



Hamartoma Mimicking Asthma in A Female Patient: A Case Report with Unusual Clinical, Radiological and Pathological Findings

Kadın Hastada Astımı Taklit Eden Hamartom: Sıradışı Klinik, Radyolojik ve Patolojik Bulguları ile Olgu Sunumu

Yadigar Dila Keş¹, Cigdem Ozdilekcan¹, Deniz Doğan², Hale Kivrak³

Derya Yenibertiz¹

Abstract

Endobronchial hamartomas are rare, but may cause bronchial obstruction, leading to persistent respiratory symptoms and radiological abnormalities such as atelectasis. A 43-year-old female patient with a diagnosis of asthma who had never smoked presented with complaints of cough, sputum and pleuritic chest pain. The initial diagnosis was community-acquired pneumonia, for which antibiotic treatment was administered. Although the patient's complaints regressed, atelectasis was a persistent finding on chest X-ray and so computed tomography (CT) was performed. A hypodense lesion obstructing the left upper lobe bronchus was detected in the thorax CT. An excisional biopsy was obtained during rigid bronchoscopy, the pathology of which confirmed the hamartoma diagnosis. We present the case of a patient with an endobronchial hamartoma and persistent atelectasis, and with treatment-resistant respiratory symptoms mimicking asthma.

Keywords: Endobronchial Hamartoma, Asthma, Bronchoscopy, Electrocoagulation.

Öz

Endobronşiyal hamartomlar ise nadir görülür ve bronş obstrüksiyonuna neden olarak, persistan solunum semptomları ile atelektazi gibi radyolojik anormalliklere yol açabilir. Sigara kullanmamış, astım tanısı olan 43 yaşındaki kadın hasta; öksürük, balgam ve plevral göğüs ağrısı şikâyetleri ile başvurdu. İlk değerlendirmede toplum kökenli pnömoni ön tanısı ile antibiyotik tedavisi başlandı. Hastanın semptomlarında gerileme olmasına rağmen, akciğer grafisinde atelektazi bulgusu devam ettiğinden toraks bilgisayarlı tomografisi (BT) yapıldı. Toraks BT'de, sol üst lob bronşunu tıkayan hipodens bir lezyon saptandı. Rijit bronkoskopi eşliğinde eksizyonel biyopsi gerçekleştirildi ve patolojik inceleme sonucunda hamartom tanısı doğrulandı. Bu olgu sunumunda, kalıcı atelektazi ve persistan solunum semptomları ile astımı taklit eden endobronşiyal hamartom tanılı bir hastayı sunmaktayız.

Anahtar Kelimeler: Endobronşiyal Hamartom, Astım, Bronkoskopi, Elektrokoagülasyon.

¹Department of Chest Diseases, University of Health Sciences, Dr. Abdurrahman Yurtaslan Ankara Oncology Training and Research Hospital, Ankara, Türkiye

²Department of Chest Diseases, University of Health Sciences, Gülhane Training and Research Hospital, Ankara, Türkiye

³Department of Pathology, University of Health Sciences, Gülhane Training and Research Hospital, Ankara, Türkiye

¹Sağlık Bilimleri Üniversitesi Dr. Abdurrahman Yurtaslan Ankara Onkoloji Eğitim Ve Araştırma Hastanesi, Göğüs Hastalıkları Kliniği, Ankara

²Sağlık Bilimleri Üniversitesi, Gülhane Eğitim Ve Araştırma Hastanesi Göğüs Hastalıkları Kliniği, Ankara

³Sağlık Bilimleri Üniversitesi, Gülhane Eğitim Ve Araştırma Hastanesi, Patoloji Kliniği, Ankara

