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Factors Associated with Diffuse Idiopathic Pulmonary Neuroendocrine Cell Hyperplasia (DIPNECH) in Patients with Bronchiectasis: A Retrospective Study

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Abstract

Background

Diffuse idiopathic pulmonary neuroendocrine cell hyperplasia (DIPNECH) is a preinvasive neuroendocrine lesion that may progress to carcinoid tumors. Chronic inflammatory airway diseases such as bronchiectasis may promote neuroendocrine proliferation; however, clinical predictors of DIPNECH in bronchiectasis remain unclear.

Methods

We retrospectively analyzed 58 patients who underwent lung resection for bronchiectasis 2022 and 2025. Demographic and clinical data, inflammatory markers, radiologic findings including thoracic computed tomography for millimeter-sized nodules, histopathologic diagnoses, and bronchiectasis severity index were assessed.

Results

DIPNECH was identified in 19 patients (32.8%). Patients with DIPNECH were older (51.6 ± 13.3 vs. 41.2 ± 14.9 years, $p = 0.013$) and had significantly higher BSI scores (median 5 vs. 3, $p = 0.022$). Pulmonary nodules on computed tomography were markedly more frequent in patients with DIPNECH (73.3% vs. 18.6%, $p < 0.001$). Inflammatory markers and sex were not significantly associated with DIPNECH. In univariate analysis, age, pulmonary nodules, and BSI showed a significant association with DIPNECH, while in multivariate analysis, only pulmonary nodules (OR 14.12, 95% CI 2.58–77.15, $p = 0.002$) and BSI (OR 1.99, 95% CI 1.13–3.50, $p = 0.017$) remained independent predictors.

Conclusion

A subset of bronchiectasis patients with high BSI and pulmonary nodules may represent a higher-risk group for DIPNECH, potentially warranting risk-adapted surgical management strategies.

Introduction

Diffuse idiopathic pulmonary neuroendocrine cell hyperplasia (DIPNECH) is a rare pulmonary disease characterized by the proliferation of pulmonary neuroendocrine cells within the bronchial and bronchiolar epithelium without invasion through the basement membrane and is considered a preinvasive precursor of carcinoid tumors. DIPNECH is often underdiagnosed preoperatively because of subtle radiologic features and symptom overlap with other chronic airway diseases[1].

Chronic airway inflammation has been implicated in neuroendocrine cell proliferation. Persistent inflammatory signaling and epithelial injury may contribute to abnormal neuroendocrine activation and proliferation of pulmonary neuroendocrine cells, particularly in chronic lung disease. Bronchiectasis, characterized by permanent bronchial dilation and chronic infection–inflammation cycles, provides a microenvironment of repeated epithelial injury and immune activation that may predispose to such proliferative processes. Patients with bronchiectasis commonly experience chronic productive cough, recurrent infections, and airflow limitation, with quality of life increasingly impacted over time. Bridging chronic airway inflammation and neuroendocrine proliferation, several case reports and small series have noted the coexistence of DIPNECH in patients with bronchiectatic changes; however, large cohort data are lacking. The mechanisms by which chronic inflammation might influence neuroendocrine cell dysregulation remain an area of active investigation[2, 3]

Bronchiectasis is primarily managed with medical therapy and pulmonary rehabilitation. Surgery is considered in cases of recurrent infections or exacerbations, persistent hemoptysis despite optimal pharmacological management, or the presence of destroyed lung. The potential development of DIPNECH has not been defined as a surgical indication in patients with bronchiectasis[4].

Several validated scoring systems have been developed to assess the severity of bronchiectasis. These scoring models can predict long-term mortality, risk of hospitalization, and exacerbation frequency, and are also associated with health-related quality of life measures[5]. Similarly, inflammatory markers, such as the neutrophil-to-lymphocyte ratio (NLR), systemic immune-inflammation index (SII), and platelet-to-lymphocyte ratio (PLR), have been shown to be associated with an increased risk of exacerbation, increased disease activity, and adverse clinical outcomes in patients with bronchiectasis. However, the relationship of these scoring systems and inflammatory parameters with the development of DIPNECH in bronchiectasis has not yet been investigated[6, 7].

