Monaldi Archives for Chest Disease

eISSN 2532-5264 https://www.monaldi-archives.org/

Monaldi Arch Chest Dis 2026 [Online ahead of print]

To cite this Article:
Sharma D, Kumar S, Prakash V, et al. **Diagnostic yield of thoracoscopic pleural biopsy in undiagnosed exudative pleural effusion and comparative analysis of pleural fluid adenosine deaminase levels**. *Monaldi Arch Chest Dis* doi: 10.4081/monaldi.2026.3910

Submitted: 29-01-2026
Accepted: 3-07-2026

Diagnostic yield of thoracoscopic pleural biopsy in undiagnosed exudative pleural effusion and comparative analysis of pleural fluid adenosine deaminase levels

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Conflict of interest: the authors declare no conflict of interest.

Ethics approval and consent to participate: the study was approved by the Institutional Ethics Committee, India. IEC approval letter No. 3177/Ethics/2025, dated **0**5 March 2025). The study

was conducted in accordance with the principles of the Declaration of Helsinki and applicable institutional and national ethical guidelines.

Informed consent: written informed consent was obtained from all participants prior to enrolment and prior to medical thoracoscopy.

Patient consent for publication: written informed consent for publication was obtained from all participants

Availability of data and materials: all data generated or analyzed during this study are included in this article.

Acknowledgments: the authors acknowledge the support of the Department of Pulmonary and Critical Care Medicine, for facilitation of patient evaluation and procedures. The authors also thank the nursing and technical staff for their assistance during thoracoscopy and specimen processing, and express gratitude to all participants for providing informed consent.

Declaration of generative artificial intelligence and artificial intelligence-assisted technologies in the writing process: not used.

Abstract

Undiagnosed exudative pleural effusion remains a frequent clinical challenge, particularly in high-burden settings where tuberculosis and malignancy predominate. Pleural fluid adenosine deaminase (ADA) is useful for suggesting tuberculous pleural effusion (TPE), but a substantial "gray zone" overlaps with malignant pleural effusion (MPE). This study evaluated the diagnostic yield of medical thoracoscopy with comparative analysis of pleural fluid ADA levels. This prospective, single-center, exploratory observational study was conducted over 1.5 years in a tertiary care hospital of north India. Consecutive adults (>18 years) with undiagnosed exudative pleural effusion (Light's criteria) were enrolled after pleural fluid CBNAAT/GeneXpert was negative for *Mycobacterium tuberculosis* and pleural fluid cytology negative for malignancy on at least two samples, taken ≥24 hours apart. A total of 150 patients were included (92 males, 58 females). All patients underwent single-port medical thoracoscopy under conscious sedation, systematic pleural inspection, and targeted biopsy (3-6 samples). Biopsies were sent for histopathology and CBNAAT. Thoracoscopic pleural biopsy established malignancy in 104/150 (69.3%) and tuberculosis in 40/150 (26.7%) patients, resulting in an overall diagnostic yield of 96%. Non-specific inflammation was observed in four patients, pyogenic inflammation in one, and one biopsy was inconclusive. Metastatic adenocarcinoma was the commonest malignancy (88/104). In biopsy-proven TPE, tissue CBNAAT positivity was low (9/40, 22.5%). ADA was significantly higher in tubercular effusions than MPE (40.17 *vs.* 20.11 IU/L; $p<0.001$). Major complications were infrequent with no procedure-related mortality.

Key words: pleural effusion, thoracoscopy, adenosine deaminase, tuberculosis.

Introduction

Pleural effusion is a common clinical problem and represents a manifestation of many underlying diseases. Infections, benign conditions and malignancies are all recognised causes, and even after thorough clinical, radiological and laboratory evaluation, about 15-20% of pleural effusions may remain undiagnosed [1,2]. In India, tuberculosis is the leading aetiology of exudative pleural effusion, followed by malignancy [3,4]. More than 40% of patients with an initially undiagnosed pleural effusion may later be diagnosed with tuberculosis, so tuberculous pleuritis should always be a strong diagnostic consideration when the cause of an exudative effusion is unclear [5]. Closed pleural biopsy can establish a diagnosis in roughly 40-80% of such undiagnosed cases [6,7].

