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REVIEW ARTICLE

Pulmonary Carcinoid Tumors as an Asthma Mimic: Diagnostic Challenges, Clinical Presentation, and Management Strategies

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ABSTRACT

BACKGROUND:
Asthma is a common chronic respiratory disease; however, some patients continue to have symptoms despite appropriate guideline-directed therapy. Pulmonary carcinoids are rare, well-differentiated neuroendocrine neoplasms that may cause cough, wheeze, dyspnea, hemoptysis, and recurrent infection through focal airway obstruction, thereby mimicking asthma. This review summarizes the pathophysiology, epidemiology, clinical presentation, diagnostic evaluation, and management of pulmonary carcinoid tumors presenting as an asthma mimic and highlights clinical features that should prompt reconsideration of an asthma diagnosis.

METHODS:
This is a narrative, non-systematic review. A structured search and screening process was used specifically to identify the case-level literature summarized in Table 1, but this does not constitute a systematic review: only one database was searched, no protocol was registered or pre-published, and findings are synthesized narratively rather than through meta-analysis. The search strategy used combinations of the terms "pulmonary carcinoid," "bronchial carcinoid," "asthma mimic," "endobronchial tumor," "refractory asthma," and "unilateral wheezing" to identify 14 English-language case-level reports of carcinoid tumors presenting with or mimicking asthma; seven of these are included in the present review (Table 1), while seven others were excluded (four for an irrelevant clinical scenario, two for an alternative diagnosis of adenoid cystic carcinoma, and one for carcinoid syndrome without asthma-like features in a patient with COPD). Inclusion/exclusion followed a priori eligibility criteria established prospectively, before screening began. Screening was performed by a single reviewer, with a second-pass re-screening of all records by the same reviewer to check consistency, since no second, independent screener was available; there were no discrepancies between the two passes. A PRISMA flow diagram was not produced, consistent with the narrative, non-systematic design of this review. The included case reports were assessed using four of the CARE reporting domains (demographics, work-up, treatment, outcome/follow-up) as a means of appraisal of reporting quality rather than formal risk-of-bias assessment; results of this informal appraisal are described rather than pooled. Summary: Pulmonary carcinoid tumors are usually indolent neuroendocrine neoplasms, but centrally located lesions can narrow a major airway and cause wheeze, cough, dyspnea, hemoptysis, and post-obstructive infection. Because this presentation can resemble asthma, diagnosis may be delayed despite escalating inhaled therapy or consideration of biologic treatment. Features that should raise suspicion for a focal endobronchial lesion include localized or unilateral wheeze, adult-onset or atypical symptoms, poor or incomplete response to appropriate asthma therapy, recurrent infection in the same lung segment, hemoptysis, asymmetric breath sounds, or unexpected imaging findings. Chest CT and bronchoscopy, followed by histopathology and neuroendocrine immunohistochemistry when tissue sampling is appropriate, are central to diagnosis and to distinguishing typical from atypical carcinoids. Complete surgical resection remains the preferred treatment for most localized, resectable pulmonary carcinoid tumors. Bronchoscopic/endoscopic treatments, including Nd:YAG laser photoresection, electrocautery or snare resection, cryotherapy, and rigid bronchoscopic debulking, may be considered as parenchyma-sparing approaches in carefully selected patients with strictly intraluminal typical carcinoids; available evidence is largely retrospective and direct comparative evidence against surgery remains limited. For unresectable or metastatic disease, treatment may include somatostatin analogues, peptide receptor radionuclide therapy, targeted therapy, and other individualized systemic options.

CONCLUSION:
Pulmonary carcinoid should be considered in patients with persistent or atypical asthma-like symptoms that do not respond as expected to guideline-directed therapy. Early recognition permits timely CT imaging, bronchoscopic evaluation, histopathologic confirmation, and

Abstract (continued)

definitive management; surgical resection remains standard for most localized resectable tumors, while selected strictly intraluminal typical carcinoids may be considered for bronchoscopic treatment.

KEYWORDS

Pulmonary carcinoid tumor; asthma mimic; refractory asthma; airway obstruction; neuroendocrine tumor; bronchoscopy; wheezing; differential diagnosis

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Syeda Alizay Fatima conceived the review, performed the literature search and screening, analyzed and interpreted the literature, prepared the tables and figures, drafted the manuscript, critically revised it for intellectual content, and approved the final version for submission.

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Pulmonary Carcinoid Tumors as an Asthma Mimic: Diagnostic Challenges, Clinical Presentation, and Management Strategies

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Summary: Pulmonary carcinoid tumors are usually indolent neuroendocrine neoplasms, but centrally located lesions can narrow a major airway and cause wheeze, cough, dyspnea, hemoptysis, and post-obstructive infection. Because this presentation can resemble asthma, diagnosis may be delayed despite escalating inhaled therapy or consideration of biologic treatment. Features that should raise suspicion for a focal endobronchial lesion include localized or unilateral wheeze, adult-onset or atypical symptoms, poor or incomplete response to appropriate asthma therapy, recurrent infection in the same lung segment, hemoptysis, asymmetric breath sounds, or unexpected imaging findings. Chest CT and bronchoscopy, followed by histopathology and neuroendocrine immunohistochemistry when tissue sampling is appropriate, are central to diagnosis and to distinguishing typical from atypical carcinoids.