This study aimed to identify clinical and radiologic factors associated with the development of DIPNECH in patients with bronchiectasis who underwent surgical resection. Recognizing the risk factors for DIPNECH may allow clinicians to identify patients at a higher risk who could benefit from earlier intervention to prevent possible progression to carcinoid tumors.

Materials and Methods

We retrospectively reviewed patients who underwent surgical resection for bronchiectasis between January 2022 and December 2025. The indications for surgery were based on established criteria in the literature, including localized bronchiectasis poorly controlled despite optimal medical management, including airway clearance and antibiotics. Recurrent or persistent infection with frequent exacerbations, hemoptysis refractory to bronchial artery embolization or life-threatening hemorrhage, presence of a destroyed lung segment leading to significant symptoms and complication prevention in anatomically limited disease, and improved quality of life.

All patients received preoperative antibiotic therapy until sputum cultures were negative. The surgical approach (video-assisted thoracoscopic surgery vs. thoracotomy) was individualized according to the adhesions and disease extent. Demographic data, clinical presentation, pulmonary function test, and surgical outcomes were analyzed. Preoperative thoracic CT scans were re-evaluated for previously unreported millimeter nodules(Fig. 1).

Bronchiectasis severity was quantified using the Bronchiectasis Severity Index (BSI), a validated multidimensional score that predicts morbidity and mortality in bronchiectasis. The index incorporates age, body mass index, FEV1% predicted, prior hospitalizations, exacerbation frequency, Medical Research Council dyspnea score, chronic colonization status, and radiological disease extent. The components of the Bronchiectasis Severity Index (BSI) with their respective scoring weights are summarized in Table 1.

Preoperative biochemical analyses included complete blood count and C-reactive protein (CRP). Systemic inflammatory status was calculated using routine preoperative complete blood count parameters. The neutrophil-to-lymphocyte ratio (NLR) was calculated as the absolute neutrophil count divided by the absolute lymphocyte count. The platelet-to-lymphocyte ratio (PLR) was calculated as platelet count/absolute lymphocyte count. The systemic immune-inflammation index (SII) was calculated as platelet count × neutrophil count/lymphocyte count.

Table 1
Bronchiectasis Severity Index (BSI) scoring system.

Variable	Category	Score
Age (years)	< 50	0
	50–69	2
	70–79	4
	≥ 80	6
Body Mass Index (BMI)	≥ 18.5	0
	< 18.5	2
FEV_1 (% predicted)	> 80	0
	50–80	1
	30–49	2
	< 30	3
Previous hospital admission	No	0
	Yes	5
Exacerbations in previous year	0	0
	1–2	0
	≥ 3	2
MRC dyspnea score	1–3	0
	4	2
	5	3
Pseudomonas colonization	No	0
	Yes	3
Colonization with other organisms	No	0
	Yes	1
Radiological severity	< 3 lobes involved	0
	≥ 3 lobes or cystic bronchiectasis	1

Statistical Analysis

Parametric variables were expressed as mean ± SD and analyzed with Student's t-test. Nonparametric variables are presented as the median [interquartile range] and compared using the Mann–Whitney U

test. Categorical variables were analyzed using the chi-squared test or Fisher's exact test. Variables that demonstrated statistical significance in the comparative analyses were first included in the univariate regression analysis. Subsequently, variables that remained significant in the univariate analysis were entered into the multivariate regression model to identify independent predictors of DIPNECH. A p-value < 0.05 was considered statistically significant.

Results

A total of 58 patients who underwent surgical resection for bronchiectasis were included in the study. The mean age of the cohort was 44.6 ± 15.1 years, and females constituted the majority (62.1%). The most common symptoms before surgery were hemoptysis (34.5%), followed by recurrent pneumonia (22.4%) and cough (20.7%), while 10.3% of patients were asymptomatic. Comorbidities were present in 43.1% of patients. The mean body mass index was 26.1 ± 4.5 kg/m². A history of smoking was reported in 25.9% of cases, with a mean exposure of 13.8 ± 7.6 pack-years.