Pleural biopsy and adenosine deaminase (ADA) together form an important part of the diagnostic work-up in exudative pleural effusion. Pleural fluid ADA values >70 U/L are highly suggestive of tuberculous pleural effusion and show good concordance with histopathological abnormalities on pleural tissue [8]. In malignant effusions, pleural fluid cytology can identify cancer in a substantial proportion of cases, while closed pleural biopsy contributes additional diagnostic value [9]. Light's criteria remain the cornerstone in distinguishing exudates from transudates, with pleural LDH performing particularly well among its components [10,11]. Pleural fluid ADA is a valuable adjunctive biomarker for differentiating tuberculous from non-tuberculous pleural effusions, although its interpretation should be considered in the appropriate clinical context [12].

Although ADA had high sensitivity and specificity for tuberculous effusions and pleural biopsy yielded good results for tuberculosis and malignancy, data on the diagnostic performance of medical thoracoscopy in undiagnosed exudative pleural effusion and its comparative analysis with ADA levels were limited. Therefore, the present study was undertaken to assess the diagnostic yield of medical thoracoscopy and analyse pleural fluid ADA in patients with undiagnosed exudative pleural effusion, with the aim of refining the diagnostic pathways and reduce the burden of unexplained effusions.

Material and Methods

Study design and setting

This was a prospective, single-centre, exploratory observational study conducted in the Department of Pulmonary and Critical Care Medicine at a tertiary care centre in India over a

period of 1.5 years. All consecutive adult patients aged >18 years presenting with undiagnosed exudative pleural effusion who fulfilled the predefined inclusion criteria were enrolled. Undiagnosed pleural effusion was defined as exudative as per Light's criteria with pleural fluid GeneXpert/CBNAAT negative for *Mycobacterium tuberculosis* and pleural fluid cytology negative for malignancy on at least two separate samples taken ≥24 hours apart. Patients were included only if they were considered surgically/anaesthetically fit for medical thoracoscopy & given consent for the procedure. Patients were excluded if they had severe uncorrected hypoxaemia despite supplemental oxygen, unstable cardiovascular or haemodynamic status, coagulation defects, obliterated or inaccessible pleural space due to dense fibrosis, suspected hydatid cysts or highly vascular pulmonary lesions, pregnancy.

Sample size

Previous studies have shown that the proportion of cases diagnosed by medical thoracoscopy is approximately 90–95% [13]. We decided to use the lower estimate for calculation of sample size. The Daniel's formula for prevalence studies has been used [Eq. 1].

$$N=Z^2\ p(1-p)/d^2 \qquad \text{[Eq. 1]}$$

Where: n=Sample size, Z statistic for level of confidence, P=Expected proportion of cases diagnosed by medical thoracoscopy (90%), d=Precision (5%).

After accounting for approximately 10% noncompliance, the final sample size of 150 was considered feasible. Hence, 150 patients of pleural effusions of undiagnosed etiology were enrolled.

After securing approval from the Institutional Ethics Committee (Ref. Code: XXXI-PGTSC-IIA/P30),Ref. No.-3177/ethics/2025 and obtaining written informed consent from each participant, detailed clinical information was recorded. All enrolled patients underwent routine laboratory investigations including complete hemogram, renal and liver function tests, blood glucose levels, bleeding profile, and viral markers (HIV ELISA, Anti-HCV antibody, HBsAg). Radiological assessment was performed using chest X-ray for initial evaluation and contrast-enhanced CT scan when deemed necessary. Pleural fluid analysis was conducted in all cases, including biochemical, cytological, and bacteriological evaluation. Subsequently, each patient underwent medical thoracoscopy under conscious sedation after fasting for six hours.