Submitted (Başvuru tarihi): 23.07.2025 **Accepted (Kabul tarihi):** 05.09.2025

Correspondence (İletişim): Yadigar Dila Keş, Department of Chest Diseases, University of Health Sciences, Dr. Abdurrahman Yurtaslan Ankara Oncology Training and Research Hospital, Ankara, Türkiye

e-mail: dilakunya@gmail.com



Pulmonary endobronchial hamartomas are rare, and typically benign tumors. They are mostly asymptomatic and are frequently detected incidentally, but can cause bronchial obstruction and present with symptoms such as chronic cough, sputum production, hemoptysis and dyspnea as they grow in size (1). Histologically, endobronchial hamartomas are composed of fibroelastic tissue, cartilage, fat tissue and epithelial components, and are usually unilateral. Endobronchial hamartomas mostly develop in people aged 50–60 years, and are more common in males. The most common radiologic presentation is a solitary lesion localized in the upper lobe of the left lung. Pulmonary hamartomas are the most common type of benign lung lesion, and endobronchial localization is rare, occurring in 1.4–10% of the reported cases (1,2). Lesions can develop with bronchial obstruction features, and with clinical and radiological findings that can be confused with malignancy. Endobronchial lesions, in particular, can present with such symptoms as persistent lung infections and fever due to airway obstruction. For this reason, endobronchial hamartomas should be kept in mind in the differential diagnosis of pulmonary diseases. Endobronchial hamartomas are diagnosed based on bronchoscopy and biopsy, while imaging methods such as CT and positron emission tomography (PET) can clarify the nature of the lesions and rule out malignancy through metabolic characterization. Early diagnosis is crucial for successful treatment management and appropriate follow-up. The treatment for endobronchial hamartomas is surgical excision, although treatments may vary depending on the size, location and symptoms of the patient. Bronchoscopic methods can be considered as a minimally invasive and effective treatment option, especially for small and accessible lesions. Our case with an endobronchial hamartoma presented with persistent respiratory symptoms mimicking asthma and atelectasis on chest X-ray. We present this less common case of a female patient to the literature, including the diagnostic and management approaches employed and the endobronchial treatment strategy applied. Aside from patient's unusual clinical and radiological presentation, the absence of cartilage, fat and bone tissue in the pathological findings was another notable feature. The patient provid-

ed written informed consent for the publication of this case report.

CASE

A 43-year-old non-smoker female patient presented to our outpatient clinic with complaints of cough, yellow sputum and pleuritic chest pain for the past 20 days, but no fever. She had been prescribed clarithromycin 500 mg at a family health center, which she had taken regularly and had completed the treatment, but her symptoms had not improved, prompting her to seek further evaluation at our pulmonology outpatient clinic. Her medical history included asthma, hypertension and hypothyroidism, and she had been treated for asthma with salmeterol/fluticasone for 4 years without significant objective benefit. Regarding the chest pain and venous thromboembolism risk, she had no recent long travels, immobilization history or hormone therapy use. Her pain was localized to the left anterior chest. A physical examination revealed normal breath sounds, no abnormalities were identified on inspection and a cardiac evaluation was also normal. As can be seen from the flow-volume curve in Figure 1 and in spirometric values in Table 1, FVC (Forced Vital Capacity) increased from 3.14 L (PRE) to 3.51 L (POST) – a >12% improvement that suggested improved lung volume after medication.

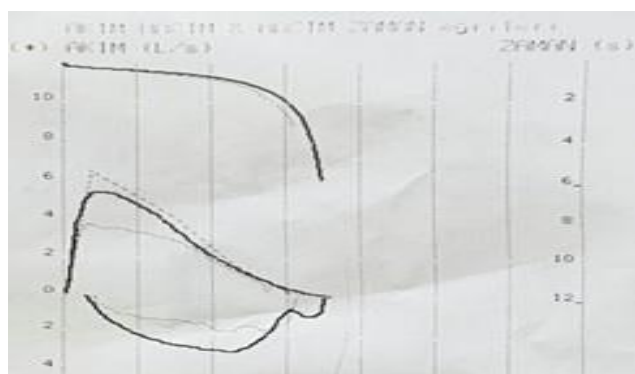


Figure 1: Reversibility spirometry flow – volume curve

FEV1 (Forced Expiratory Volume in 1 second) improved by +9% post-bronchodilator, which is considered negative reversibility as it does not meet the commonly applied threshold of $\geq 12\%$ and 200 mL for significant bronchodilator response. The FEV1/FVC ratio decreased slightly after bronchodilator use (from 78.3% to 76.0%), but remained close to the normal range (normal: typically >70%). Partial post-bronchodilator reversibil-