Complete surgical resection remains the preferred treatment for most localized, resectable pulmonary carcinoid tumors. Bronchoscopic/endoscopic treatments, including Nd:YAG laser photoresection, electrocautery or snare resection, cryotherapy, and rigid bronchoscopic debulking, may be considered as parenchyma-sparing approaches in carefully selected patients with strictly intraluminal typical carcinoids; available evidence is largely retrospective and direct comparative evidence against surgery remains limited. For unresectable or metastatic disease, treatment may include somatostatin analogues, peptide receptor radionuclide therapy, targeted therapy, and other individualized systemic options.

Conclusion: Pulmonary carcinoid should be considered in patients with persistent or atypical asthma-like symptoms that do not respond as expected to guideline-directed therapy. Early recognition permits timely CT imaging, bronchoscopic evaluation, histopathologic confirmation, and definitive management; surgical resection remains standard for most localized resectable tumors, while selected strictly intraluminal typical carcinoids may be considered for bronchoscopic treatment.

Keywords

Pulmonary carcinoid tumor; asthma mimic; refractory asthma; airway obstruction; neuroendocrine tumor; bronchoscopy; wheezing; differential diagnosis

Introduction

Asthma is highly prevalent and imposes a substantial burden on patients and healthcare systems. Management is based on inhaled corticosteroid-containing therapy, with additional controller treatments and biologic agents reserved for appropriately selected patients with difficult-to-treat or severe disease [7,8]. Before escalating therapy, particularly to biologics, clinicians should confirm the diagnosis of asthma and evaluate alternative causes of persistent respiratory symptoms [7,8].

Pulmonary carcinoid tumors are well-differentiated pulmonary neuroendocrine neoplasms classified as typical or atypical carcinoids. Although they generally have a more favorable prognosis than high-grade pulmonary neuroendocrine carcinomas, centrally located tumors may produce a wide range of respiratory manifestations and can be diagnostically challenging. Persistent asthma-like symptoms may reflect uncontrolled asthma, an incorrect diagnosis, comorbid disease, inadequate treatment, or an alternative process such as

inducible laryngeal obstruction, bronchiectasis, COPD, foreign-body aspiration, or an endobronchial tumor.

Pulmonary carcinoid tumors may cause cough, wheezing, dyspnea, and recurrent pneumonia through partial or complete obstruction of a major airway. This review summarizes mechanisms underlying asthma-like presentations, identifies practical clinical clues that should prompt investigation for a focal endobronchial lesion, and reviews diagnostic and management strategies for pulmonary carcinoid tumors.

For this review, two clinically distinct scenarios should be separated. In a true asthma mimic, asthma-like symptoms arise from focal mechanical airway obstruction caused by the tumor and may improve substantially after the obstruction is relieved. In contrast, a carcinoid tumor may coexist with objectively confirmed asthma, in which case inflammatory airway symptoms can persist after tumor treatment. This distinction matters because asthma-directed anti-inflammatory therapy and biologic agents do not correct fixed mechanical obstruction from an endobronchial mass.

Prior reports on this topic have primarily emphasized diagnosis and surgery, with less attention to the accumulating literature supporting bronchoscopic (endoscopic) treatment as a parenchyma-sparing option in appropriately selected patients. The current review extends the existing literature by (1) synthesizing relevant red flags that help differentiate carcinoid tumor from asthma in a single table (Table 2), (2) presenting an algorithm that integrates these features into a logical diagnostic and therapeutic work-up (Figure 3), and (3) emphasizing that bronchoscopic treatment may be considered alongside surgical resection as a therapeutic option, an issue not previously addressed in asthma-mimic reviews.

Review Methods

Search Strategy and Scope

A structured PubMed search was used specifically to identify the case-level literature summarized in Table 1. No publication-year restriction was applied; the reports identified for this case-level synthesis were published between 1993 and 2020. Search terms included combinations of “pulmonary carcinoid,” “bronchial carcinoid,” “asthma mimic,” “endobronchial tumor,” “refractory asthma,” and “unilateral wheezing,” and the search was limited to English-language reports. One reviewer screened all identified records against prospectively defined eligibility criteria (see Eligibility Criteria). Fourteen case-level reports, case series, or case reviews were identified; seven were included in Table 1 and seven were excluded (four for a clinically irrelevant scenario, two for adenoid cystic carcinoma of the trachea, and one for carcinoid syndrome without asthma-like features in a patient with COPD). Additional references used for epidemiology, diagnosis, and management were selected narratively. No protocol was registered, no formal systematic risk-of-bias assessment was performed, and no quantitative synthesis was undertaken. The search and screening process should therefore be interpreted as a transparent component of a narrative review rather than as a systematic review.

Eligibility Criteria

Eligible records were those that described patients with (a) a carcinoid tumor located within the pulmonary, bronchial, or tracheal region; (b) accompanying respiratory symptoms (wheeze, cough, dyspnea, and/or recurrent infection) diagnosed as, or clinically suspected to represent, asthma; and (c) a sufficient case description. Records were excluded if the described patients had a non-carcinoid endobronchial tumor, carcinoid syndrome without

asthma-like features, non-English language, or abstract/conference presentations without a case description. The above criteria were generated prospectively, prior to commencing with the screening process.

Second-Pass Screening Consistency Check

Because no second independent screener was available, the same reviewer re-screened all 14 records in a second pass using the same prespecified eligibility criteria; classifications were identical on both passes. This repeat-screening approach provides an internal consistency check but cannot replace independent screening by a second reviewer and remains susceptible to selection bias.