Following ultrasound assessment of the pleural cavity for loculations, adhesions, and pleural thickening, the procedure site was marked and prepared aseptically. Under local anesthesia and conscious sedation using midazolam and fentanyl, thoracoscopic examination of the pleural cavity was performed through a single port. The pleural surfaces were systematically inspected, loculations were gently released, and 3–6 pleural biopsies were taken from suspicious or representative areas. The obtained tissue samples were sent for histopathology and GeneXpert (CBNAAT). After completion of the procedure, a chest tube was placed through the same port and maintained until lung re-expansion achieved, air leak stopped, and pleural drainage reduced to <50–100 mL/day. All patients were monitored clinically and radiologically until complete recovery and chest tube removal, ensuring standardized and systematic data acquisition throughout the study.

All collected data were entered into Microsoft Excel and analysed using SPSS version 25. Quantitative variables were summarised as mean ± standard deviation (SD), while qualitative variables were presented as frequencies and percentages. Categorical variables were compared using the Chi-square test or Fisher's exact test, as appropriate. A p-value of <0.05 was considered statistically significant.

Results

Out of 150 participants, males (n=92) had a higher mean age (58.79 ± 11.39 years) compared with females (n=58; 54.55 ± 12.35 years). Most patients were in the 41–60 years (44 males, 31 females) and 61–80 years (40 males, 21 females) age groups, with very few ≤20 or >80 years of age (Figure 1).

Effusion was predominantly unilateral, occurring slightly more often on the right side (48.7%) than on the left (46.7%). A smaller proportion of patients (4.7%) had bilateral effusions. Pleural fluid was more often haemorrhagic (57.3%) than straw coloured (42.7%), indicating that a substantial proportion of patients presented with blood-stained effusions, which is frequently associated with malignant or more severe pleural pathology.

Baseline blood tests showed mild anaemia (mean haemoglobin 10.82 g/dL). Pleural fluid analysis demonstrated exudative, lymphocyte-predominant effusion, with high protein levels (mean 4.66 g/dL) and markedly elevated lymphocytes (80.07%), while ADA levels (mean 23.53 IU/L) were in the intermediate range, underscoring the need for further diagnostic evaluation such as thoracoscopy and biopsy (Table 1).

Cough (n=118), loss of appetite (n=110), breathlessness (n=86), loss of weight (n=82) and chest pain (n=80) were the predominant presenting complaints, whereas fever was less common. Among co-morbidities, diabetes mellitus (n=30) were most frequent, followed by hypertension (n=23), chronic kidney disease and coronary artery disease were rare. On examination, pallor (n=52) was the leading clinical sign, with fewer patients showing clubbing (n=28) or peripheral lymphadenopathy (n=22). Thoracoscopic examination most commonly revealed pleural thickening (n=100) and nodules (n=88), followed by puckering of the pleura (n=62) and loculations/adhesions (n=58). Mass lesions were seen less frequently (n=32) (Figure 1).

Thoracoscopic pleural biopsy most commonly demonstrated malignancy which was identified in 104 of 150 patients, followed by tubercular pleuritis (40 cases). Non-specific inflammation reported in four cases and one case reported as pyogenic inflammation. Biopsy was inconclusive in one patient. These finding indicates a high diagnostic yield for definitive etiology. Among malignant effusions, metastatic adenocarcinoma overwhelmingly predominated (88 cases), with far fewer squamous cell carcinoma (8), mesothelioma (3), non-Hodgkin's lymphoma (2), undifferentiated carcinoma (2) and small cell carcinoma (1) (Figure 2). CBNAAT on pleural biopsy tissue was positive for *Mycobacterium tuberculosis* with rifampicin sensitivity in 22.5% cases and none showed rifampicin resistance.

