characteristics of the study population were summarized using descriptive statistics, with mean and standard deviation or median (IQR) for continuous variables and frequency and percentage for categorical variables. The distribution of continuous variables was assessed using the Shapiro–Wilk test. Continuous variables with a non-normal distribution were compared across etiological groups using the Kruskal–Wallis test, while categorical variables were analyzed using the Chi-square test or Fisher's exact test, as appropriate Statistical significance was set at a p-value of <0.05 for all tests.

Results

A total of 133 children with pleural effusion were included in the study. The demographic and clinical characteristics are summarized in Table 1. The mean age at presentation was 8.67 ± 4.93 years with 72 (54.1%) males. Common symptoms included fever (85.71%), cough (67.66%), and shortness of breath (53.38%). The predominant clinical signs were decreased breath sounds (90.22%) and dull percussion note (87.96%). 78.2% patients were found to be anaemic.

Table 1: Baseline Characteristics of the study Population (N=133).

Characteristic	Value {mean ± SD / n(%)}
Age (years)	8.67 ± 4.93 years
Male	72 (54.1%)
Clinical features	
Fever	114 (85.71)
Cough	90 (67.66)
Chest pain	34 (25.56)
Shortness of breath	71 (53.38)
Generalized swelling	22 (16.54)
Ascites	18 (13.53)
Pedal edema	9 (6.76)
Localized swelling	5 (3.75)
Jaundice	8 (6.01)
Vomiting	24 (18.04)
Urinary complaints	11 (8.27)

Characteristic	Value {mean ± SD / n(%)}
Clinical Signs	
Tachypnea	91 (68.42)
Tachycardia	71 (53.38)
Anemia	105 (78.9)
Lymphadenopathy	34 (25.56)
Dull percussion	117 (87.96)
Bronchial breathing	15 (11.27)
Decreased breath sounds	120 (90.22)
Immunization Status	
Completely immunized	90 (67.7)
Partially immunized	14 (10.5)
Unimmunized	29 (21.8)
Socioeconomic Status (Modified Kuppuswamy Scale)	
Lower class (<10)	62 (46.61)
Middle class (1125)	46 (34.58)
Upper class (2629)	25 (18.79)

Out of the total patients enrolled in the study, 105 (78.9%) were anaemic as per the World Health Organization (WHO) criteria, with a mean haemoglobin level of 8.96 ± 1.95 gm/dL. Among these anaemic children, 17 had severe anaemia, 86 had moderate anaemia, and 2 had mild anaemia. The mean total leucocyte count (TLC) was 11,962.33 ± 6,949.28 cells/mm³. Based on age-appropriate laboratory reference ranges, 5 (3.8%) children had a decreased TLC, 75 (56.4%) had a TLC within the normal range, and 53 (39.8%) had an elevated TLC.
Chest radiographs and ultrasound revealed that right-sided pleural effusion was the most common, occurring in 66 (49.6%) children, followed by bilateral effusion in 37 (27.8%) and left-sided effusion in 30 (22.6%). Underlying lung parenchymal abnormalities were found in 69 (51.87%) children, including consolidation in 65 (48.8%) and necrotizing pneumonia in 4 (3%). Thick septation with loculation was observed in 14 (10.5%) children. CT thorax was performed in 17 (12.8%) children, providing detailed anatomical information that facilitated decortication planning in 14 (10.5%) cases and guided conservative management in 3 (2.3%) cases with necrotizing pneumonia. Additional findings on chest x-ray included mediastinal lymphadenopathy in 27 (20.3%) children, with 25 cases diagnosed as tuberculosis and 2 as haematological malignancy.
Diagnostic aspiration of PF was performed in 101(75.9%) cases, as 32 (24.06%) children had minimal effusion not amenable to tapping even with ultrasound guidance. Analysis according to Light's criteria classified 28.7% (n=29) of the effusions as transudative and 71.3% (n=72) as exudative. PF analysis showed low glucose (<60 mg/dl) in 43.5% (n=44/101) of samples, and elevated PF to serum protein ratio (>0.5), PF to serum LDH ratio (>0.6), and PF LDH (>2/3rd serum LDH) were observed in 54.4% (n=55/101), 66.3% (n=67/101), and 60.3% (n=61/101) of the samples, respectively.
Microbiological cultures of PF identified Staphylococcus aureus in 5 (3.8%) cases and Streptococcus pneumoniae in 3 (2.3%) cases. PF CBNAAT detected M.tb in only 2 of the 28 patients (7.1%) diagnosed with tuberculosis.