Pathophysiology: Mechanisms Underlying Asthma-Like Symptoms

Pulmonary carcinoid tumors are relatively indolent, well-differentiated neuroendocrine tumors with less aggressive behavior than high-grade pulmonary neuroendocrine carcinomas. According to the World Health Organization (WHO) classification of pulmonary neuroendocrine tumors, last updated in 2021, pulmonary carcinoids are divided into typical and atypical carcinoids based on mitotic counts and the presence or absence of necrosis: while typical carcinoids have less than 2 mitoses per 2 mm² and no necrosis, atypical carcinoids have 2–10 mitoses per 2 mm² and/or foci of necrosis; the latter also exhibit a higher likelihood of lymph node infiltration, recurrence, and metastasis [6,31].

The principal mechanism by which an endobronchial carcinoid can mimic asthma is focal mechanical narrowing of a major airway. Wheeze caused by a central lesion is often localized or unilateral, whereas asthma more typically produces diffuse, bilateral expiratory wheeze [9,10]. Reduced breath sounds on the affected side and recurrent infection distal to the same obstructed bronchus can provide additional clues. Importantly, unilateral wheeze

is not specific to carcinoid; foreign bodies, mucus plugging, airway strictures, and other endobronchial masses can produce the same finding. Accordingly, persistent localized wheeze should prompt investigation for a focal structural lesion rather than be interpreted as a tumor-specific sign [1-3].

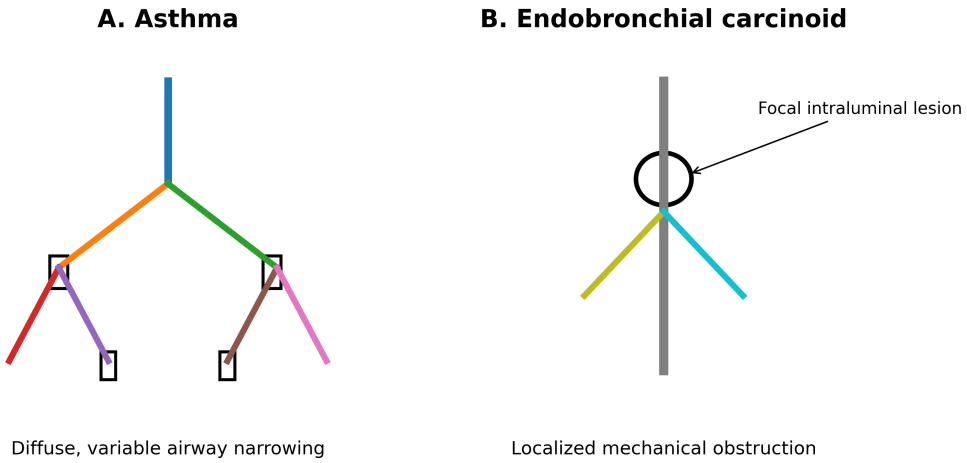

Figure 1. *Schematic comparison of diffuse airway involvement in asthma (A) versus focal, unilateral endobronchial obstruction caused by a centrally located carcinoid tumor.*

Because tumor-related airway obstruction is structural rather than purely bronchospastic, symptoms may respond poorly or incompletely to bronchodilator therapy; however, partial symptomatic improvement does not exclude an endobronchial carcinoid. Improvement can also occur after bronchoscopic or surgical removal of the obstructing lesion, independent of any effect on airway inflammation.

In addition, pulmonary carcinoid tumors can occasionally release serotonin and other bioactive mediators that contribute to bronchoconstriction; however, classic carcinoid syndrome is uncommon in pulmonary carcinoid tumors. These mediators can cause bronchospasm, flushing, diarrhea, or dyspnea and can exacerbate asthma symptoms when

released [12]. Thus, because bronchial stenosis, superimposed infection, and release of mediators can each cause wheezing that is partially responsive to bronchodilators, carcinoid tumors cannot be reliably distinguished from asthma on the basis of clinical response to therapy [4].

Epidemiology of Pulmonary Carcinoid Tumors

Pulmonary carcinoid tumors are uncommon neoplasms that account for approximately 1-2% of primary lung cancers and 20-30% of pulmonary neuroendocrine tumors in commonly cited series [5]. Their reported incidence has increased over time, likely in part because of greater use of cross-sectional imaging and bronchoscopy [5]. Compared with many other primary lung cancers, pulmonary carcinoids occur in relatively younger patients and have a weaker association with cigarette smoking; absence of a smoking history should therefore not lower clinical suspicion when other features suggest an endobronchial lesion [5,13].