Patients with malignant pleural effusion (n=104) and tuberculous pleural effusion (n=40) were similar in age, but TB cases showed a strong male predominance (90% vs 52.8%, $p<0.001$). Chronic kidney disease was slightly more frequent in TB, while other co-morbidities did not differ significantly. Haemorrhagic pleural fluid was significantly more common in malignancy (64.4% vs 40%, $p=0.002$). Mean pleural fluid protein was similar, but ADA levels were markedly higher in tuberculosis (40.17 vs 20.11 IU/L, $p<0.001$). Thoracoscopically, nodules and puckering of pleura were more commonly seen in malignancy, while pleural thickening and loculations/adhesions were more frequent in TB (Table 1).

Receiver operating characteristic (ROC) analysis was performed to assess the diagnostic performance of pleural fluid ADA in differentiating tuberculous pleural effusion from malignant pleural effusion. ADA demonstrated good discriminatory ability, with an area under the ROC curve (AUC) of 0.79 (95% CI: 0.71–0.86; $p<0.001$). Using the conventional cut-off value of 40 IU/L, sensitivity, specificity, positive predictive value, and negative predictive value were 67.5%, 92.3%, 77.1%, and 88.1%, respectively. Although ADA levels were significantly higher in tuberculosis, substantial overlap between tuberculous and malignant effusions was observed,

indicating that ADA should be interpreted alongside thoracoscopic and histopathological findings rather than a standalone diagnostic test. Among the 40 biopsy-confirmed tuberculous pleural effusion cases, 13 patients (32.5%) had pleural fluid ADA levels <40 IU/L. Compared with patients having ADA ≥40 IU/L, those with lower ADA levels tended to be older and had a higher frequency of prior anti-tubercular treatment exposure; however, these differences did not reach statistical significance.

Major complications were infrequent and occurred in 14 patients, including persistent bronchopleural fistula beyond 72 hours (n=5), non-expansion of the lung (n=7), and postoperative empyema (n=2). No cases of major bleeding requiring blood transfusion, re-expansion pulmonary edema necessitating intensive care unit admission, or procedure-related mortality were observed. Minor complication events included post-procedure pain (n=51), hypoxia during the procedure (n=5), and subcutaneous emphysema (n=4), with some patients experiencing more than one minor complication. Overall, medical thoracoscopy with pleural biopsy was a safe procedure in this cohort, with predominantly manageable minor adverse events and a low frequency of serious complications (Table 2)

Discussion

In the present study of 150 patients with exudative pleural effusion undergoing thoracoscopy and pleural biopsy, the demographic, biochemical and thoracoscopic characteristics, as well as the diagnostic yield, were analysed and compared with the findings of previously published literature.

Our cohort comprised 61.3% males and 38.7% females, with pleural disease predominantly affecting middle-aged and elderly individuals, consistent with the demographic trends reported in multiple studies. Mittal et al. and Kumar et al. similarly noted that exudative pleural effusions commonly present in the fifth to seventh decades of life [14,15]. The mean age of our patients was 57.15 years, comparable to other series. Although malignancy generally increases with age, no significant age difference was observed between malignant pleural effusion (MPE) and tuberculous pleural effusion (TPE), a pattern that may reflect referral bias from empirical anti-tubercular therapy at peripheral centres.

Biochemically, our cohort demonstrated lymphocyte-predominant exudates with intermediate ADA levels (mean 23.53 IU/L). This overlaps with the “gray zone” ADA range described by Yang et al. [16], while Mittal et al. proposed an ADA cut-off of >40 IU/L with high sensitivity