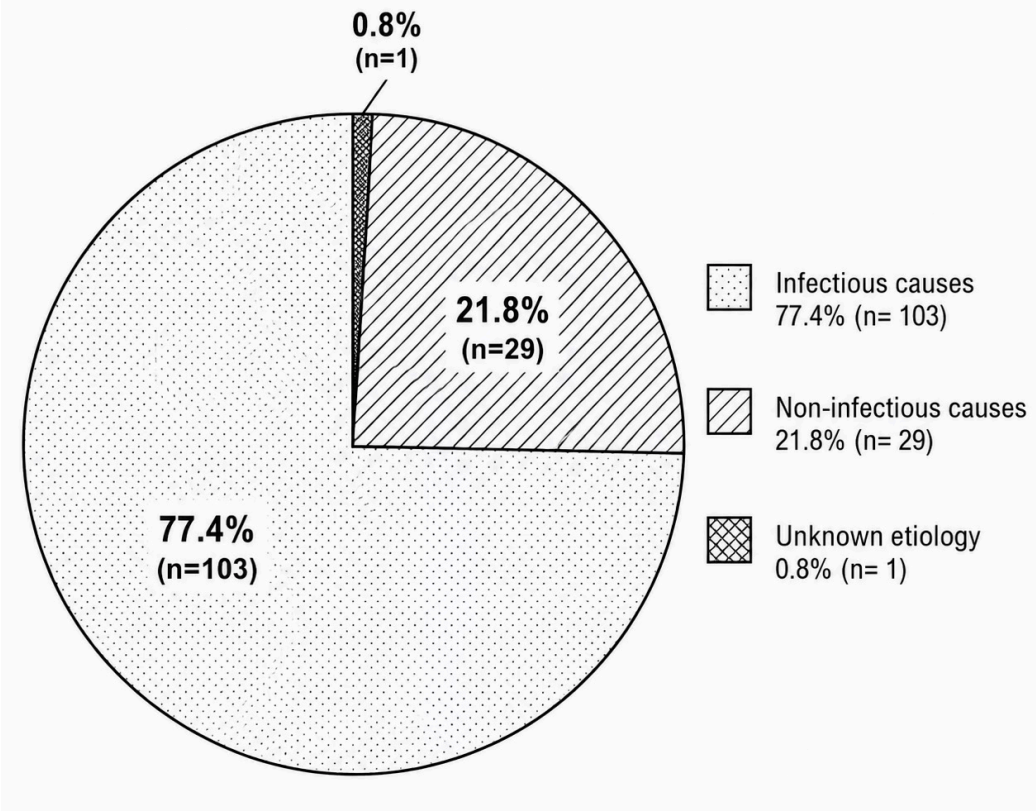
Final Diagnosis

Table 2 and Figure 1 summarize the distribution of the final etiological diagnoses based on detailed clinical evaluation.

Table 2: Final Diagnosis of Pleural Effusion Cases (N=133).

Etiological category	Diagnosis	n (%)
Infectious causes (n=103, 77.4%)		
	Tuberculosis	28 (21.1)
	Empyema (complicated parapneumonic effusion)	27 (20.3)
	Simple parapneumonic effusion	20 (15.0)
	Sepsisassociated pleural effusion	22 (16.5)
	Dengue fever	6 (4.5)
Noninfectious causes (n=29, 21.8%)		
	Nephrotic syndrome	12 (9.0)
	Acute hepatitis	11 (8.3)
	Malignancy	4 (3.0)
	Acute pancreatitis	2 (1.5)
Undetermined	Unknown etiology	1 (0.8)

Figure 1: Distribution of the etiological categories of pleural effusion in the study population (N = 133).