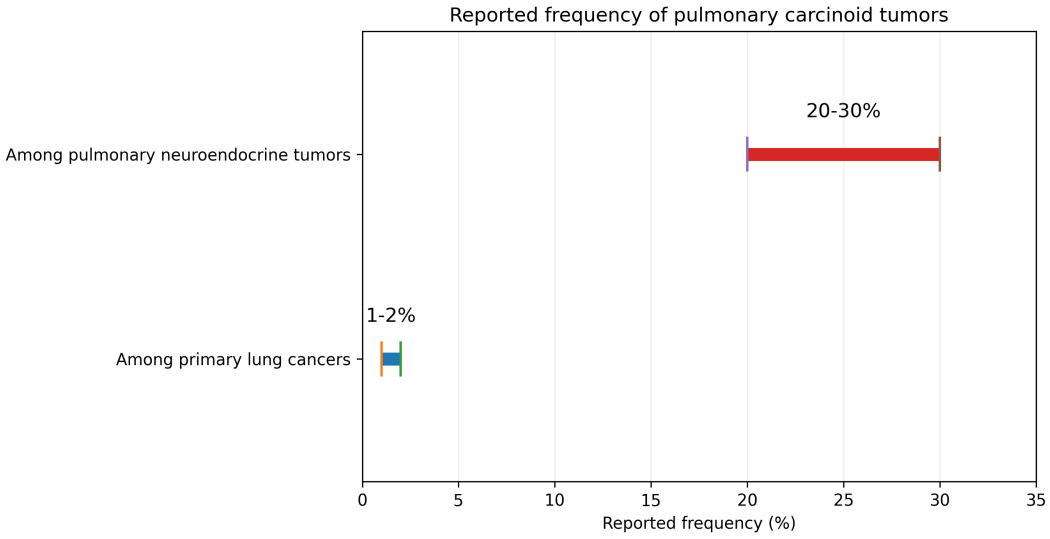

Figure 2. *Reported frequency of pulmonary carcinoid tumors (A) among all primary lung cancers and (B) among pulmonary neuroendocrine tumors.*

Typical carcinoids are generally indolent tumors with low mitotic activity, absent necrosis, and a lower frequency of lymph-node involvement than atypical carcinoids. In contrast, atypical carcinoids are associated with increased risk of lymph node metastases, tumor recurrence, and distant spread of the disease; therefore, correct histopathological diagnosis is critical [14].

Pulmonary carcinoids are often diagnosed in mid-adulthood but can occur across a broad age range. Their indolent growth and central airway location can lead to prolonged treatment for presumed asthma or recurrent pneumonia before endoscopic and pathologic evaluation establishes the diagnosis. More recent case literature continues to illustrate this diagnostic pattern [11].

Published Cases Demonstrating Pulmonary Carcinoid Tumors as an Asthma Mimic

Endobronchial carcinoids may cause cough, wheeze, dyspnea, recurrent infection, and airflow limitation through bronchial obstruction. Published reports also show that a carcinoid tumor and true asthma can coexist, further complicating interpretation of persistent respiratory symptoms.

Table 1. Summary of published cases in which pulmonary carcinoid tumors mimicked asthma

Report	Presentation	Outcome

Report	Presentation	Outcome
Aydin et al. [20]	48-year-old woman with a 12-year asthma history and persistent wheeze/dyspnea despite high-dose ICS/LABA, montelukast, and systemic corticosteroids; being considered for anti-IgE therapy	Bronchoscopy and CT revealed a left lower lobe endobronchial mass; typical carcinoid confirmed on biopsy; symptoms resolved after left lower lobectomy, although the patient later had a separate acute asthma exacerbation
Becher et al. [2]	Unilateral wheezing as the presenting and dominant clinical sign	Bronchoscopy identified an endobronchial carcinoid tumor as the cause of localized wheeze
Santra et al. [21]	Refractory dyspnea and wheeze initially managed as asthma	Bronchial carcinoid identified after failure to respond to asthma therapy prompted further investigation
Biçer et al. [22]	Uncontrolled severe asthma with a coexisting carcinoid tumor, presenting with pneumomediastinum	Illustrates that carcinoid tumor and true asthma can coexist, complicating the clinical picture
Davila et al. [23]	Case series/review describing bronchial carcinoid presentations including cough, wheeze, hemoptysis, recurrent infection, and dyspnea	Reinforces that this overlapping symptom pattern should prompt consideration of bronchial carcinoid in the differential diagnosis
Emeryk et al. [24]	39-year-old man with 10-11 months of persistent wheeze, cough, and hemoptysis unresponsive to inhaled β_2-agonists and steroids	Bronchoscopy revealed an occluding endobronchial mass in the left main bronchus; biopsy confirmed carcinoid
Rizk et al. [25]	25-year-old woman with dyspnea and wheeze unresponsive to inhaled β_2-agonists and steroids for about 1 year	CT showed a polypoid lesion in the left main bronchus; rigid bronchoscopic excision confirmed a typical carcinoid

Aydin et al. reported a 48-year-old woman with a 12-year history of asthma who had persistent wheeze and dyspnea despite high-dose inhaled corticosteroid/long-acting beta2-agonist therapy, montelukast, and systemic corticosteroids and was being considered for anti-IgE therapy [20]. Bronchoscopy and CT identified a left lower lobe endobronchial mass, and biopsy confirmed a typical carcinoid. Lobectomy relieved the tumor-related symptoms, although a later acute asthma exacerbation showed that asthma and carcinoid can coexist.

Becher et al. described unilateral wheeze as the presenting manifestation of a bronchial carcinoid confirmed by bronchoscopy [2]. Santra et al. reported severe dyspnea and

wheeze initially managed as asthma before failure of conventional therapy prompted bronchoscopy and diagnosis of a bronchial carcinoid [21]. Biçer et al. described a patient with severe asthma and a coexisting bronchial carcinoid who presented with pneumomediastinum [22].

Davila et al. reported a broader series of bronchial carcinoid presentations that included cough, wheeze, hemoptysis, recurrent respiratory infections, and dyspnea [23]. Emeryk et al. described a 39-year-old man with approximately 10-11 months of persistent wheeze, hemoptysis, and cough despite inhaled beta2-agonists and corticosteroids; bronchoscopy demonstrated an obstructing left-main-bronchus carcinoid [24]. Rizk et al. reported a 25-year-old woman treated for presumed asthma for approximately 1 year before CT and rigid bronchoscopy identified and removed a polypoid left-main-bronchus lesion, which was confirmed as a typical carcinoid [25].