(96.88%) [14]. However, more recent studies, including Yang et al. and Kim et al., have shown that rigid reliance on this threshold can result in misclassification—particularly in elderly patients or in regions with shifting TB epidemiology [8,16]. Our findings reinforce this evolving understanding: the mean ADA in TPE (40.17 IU/L) was significantly higher than in MPE (20.11 IU/L), yet 32.5% of TB cases in our series had ADA <40 IU/L and 7.66% of malignancies had ADA >40 IU/L. Similar observations have been reported in other series [15,17], underscoring that ADA alone cannot reliably distinguish TB from malignancy. Receiver operating characteristic analysis demonstrated good discriminatory performance of ADA, with an area under the curve (AUC) of 0.79 (95% CI: 0.71–0.86). At the conventional ADA cut-off value of 40 IU/L, sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) were 67.5%, 92.3%, 77.1%, and 88.1%, respectively. Exploratory analysis using the Youden index identified an optimal ADA threshold of 35 IU/L, which improved sensitivity to 72.5% while maintaining good specificity (89.4%), with corresponding PPV and NPV of 72.5% and 89.4%, respectively. However, the modest improvement in sensitivity was achieved at the expense of reduced specificity. Collectively, these findings suggest that although pleural fluid ADA remains a valuable adjunctive biomarker, it should not be used in isolation and is best interpreted in conjunction with thoracoscopic findings and histopathological examination for definitive diagnosis. The exploratory 35 IU/L threshold identified in this study warrants validation in larger, independent cohorts before consideration for routine clinical application. Among the 13 biopsy-confirmed tuberculous pleural effusion patients with ADA levels <40 IU/L, subgroup analysis suggested a tendency toward older age and previous anti-tubercular treatment exposure. Although the small sample size precluded definitive conclusions, these findings support previous reports indicating that factors such as immunosuppression, malnutrition, and altered cellular immune responses may contribute to falsely low ADA levels.

Thoracoscopic findings in our study—predominantly pleural thickening, nodules, puckering, and adhesions—are consistent with patterns described in the literature. Pleural thickening was the most frequent abnormality observed in the low-ADA TB population of Kumar et al. and in the study by Hussein et al. [15,18]. Nodularity and pleural masses were more frequently associated with malignancy in our cohort, supporting the observations of Lee et al. [19], who demonstrated that pleural nodularity—especially > eight focal nodules—strongly predicted malignant etiology. In our study, TB commonly presented with diffuse pleural thickening and

septations/loculations, whereas malignancy typically showed pleural nodules, puckering and thickening, comparable with previous studies [5,15,18,19].

Considering only malignancy and tuberculosis as definitive etiological diagnoses. The diagnostic yield of thoracoscopic pleural biopsy in our study was 96%, identifying malignancy in 104 cases (69.33%), TB in 40 cases (26.66%), placing our results in the range of previously reported diagnostic yield (74–99%), in earlier studies. This aligns with the diagnostic yields reported by Arif M et al, Kumar et al. [5,15], who demonstrated that thoracoscopy provides superior diagnostic accuracy, particularly in cytology-negative, low-ADA exudates. The predominance of metastatic adenocarcinoma as the leading cause of MPE in our population (84.6% of all malignant cases) mirrors the malignant profile described by Kim et al. [8] and is consistent with global trends showing rising lung adenocarcinoma metastasis to the pleura. Lung was the most common primary site, followed by breast, female genital tract, oropharyngeal and gastrointestinal malignancies, and in 13.6% of patients the primary tumour remained occult. Mesothelioma was relatively uncommon, likely reflecting regional differences in environmental exposure.

The low positivity of CBNAAT among biopsy-proven TB cases in our study (22.5%) reaffirms the well-recognized paucibacillary nature of pleural TB. This is in keeping with Lee et al. [19], who emphasized that microbiological confirmation rates remain modest despite strong clinical and histopathological evidence. All our TB cases showed granulomatous inflammation on histopathology, and most were CBNAAT-negative, underscoring the continued reliance on histology and composite clinical–biochemical assessment for definitive diagnosis. Notably, the relatively lower proportion of TB cases in our study as compared to older Indian thoracoscopic studies likely reflects evolving epidemiology, with increasing lung cancer incidence and widespread empirical ATT at peripheral centres delaying timely recognition of malignant pleural effusion.