One child died within 4 hours of presentation in the emergency room, and a complete evaluation to determine the underlying cause could not be performed. Out of the 28 patients with tuberculosis, 16 (57.1%) had M.tb detected on CBNAAT in sputum or gastric aspirate. Among the two children with M.tb detected in PF, one had M.tb detected in both gastric aspirate and PF. Additionally, one child had M.tb detected in FNAC of cervical lymph node. The remaining 10 (35.7%) patients were diagnosed based on clinico-radiological findings.

Out of the 133 children included in the study, 83 (62.4%) were managed conservatively, while 27 (20.3%) children with empyema required intercostal tube (ICT) drainage, with fibrinolytics administered to 13 (48.1%) out of 27 children with empyema. Decortication was needed for rest of 14 (51.9%) children. The median duration of ICT drainage was 239 (168-300) hours, and the median length of hospital stay was 342 (236-508) hours. Nine (6.8%) children died due to septicaemia and multi-organ dysfunction syndrome.

Of the 124 children who survived, 9 (7.3%) were lost to follow-up. The remaining children were clinically well with no new complaints. Among those followed up, children with dengue fever, septicaemia, acute hepatitis, nephrotic syndrome and acute pancreatitis had normal chest X-rays and clear costophrenic angles on ultrasound at the 6-week follow-up. Of the 28 children with tuberculosis, 3 (10.7%) had mild blunting of the costophrenic angle on ultrasound, and 4 (14.3%) had persistent mediastinal widening on chest X-rays. Among the 11 children with empyema who underwent decortication and completed follow-up none showed residual effusion or recurrence at 6 weeks post-discharge. Of the 27 children managed with intercostal drainage, 2 died during their hospital stay, and 3 of the remaining 25 (12%) had minimal pleural fluid at 6 weeks but were asymptomatic. Additionally, 4 out of 20 children (20%) with simple parapneumonic effusion had some pulmonary infiltrates on chest X-ray but no evidence of effusion on either chest X-ray or ultrasound.

To evaluate the relationship between clinical and pleural fluid (PF) parameters and the etiology of pleural effusion, statistical analyses were conducted to compare important variables across the different etiological groups. One child, for whom an underlying diagnosis could not be established, was excluded from this analysis. We focused on parameters such as age, hemoglobin (Hb) levels, total leukocyte count (TLC), and specific PF characteristics, given their relevance in differentiating the causes of effusion. These analyses aimed to identify significant patterns that could better inform diagnostic and treatment strategies. The findings, summarized in Table 3, highlight key associations that reflect varying disease characteristics and their clinical impacts.

Table 3: Association of various clinical or laboratory parameters with the etiology of Pleural Effusion (N=132).