Taken together, these reports show a recurring pattern: persistent or atypical asthma-like symptoms, especially when accompanied by localized wheeze, hemoptysis, recurrent focal infection, unexpected imaging, or inadequate response to appropriate therapy, should prompt reconsideration of the diagnosis. Targeted chest imaging and, when indicated, bronchoscopy are more appropriate than simply escalating asthma therapy when a focal structural lesion is suspected.

Reporting Completeness Appraisal of Included Case Reports

Because case reports are inherently susceptible to selective reporting, six individually reported cases in Table 1 were appraised against four reporting domains adapted from the CARE case-report guideline [29]: patient demographics, diagnostic work-up, treatment, and post-treatment outcome/follow-up. Davila et al. is an aggregate case series/review and was

therefore not assessed with this single-case reporting checklist. This appraisal reflects the information available during screening (primarily abstract-level for several reports) and was intended to describe reporting completeness rather than methodological quality or risk of bias; it should not be interpreted as a substitute for a formal risk-of-bias tool.

Report	Demographics reported	Diagnostic work-up detailed	Treatment described	Outcome/follow-up reported	Overall completeness
Aydin et al. [20]	Yes	Yes	Yes	Yes	High
Becher et al. [2]	Not stated in available abstract	Yes	Not stated in available abstract	Not stated in available abstract	Low
Santra et al. [21]	Not stated in available abstract	Yes	Not stated in available abstract	Not stated in available abstract	Low
Biçer et al. [22]	Not stated in available abstract	Partial	Not stated in available abstract	Not stated in available abstract	Low
Emeryk et al. [24]	Yes	Yes	Not stated in available abstract	Not stated in available abstract	Moderate
Rizk et al. [25]	Yes	Yes	Yes	Not stated in available abstract	Moderate

Among the six individually appraised cases, only Aydin et al. reported a clearly described post-treatment outcome/follow-up in the material available for this review. The remaining reports primarily documented diagnosis and treatment without detailed long-term follow-up. This limitation in outcome reporting is addressed further in the Limitations section.

Clinical Presentation: Recognizing Pulmonary Carcinoid Tumors as an Asthma Mimic

Symptoms of pulmonary carcinoid tumors depend on tumor location, degree of bronchial obstruction, and disease extent. Tumors may be entirely asymptomatic and found

incidentally, or they may produce obstructive symptoms of the lower respiratory tract, including:

- Chronic cough
- Wheezing
- Shortness of breath
- Frequent respiratory infections
- Hemoptysis
- Airway obstruction

Wheezing is particularly misleading because it readily suggests asthma. Tumor-related wheeze, however, is often localized and may have a focal correlate on imaging or bronchoscopy. It often responds incompletely to standard asthma treatment, although partial improvement with bronchodilators does not exclude an endobronchial carcinoid.

Clinical features that should raise suspicion for a carcinoid tumor rather than asthma include: obstructive symptoms presenting for the first time in adulthood; persistent wheeze that is unresponsive or only partially responsive to asthma medications; localized wheeze, asymmetric decreased breath sounds, or recurrent infection confined to the same lung region; and unilateral wheezing.

Table 2. Clinical features that help differentiate asthma from an endobronchial carcinoid tumor

Clinical feature	Asthma	Endobronchial carcinoid tumor
Distribution of airway involvement	Typically diffuse, generalized airway inflammation	Focal, localized bronchial obstruction
Wheezing pattern	Usually bilateral, diffuse expiratory wheeze	Often unilateral or localized wheeze

Clinical feature	Asthma	Endobronchial carcinoid tumor
Breath sounds on auscultation	Usually symmetric	May be asymmetric or decreased on the affected side
Onset	Often childhood or early life; variable	Frequently adult-onset when tumor-related
Response to bronchodilators/ICS	Generally effective disease control when asthma is correctly diagnosed and adequately treated	Often refractory or only partially responsive
Infections	Not typically localized to the same lung segment	Recurrent infection in the same lung segment
Additional signs	Hemoptysis is uncommon	May include hemoptysis or dysphonia
Course	Chronic and fluctuating	May progressively worsen if obstruction increases

A progressive course of asthma-like symptoms that does not respond to treatment can be a reason for suspicion itself. While asthma is a chronic disease with a variable course, a carcinoid tumor can lead to progressive obstruction if left untreated. Unilateral or localized wheeze is a sign of focal airway obstruction of any cause (a finding not specific to carcinoid tumor) and the value of such a discovery lies in obtaining imaging and bronchoscopy, not in the confirmation of a specific etiology.

Diagnostic Approach

Diagnosis of a pulmonary carcinoid tumor relies on clinical assessment together with pulmonary function testing, radiographic imaging, bronchoscopy, and histopathology (Table 3).

Table 3. Summary of diagnostic modalities used to evaluate suspected pulmonary carcinoid tumor

Diagnostic modality	Purpose / key findings	Notes

Diagnostic modality	Purpose / key findings	Notes
Spirometry ± bronchodilator testing	Assesses variable airflow obstruction consistent with asthma	Helps objectively confirm or refute asthma before escalation to advanced therapy
Chest radiograph	May show hilar opacity, atelectasis, or recurrent focal infiltrates	A normal radiograph does not exclude an endobronchial lesion
Contrast-enhanced chest CT	Identifies an endobronchial mass, bronchial stenosis, distal atelectasis, or post-obstructive change	Carcinoids are often hypervascular and enhance intensely; small endobronchial lesions may still be missed
Bronchoscopy	Direct visualization of a smooth, vascular, protruding endobronchial mass and assessment of airway obstruction	Should be considered when imaging is inconclusive or clinical findings suggest a focal endobronchial lesion
Endobronchial biopsy	Provides tissue for histologic confirmation	Perform cautiously because of the potential for significant bleeding
Histopathology + immunohistochemistry	Confirms neuroendocrine differentiation and distinguishes typical from atypical carcinoid	Chromogranin A and synaptophysin are commonly used neuroendocrine markers; classification informs prognosis and management