Comparing biochemical and thoracoscopic predictors between MPE and TPE, our findings show close similarity to previously published models. Elevated pleural fluid protein was nondiscriminatory, while ADA was significantly higher in TB. Pleural fluid appearance varied significantly, with haemorrhagic effusions strongly associated with malignancy—consistent with established literature. Thoracoscopically, nodularity and puckered pleura favored malignancy, while diffuse thickening and adhesions were more common in TB, echoing the predictive trends described by Hussein et al. and Lee et al. [18,19].

Finally, the safety profile of thoracoscopy in our study was highly favorable. Major complications were rare and comparable to those reported by Kumar et al. [15], and Kim et al. [8], who demonstrated that thoracoscopy, when performed in experienced centres, is associated with low morbidity and no procedure-related mortality. In our cohort, major complications occurred in 14 patients and comprised persistent bronchopleural fistula beyond 72 hours (n=5), postoperative empyema (n=2), and non-expansion of the lung (n=7). Minor complication events included post-procedure pain (n=51), transient hypoxia during the procedure (n=5), and subcutaneous emphysema (n=4), with some patients experiencing more than one minor complication. No cases of major bleeding requiring blood transfusion, re-expansion pulmonary edema necessitating intensive care unit admission, or procedure-related mortality were observed. Overall, the predominance of minor, self-limited adverse events and the low frequency of serious complications underscore the safety of thoracoscopy when performed in appropriately selected patients.

In summary, our findings are largely concordant with published evidence and highlight three major themes emerging in modern pleural diagnostics: (1) Limitations of fixed ADA cut-offs, (2) important role of thoracoscopic morphology and pleural biopsy, and (3) the growing diagnostic complexity within the ADA gray zone due to shifting epidemiology. This study has several limitations. As it was a single centre study, findings may not be generalizable in other healthcare settings. In addition. The absence of blinded thoracoscopic interpretation may have introduced observer bias.. The study also did not include cost-effectiveness analysis, hence economic implications could not be assessed.

Conclusions

Medical thoracoscopy was found to be a safe and effective modality for the evaluation of undiagnosed exudative pleural effusions, achieving an excellent diagnostic yield and accurately distinguishing between malignancy and tuberculosis. Given the limitations of fixed ADA cut-offs across diverse epidemiological settings and limited sensitivity of molecular diagnostic tests in pleural TB, early referral for thoracoscopy and integration of biochemical, radiological and thoracoscopic findings may improve diagnostic accuracy. Larger multicentric studies and improved molecular diagnostic tools are needed to further refine diagnostic pathways, facilitating early and accurate etiological classification of pleural effusions.

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Table 1. Comparison of clinical, pleural fluid and thoracoscopic findings between malignancy and tuberculosis.

Variables		Malignancy(n-104)	Tuberculosis(n-40)	P-value
Age in Years (Mean± SD)		56.85 ± 12.33	58.88 ± 8.90	0.276
Sex	**Male(n%)**	55 (52.8%)	36 (90%)	<0.001
	Female(n%)	49 (47.11%)	04 (10%)	
History of ATT [Yes(n%)]		21(20.1%)	14 (35%)	0.06
Side of Pleural Effusion	Left side	47(45.19%)	21(52.5%)	0.590
	Right side	51(49.03%)	18(45%)	
	Bilateral	6(5.8%)	01(2.5%)	
Colour of Pleural Fluid	Haemorrhagic	67(64.42%)	16(40%)	0.002
	Straw Colour	37(35.58)	24(60%)	
Pleural Fluid Protein (Gm%) (Mean±SD)		4.61±1.08	4.78±1.05	0.286
Pleural Fluid ADA (IU/L)		20.11±10.33	40.17 ±23.00	<0.001
Pleural Fluid Lymphocyte (%)		80.85±18.64	75.17±24.71	0.688
Thoracoscopic Findings	**Loculations/Adhesions**	36(34.6%)	22(55%)	0.040
	Nodules	74(71.1%)	14(35%)	<0.001
	Mass	27(25.96%)	05(12.5%)	0.129
	Puckered Pleura	55(52.88%)	07(17.5%)	<0.001
	Pleural Thickening	66(63.46%)	34(85%)	0.020
	Sagograins nodules	0(0%)	02 (5%)	0.133

Table 2. Complications of rigid thoracoscopy.

