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Acute dyspnea and stridor in primary care: Tracheal adenoid cystic carcinoma in a non-smoker

Abstract

Background: Tracheal adenoid cystic carcinoma is a rare cause of airway obstruction and may initially mimic common respiratory conditions. In primary care, recognition is particularly challenging because chest radiography may be normal and symptoms may temporarily improve after corticosteroid or bronchodilator treatment.

Case presentation: A 42-year-old non-smoking woman with no history of pulmonary disease presented to primary care with rapidly progressive dyspnea, orthopnea, dry cough, and poor response to inhaled bronchodilator therapy. Examination revealed respiratory distress, accessory muscle use, wheezing, and subsequently clearly audible stridor. Initial chest radiography was unremarkable, and the symptoms were initially attributed to acute laryngitis. Recurrent deterioration and persistent stridor prompted hospitalization and computed tomography of the neck and chest, which demonstrated a tracheal mass causing critical airway narrowing. Endoscopic airway relief and biopsy confirmed adenoid cystic carcinoma. Complete surgical resection was subsequently achieved.

Conclusion: Persistent stridor, rapidly progressive dyspnea, repeated clinical deterioration, and poor response to bronchodilator therapy should prompt reconsideration of common respiratory diagnoses. A normal chest radiograph does not exclude clinically significant central airway obstruction. Early cross-sectional imaging should be considered when symptoms and clinical findings remain unexplained.

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Introduction

Tracheal ACC represents the second most common histological type of tracheal tumor. These are slow-growing tumors arising from bronchial glands with infiltrative growth along the airway [1-3]. The reported incidence is 0.1 per 100,000 inhabitants per year [4]. Due to non-specific symptoms, the diagnosis of ACC is usually delayed for more than one year after symptom onset. The disease is frequently misdiagnosed and treated as asthma, COPD, or pneumonia [1-7].

This case is considered clinically relevant due to both the rarity of this tumor and the unusually short duration of symptoms, which contrasts with the majority of previously reported cases. According to the Slovenian Cancer Registry (Slora), approximately two patients are diagnosed with this type of cancer in Slovenia every decade [8]. The relevance of this case extends beyond the rarity of the underlying tumor. It illustrates the diagnostic challenge of distinguishing upper airway obstruction from more common causes of acute dyspnea in primary and emergency care. The report therefore focuses on the clinical warning signs, repeated reassessment, and diagnostic escalation that led to the recognition of a critical tracheal obstruction despite initially non-specific symptoms and unremarkable chest radiography.

Case presentation

Patient information

A 42-year-old non-smoking woman was evaluated in a primary care outpatient clinic in winter due to dyspnea and

cough. Symptoms started one week earlier with a dry cough without fever, sore throat, or rhinorrhea. Over the weekend, progressive dyspnea and orthopnea developed. The day before, she was evaluated in an emergency outpatient clinic and prescribed inhaled ipratropium/fenoterol 0.05 mg/0.02 mg (two inhalations every 8 hours) without clinical improvement. Her medical history included arterial hypertension, for which she discontinued treatment one year earlier, gestational diabetes, hemorrhoids, and obesity. She denied allergies, asthma, other pulmonary disease, and oral contraceptive use. Family history was negative for malignancy and asthma.

Clinical examination

The patient was visibly distressed, tachypneic, and leaning forward. Dyspnea worsened after walking a few steps. Accessory respiratory muscle use, jugular and supraclavicular retractions, expiratory wheezing, and audible gasping were observed. She spoke in short sentences with pauses for breath. Vital signs were: T 36.6°C, BP 200/109 mmHg, HR 101/min, and SpO_2 95% at rest, and 91–93% during speech. Lung auscultation revealed diminished breath sounds with occasional expiratory wheezes. Cardiac examination showed regular tachycardia. Nebulized ipratropium/fenoterol 2 mL (0.5 mg/0.261 mg) diluted with 2 mL 0.9% NaCl was administered. Only mild subjective improvement was reported, while vital signs remained unchanged. The combination of rapidly progressive symptoms, marked respiratory effort, oxygen desaturation during speech, and minimal response to bronchodilator therapy was considered inconsistent with uncomplicated viral respiratory disease. Although mild expiratory wheezing was present, the absence of a history of asthma or other pulmonary disease and the severity of the clinical presentation supported urgent secondary-care assessment. The patient was referred to the internal medicine emergency service.