Preoperative inflammatory markers showed a median CRP level of 2 [1–8.5], a white blood cell count of 7.6 $\times 10^9$/L [6.2–9.2], and a median bronchiectasis severity index of 3 [2–5]. The mean NLR, PLR, and SII values were 2.3 ± 1.0, 129 ± 44, and 586 [259–875], respectively. With respect to disease distribution, bronchiectasis was classified as lobar in 56.8% of patients, multilobar in 16.2%, segmental in 10.8%, and multisegmental in 16.2%.

Minimally invasive surgery was performed in 30 patients (51.7%), while 28 patients (48.3%) underwent thoracotomy. Postoperative complications occurred in 6 patients (10.3%). The median chest drain removal time was 4 [3–6] days, and the median length of hospital stay was 5 [4–7] days. The mean follow-up duration was 20 ± 11.3 months. DIPNECH was histopathologically diagnosed in 19 patients, corresponding to a prevalence of 32.8% in the study cohort (Table 2).

Table 2
Baseline Demographic, Clinical, and Operative Characteristics of the Study Population

Variable	n (%) / mean ± SD / median[IQR]
Number of Patients	58
Age	44.6 ± 15.1
Sex	Male: 22 (27.9) Female: 36 (62.1)
Comorbidities	Present: 25 (43.1) Absent: 33 (56.9)
Body Mass Index (kg/m^2)	26.1 ± 4.5
Smoking history	Present: 15 (25.9) Absent: 43 (74.1)
Smoking duration (pack/year)	13.8 ± 7.6
Preoperative CRP (Median[IQR])	2 [1-8.5]
Preoperative WBC (×10^9/L)	7.600 [6.200–9200]
Severity İndex	3 [2–5]
NLR	2.3 ± 1
PLR	129 ± 44
SII	586 [259–875]
Operation Type	Minimally invasive: 30 (51.7) Toracotomy: 28 (48.3)
Complication	6 (%10.3)
Drain Removal (days)	4 [3–6]
Length of stay (days)	5 [4–7]
DIPNECH* Diagnosis	19 (%32.8)
Follow-up (Month)	20 ± 11.3

DIPNECH: Diffuse Idiopathic Pulmonary Neuroendocrine Cell Hyperplasia; BSI: Bronchiectasis Severity Index; NLR: Neutrophil-to-Lymphocyte Ratio; PLR: Platelet-to-Lymphocyte Ratio; SII: Systemic Immune-Inflammation Index; WBC: White blood cell count.

Patients with DIPNECH were significantly older than those without (51.6 ± 13.3 vs. 41.2 ± 14.9 years, p = 0.013). There were no significant differences between the groups in terms of sex distribution (p = 0.906), body mass index (p = 0.121), smoking history (p = 0.956), or extent of bronchiectasis (p = 0.745). Inflammatory parameters, including NLR (p = 0.583), PLR (p = 0.661), and SII (p = 0.602), were also similar between the groups.

The Bronchiectasis Severity Index was significantly higher in the DIPNECH group (median 5 [4–7] vs. 3 [2–4], p = 0.022). Pulmonary nodules on computed tomography were significantly more frequent in patients with DIPNECH, observed in 11 (73.3%) compared with 8 (18.6%) patients without DIPNECH (p < 0.001) (Table 3).

Univariate regression analysis identified age (OR 1.05, 95% CI 1.00–1.09, p = 0.018), pulmonary nodules (OR 12.51, 95% CI 3.03–47.73, p < 0.001), and bronchiectasis severity index (OR 1.88, 95% CI 1.29–2.74, p = 0.001) as factors associated with DIPNECH. In multivariate analysis, pulmonary nodules (OR 14.12, 95% CI 2.58–77.15, p = 0.002) and bronchiectasis severity index (OR 1.99, 95% CI 1.13–3.50, p = 0.017) remained independent predictors, whereas age was no longer statistically significant (p = 0.976) (Table 4).

Table 3
Demographic, Clinical, and Radiological Characteristics Categorized by the Presence of Coexisting DIPNECH.

