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# Site-Specific Thoracoscopic Findings, Diagnostic Yield, and Predictive Morphology for Malignant versus Tubercular Pleural Disease

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## ABSTRACT

**Background**: Undiagnosed exudative pleural effusion frequently requires tissue diagnosis, particularly where tuberculosis and malignancy coexist. Medical thoracoscopy permits direct visualisation and targeted biopsy, but the diagnostic relevance of site-specific pleural morphology remains incompletely defined.
**Aims**: To determine the diagnostic yield of medical thoracoscopy and evaluate whether site-specific thoracoscopic morphology can differentiate malignant from tubercular pleural disease.
**Materials and Methods**: This prospective observational study enrolled 30 adults with exudative pleural effusion that remained unexplained after clinical, radiological, biochemical, microbiological and cytological assessment. Thoracoscopy was performed under local anaesthesia with conscious sedation. Apical, mid-costal, lower-costal and diaphragmatic findings were recorded, targeted biopsies were obtained, and final diagnoses were established by histopathology with ancillary testing. Associations were examined using Fisher exact or chi-square tests, Mann-Whitney U tests, Spearman correlation and exploratory penalised regression.
**Results**: Thoracoscopy established a conclusive diagnosis in 29 of 30 patients (96.7%). Tubercular pleuritis accounted for 12 cases (40.0%), malignant disease for 14 (46.7%) and other benign disease for four (13.3%). Inflammation or hyperaemia (73.3%), pleural thickening (66.7%), nodules (56.7%) and adhesions (56.7%) were common. Apical disease most often appeared as inflammation or nodules (30.0% each), lower-costal disease as adhesions (33.3%) or thickening (30.0%), and diaphragmatic disease as inflammation (50.0%). Parietal pleural nodules were present in all malignant cases but only 3 of 12 tubercular cases and no other benign cases ($p < 0.001$); sensitivity, specificity, positive predictive value and negative predictive value for malignancy were 100%, 81.2%, 82.4% and 100%, respectively. Mean pleural-fluid adenosine deaminase was higher in tuberculosis than malignancy (66.4 versus 25.3 U/L; $p < 0.001$). Most patients (60.0%) had no complication; recorded adverse events were minor and self-limited.
**Conclusion**: Medical thoracoscopy provided very high diagnostic yield with acceptable safety. Pleural nodularity strongly favoured malignancy, whereas diffuse inflammation or hyperaemia and high pleural-fluid adenosine deaminase favoured tubercular or inflammatory disease. These visual patterns can guide targeted biopsy but should complement, not replace, histopathological confirmation.

***Keywords***: *Exudative pleural effusion; medical thoracoscopy; pleural malignancy; pleural tuberculosis; pleural nodules; diagnostic yield.*

## INTRODUCTION

Pleural effusion is a common manifestation of local pleural disease and systemic illness. Once an effusion is classified as exudative by Light's criteria, the principal diagnostic task is to distinguish infection, tuberculosis, malignancy and less common inflammatory causes.[1,2] Thoracentesis frequently provides only an incomplete answer: cytology may miss focal or predominantly pleural malignancy, whereas smear, culture and nucleic-acid testing have limited sensitivity in

paucibacillary tuberculous pleuritis. Contemporary pleural guidance therefore recommends tissue sampling when a unilateral exudative effusion remains unexplained after appropriate fluid analysis and imaging.[3,4]

Closed pleural biopsy is inexpensive and remains useful in selected settings, but blind sampling can miss patchy metastatic deposits, mesothelioma and focal granulomatous disease.[5] Medical thoracoscopy overcomes this limitation by permitting direct inspection of the pleural cavity and biopsy of visually abnormal areas under local anaesthesia and conscious sedation. Across systematic reviews and recent cohorts, diagnostic yields generally range from approximately 80% to more than 95%, with major complications uncommon when the procedure is performed by trained teams.[6-10]