Diagnostic workup

At presentation, inspiratory and expiratory stridor over the trachea, accessory respiratory muscle use, and oxygen saturation of 92% were noted. Arterial blood gas analysis revealed chronic compensated respiratory acidosis with mild hypoxemia. Laboratory findings showed mildly elevated CRP, neutrophilia, and lymphopenia, while D-dimer and ECG were normal. Chest radiography revealed no significant abnormalities. Nebulized therapy was administered: dexamethasone 2 mL (4 mg/mL) diluted with 2 mL Aqua, ipratropium /fenoterol 2 mL (0.5 mg/0.261 mg) diluted with 2 mL Aqua, and adrenaline 0.5 mg (1 mg/mL) diluted with 2 mL 0.9% NaCl. Additionally, methylprednisolone 40 mg was administered intravenously. Following treatment, stridor almost completely resolved. A diagnosis of viral laryngitis was made, and the patient was discharged with beclomethasone/formoterol 100 mcg/6 mcg (two inhalations every 12 hours), as-needed inhaled ipratropium/fenoterol 0.05 mg/0.02 mg, and oral methylprednisolone 32 mg daily for 5 days. In retrospect, the differential diagnosis included acute laryngitis with upper airway edema, new-onset obstructive airway disease, inducible laryngeal obstruction, subglottic or tracheal stenosis, an aspirated foreign body, external airway compression, and an intraluminal airway lesion. Pulmonary embolism was considered less likely because of the normal D-dimer result and the predominance of upper airway signs. The persistence of stridor and the lack of sustained improvement made a structural airway abnormality increasingly likely.

Several hours after discharge, the patient's condition deteriorated. She returned to the internal medicine emergency service the following morning. Clinical status and arterial blood gas analysis were unchanged compared with the previous day. Laboratory tests showed an increased leukocyte count, most likely related to corticosteroid therapy. The patient was admitted for further evaluation.

During hospitalization, treatment included nebulized dexamethasone 2 mL (4 mg/mL) diluted with 2 mL Aqua every 8 hours, nebulized salbutamol 2 mL (0.5 mg/mL) diluted with 2 mL Aqua every 8 hours, oral methylprednisolone 32 mg daily, oral esomeprazole 40 mg daily, and oral amlodipine/perindopril/indapamide 4 mg/10 mg/1.25 mg daily. As needed, nebulized adrenaline 0.5 mg (1 mg/mL) diluted with 2 mL 0.9% NaCl was administered. Repeated presentation, persistent stridor, and the absence of a sustained response to corticosteroids, adrenaline, and bronchodilator therapy were considered diagnostic warning signs. Chest CT and contrast-enhanced CT of the neck were performed, revealing an irregular soft tissue mass in the upper trachea (Figure 1). The residual airway lumen measured approximately 4 mm at the narrowest point.

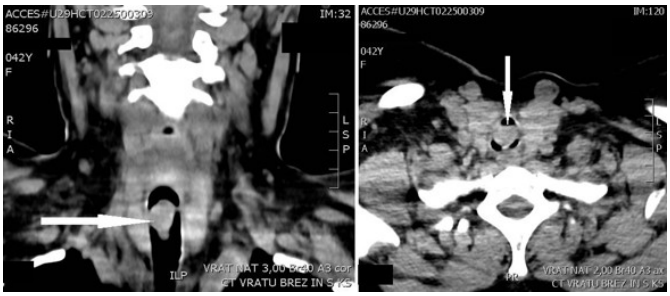

Figure 1: Contrast-enhanced computed tomography of the neck in the coronal plane (left) and axial plane (right), demonstrating an irregular soft-tissue mass in the upper trachea below the vocal cords. The lesion caused critical narrowing of the tracheal lumen to approximately 4 mm at its narrowest point (arrows).