Complications		n
Major Complications*	Persistent Bronchopleural fistula beyond 72 hours	**5**
	Non-expansion of lung	**7**
	Post-operative infections (Empyema)	**2**
	Major bleeding requires blood transfusion	**0**
	Re-expansion pulmonary edema requiring ICU	**0**
	Death	**0**
Minor Complications†	Post procedure pain	**51**
	Hypoxia during the procedure	**5**
	Subcutaneous emphysema	**4**

*Major complications are reported as the number of affected patients. †Minor complications are reported as events; some patients experienced more than one minor complication.

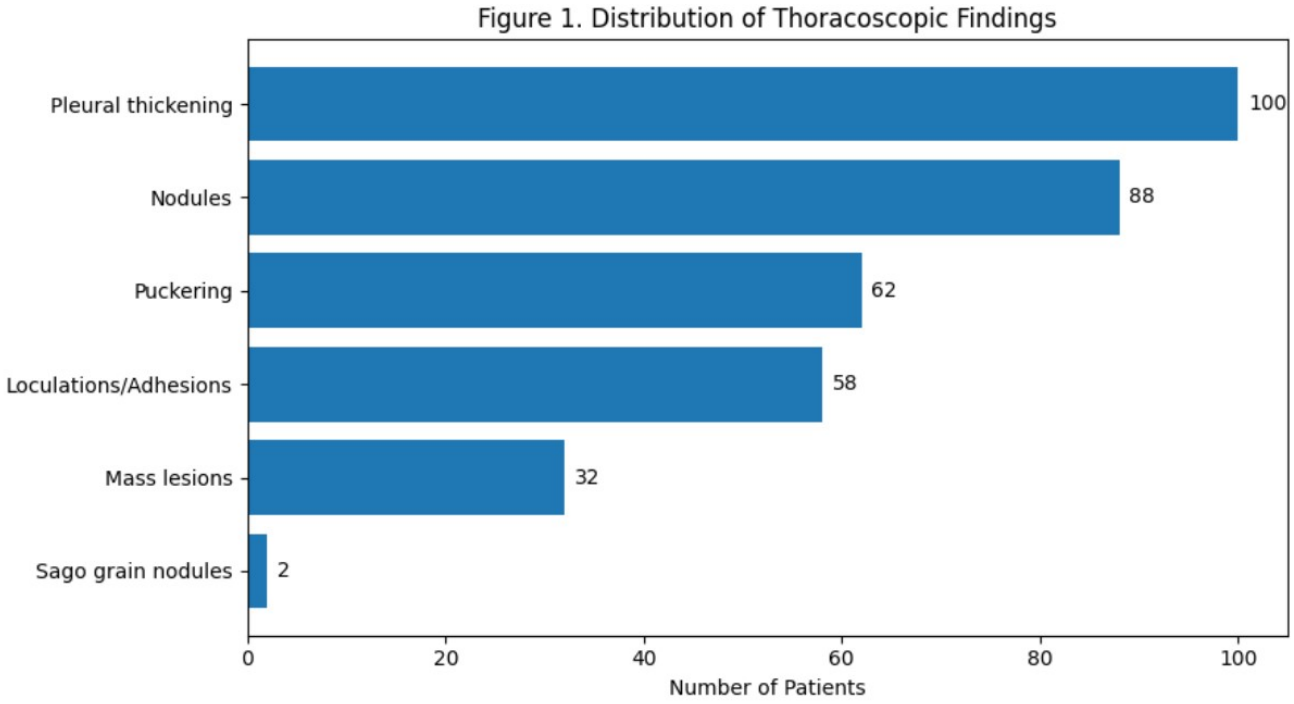

Figure 1. Distribution of thoracoscopic findings during medical thoracoscopy. Values represent the number of patients demonstrating each thoracoscopic feature.