Variable	Tuberculosis (n=28)	Empyema (n=27)	Simple parapneumonic effusion (n=20)	Sepsis (n=22)	Dengue (n=6)	Nephrotic syndrome (n=12)	Acute hepatitis (n=11)	Malignancy (n=4)	Acute pancreatitis (n=2)	Statistical test	p-value*
Age (years), mean ± SD	11.64 ± 4.29	7.08 ± 4.58	6.6 ± 2.0	9.6 ± 2.8	3.6 ± 4.0	12.5 ± 1.5	5.8 ± 3.2	11.7 ± 2.0	10.2 ± 1.7	One-way ANOVA	0.011
Male sex, n (%)	16 (57.1%)	12 (44.4%)	14 (70.0%)	9 (40.9%)	2 (33.3%)	8 (66.7%)	6 (54.5%)	3 (75.0%)	1 (50.0%)	Chi-square†	0.115
Duration of fever (days), median (IQR)	6 (5-8)	9 (7-12)	2 (1-4)	9 (7-13)	4 (3-7)	5 (3-7)	6 (4-9)	3 (1-5)	5 (4-7)	Kruskal-Wallis	0.018
Duration of cough (days), median (IQR)	4 (3-7)	7 (6-9)	7 (6-11)	9 (8-11)	8 (7-9)	3 (2-5)	4 (2-5)	7 (5-11)	9 (8-13)	Kruskal-Wallis	0.324
Right-sided pleural effusion, n (%)	15 (53.5%)	16 (59.2%)	11 (55.0%)	12 (54.5%)	4 (66.6%)	4 (33.3%)	3 (27.3%)	1 (25.0%)	0 (0.0%)	Chi-square†	0.005
BMI, kg/m²	16.3 ± 1.3	17.1 ± 2.3	19.2 ± 3.4	17.1 ± 3.6	19.6 ± 3.2	20.6 ± 2.6	18.1 ± 1.7	17.0 ± 1.7	18.9 ± 2.1	Kruskal-Wallis	0.491
Hemoglobin, gm/dL	11.4 ± 1.0	10.7 ± 1.9	9.6 ± 1.4	10.3 ± 1.5	10.8 ± 1.8	12.1 ± 1.8	10.7 ± 1.3	8.5 ± 2.2	11.7 ± 2.0	Kruskal-Wallis	0.002
TLC, cells/cumm	10079 (8661-11053)	18249 (10721-19537)	16546 (8126-17629)	16949 (16316-17212)	15775 (15496-16159)	10924 (9614-13282)	10106 (9915-12005)	12492 (11937-12515)	12600 (10540-14856)	Kruskal-Wallis	<0.001
PF TLC, cells/cumm	2723 (2186-3881)	4248 (3268-4348)	3480 (1864-3804)	4556 (3428-4667)	1618 (1261-2330)	2967 (2926-3187)	1515 (1392-1751)	1901 (1656-2823)	–	Kruskal-Wallis	<0.001
PF Neutrophil Count, cells/cumm	931 (923-1838)	2464 (1731-2657)	2219 (1861-2464)	1978 (1599-2261)	888 (860-894)	913 (815-1936)	1110 (622-1808)	197 (150-1148)	–	Kruskal-Wallis	<0.001
PF Lymphocyte Count, cells/cumm	1008 (967-1058)	636 (351-787)	623 (536-671)	799 (591-926)	453 (270-671)	802 (152-928)	763 (736-1864)	1371 (1062-1880)	–	Kruskal-Wallis	<0.001
PF Protein, gm/dL	4.6 ± 1.0	4.7 ± 1.2	2.5 ± 1.0	2.4 ± 1.2	3.1 ± 0.9	1.2 ± 1.4	4.2 ± 0.9	3.1 ± 1.2	–	Kruskal-Wallis	<0.001
PF Glucose, mg/dL	80 ± 20	50 ± 16	68 ± 10	74 ± 15	85 ± 8	72 ± 20	70 ± 11	47 ± 17	–	Kruskal-Wallis	0.007
PF LDH, U/L, median (IQR)	787 (526-957)	1090 (834-3335)	423 (112-853)	186 (123-288)	484 (303-664)	100 (98-102)	111 (102-123)	635 (377-892)	–	Kruskal-Wallis	<0.001
Duration of hospital stay (days), median (IQR)	12 (10-20)	18 (14-28)	06 (03-09)	15 (14-21)	6 (5-21)	08 (6-17)	5 (4-8)	27 (23-30)	12 (10-17)	Kruskal-Wallis	<0.001

Significant associations were observed between age, hemoglobin (Hb) levels, pleural fluid (PF) analysis, and the etiology of pleural effusion. Tuberculosis (TB) was more common in adolescents, while empyema affected younger children, and malignancies were prevalent in older children. The mean ages of presentation for empyema and tuberculosis were 7.08±4.58 years and 11.64±4.29 years respectively. While we observed a trend of lower BMI in conditions like empyema, TB, and sepsis, and higher BMI in nephrotic syndrome, dengue, and parapneumonic effusions, this difference did not reach statistical significance.

PF total leukocyte count (TLC), neutrophil, and lymphocyte profiles were key in distinguishing etiologies. TLC and neutrophil counts were highest in empyema and simple parapneumonic effusions, indicative of infections, while lymphocyte counts were elevated in malignancies and TB. PF protein was highest in TB and empyema, while glucose was lowest in malignancies and empyema. PF LDH levels also varied significantly across the different etiological groups, with higher values observed in infective pleural effusions.

The duration of hospital stay varied significantly with etiology, with malignancies and empyema leading to longer stays, while conditions like dengue and acute hepatitis resulted in shorter hospitalizations. These findings highlight the influence of underlying etiology and nutritional status on disease severity and clinical outcomes.

Discussion

Pleural effusion in children is a complex condition with a wide spectrum of causes, varying based on age, geography, and clinical presentation. Our study, which included 133 children, aimed to describe the clinical profile and outcomes of paediatric pleural effusion in a tertiary care setting.