ity was also noted, especially in FVC. The overall spirometry pattern did not indicate a clear obstruction or restriction, though mild obstruction could not be ruled out depending on clinical context and full PFTs. Among the laboratory test results, C-reactive protein (CRP) was 148 mg/L, and community-acquired pneumonia was initially considered due to the elevated inflammatory markers. The patient, who had previously been taking clarithromycin, was started on a respiratory quinolone monotherapy. A follow-up examination 10 days later revealed a decrease in complaints and inflammatory marker response, while her CRP level had regressed to 5 mg/L. All other routine blood workups were within the normal range.

Table 1: Spirometry values

Parameter	Predicted	Pre (Before Meds)	Post (After Meds)	% Change (Δ)
FVC (L)	3.17	3.14	3.51	+12%
FEV1 (L)	2.72	2.46	2.67	+9%
FEV1/FVC (%)	80.0	78.3	76.0	-3%
PEF (L/s)	6.53	3.74	5.44	+45%
FEF25-75% (L/s)	3.35	2.33	2.82	+21%

A linear band formation was observed extending from the left hilum to the periphery, as well as an area of heterogeneous infiltration in the left apex associated with the atelectasis noted in the patient's postero-anterior chest X-ray (Figure 2). The atelectasis noted during the chest X-ray had persisted, and a Chest CT scan was planned for further evaluation (Figure 3). The CT scan revealed a hypodense 7 mm-diameter lesion within the left upper lobe bronchus (secretion, tumor, foreign body), and a distal nodular atelectasis area measuring approximately 77x37 mm with air-fluid bronchograms. The size of the described area was noted to have increased significantly in a comparison with the previous examination, prompting a bronchoscopic evaluation with a subsequent PET CT correlation, if necessary. A subsequent PET CT scan was requested by radiology for the metabolic characterization of the lung lesion, and fiberoptic bronchoscopy and PET/CT for further imaging were planned. The patient's PET/CT imaging revealed no distinguishable pathological FDG uptake in the millimetric

densification area within the left upper lobe bronchus. Increased FDG uptake (SUVmax: 3.58) was observed in the area of atelectasis and consolidation, extending from the left lung apicoposterior segment to the suprahilar region, adjacent to the fissure and between the mediastinal pleura and costal pleura, where some areas were identified to contain air bronchograms.



Figure 2: Chest radiograph obtained prior to treatment revealing an area of atelectasis with a linear band formation (A). Post-treatment image showing the lack of complete radiological resolution (B)

The left lung findings primarily suggested an inflammatory process; however, it was recommended that these findings be evaluated in conjunction with clinical data for differential diagnosis, along with close follow-up. The bronchoscope was passed through the right nasal cavity into the trachea. The trachea, carina and right airways were inspected up to the segmental bronchi, and were within normal limits. A bright, regular-surfaced, white-colored endobronchial lesion with lobulations was observed at the entrance to the left upper lobe, completely obliterating it, and so a punch biopsy was performed to obtain lavage from the area. The collected samples were sent for cytological and pathological examination. The endobronchial mass was observed to shrink during the punch biopsy procedure, and the obliterated bronchus was slightly opened. The patient tolerated the procedure well, with oxygen saturation remaining above 93% throughout the procedure. He was hemodynamically stable and experienced no complications (Figure 4). The pathological analysis of the punch biopsy revealed "findings of chronic inflammation, with blood, fibrin, and bronchial epithelial cells observed in a mucous substrate in the cell block".