In tuberculosis-endemic regions, the clinical overlap between tubercular and malignant pleural disease creates an additional challenge. Fever, weight loss, lymphocyte-predominant exudate and pleural thickening are not specific for tuberculosis; conversely, malignant effusions may have apparently non-malignant biochemistry and negative cytology. Tissue confirmation prevents empirical treatment from delaying cancer diagnosis and establishes the malignant subtype required for oncological management.[11-13]

Thoracoscopy also provides immediate morphological information. Pleural nodules, irregular thickening, masses, plaques, adhesions, hyperaemia and sago-grain-like lesions have been described, although their predictive value varies across cohorts and operators.[14-16] Most reports describe morphology globally rather than by pleural site. Systematic recording of the apical, mid-costal, lower-costal and diaphragmatic surfaces may improve lesion targeting and provide reproducible descriptors for clinicopathological correlation.

In our study, we evaluated the diagnostic yield and safety of medical thoracoscopy in undiagnosed exudative pleural effusion and examined whether site-specific thoracoscopic morphology, interpreted alongside imaging and pleural-fluid adenosine deaminase (ADA), could distinguish malignant from tubercular pleural disease.

## MATERIALS AND METHODS

A prospective observational study was conducted in the Department of Respiratory Medicine, Kamla Nehru Chest Hospital, Dr. S. N. Medical College, Jodhpur, Rajasthan. Adults older than 18 years with exudative pleural effusion of undetermined aetiology after initial diagnostic assessment were enrolled after Institutional Ethics Committee approval and written informed consent. Patients who were medically unsuitable for thoracoscopy or unable or unwilling to provide consent were excluded. The initial work-up comprised clinical examination, chest radiography, thoracic ultrasonography and/or contrast-enhanced computed tomography (CECT), pleural-fluid biochemistry, differential cell count, cytology, bacterial studies, acid-fast bacillus testing and cartridge-based nucleic acid amplification testing (CBNAAT), as clinically indicated.

Pleural fluid was classified as exudative when at least one Light criterion was fulfilled: a pleural-fluid/serum protein ratio greater than 0.5, a pleural-fluid/serum lactate dehydrogenase (LDH) ratio greater than 0.6, or pleural-fluid LDH greater than two-thirds of the upper reference limit for serum LDH.[2] The planned sample size was based on an expected thoracoscopic sensitivity of approximately 90% and an estimated tuberculosis prevalence of 40% among patients undergoing medical thoracoscopy in a high-burden setting.[11] At 95% confidence and an absolute precision of 20%, the minimum calculated sample was 26; this was increased to 30 participants.

After pre-procedure evaluation of haemogram, coagulation profile and relevant viral markers, patients were positioned in the lateral decubitus position with the affected side uppermost. Thoracic ultrasonography and imaging were used to select a safe entry site. Local anaesthesia with lignocaine and conscious sedation were administered. A small incision was made immediately above the rib, a trocar was introduced and pleural fluid was evacuated. The pleural cavity was inspected systematically, with separate documentation of the apical costal, mid-costal, lower-costal and diaphragmatic pleura. Abnormalities were recorded as nodules, plaques, mass lesion, thickening, adhesions, inflammation or hyperaemia, or abnormal vessels. Multiple targeted biopsies were obtained from representative abnormal areas and sent for histopathology, microbiology and immunohistochemistry when indicated. A chest drain was inserted after the procedure, and patients were monitored for immediate complications.

The primary outcome was diagnostic yield, defined as the proportion of patients in whom thoracoscopy and pleural biopsy established a conclusive final diagnosis. Secondary outcomes included the histopathological spectrum, site-specific morphology, relationship of morphology and imaging with final diagnosis, diagnostic performance of candidate malignant markers and procedure-related complications. Continuous variables were summarised as mean with standard deviation or median with range, and categorical variables as number and percentage. Between-group ADA values were compared using the Mann-Whitney U test. Categorical associations were assessed with the chi-square or Fisher exact test, and Spearman rank correlation was used for binary or ordinal associations. Sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV) and accuracy were calculated from 2×2 tables. Because several predictors showed complete or near-complete separation in a small cohort, adjusted analyses were exploratory and used penalised logistic regression with a restricted number of clinically relevant variables. A two-sided p value below 0.05 was considered statistically significant.