Confirmation of the Asthma Diagnosis

Before diagnosing severe or refractory asthma, clinicians should confirm the diagnosis objectively when feasible, since misdiagnosis exposes patients to unnecessary corticosteroids, biologics, and repeated emergency visits. Studies of patients referred for severe asthma evaluation have found that a meaningful proportion did not, in fact, have asthma, which explained their lack of response to treatment [15]. Objective testing includes spirometry to demonstrate variable airflow obstruction, bronchodilator response, peak expiratory flow variability, and assessment of airway inflammation where applicable. Severe asthma should be diagnosed only after other conditions that could account for the symptoms have been considered.

Imaging Evaluation

Chest radiography may be normal or may show hilar airspace disease, atelectasis, recurrent focal infiltrates, or signs of post-obstructive infection; a normal film does not exclude an endobronchial lesion. Contrast-enhanced chest CT is an appropriate next step when a focal structural lesion is suspected and can demonstrate pulmonary carcinoid tumors, which are often hypervascular and enhance intensely. Characteristic CT findings include a well-defined hilar or perihilar mass, bronchial obstruction with distal atelectasis, mucus plugging, post-obstructive pneumonia, and, when present, regional lymphadenopathy. Small endobronchial lesions can nonetheless be missed on CT, so bronchoscopy remains necessary in selected patients [16].

Bronchoscopy and Histopathological Diagnosis

Bronchoscopy is central to the evaluation of suspected endobronchial carcinoid tumors because it permits direct visualization of the airway, assessment of the extent of obstruction, and tissue sampling when clinically appropriate. Typical carcinoids appear as smooth, rounded, highly vascular endobronchial masses; because of this vascularity, biopsy should be performed cautiously to avoid significant bleeding. Histopathology with immunohistochemistry for neuroendocrine markers such as chromogranin A and synaptophysin confirms the diagnosis and distinguishes typical from atypical carcinoids [17], a distinction that carries direct implications for treatment and prognosis.

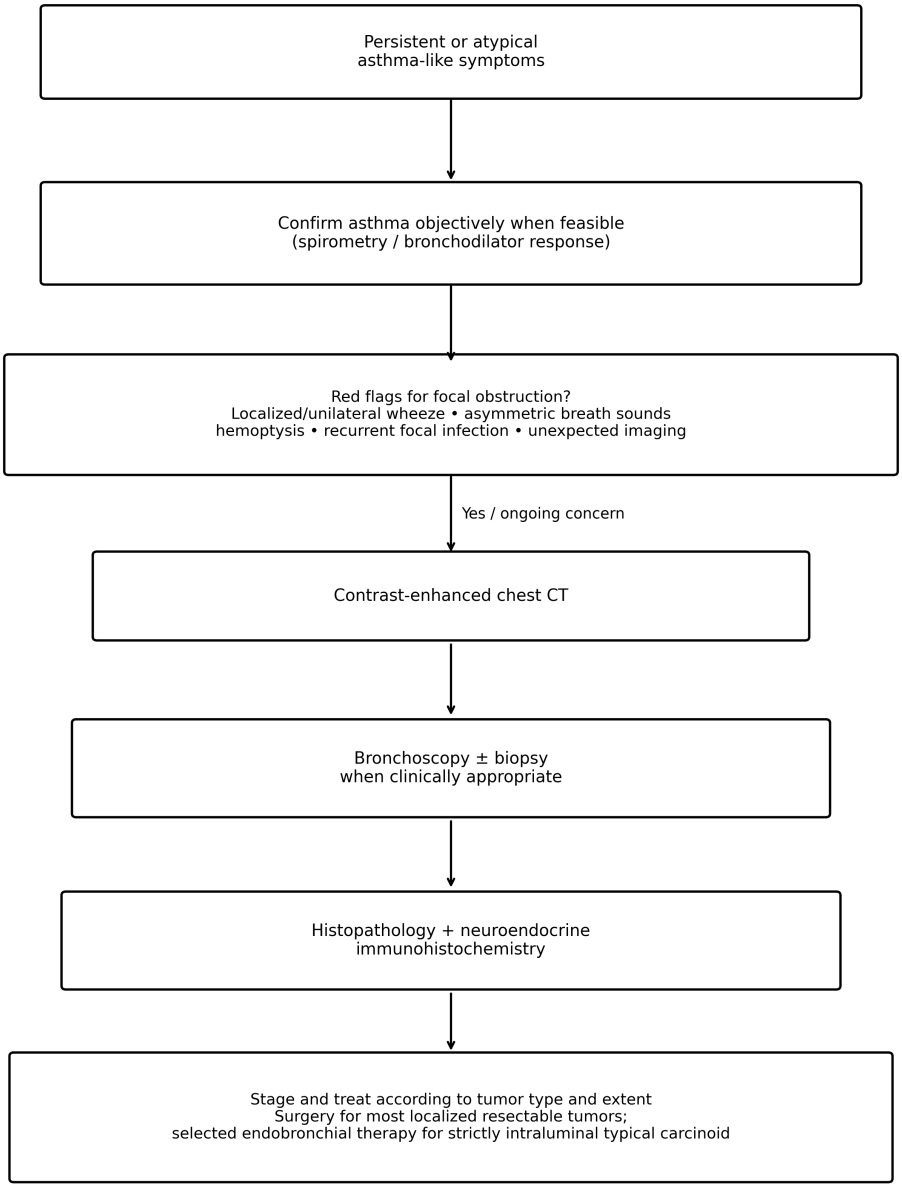

Figure 3. *Suggested diagnostic pathway for evaluating a patient with an asthma diagnosis and persistent or atypical respiratory symptoms.*

Note: The final step of treatment depicted in this figure (surgical resection) is the reference standard; the management section below describes an additional therapeutic option,

namely bronchoscopic/endoscopic therapy for strictly intraluminal, typical carcinoids in carefully selected patients.