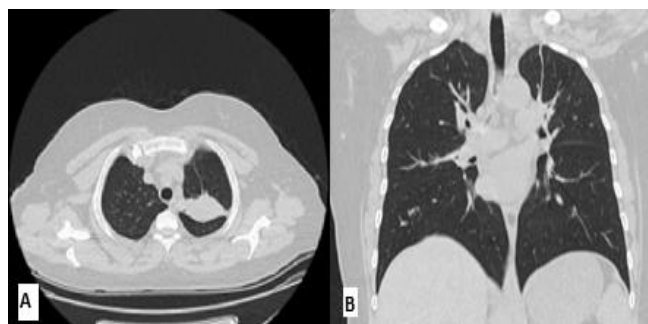


Figure 3: Chest-lung window (A) and chest-mediastinal window (B) showing post-obstructive atelectasis secondary to endobronchial obstruction

Due to the lack of diagnostic value of the biopsy obtained via flexible bronchoscopy, an excisional biopsy of the endobronchial lesion obliterating the bronchus was planned, for which the patient was referred to an interventional pulmonology center. The endobronchial lesion was excised via cryotherapy and was identified as a hamartoma in the pathology report. The biopsy specimen revealed a polypoid mass (A, X2) with spindle cells in the fibrotic and edematous stroma without a myxoid component, and the surface was lined with benign airway epithelium (B, X20). Immunohistochemically, the spindle cells expressed SMA (C, X10) and Desmin (D, X10). No necrosis or increased mitotic activities were observed; immunohistochemically S100, PanCK and STAT6 were negative, and no loss of expression was observed with Rb (Figure 5).

DISCUSSION

A comprehensive review of endobronchial hamartoma cases reported between 2010 and 2025 was made for the case report, the relevant details of which are presented in Table 2, with references provided in the first column (3–16). Hamartomas of the respiratory tract are rare benign lesions that are primarily composed of mature mesenchymal tissue such as cartilage, adipose tissue and smooth muscle. They are usually clinically silent and discovered incidentally (14). In a series assessed by Van den Bosch et al. (17), segmental atelectasis was the most frequently observed radiological finding, in alignment with the segmental atelectasis of the upper lobe that was predominant on computed tomography in the presented case. Pulmonary hamartoma were first described in 1904 by German pathologist Eugen Albrecht (15). They are generally composed of mature mesenchymal tissue commonly

found in the lung, but without the preservation of the normal architecture (18). The histological makeup of these tumors usually includes a mix of mesenchymal tissue, such as adipose tissue, cartilage, bone or smooth muscle bundles, as well as fibromyxoid tissue, in varying proportions. They are noninvasive, slow-growing, nodular lesions that sometimes feature cleft-like spaces lined with respiratory epithelium (19). Unlike in previously reported cases, no cartilage, fat or bone tissue was observed in our case, while scattered smooth muscle cells without fascicular organization were observed in the vascularized fibrotic stroma. Small hamartomas typically do not require intervention but only follow-up for growth. When larger, however, they may obstruct the bronchial lumen, leading to symptoms such as cough, or pleuritic chest pain, as observed in our patient. Our case underlines the importance of differential diagnosis when evaluating endobronchial lesions, especially in patients with atypical presentations. This case report presents a rare case of endobronchial hamartoma with asthma-like symptoms to the literature, and discusses the diagnostic and therapeutic approaches followed. Our patient was 43 years old, which was not significantly different from the mean age reported in the cases summarized in Table 2. The fact that the patient was female is particularly noteworthy, given the predominance of males featured in earlier studies. Of the reported cases detailed in Table 2, eight were identified as non-smokers (4,5,7,8,11,13,14) and five were current or former smokers (1,9,10,15,16), while the smoking status of the remaining two cases was not reported (3,6). Consistent with the majority of these cases, our patient was a non-smoker, and her symptoms were strikingly similar to those usually associated with asthma, which led to a misdiagnosis and the initiation of asthma treatment.