## RESULTS

Thirty consecutive patients underwent medical thoracoscopy. The largest age group was 55-64 years (36.7%), followed by 45-54 years (26.7%); 18 patients (60.0%) were men. Dyspnoea (96.7%), cough (83.3%), chest pain (60.0%) and fever (56.7%) were the dominant symptoms, and 46.7% had a smoking history. Right-sided effusion was present in 66.7%. Moderate effusion was the commonest chest-radiograph pattern (50.0%), while CECT demonstrated pleural thickening in 53.3%, pleural nodules in 33.3%, mass lesions in 30.0% and mediastinal lymph nodes in 30.0% (Table 1).

**Table 1. Baseline clinical and radiological profile (N = 30)**

| Characteristic | Finding |
|---|---|
| Age group, n (%) | ≤34: 4 (13.3); 35–44: 5 (16.7); 45–54: 8 (26.7); 55–64: 11 (36.7); ≥65: 2 (6.7) |
| Male sex | 18 (60.0%) |
| Smoking history | 14 (46.7%) |
| Presenting symptoms | Dyspnoea 29 (96.7%); cough 25 (83.3%); chest pain 18 (60.0%); fever 17 (56.7%); haemoptysis 5 (16.7%) |
| Effusion side | Right 20 (66.7%); left 10 (33.3%) |
| Chest radiograph | Moderate effusion 15 (50.0%); massive effusion 7 (23.3%); collapse/consolidation 5 (16.7%); pleural thickening 3 (10.0%) |
| Ultrasonography | Pleural thickening 14 (46.7%); nodules 11 (36.7%); echoic fluid 7 (23.3%); septations 5 (16.7%) |
| CECT chest | Pleural thickening 16 (53.3%); nodules 10 (33.3%); mass lesion 9 (30.0%); lymph nodes 9 (30.0%); volume loss 5 (16.7%) |

*Values are n (%) unless otherwise stated. CECT: contrast-enhanced computed tomography.*

Pleural fluid was lymphocyte predominant in 26 patients (86.7%). Cytology was positive or suspicious for malignant cells in eight (26.7%), CBNAAT was positive in six (20.0%) and culture in two (6.7%). Mean pleural-fluid ADA was 42.5 ± 21.2 U/L, LDH 682.7 ± 208.3 U/L, protein 4.5 ± 0.7 g/dL and glucose 63.8 ± 16.4 mg/dL. The most frequent overall thoracoscopic findings were inflammation or hyperaemia (73.3%), thickening (66.7%), nodules (56.7%) and adhesions (56.7%) (Table 2).

**Table 2. Pleural-fluid profile and overall thoracoscopic findings**

| Domain | Finding |
|---|---|
| Pleural-fluid appearance | Straw coloured 11 (36.7%); serosanguineous 8 (26.7%); haemorrhagic 5 (16.7%); serous 3 (10.0%); turbid 2 (6.7%); purulent 1 (3.3%) |
| Differential cell count | Lymphocyte predominant 26 (86.7%); neutrophil predominant 2 (6.7%); mixed 2 (6.7%) |
| Cytology positive/suspicious | 8 (26.7%) |
| Pleural-fluid CBNAAT positive | 6 (20.0%) |
| Pleural-fluid culture positive | 2 (6.7%) |
| Biochemistry, mean ± SD | ADA 42.5 ± 21.2 U/L; LDH 682.7 ± 208.3 U/L; protein 4.5 ± 0.7 g/dL; glucose 63.8 ± 16.4 mg/dL |
| Overall thoracoscopic morphology | Inflammation/hyperaemia 22 (73.3%); thickening 20 (66.7%); nodules 17 (56.7%); adhesions 17 (56.7%); plaques 3 (10.0%); mass lesion 3 (10.0%) |
| Intra-pleural fluid appearance | Clear/straw coloured 15 (50.0%); haemorrhagic 13 (43.3%); purulent 2 (6.7%) |

*ADA: adenosine deaminase; CBNAAT: cartridge-based nucleic acid amplification test; LDH: lactate dehydrogenase.*

Thoracoscopic morphology varied by pleural site. At the apical costal pleura, inflammation and nodules were equally common (30.0% each). The mid-costal surface showed a mixed pattern, with inflammation (23.3%) and nodules (20.0%) predominating. Lower-costal disease was more often organised, with adhesions (33.3%) and thickening (30.0%). The diaphragmatic pleura most frequently showed inflammation (50.0%), followed by nodules (30.0%) and plaques (20.0%) (Table 3 and Figure 1).