	DIPNECH (+)	DIPNECH(-)	p
	N(%) / mean ± SD / median [IQR]		
Number of patients	39 (67.2)	19 (32.8)	
Age (year)	41.2 ± 14.9	51.6 ± 13.3	**0.013***
Sex	15 (68.2)	7 (31.8)	0.906
Male	24 (66.7)	12 (33.3)	
Female			
Body Mass Index (kg/m^2)	25 ± 4.7	27 ± 2.5	0.121
Smoking history	10 (66.7)	5 (33.3)	0.956
Present	29 (67.4)	14 (32.6)	
Absent			
Severity index	3 [2–4]	5 [4–7]	**0.022***
Pulmonary Nodule	4 (26.7)	11 (73.3)	**< 0.001***
Present	35 (81.4)	8 (18.6)	
Absent			
Extend of bronchiectasis	25 (65.8)	13 (34.2)	0.745
One lob or less	14 (70)	6 (30)	
More than a lob			
NLR	2.4 ± 1.1	2.3 ± 0.8	0.583
PLR	129 ± 44	138 ± 41	0.661
SII	681 ± 469	623 ± 248	0.602

DIPNECH: Diffuse Idiopathic Pulmonary Neuroendocrine Cell Hyperplasia; BSI: Bronchiectasis Severity Index; NLR: Neutrophil-to-Lymphocyte Ratio; PLR: Platelet-to-Lymphocyte Ratio; SII: Systemic Immune-Inflammation Index.

Table 4
Univariate and Multivariate Regression Analysis for Predictors of DIPNECH

	Univariate Analysis			Multivariate Analysis		
	OR	**CI (%95)**	**P**	**OR**	**CI (%95)**	**P**
Age (per year)	1.05	1.00-1.09	**0.018***	1.00	0.94–1.06	0.976
Pulmonary Nodule	12.51	3.03–47.73	**< 0.001***	14.12	2.58–77.15	**0.002***
Bronchiectasis Severity Index	1.88	1.29–2.74	**0.001***	1.99	1.13–3.50	**0.017***

DIPNECH: Diffuse Idiopathic Pulmonary Neuroendocrine Cell Hyperplasia

Discussion

In this study of patients undergoing surgical resection for bronchiectasis, DIPNECH was identified in 32.8% cases, and two independent predictors were demonstrated: a higher Bronchiectasis Severity Index (BSI) and the presence of pulmonary nodules on computed tomography. These findings define a clinically identifiable high-risk subgroup within bronchiectasis patients and suggest a measurable link between disease severity and neuroendocrine proliferation.

DIPNECH is generally considered a rare entity, it has been increasingly recognized in histopathological specimens obtained from lung resections performed for other indications. Previous studies have reported DIPNECH either as an incidental finding or in association with tumorlets and carcinoid tumors, often without clear clinical predictors[2, 8–11]. In contrast to these heterogeneous cohorts, our study specifically evaluates a population characterized by bronchiectasis. The markedly higher prevalence observed in our cohort supports the hypothesis that persistent airway inflammation may contribute to the development of neuroendocrine cell hyperplasia. Moreover, the use of surgical specimens likely facilitates the detection of subclinical DIPNECH, which may otherwise remain unrecognized [2, 12, 13].

Despite the well-established female predominance reported in the literature, no significant sex difference was observed in our study. Previous series have reported a strong female predominance, with ratios as high as 10:1[10, 14]. The absence of this pattern in our cohort may reflect the distinct pathophysiological context of bronchiectasis-related inflammation rather than classical DIPNECH.

Chronic inflammation is a central feature of bronchiectasis and is characterized by recurrent infection, neutrophilic inflammation, and progressive airway damage. Pulmonary neuroendocrine cells are known to respond to hypoxia, epithelial injury, and inflammatory mediators and may participate in airway repair processes [15]. Persistent inflammatory stimulation may therefore result in dysregulated neuroendocrine proliferation. Previous studies have suggested an association between chronic lung disease and DIPNECH; however, objective clinical predictors have not been clearly defined[2, 16]. Our findings provide quantitative evidence supporting this relationship.

One of the most notable findings of this study is the independent predictive role of BSI. As a validated multidimensional scoring system, the BSI reflects not only disease severity but also cumulative inflammatory burden and structural lung damage [5]. The observed association between higher BSI scores and DIPNECH suggests that neuroendocrine proliferation may be more likely to occur in patients with advanced or biologically active diseases. This expands the clinical relevance of the BSI beyond its established prognostic role and raises the possibility that it may also serve as a marker of oncogenic potential in chronic inflammatory lung conditions.