Figure 4: Bright polypoid lesion obliterating the left main bronchus

Previous studies have reported cases with symptoms such as cough and dyspnea, similar to our case; however, in the cases described by Habib et al. (15) and Minalyan et al. (9) in 2024, hemoptysis was also reported in addition to cough. Our evaluation of the bronchoscopic findings in the presented case revealed a lesion that was completely obliterating the left upper lobe. In the cases summarized in Table 2, the reported endobronchial hamartomas had various localizations, including the upper lobes in six cases (5,7,9,10,11,13), the lower lobes in two cases (7,15), the main bronchi in three cases (3,4,16), the intermediate bronchus in one case (12) and the trachea in one case (14). Our case had left upper lobe involvement, consistent with the most commonly reported sites. Histopathological evaluation is crucial for a definitive diagnosis. In our case, standard bronchoscopic procedures failed to yield sufficient histopathological data to support our preliminary differential diagnoses of lipoma, hamartoma, carcinoid tumor and malignancy, and so the patient underwent a rigid bronchoscopy for both diagnostic and therapeutic purposes. The endobronchial lesion was excised via rigid bronchoscopy, which allowed for both confirmation of the diagnosis and initiation of appropriate treatment. The histological appearance of such tumors is generally a mixture of mature mesenchymal tissue, including adipose tissue, cartilage, bone and smooth muscle bundles, and fibromyxoid tissue, in varying proportions. Atypically, no cartilage, fat or bone tissue was observed in our case; instead, scattered smooth muscle cells without fascicular organization in vascularized fibrotic stroma.

Our review of the literature revealed that similar diagnostic bronchoscopies were often followed by rigid bronchoscopies for differential diagnosis and treatment purposes, and then by cryotherapy, argon plasma coagulation, electrocautery and laser resection procedures. In most reported cases, definitive diagnosis was obtained following resection. In our case, the team applied electrocautery in combination with a snare probe for lesion removal, the patient underwent fiberoptic bronchoscopy (FOB). Among the preliminary differential diagnoses for the observed smooth-surfaced endobronchial lesion was carcinoid tumor, and so a bronchoscopic biopsy was performed with great caution, and after confirming the absence of bleeding with the first sample, the

procedure continued safely. As our center lacked the necessary facilities for histopathological verification, the patient was referred to a specialized endobronchial treatment center for definitive diagnosis and treatment. Although rigid bronchoscopy may be considered preferable for lesions with a high risk of bleeding, our approach was guided by a clinical assessment and the available resources, and the procedure was completed without complications. The main differential diagnoses are other benign lesions, such as bronchial tuberculosis, bronchial lipoma, leiomyoma, fibroma, chondroma and neurogenic tumors, although malignant airway tumors such as a carcinoid tumor and bronchial metastasis, should also be kept in mind (12). This case highlights the importance deeper diagnostic evaluation in patients who do not exhibit objective or subjective improvement during treatment, as pulmonary endobronchial hamartomas can be clinically misdiagnosed as other conditions. In the early stages, both benign and malignant endobronchial tumors may have similar signs and symptoms, which can be misdiagnosed as asthma, COPD or pulmonary infection. Most commonly, patients seek treatment for cough and hemoptysis, chest pain, dyspnea, localized wheezing, recurrent pneumonia or atelectasis due to bronchial obstruction. In the absence of airway obstruction, the patient may be asymptomatic. This case emphasizes the need of further investigation in patients who do not show objective or subjective benefit during treatment, as pulmonary endobronchial hamartomas may be confused with other pathologies. If the tumor is in the lumen of the trachea or bronchi, the patient's complaints may mimic respiratory diseases such as bronchial asthma, chronic obstructive pulmonary disease or recurrent pneumonia.