**Table 3. Site-specific thoracoscopic morphology**

| Pleural site | Thoracoscopic findings, n (%) |
|---|---|
| Apical costal | Inflamed pleura 9 (30.0); nodules 9 (30.0); nodules with hyperaemia 5 (16.7); thickening 4 (13.3); hyperaemic vessels 3 (10.0) |
| Mid-costal | Inflamed pleura 7 (23.3); nodules 6 (20.0); thickening 5 (16.7); adhesions 4 (13.3); mass lesion 4 (13.3); nodules with hyperaemia 4 (13.3) |
| Lower-costal | Adhesions 10 (33.3); thickening 9 (30.0); nodules 7 (23.3); inflamed pleura 4 (13.3) |
| Diaphragmatic | Inflammation 15 (50.0); nodules 9 (30.0); plaques 6 (20.0) |

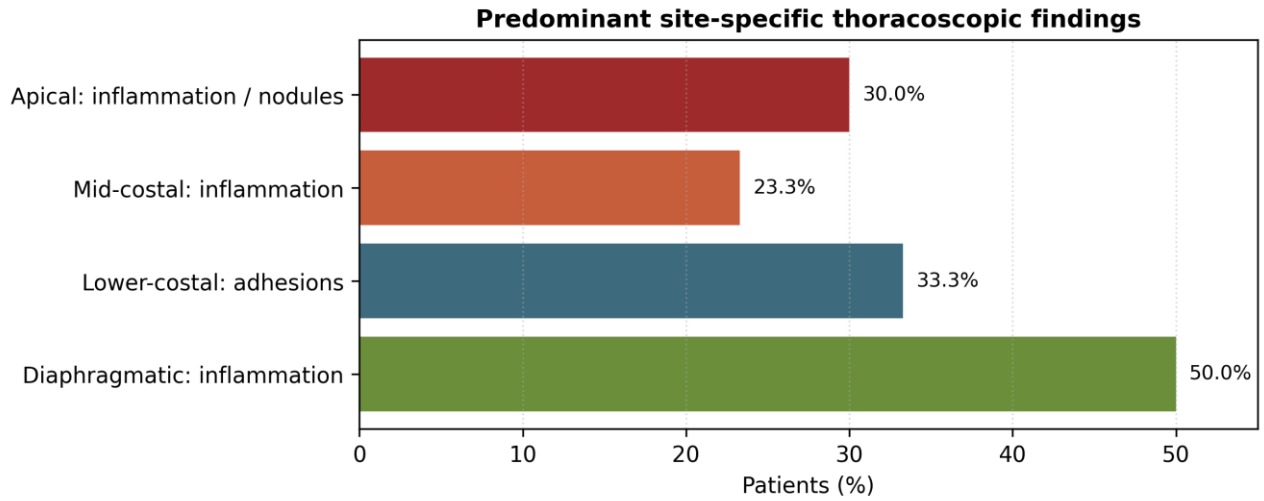


**Figure 1. Predominant site-specific thoracoscopic findings.**

Pleural biopsy was obtained in every patient. Histopathology showed necrotising granulomatous pleuritis in 12 (40.0%), metastatic adenocarcinoma in nine (30.0%), malignant mesothelioma in two (6.7%), squamous cell carcinoma in two (6.7%), acute suppurative pleuritis in two (6.7%), and one case each of lymphoproliferative malignancy, chronic fibrinous pleuritis and chronic non-specific pleuritis. Final clinicopathological diagnoses comprised tubercular pleuritis in 12 (40.0%), malignant pleural disease in 14 (46.7%) and benign or other disease in four (13.3%). Thoracoscopy was conclusive in 29 patients, giving a diagnostic yield of 96.7%. Eighteen patients (60.0%) had no complication; mild chest pain, subcutaneous emphysema, transient fever and minor bleeding occurred in three patients each (10.0%), without major haemorrhage, respiratory deterioration or procedure-related death (Table 4).