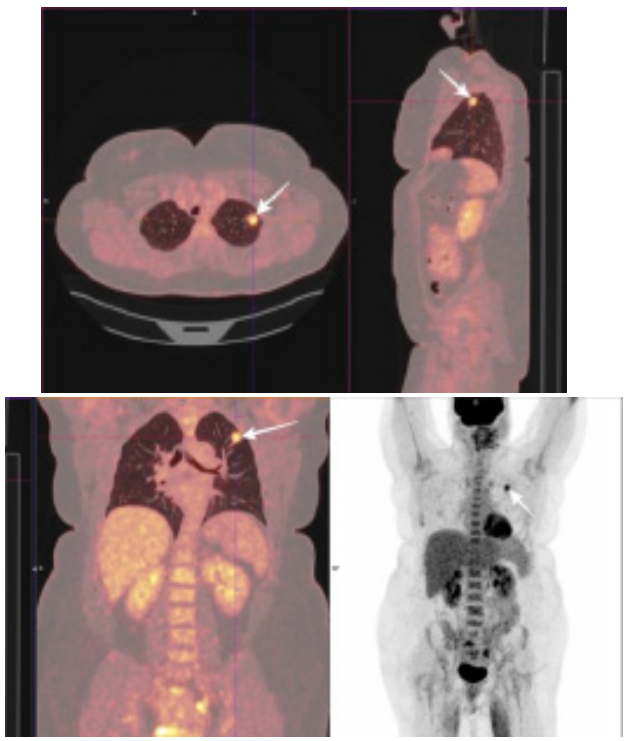

Figure 2: PET/CT images in different planes showing a suspected lesion in the left upper lobe (arrows) with increased radiotracer uptake.

The patient was transferred to the intensive care unit of a tertiary center, where rigid tracheoscopy with tumor coring and biopsy was performed. Histopathological analysis confirmed ACC. Staging brain CT revealed no metastatic disease. PET/CT showed increased radiotracer uptake in the left upper pulmonary lobe, suspicious for metastasis or inflammation (Figure 2), while the tracheal tumor demonstrated low uptake.

No suspicious lymph nodes were identified. Radical surgical resection of the tracheal tumor, with possible left upper lobectomy, was planned.

Treatment

The patient was readmitted for surgical treatment. Follow-up chest CT showed regression of the previously observed left upper lobe lesion. Tracheal resection with end-to-end anastomosis was performed, and frozen section confirmed tumor-free margins.

Follow-up

Histopathological examination of the resected specimen showed no lymph node metastases and confirmed the presence of perineural invasion. The tumor was staged as pT1N0 according to the Bhattacharyya staging system. Six weeks after resection, follow-up examination by the surgeon was performed. Chest radiography showed no abnormalities. The patient was in excellent clinical condition. Annual follow-up was recommended.

Discussion

Primary malignant tracheal tumors are rare, accounting for less than 0.5% of all cancers. In adults, approximately 90% of tracheal tumors are malignant, with squamous cell carcinoma being the most common, followed by ACC, which accounts for 10–20% of cases [1,4,6,9]. ACC is a slow-growing malignancy arising from submucosal bronchial glands, characterized by submucosal growth along the airway [1-3]. It affects both sexes equally, most commonly between the fourth and fifth decades, and is not associated with smoking, alcohol consumption, or known risk factors [1,5,7]. The patient described in this report was consistent with these epidemiological characteristics.

Clinical presentation usually includes progressive dyspnea, cough, hemoptysis, orthopnea, wheezing and stridor [1,3-5,10-18]. In this case, the patient presented with dyspnea at rest, dry cough, and orthopnea, with stridor and faint wheezing on auscultation. Diagnosis is often delayed due to misinterpretation of symptoms as asthma, COPD, or pneumonia [1-7]. Unlike many reported cases, where symptoms persisted for months or years before diagnosis [3,11-13,15,16,19], this patient presented after only a few days of dyspnea. A focused PubMed search conducted in July and August 2025 identified only two English-language open-access case reports published within the preceding 10 years that described progressive dyspnea lasting less than one week before presentation [14,17]. In the case reported by Kapatia et al., the diagnosis of ACC was established only at autopsy [14]. From a primary care perspective, several features in this case represented diagnostic warning signs. The patient had no previous history of asthma or chronic pulmonary disease, symptoms progressed rapidly, bronchodilator treatment produced little benefit, and respiratory distress was disproportionate to the initially unremarkable chest radiograph. Most importantly, inspiratory and expiratory stridor suggested central or upper airway obstruction rather than isolated lower airway bronchospasm. These findings demonstrate the importance of reassessing the working diagnosis when the clinical course is atypical or treatment response is insufficient [20]. Standard chest radiography may fail to identify lesions of the upper or central airway. Consequently, a normal chest radiograph should not be considered sufficient to exclude tracheal obstruction when stridor or other signs of central airway compromise are present [1,4].