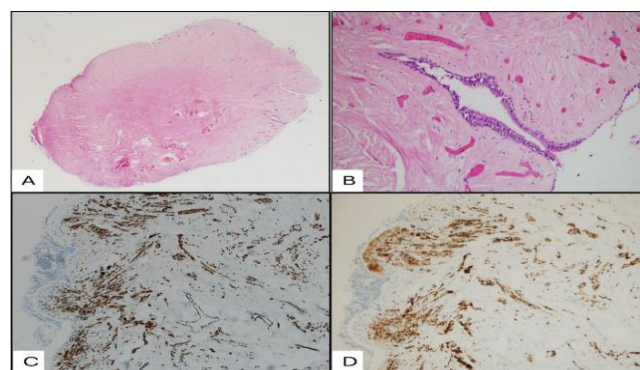


Figure 5: Histopathological examination ((A) H&E, x2, (B) H&E, x20, (C) SMA, x10, (D) Desmin, x10))

Our resected endobronchial lesion measured 12 × 11 mm in diameter, and lobulation was observed. As can be seen from the summary of earlier cases in the literature presented in Table 2, lesion sizes ranged from 10 × 7 × 5 mm (16) to 40 × 20 × 10 mm (11). Our lesion, therefore, can be considered of average size among the other reported cases. The larger reported lesions in the summary were generally managed with surgical resection. Endobronchial hamartomas are generally associated with a favorable prognosis; however, recurrence rates of approximately 10% following bronchoscopic resection have been documented (20). There is currently a lack of consensus on the optimal surveillance interval or follow-up duration. Notably, the malignant transformation of such lesions has rarely been observed on long-term follow-up. Our case remains asymptomatic and her clinical, radiological and spirometric follow-up is continuing, with no evidence of relapse observed to date. However, serial computed tomography (CT) imaging will be used for postoperative follow-up to monitor for possible recurrence annually. The patient's asthma-like symptoms resolved and radiological improvement was confirmed on CT imaging (Figure 6), indicating a marked regression in the atelectasis and obstruction findings; consequently, inhaler therapy was discontinued. The patient will

continue to be monitored for potential future relapses. One limitation of this report is the relatively short follow-up period, which prevents the assessment of long-term recurrence. Furthermore, while the diagnosis of hamartoma was made based on a histopathological analysis, the absence of cartilage and adipose tissue may pose diagnostic challenges in similar cases in the future. Finally, a more comprehensive account of the patient's long-term functional and radiological outcomes would enhance the clinical significance of this case.



Figure 6: CT image demonstrating marked regression of atelectasis following the removal of the lesion through electrocautery in combination with a snare probe

Table 2: The Review and The Summary of Cases between the Years 2010- 2025

Year	Author	Age	Gender	Smoking	Symptoms	Diameters of Lesion	Location of Lesion	Treatment	Relapse
2010	Rai et al. (3)	40	Male	Unknown	Cough, Fever, Anorexia	Unknown	Left Main Bronchus	Diode Laser	No
2011	Mon-dello et al. (4)	65	Male	Non-Smoker	Asymptomatic	Unknown	Left Main Bronchus	Electrosurgical Snaring	No
2012	Guarino et al.(5)	35	Female	Non-Smoker	Dyspnea, Cough, Chest Pain	Unknown	Left Upper Obe	Fenestrated Crocodile Biopsy Forceps	No
2013	Ga-yathri et al.(6)	65	Male	Unknown	Cough	Unknown	Right Upper Lobe	Conservative Follow Up	No
2013	Seğmen et al. (7)	59	Male	Non-Smoker	Cough and Dyspnea	30 Mm	Left Lower Lobe	APC and Electrocautery	No
2015	Mertoğlu et al. (8)	45	Male	Non-Smoker	Cough, Fever	Unknown	Right Main Bronchus	APC and Electrocautery	No
2017	Ahmed et al. (1)	53	Male	Former Smoker	Cough, Dizziness	Unknown	Left Upper Lobe	Laser Resection	Yes
2019	Mi-nalyan et al. (9)	49	Male	Former Smoker	Fever, Cough, Hemoptysis	14mm	Left Upper Lobe	Forceps Debulking and Cauterization	No
2022	Suzuki et al(10)	51	Male	5 Pack/Year	Asymptomatic	10 Mm	Right Main Bronchus	High-Frequency Electrocautery	No