Management of Pulmonary Carcinoid Tumors

Treatment depends on tumor type, location, stage, and the presence or absence of metastasis.

Bronchoscopic (Endoscopic) Management of Endobronchial Carcinoid Tumors

Surgical resection remains the preferred treatment for most localized, resectable pulmonary carcinoid tumors. In carefully selected patients, bronchoscopic techniques may serve as a parenchyma-sparing approach, particularly for strictly intraluminal typical carcinoids without radiologic evidence of bronchial-wall or parenchymal invasion. Approximately 20% of bronchial carcinoids have been described as purely intraluminal polypoid lesions potentially suitable for endobronchial treatment [10]. In the systematic review by Reuling et al., nine endobronchial-treatment studies and 43 surgical studies were identified; outcomes were favorable in selected patients, but the evidence was retrospective, non-randomized, and subject to selection bias [10]. Current guidance continues to favor complete surgical resection for most operable localized tumors, while reserving endobronchial treatment for selected situations, including patients unsuitable for surgery or as part of a staged strategy [10,30].

The bronchoscopic techniques that have been reported in the literature include rigid bronchoscopy with mechanical debulking, neodymium:YAG (Nd:YAG) laser photoresection, electrocautery or snare resection, argon plasma coagulation, cryotherapy, including cryorecanalization, and, more rarely, photodynamic therapy [26] (Table 4). Brokx et al. reported on long-term outcomes (follow-up duration of up to 5 years) after initial

bronchoscopic therapy (with or without subsequent surgery) in patients with typical carcinoids and found that local tumor control was achieved in all patients who were appropriately selected [26]. In a series of 10 patients who underwent flexible bronchoscopic Nd:YAG laser photoresection of a carcinoid tumor under conscious sedation, no treatment-related complications were reported, and with a follow-up duration of 29 months, no recurrences were detected, albeit with multiple procedures being needed for tumor clearance [27]. The review by Reuling et al. cites a figure from another study reporting an 8% recurrence rate at 10 years after EBT and suggests that surgical salvage can be an option if recurrence develops [10] (Table 5).

Table 4. Endobronchial (bronchoscopic) treatment modalities reported for carcinoid tumors

Modality	Mechanism / use	Notes
Rigid bronchoscopy with mechanical debulking	Direct mechanical removal of tumor tissue to restore airway patency	Often used to achieve rapid airway control before definitive treatment
Nd:YAG laser photoresection	Thermal ablation and vaporization of tumor tissue	Widely reported; repeat sessions may be required
Electrocautery / snare resection	Electrosurgical excision, particularly for pedunculated lesions	Useful for polypoid tumors with a discrete base
Argon plasma coagulation	Non-contact thermal coagulation	May be used for hemostasis or residual tumor ablation
Cryotherapy / cryorecanalization	Freeze-thaw injury causing tumor necrosis or immediate cryoextraction	May be combined with other bronchoscopic modalities
Photodynamic therapy	Light-activated cytotoxic effect after photosensitizer administration	Reported in selected cases and less commonly used

Table 5. Reported outcomes of endobronchial treatment strategies for pulmonary carcinoid tumors

Source	Population / design	Reported outcome
Reuling et al., 2019 [10]	Systematic review identifying 9 studies of endobronchial treatment and 43 surgical studies	Favorable recurrence, complication, and survival outcomes were reported in selected patients, but the evidence was retrospective and non-randomized; long-term recurrence remained possible and surgical salvage could be used when needed
Brokx et al., 2015 [26]	Long-term follow-up after initial bronchoscopic therapy, with or without later completion surgery	Durable local control was reported in appropriately selected typical carcinoids
Fruchter et al., 2009 [27]	10 patients treated with flexible-bronchoscopic Nd:YAG laser photoresection under conscious sedation	No major treatment-related complications and no recurrence at a median 29-month follow-up; 2-4 procedures were required per patient

Successful endobronchial management depends on careful patient selection. Favorable features include histologic confirmation of a typical carcinoid, a strictly intraluminal lesion with a clearly defined base or stalk, no radiologic or endobronchial-ultrasound evidence of extraluminal extension, no nodal or distant metastases, and the ability to adhere to bronchoscopic and radiologic surveillance [26-28]. Because small biopsies can underestimate tumor extent or histologic aggressiveness, bronchoscopic treatment may also function as an initial diagnostic and local-control strategy while preserving the option of definitive surgery if atypical histology, wall invasion, incomplete clearance, or recurrence is identified [26-28].

Surgical Treatment

Complete surgical resection is the treatment of choice for localized pulmonary carcinoid tumors and is associated with excellent survival. The surgical approach is tailored to tumor size, location, airway involvement, and overall pulmonary function; centrally located tumors

can often be managed with parenchyma-sparing techniques such as sleeve resection or bronchoplastic reconstruction rather than more extensive resection [18]. Options include:

- Sleeve resection
- Bronchoplastic procedures
- Lobectomy
- Other resections tailored to tumor extent

Evaluation of regional lymph nodes is important, particularly for atypical carcinoids, to help establish disease stage. Table 6 summarizes the recommended treatment approach by clinical setting.