CT is the preferred imaging modality for evaluating tracheobronchial tumors, as it enables assessment of intraluminal and extraluminal involvement as well as metastases [1,4,7,9]. In this case, computed tomography was the decisive investigation because it demonstrated both the lesion and the severity of airway narrowing. Bronchoscopy provides more detailed tumor visualization, allows tissue sampling, and may relieve airway obstruction [1,4], while PET/CT may assist in assessing disease extent, although radiotracer uptake in ACC is variable [1]. Accordingly, CT, bronchoscopy, and PET/CT were all performed as part of the diagnostic work-up.

ACC is characterized by perivascular and perineural invasion, the latter being associated with local recurrence, metastases, and poorer prognosis [7]. Both lymphatic and hematogenous metastases may occur [3,5].

Due to the rarity of ACC, no evidence-based treatment guidelines have been established [5-7]. Surgical resection with primary anastomosis remains the preferred treatment for resectable tumors [1,3,4]. Positive margins are reported in 45.8–63% of cases, reflecting the infiltrative growth pattern of ACC and the challenges associated with achieving complete resection [3,6,7]. Adjuvant radiotherapy is recommended after incomplete resection [1,4,5]. In the present case, complete (R0) resection was achieved.

ACC may recur even 10–20 years after treatment, either locally or as distant metastases. Therefore shorter follow-up periods may underestimate recurrence risk and limit prognostic assessment [1,6,7]. Another challenge is the lack of a universally accepted staging system for primary tracheal tumors; however, the Bhattacharyya staging system is commonly used and was applied in this case [6,21].

Complete (R0) resection is associated with better survival outcomes than incomplete resection; however, R1 resection followed by postoperative radiotherapy may achieve comparable survival rates [2,3]. A systematic review reported 5-year overall survival rates of 88.7–95% after R0 resection and 84–88.4% after R1 resection with postoperative radiotherapy [6]. Although definitive diagnosis and treatment required specialized endoscopic, pathological, and surgical assessment, the principal educational value of this report lies in the earlier stages of the clinical pathway. The case highlights symptom interpretation, recognition of upper airway warning signs, repeated reassessment, and timely escalation from primary and general emergency care to advanced imaging and specialist management.

The principal limitation of this report is the short duration of postoperative follow-up, which does not allow conclusions regarding long-term recurrence. This is particularly relevant because tracheal adenoid cystic carcinoma may recur many years after treatment. Nevertheless, the purpose of this report is not to evaluate long-term oncological outcomes but to illustrate the diagnostic pathway and clinical warning signs

associated with critical tracheal obstruction presenting as acute dyspnea.

Clinical implications for primary care

In patients presenting with acute or rapidly progressive dyspnea, the following findings should prompt consideration of upper or central airway obstruction:

1. Inspiratory or biphasic stridor, particularly when initially interpreted as wheezing.
2. Severe respiratory distress despite relatively preserved oxygen saturation at rest.
3. Poor or short-lived response to bronchodilators, corticosteroids, or adrenaline.
4. Repeated presentation or rapid deterioration after initial treatment.
5. A normal chest radiograph despite persistent signs of airway obstruction.

When these features are present, reassessment of the initial diagnosis and early imaging of the neck and central airway should be considered.

Conclusion

This case illustrates an uncommon but clinically important cause of acute dyspnea encountered in primary care. Tracheal adenoid cystic carcinoma may present with symptoms resembling laryngitis or asthma, while chest radiography remains inconclusive. Persistent stridor, poor response to standard respiratory treatment, repeated deterioration, and discordance between clinical severity and initial investigations should prompt reconsideration of the diagnosis and early evaluation of the central airway. The case emphasizes the importance of clinical reassessment and timely escalation rather than the recognition of a rare diagnosis at the first presentation.

Learning points

- Stridor may be mistaken for wheezing, particularly in a distressed patient.
- A normal chest radiograph does not exclude significant tracheal obstruction.
- Poor response to bronchodilator therapy should prompt reassessment of the initial diagnosis.
- Repeated presentation and rapid deterioration are important diagnostic warning signs.
- The role of primary care is not necessarily to identify the rare tumor immediately, but to recognize an atypical course and ensure timely diagnostic escalation.

Declarations

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