Table 2: Continued

2023	Fernandez-Trujillo et al. (11)	44	Female	Non-Smoker	Cough, Expectoration	40x20x10mm	Right Main Bronchus	Cryotherapy	No
2024	Bouanani et al. (12)	57	Male	Former Smoker	Cough	Unknown	Right Intermediate Bronchus	Surgical Resection	No
2024	Takigawa et al. (13)	82	Male	Non-Smoker	Dyspnea	40x20 Mm	Left Upper Lobe	Cryotherapy	Unknown
2024	Kazıróđ et al. (14)	53	Male	Non-Smoker	Dyspnea, Wheezing	18 Mm	Trachea	Endoscopic Electroresection	No
2024	Habib et al. (15)	71	Male	20 Pack/Year	Cough, Hemoptysis	13x4 Mm	Right Middle Lobe Segmental Bronchus	Cryotherapy and APC	Unknown
2025	Daboussi et al. (16)	64	Male	Former Smoker	Cough	10x7x5mm	Left Main Bronchus	Segmentectomy	No

CONCLUSION

This case highlights the importance of deeper diagnostic evaluation in patients who do not exhibit objective or subjective improvement during treatment, as pulmonary endobronchial hamartomas can be clinically misdiagnosed as other conditions. Patients with undiagnosed dyspnea should be further evaluated using CT imaging and bronchoscopy. Endobronchial hamartomas can be treated effectively and safely using interventional bronchoscopic methods. After a complete resection, additional therapeutic options (such as cryotherapy) applied to the root of the lesions may help prevent recurrence. Successful results have been achieved with endobronchial resection approaches both in our case and in the cases reviewed in Table 2. All patients should be followed up for recurrent disease.

CONFLICTS OF INTEREST

None declared.

AUTHOR CONTRIBUTIONS

Concept - Y.D.K., Ç.Ö., D.Y.; Planning and Design - Y.D.K., Ç.Ö.; Supervision - Y.D.K., Ç.Ö.; Funding - Y.D.K., Ç.Ö., D.D., H.K.; Materials - Y.D.K., Ç.Ö., D.D., H.K.; Data Collection and/or Processing - Y.D.K.; Analysis and/or Interpretation - Y.D.K., Ç.Ö.; Literature

Review - Y.D.K., Ç.Ö.; Writing - Y.D.K.; Critical Review - Y.D.K., Ç.Ö.

REFERENCES

1. Ahmed S, Arshad A, Mador MJ. Endobronchial Hamartoma; A Rare Structural Cause of Chronic Cough. *Respir Med Case Rep* 2017; 22:224-7. [CrossRef]
2. Gong Y, Lu L, Long F, Yang H, Chen X, Li S. A Case of COPD with Left Endobronchial Hamartoma by Bronchoscopic Intervention. *Case Rep Clin Med* 2018; 7:550. [CrossRef]
3. Rai SP, Patil AP, Saxena P, Kaur A. Laser Resection of Endobronchial Hamartoma Via Fiberoptic Bronchoscopy. *Lung India* 2010; 27:170-2. [CrossRef]
4. Mondello B, Lentini S, Buda C, Monaco F, Familiari D, Sibilio M, et al. Giant Endobronchial Hamartoma Resected by Fiberoptic Bronchoscopy Electrosurgical Snaring. *J Cardiothorac Surg* 2011; 6:97. [CrossRef]
5. Guarino C, Cesaro C, La Cerra G, Lucci R, Cesaro F, Zamparelli E, et al. Endobronchial Hamartoma in a Young COVID-19 Symptomatic Woman. Radical Endoscopic Treatment with a Disposable Bronchoscope. *Case Report. Monaldi Arch Chest Dis* 2021; 92(3). [CrossRef]