Treatment of Advanced and Metastatic Disease

For unresectable tumors or distant metastases, options include somatostatin analogues, targeted therapy, and peptide receptor radionuclide therapy (PRRT) guided by somatostatin receptor imaging and individual tumor biology. The pivotal randomized trial that established PRRT efficacy was performed in midgut neuroendocrine tumors [19]; prospective and retrospective series specific to bronchial typical and atypical carcinoids have separately reported objective responses and disease control with ^{177}Lu-DOTATATE, with higher response rates in typical than in atypical carcinoids [32]. Care should be coordinated through a multidisciplinary team experienced in neuroendocrine lung tumors, and observation may be appropriate for select asymptomatic patients. Table 7 summarizes the key histologic and prognostic differences between typical and atypical carcinoids described throughout this review.

Table 6. Overview of treatment approaches by clinical setting

Clinical setting	Treatment approach

Clinical setting	Treatment approach
Strictly intraluminal typical carcinoid without wall invasion	Bronchoscopic/endoscopic treatment may be considered as a parenchyma-sparing option in carefully selected patients, particularly when the lesion is strictly intraluminal or surgery is unsuitable; complete surgical resection remains standard for most localized resectable tumors
Localized, resectable tumor	Complete surgical resection (e.g., sleeve resection, bronchoplastic reconstruction, or lobectomy) while preserving normal lung tissue when feasible; regional lymph-node evaluation is important, especially for atypical carcinoids
Unresectable disease / distant metastases	Somatostatin analogues, targeted therapy, peptide receptor radionuclide therapy when appropriate, and individualized systemic therapy based on receptor status and tumor biology
Across disease settings	Multidisciplinary discussion involving thoracic surgery, pulmonology, oncology, radiology, and pathology; surveillance should be individualized according to stage, histology, and treatment

Table 7. Comparison of typical and atypical pulmonary carcinoid tumors

Feature	Typical carcinoid	Atypical carcinoid
Relative frequency	More common subtype	Less common
Mitotic count	<2 mitoses per 2 mm²	2-10 mitoses per 2 mm²
Necrosis	Absent	Present as punctate/focal necrosis
Growth pattern	Usually slow-growing and indolent	More aggressive
Lymph-node involvement	Less frequent	More frequent
Risk of recurrence/metastasis	Lower	Higher
Overall prognosis	Generally favorable after complete treatment	Less favorable than typical carcinoid

Limitations

This review is based primarily on case reports, small case series, and narrative reviews rather than large comparative cohorts, reflecting the low overall incidence of pulmonary carcinoid tumors and the resulting scarcity of higher-quality evidence on their overlap with asthma. Publication bias likely favors atypical or diagnostically striking presentations over

routine ones, which may overstate how distinctive the clinical features described here are in unselected populations. The recommendations on when to suspect an endobronchial tumor in a patient with presumed asthma are therefore based on expert opinion and accumulated case experience rather than prospective validation.

The evidence supporting bronchoscopic (endoscopic) treatment specifically is similarly limited to small, often single-center studies and one systematic review of a small number of studies; randomized comparisons of bronchoscopic and surgical treatments are lacking, and therefore the comparisons of outcomes described in this review should be regarded with caution.

Conclusion

Pulmonary carcinoid tumors are rare but should be part of the differential diagnosis in patients with asthma-like symptoms, since endobronchial obstruction can produce wheeze, cough, dyspnea, recurrent infection, and hemoptysis that closely mimic asthma. Because asthma is far more common than carcinoid tumors, the latter are often overlooked, and patients, including several described in this review, may be treated for years with escalating inhaled and systemic corticosteroids, or even considered for biologic therapy, before the correct diagnosis is made.

Clinicians should reassess the diagnosis in patients with treatment-resistant asthma rather than assuming refractory symptoms simply reflect more severe airway inflammation. Unilateral wheeze, asymmetric breath sounds, recurrent infection localized to one lung segment, unexpected radiographic findings, or hemoptysis should prompt further evaluation, typically with chest CT followed by bronchoscopy, although none of these findings is specific enough to confirm or exclude asthma on its own. Notably, asthma and a carcinoid tumor can

coexist, and symptoms may persist after tumor resection if underlying asthma is also present, underscoring the need to distinguish obstruction from tumor mass from inflammation-driven airway disease, since the two require different treatment.

Diagnosis requires a multidisciplinary approach involving pulmonology, radiology, pathology, and thoracic surgery, with chest CT to identify endobronchial masses and post-obstructive changes and bronchoscopy with biopsy to confirm the diagnosis and distinguish typical from atypical carcinoid, which differ in prognosis and treatment.

Complete surgical resection remains the principal treatment for most localized, resectable disease, offering a generally favorable prognosis, but bronchoscopic (endoscopic) therapy appears to be a reasonable parenchyma-sparing option or initial approach for strictly intraluminal typical carcinoids in selected patients, with completion surgery for those with incomplete resection or atypical histology, and individualized systemic therapy for more advanced disease. Increased recognition of pulmonary carcinoid tumor as an entity that may mimic asthma and awareness of the therapeutic options for these tumors when the diagnosis is established may hasten the diagnostic process and improve patient care by diminishing the likelihood of both inappropriate escalation of therapy for asthma and unnecessary extent of surgery when the diagnosis is established.

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