**Table 4. Histopathology, final diagnosis, diagnostic yield and safety**

| Outcome | n (%) |
|---|---|
| Histopathology | Necrotising granulomatous pleuritis 12 (40.0); metastatic adenocarcinoma 9 (30.0); mesothelioma 2 (6.7); squamous cell carcinoma 2 (6.7); acute suppurative pleuritis 2 (6.7); lymphoproliferative malignancy 1 (3.3); chronic fibrinous pleuritis 1 (3.3); chronic non-specific pleuritis 1 (3.3) |
| Final diagnosis | Tubercular pleuritis 12 (40.0); lung adenocarcinoma 7 (23.3); breast adenocarcinoma 2 (6.7); mesothelioma 2 (6.7); squamous-cell lung carcinoma 2 (6.7); parapneumonic effusion 2 (6.7); lymphoma 1 (3.3); rheumatoid pleuritis 1 (3.3); non-specific chronic pleuritis 1 (3.3) |
| Diagnostic yield | Conclusive 29 (96.7); non-conclusive 1 (3.3) |
| Diagnostic category | Malignant 14 (46.7); non-malignant 16 (53.3) |
| Complications | None 18 (60.0); mild chest pain 3 (10.0); subcutaneous emphysema 3 (10.0); transient fever 3 (10.0); minor bleeding 3 (10.0) |

Morphological and imaging patterns differed across diagnostic groups. Thoracoscopic parietal nodules were seen in every malignant case, compared with 25.0% of tubercular cases and none of the other benign cases ($p < 0.001$). Inflammation or hyperaemia was universal in tubercular and other benign disease but occurred in 42.9% of malignant cases ($p < 0.001$). USG and CECT pleural nodules were confined to the malignant group. Mean ADA was 66.4 ± 9.4 U/L in tuberculosis, 25.3 ± 6.8 U/L in malignancy and 31.1 ± 2.9 U/L in other benign disease; the tuberculosis-malignancy difference was highly significant ($p < 0.001$) (Table 5).

**Table 5. Morphological, imaging and ADA findings according to final diagnosis**

| Finding | Tuberculosis (n=12) | Malignant (n=14) | Other benign (n=4) | p value |
|---|---|---|---|---|
| Thoracoscopic parietal nodules | 3/12 (25%) | 14/14 (100%) | 0/4 (0%) | <0.001 |
| Inflammation / hyperaemia | 12/12 (100%) | 6/14 (43%) | 4/4 (100%) | <0.001 |
| USG pleural nodules | 0/12 (0%) | 11/14 (79%) | 0/4 (0%) | <0.001 |
| CECT pleural nodules | 0/12 (0%) | 10/14 (71%) | 0/4 (0%) | <0.001 |
| CECT pleural thickening | 6/12 (50%) | 10/14 (71%) | 0/4 (0%) | 0.067 |
| Thoracoscopic mass lesion | 0/12 (0%) | 3/14 (21%) | 0/4 (0%) | 0.053 |
| USG septations | 4/12 (33%) | 0/14 (0%) | 1/4 (25%) | 0.021 |
| Thoracoscopic pleural thickening | 8/12 (67%) | 8/14 (57%) | 4/4 (100%) | 0.317 |
| Pleural adhesions | 7/12 (58%) | 7/14 (50%) | 3/4 (75%) | 0.508 |
| ADA, mean ± SD (U/L) | 66.4 ± 9.4 | 25.3 ± 6.8 | 31.1 ± 2.9 | <0.001* |