In our study, tuberculosis (21.1%) and empyema (20.3%) were the most common etiologies of pleural effusion. This finding aligns with Eastham KM et al. and Efrati O et al, who reported that infectious causes dominate pediatric pleural effusion cases, particularly in low- and middle-income countries [1,15]. However, our findings differ slightly from those of Das I et al., who reported empyema as the most frequent cause in their cohort from West Bengal [3]. Similarly, in the study by Saliya MP et al. in Gujarat, parapneumonic effusion was the most common etiology, followed by TB and empyema [16]. The predominance of tuberculosis and empyema in our cohort reflects the unique epidemiological profile of pleural effusion in low- and middle-income countries. Unlike studies from high-income settings, tuberculosis continues to represent a major cause of pediatric pleural effusion in India. This finding highlights the importance of maintaining a high index of suspicion for tuberculosis, particularly among older children and adolescents presenting with pleural effusion.

Our study identified a male predominance in pleural effusion cases, with males constituting 54.1% and a male-to-female ratio of 1.18:1, similar to the observations by Eastham KM et al. and Saliya MP et al., who also noted a higher prevalence in males [15,16]. This gender predisposition may indicate a biological susceptibility in boys or differences in health-seeking behaviour, though further research is needed to explore this factor.

The age distribution in our study showed the highest incidence in children aged 6–12 years (36.8%), with a mean age of 8.6 years. These results are in line with Saliya MP et al. , who also reported a predominance of pleural effusion in older children [16]. However, Das I et al. found that the majority of cases occurred in children aged 1–5 years [3,15]. This variation may be explained by differences in study populations and local healthcare access, particularly for younger children.

The clinical presentation in our cohort was dominated by fever (85.7%), cough (67.6%), and shortness of breath (53.3%). Lingayat AM et al. reported similar symptoms in their study [8]. Das I et al and Satpathy et al. observed a highest prevalence of shortness of breath and fast breathing as a presenting complaint [3,17]. Differences in symptom frequency may be attributed to the underlying etiology and the timing of presentation to healthcare facilities. In our study, shortness of breath was more prevalent in children with empyema, which often presents with rapid respiratory compromise.

Tachypnea, dullness on percussion, and diminished breath sounds were the most prevalent clinical signs in our study. Efrati O et al. emphasized the challenge of identifying pleural effusion-specific signs in younger children due to their smaller chest surfaces [1]. Additionally, Merino JM et al. noted the overlapping physical findings in tuberculous and bacterial effusions [18]. Our findings underscore the importance of using adjunct imaging studies, such as ultrasound, to assist in the early diagnosis and management of pleural effusion, especially in younger children where physical examination alone may be inadequate.

Although not statistically significant (p=0.491), patients with empyema, TB, and sepsis tended to have lower BMI compared to those with nephrotic syndrome, dengue, and parapneumonic effusions. Nutritional status thus plays a critical role in the pathophysiology of pleural effusion, and addressing malnutrition may prevent complications in at-risk populations.

The yield of pleural fluid cultures in our study was low [n=8/101 (7.9%)], including cases of empyema, septicemia, and parapneumonic effusion. This result is comparable to Saliya MP et al.'s finding of 11.67%, though significantly lower than the 40% isolation reported by Narayanaapa et al. [16,19] The low culture yield may be attributed to prior antibiotic use, delayed presentation to tertiary care, and the persistence of inflammation even after pathogen clearance. Lingayat AM et al. similarly noted that prior antibiotic administration reduced culture positivity rates [8].The widespread administration of empirical antibiotics before referral, observed in all children in our study, is likely to have reduced the microbiological yield of pleural fluid cultures. This finding emphasizes the need for judicious antibiotic stewardship and early referral to tertiary