The second major finding is the strong association between pulmonary nodules and DIPNECH. Small, peribronchial or centrilobular nodules are recognized radiologic features in patients with neuroendocrine cell proliferations, including DIPNECH and tumorlets [17]. In our cohort, these nodules were predominantly located adjacent to, rather than within, bronchiectatic segments. This distribution pattern suggests that the inflammatory process may extend beyond structurally affected airways and influence the surrounding lung parenchyma, potentially through paracrine signaling mechanisms or localized alterations in ventilation. Such a perilesional pattern has not been widely emphasized in previous studies and may represent an early radiologic indicator of neuroendocrine proliferation.

Current surgical indications for bronchiectasis are primarily based on symptom burden, recurrent infection, hemoptysis, and failure of medical therapy [18]. The presence of small pulmonary nodules alone is not typically considered an indication for surgical intervention. However, our findings suggest that in patients with high disease severity and radiologic evidence of nodularity, surgical resection may have an additional role in removing tissue at risk for neuroendocrine proliferation. This concept introduces a potential shift from purely symptom-based surgical decision-making toward a more risk-adapted approach, although prospective validation is required.

In contrast to BSI, systemic inflammatory markers such as NLR, PLR, and SII were not associated with DIPNECH. This finding suggests that local airway inflammation, rather than systemic inflammatory status, may be the dominant factor driving neuroendocrine cell proliferation. It is also possible that single preoperative measurements obtained during clinically stable periods do not adequately capture the cumulative inflammatory burden experienced by these patients. Longitudinal studies incorporating serial measurements may be necessary to better define this relationship.

This study has several limitations. Its retrospective design and single-center setting may limit generalizability. In addition, the inclusion of only surgically treated patients introduces selection bias, as these patients may represent a subgroup with more severe or localized disease. However, surgical specimens provide a unique opportunity for histopathological identification of DIPNECH, which is frequently underdiagnosed in non-surgical populations. The relatively homogeneous nature of the cohort and the integration of clinical, radiologic, and pathological data strengthen the validity of the findings.

Conclusion

The frequency of DIPNECH in patients undergoing surgery for bronchiectasis supports the role of chronic airway inflammation as a driver of pulmonary neuroendocrine cell proliferation. The bronchiectasis severity index and the presence of pulmonary nodules on computed tomography were identified as independent predictors. While surgical treatment in bronchiectasis is traditionally guided by infection control and symptom burden, our results suggest a potential preventive oncologic implication. Patients with severe diseases and radiologic nodules may represent a subgroup at increased risk for preinvasive neuroendocrine lesions, and earlier surgical consideration in selected cases could theoretically limit progression along the neuroendocrine tumor spectrum. Prospective studies are needed to clarify the long-term impact of this strategy.

Abbreviations

BSI – Bronchiectasis Severity Index

CI – Confidence Interval

CRP – C-reactive Protein

CT – Computed Tomography

DIPNECH – Diffuse Idiopathic Pulmonary Neuroendocrine Cell Hyperplasia

FEV1 – Forced Expiratory Volume in 1 Second

NLR – Neutrophil-to-Lymphocyte Ratio

OR – Odds Ratio

PLR – Platelet-to-Lymphocyte Ratio

SD – Standard Deviation

SII – Systemic Immune-Inflammation Index

Declarations

Ethics approval and consent to participate

This study was approved by the Başakşehir Çam and Sakura City Hospital Ethics Committee (Protocol no: 2025-253). The study was conducted in accordance with the principles of the Declaration of Helsinki. Due to the retrospective nature of the study, the requirement for informed consent was waived by the ethics committee.

Clinical trial number

Not applicable.

Conflict of interest

None declared.

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Competing interests

The authors declare that they have no competing interests.

Author contributions: E.E. conceived and designed the study, performed data analysis, and drafted the manuscript. M.K. and A.C.K. contributed to data collection. A.Y. supervised the study. H.A. contributed to data interpretation and critically revised the manuscript. T.D. supervised the study and approved the final version of the manuscript. All authors reviewed and approved the final manuscript.