6. Gayathri Devi H. Lipomatous Hamartoma: A Rare Cause For Endobronchial Obstruction. *J Pulmon Resp Med* 2013; 14:007. [CrossRef]
7. Seğmen F, Aktaş Z, Yılmaz A, Özadin E, Kaya A. Girişimsel Bronkoskopi ile Tedavi Edilen Endobronşiyal Hamartom Olgusu. *Tuberk Toraks* 2013; 61:348-50. Re
8. Mertoğlu A, Tellioglu E, Yücel N. Multiple Endobronchial Hamartoma. *Clin Respir J* 2017; 11:263-6. [CrossRef]
9. Minalyan A, Gopisetti N, Estepa A, Grover H, Patel R, Patel RK. Endobronchial Hamartoma as a Rare Cause of Recurrent Respiratory Symptoms: Case Report and Literature Review. *Cureus*. 2019; 11:e5489. [CrossRef]
10. Suzuki M, Watanabe H, Hashimoto M, Ishii S, Naka G, Ikura M, et al. Endobronchial Hamartoma Resected via Bronchoscopy using High-Frequency Electrosurgical Snare-Preoperative Strategies using Virtual Bronchoscopy. *Radiology Case Reports* 2022; 17:4232-8.
11. Fernandez-Trujillo L, Castrillón AI, Morales EI, Diaz Y, Sua LF. Severe Central Airway Obstruction Secondary to a Giant Endobronchial Hamartoma: A Case Report. *J Investig Med High Impact Case Rep* 2023; 11:23247096231158951. [CrossRef]
12. Bouanani Z, Raïs A, Benbrahim FZ, Akammar A, Bouardi NE, Haloua M, et al. A Rare Cause of Bronchial Obstruction: Endobronchial Hamartoma Case Report. *Radiol Case Rep* 2024; 19:3473-7. [CrossRef]
13. Takigawa Y, Sato K, Inoue T, Takada M, Fujiwara K. Image of a Thawed Frozen Specimen Obtained Using a Cryoprobe Floated with Oil Droplets in Normal Saline: An Endobronchial Lipomatous Hamartoma Image. *Respirol Case Rep* 2024; 12:e01318. [CrossRef]
14. Kaziród T, Tokarski S, Kaznowska E, Rzeszutko-Grabowska M. Benign Endotracheal Tumor (Hamartoma) Mimicking Bronchial Asthma. *Eur J Clin Exp Med* 2024; 22:702-7. [CrossRef]
15. Habib DS, Jani P, Zhao B, Jayaraman EA, Kesavan R. Pulmonary Endobronchial Hamartoma Presenting With Post-obstructive Pneumonia. *Cureus* 2024; 16:e60916. [CrossRef]
16. Daboussi S, Saidane A, Syrine A, Mhamdi S, Gargouri F, Messaoudi H, et al. Endobronchial Hamartomas as a Rare Cause of Chronic Cough. *Respir Med Case Rep* 2025; 55:102210. [CrossRef]
17. Van den Bosch J, Wagenaar SS, Corrin B, Elbers J, Knaepen P, Westermann C. Mesenchymoma of the Lung (so called hamartoma): A Review of 154 Parenchymal and Endobronchial Cases. *Thorax* 1987; 42:790-3. [CrossRef]
18. Matos R, Carvalho L. Pulmonary Hamartoma. *Acta Med Port* 2002; 15:165-8.
19. Saadi MM, Barakeh DH, Husain S, Hajjar WM. Large Multicystic Pulmonary Chondroid Hamartoma in a Child Presenting as Pneumothorax. *Saudi Med J* 2015; 36:487. [CrossRef]
20. Chen S-S, Zhou H, Tong B, Yu L-L, Fan S-S, Xiao Z-K. Endobronchial Hamartoma Mimicking Malignant Lung Tumor Contralateral Endobronchial Metastasis: A Case Report. *Medicine (Baltimore)* 2017; 96:e9085.[CrossRef]