**Mann-Whitney U test for tuberculosis versus malignant disease. Other p values are from categorical association tests. USG: ultrasonography.*

Parietal pleural nodules had a sensitivity of 100%, specificity of 81.2%, PPV of 82.4%, NPV of 100% and overall accuracy of 90.0% for malignancy. Radiological nodules were less sensitive but completely specific in this cohort. ADA below 40 U/L also showed 100% sensitivity for malignancy, although specificity was 75.0%. The absence of inflammation or hyperaemia was completely specific but only moderately sensitive for malignancy (Table 6). In exploratory penalised regression, thoracoscopic parietal nodules (adjusted odds ratio 136.05; 95% CI 3.12–5938.30; p = 0.011) and ADA below 40 U/L (adjusted odds ratio 105.65; 95% CI 1.95–5724.94; p = 0.022) remained independently associated with malignant diagnosis. ADA above 40 U/L was the strongest adjusted predictor of tubercular pleuritis (adjusted odds ratio 270.83; 95% CI 6.42–11,433.45; p = 0.003). These estimates were considered exploratory because of the small cohort and wide confidence intervals.

**Table 6. Diagnostic performance of selected markers for malignant pleural disease**

| Marker | Sensitivity % | Specificity % | PPV % | NPV % | Accuracy % | p value |
|---|---|---|---|---|---|---|
| Thoracoscopic parietal nodules | 100.0 | 81.2 | 82.4 | 100.0 | 90.0 | <0.001 |
| USG pleural nodules | 78.6 | 100.0 | 100.0 | 84.2 | 90.0 | <0.001 |
| CECT pleural nodules | 71.4 | 100.0 | 100.0 | 80.0 | 86.7 | <0.001 |
| ADA <40 U/L | 100.0 | 75.0 | 77.8 | 100.0 | 86.7 | <0.001 |
| Absence of inflammation / hyperaemia | 57.1 | 100.0 | 100.0 | 72.7 | 80.0 | <0.001 |

*Performance estimates are exploratory and specific to this 30-patient cohort; histopathology remained the reference standard.*

## DISCUSSION

In our study, medical thoracoscopy established a conclusive diagnosis in 29 of 30 patients, producing a diagnostic yield of 96.7%. This result lies at the upper end of published experience and is consistent with the high yields reported when thoracoscopy is performed after disciplined patient selection and targeted biopsy. Augustine et al. reported a 100% yield after using a structured diagnostic algorithm,[17] while Rawat et al. reported 98.1% in a substantially larger rigid-thoracoscopy series.[9] The pooled and contemporary literature similarly supports thoracoscopy as one of the most reliable investigations for unresolved exudative pleural effusion.[6-8]

The age and sex distribution reflected the expected referral pattern of pleural disease at a tertiary respiratory centre. Nearly two-thirds of participants were aged 45-64 years and 60.0% were men. Rai et al. also described an older adult cohort with a mean age of approximately 58 years and a predominance of malignant pleural disease.[18] Dyspnoea and cough were the most frequent symptoms in our study, but clinical symptoms alone offered limited discrimination. Fever favoured tubercular or inflammatory disease, whereas haemoptysis showed a trend towards malignant morphology; cough, dyspnoea and chest pain were non-specific. Thomas et al. similarly demonstrated that the clinical profile of patients selected for thoracoscopy varies with local epidemiology, particularly the relative burdens of tuberculosis and malignancy.[19]

Pleural-fluid examination confirmed exudation but did not reliably establish the aetiology. Cytology was positive or suspicious in only 26.7%, pleural-fluid CBNAAT in 20.0% and culture in 6.7%. Lymphocyte predominance was common in both malignant and tubercular disease. These findings reinforce the limitations of fluid-only assessment in selected unresolved effusions. Kumar et al. showed that thoracoscopy remains valuable even when ADA is low or otherwise discordant with clinical suspicion,[12] and Mehfooz et al. demonstrated that pleural biopsy substantially improves microbiological confirmation of pleural tuberculosis.[13]

Tuberculosis and malignancy were the dominant competing diagnoses, accounting for 40.0% and 46.7% of the cohort, respectively. This near-balanced distribution closely resembles the high-burden experience of Kho et al., in which malignant pleural effusion and tuberculosis constituted 49.4% and 46.4% of cases.[11] Such overlap is clinically important because empirical antitubercular treatment can delay the recognition of malignancy, whereas a tissue diagnosis simultaneously identifies granulomatous pleuritis and provides the histological subtype of malignant disease.