care centres for appropriate diagnostic evaluation.
Notably, all children in our study, irrespective of their underlying etiology, had received antibiotics prior to their hospital admission, reflecting a concerning trend of antibiotic misuse in India. Staphylococcus aureus and Streptococcus pneumoniae were the most commonly identified pathogens in our study, consistent with findings by Lingayat AM et al. and Monnier LA et al., underscoring the importance of empiric antibiotic therapy to cover these organisms when managing pleural effusion [4,8]. However, both blood and pleural fluid cultures for Mycobacterium tuberculosis were negative in all patients, a finding similar to those reported by Saliya MP et al. [16]
In our study, pleural fluid analysis highlighted total leukocyte count (TLC), neutrophil count, and lymphocyte count as key indicators for distinguishing the etiology of pleural effusion. We found that TLC and neutrophil counts were highest in cases of empyema and parapneumonic effusions, suggesting a bacterial etiology. In contrast, elevated lymphocyte counts were more commonly associated with malignancies and tuberculosis (TB). Additionally, pleural fluid protein levels were elevated in both TB and empyema, while glucose levels were lowest in cases of malignancy and empyema. These findings are consistent with those of Saliya MP et al., who also demonstrated a significant relationship between pleural fluid glucose levels and the underlying cause. Our study also demonstrated a significant association between pleural fluid LDH levels and the underlying etiology of pleural effusion. Higher LDH levels were observed predominantly in infective pleural effusions, reflecting greater pleural inflammation and cellular injury.[16]. Similar findings were observed by Das I et al., who noted correlations between neutrophil and lymphocyte counts with the underlying cause, but their study did not reveal a strong association between protein, LDH, or glucose levels with specific etiologies [3]. These findings suggest that pleural fluid biochemical and cellular parameters remain valuable adjuncts in differentiating the underlying etiology of pediatric pleural effusion, particularly in resource-limited settings where advanced molecular diagnostics may not be readily available.
The overall case fatality rate in our study was 6.8%, which is higher compared to the 1.5% reported by Lingayat AM et al. (n=1/67) and the 3.8% reported by Satpathy et al. (n=2/53) in 2015 [8,17]. However, their studies focused exclusively on children with empyema, whereas our study included all cases of pleural effusion. The majority of deaths were observed in children with septicemia and multi-system derangements, highlighting the increased severity of pleural effusion when associated with systemic infections or malignancies.
Our study's strength lies in its comprehensive prospective analysis of pediatric pleural effusion using clinical, radiological, and microbiological data, offering new insights into the etiology of the condition in Northern India. However, limitations include the single-centre design, which may not represent the broader population, and low pleural fluid culture yields due to prior antibiotic use, highlighting the need for advanced diagnostic tools. Additional limitations include the relatively small number of patients in certain etiological categories, which may have limited the statistical power for subgroup analyses. Furthermore, advanced pleural fluid biomarkers such as adenosine deaminase (ADA), interferon-γ, and molecular pathogen detection assays were not uniformly available, which may have influenced the diagnostic yield in selected cases.

Conclusion

Our study underscores the diverse etiologies of pediatric pleural effusion, with tuberculosis and empyema being the most common causes in this region. Significant associations of age and pleural fluid laboratory parameters with the underlying etiology provide valuable diagnostic insights. Despite challenges such as late referrals and low culture yields, early and appropriate management, including ICT drainage and decortication, when necessary, generally results in favourable outcomes. Notably, our study highlights the presence of various non-infectious causes of pleural effusion and reflects a concerning trend of antibiotic misuse in India, as all children had received antibiotics before hospital admission. Further research with larger cohorts and extended follow-up is needed to better understand the impact of nutritional and other risk factors on the development and outcomes of pleural effusion in children.

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Nil.

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Dr. Ashna Kumar: Contributed to the conception of the study, data acquisition, and drafting the manuscript.
Dr. Sugandhi Malgotra: Provided critical radiological expertise for data interpretation and contributed to revising the manuscript for intellectual content.
Dr. Preeti Sharma and Dr. Keshav Mehra: Played vital roles in data acquisition, patient management, and revising the manuscript for important intellectual content.
Dr. Mohd. Razaq: Offered senior guidance in study design,

data analysis, and provided critical revisions to ensure the manuscript's quality.
Dr. Nikhil Rajvanshi: As the corresponding author and a specialist in pediatric pulmonology, was involved in interpretation of data, critical revisions, and final approval of the manuscript.
Each contributor has substantially contributed to the conception and design, drafting or revising the manuscript, and final approval of the version to be published.
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