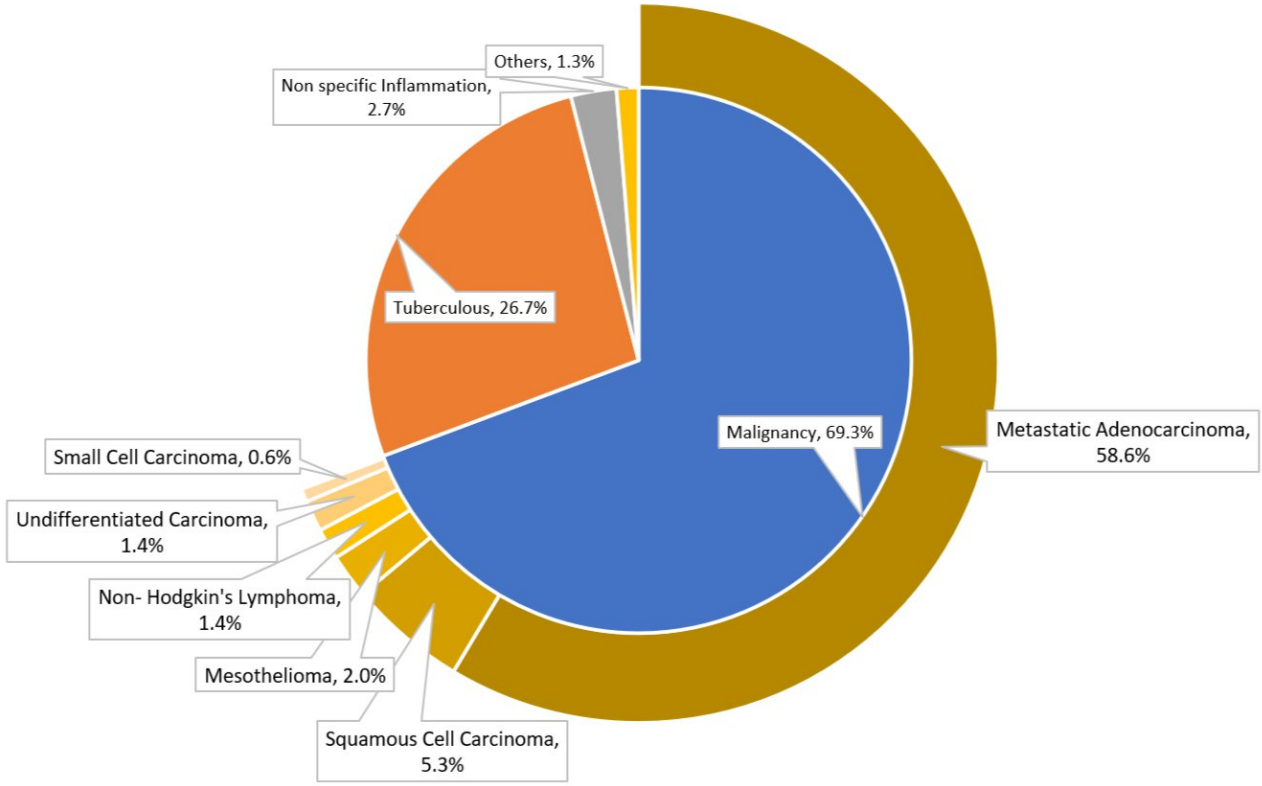

Figure 2. Histopathological Spectrum of Thoracoscopic Pleural Biopsies and Malignant Subtypes. The inner chart represents the overall histopathological diagnoses established by thoracoscopic pleural biopsy. The outer chart depicts the distribution of malignant histological subtypes among patients diagnosed with malignant pleural effusion.