The central morphological finding was the strong association between parietal pleural nodularity and malignancy. Nodules were visualised in every malignant case but in only three tubercular cases and no other benign case. The resulting sensitivity of 100%, specificity of 81.2% and NPV of 100% indicate that the absence of nodules made malignancy less likely in this cohort, while their presence strongly directed biopsy towards malignant tissue. Kuwal et al. likewise found that nodules, mass lesions and haemorrhagic pleural fluid increased the probability of malignant diagnosis.[14] Prospective multicentre work by Grosu et al. and practice surveys by Hallifax et al. have also shown that pleuroscopic appearance can inform immediate suspicion, although visual prediction alone is imperfect and remains operator dependent.[15,16]

Site-specific documentation added anatomical detail. Apical and mid-costal surfaces showed mixed inflammatory and nodular patterns, lower-costal pleura more often showed adhesions and thickening, and diaphragmatic inflammation was particularly common. The predominance of adhesions and organised thickening in dependent pleura may reflect chronicity and prior pleural organisation. Chen et al. reported a high prevalence of adhesions and noted their association with benign pleural disease.[20] Importantly, adhesions did not prevent a high diagnostic yield in our cohort, suggesting that careful inspection and targeted sampling can remain productive in partially organised pleural spaces.

ADA provided strong complementary discrimination. Mean ADA was 66.4 U/L in tubercular pleuritis compared with 25.3 U/L in malignant disease, and all tubercular cases exceeded 40 U/L. Conversely, ADA below 40 U/L showed 100% sensitivity and 75.0% specificity for malignancy in this dataset. The very large adjusted odds ratios for nodules and ADA reflect complete or near-complete separation in a small sample and should not be interpreted as stable effect sizes. The clinically relevant message is that morphology and ADA were concordant in most cases: nodularity with low ADA favoured malignancy, whereas diffuse inflammation with high ADA favoured tuberculosis. Neither feature replaced histopathology.

Safety was acceptable. Sixty per cent of patients had no adverse event, and recorded complications were limited to mild pain, transient fever, subcutaneous emphysema and minor bleeding. No major haemorrhage, respiratory deterioration or procedure-related death occurred. This profile is consistent with the favourable safety outcomes reported by Liu et al. and Behera et al.[8,10] Indian thoracoscopy series have likewise shown that direct visualisation with pleural biopsy can be delivered with high yield and manageable morbidity in appropriately selected patients.[21]

The strengths of our study were prospective data collection, systematic inspection of four pleural regions, complete biopsy acquisition and direct clinicoradiological-pathological correlation. Limitations include the single-centre design, small sample size, absence of blinded independent morphology review and wide confidence intervals in exploratory regression. The diagnostic-performance estimates may therefore be optimistic and require external validation. Nevertheless, the results support early thoracoscopy when initial evaluation fails, particularly in tuberculosis-endemic settings where malignant and tubercular pleural disease coexist.[5]

## CONCLUSION

Medical thoracoscopy achieved a diagnostic yield of 96.7% in adults with undiagnosed exudative pleural effusion and was associated only with minor, self-limited complications. Tuberculosis and malignancy were the dominant competing aetiologies. Site-specific inspection added clinically useful information: parietal pleural nodules strongly predicted malignant disease, while diffuse inflammation or hyperaemia and elevated pleural-fluid ADA favoured tubercular or benign inflammatory disease. These visual and biochemical patterns can guide lesion targeting and immediate clinical suspicion, but histopathological confirmation remains essential. Larger multicentre studies with blinded morphology assessment are required before the exploratory predictive estimates can be applied as a formal diagnostic model.

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