Data availability: The datasets analyzed during the current study are available from the corresponding author on reasonable request.

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Figures

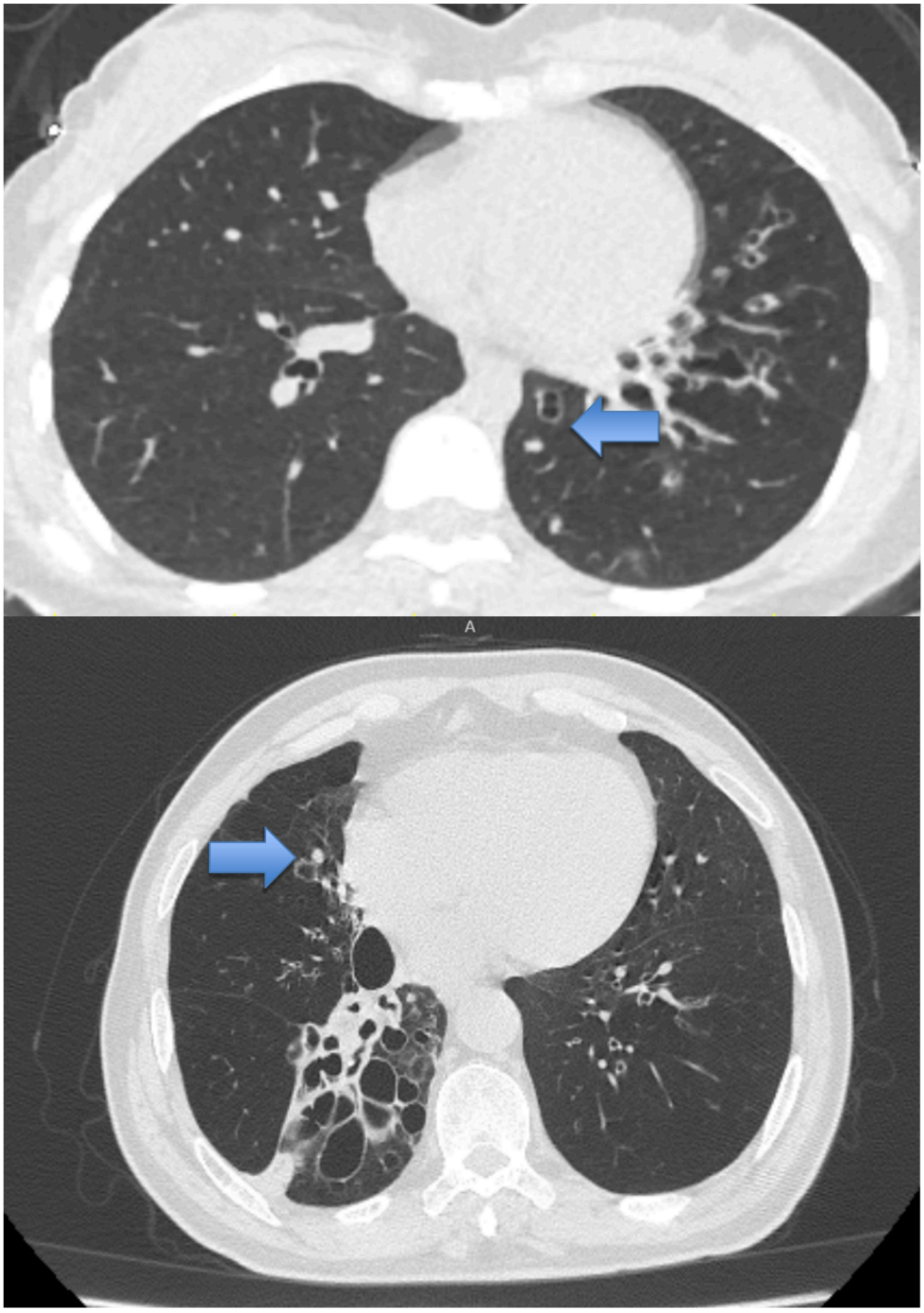


Figure 1

Representative axial chest CT image demonstrating a pulmonary nodule (arrows) adjacent to bronchiectatic airways.