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(CASE REPORT)

Primary pulmonary adenoid cystic carcinoma: A case report of prolonged survival with late metastatic disease and a brief review of the literature

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Abstract

Primary pulmonary adenoid cystic carcinoma (PACC) is a rare salivary gland–type tumor originating in the central airways. Although indolent, it is nonetheless associated with late local recurrence and distant metastasis.

A 51-year-old woman presented with a 14-month history of intermittent dry cough and progressive exertional dyspnea, followed by wheezing refractory to antibiotics and bronchodilators. Contrast-enhanced chest computed tomography revealed a 2.8-cm well-circumscribed, homogeneous, lobulated intraluminal mass arising from the right mainstem bronchus without mediastinal invasion. Positron emission tomography–computed tomography demonstrated mild-to-moderate fluorodeoxyglucose avidity without nodal or distant metastases. Bronchoscopic biopsy confirmed classic adenoid cystic carcinoma with solid, tubular, and cribriform patterns, pseudocystic spaces, and perineural invasion. Immunohistochemistry (IHC) showed positivity for CK7, p63, SOX10, S100 (myoepithelial markers), and strong membranous CD117 (c-Kit); fluorescence in situ hybridization verified MYB–NFIB gene fusion.

Treatment comprised lung-sparing carinal resection with airway reconstruction, followed by adjuvant external-beam radiotherapy (60 Gy in 30 fractions) for positive margins. Seven years postoperatively, metastatic pulmonary nodules and an iliac bone lesion emerged, initially managed with active surveillance, then lenvatinib systemic therapy and stereotactic body radiotherapy to symptomatic bone metastasis. This case illustrates the diagnostic complexity, protracted natural history, and need for extended multidisciplinary surveillance in PACC.

Keywords: Adenoid cystic carcinoma; Pulmonary; Local recurrence; Neural invasion; Immunohistochemistry; Lung-sparing carinal resection; External-beam radiotherapy

1. Introduction

Primary pulmonary adenoid cystic carcinoma (PACC) is a rare malignant salivary gland–type tumor arising from the submucosal glands of the tracheobronchial tree. It accounts for approximately 0.04–0.2% of all primary pulmonary malignancies and is recognized in the World Health Organization Classification of Thoracic Tumors as a distinct entity among salivary gland–type neoplasms. [1] Unlike conventional non–small cell lung carcinoma, PACC is not strongly linked to tobacco exposure and may present in younger or nonsmoking patients. [1] Despite its generally slow growth and low-grade histology, PACC demonstrates insidious submucosal infiltration, perineural invasion, and a high

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propensity for local recurrence and late distant metastasis, resulting in a prolonged yet unpredictable clinical course. [2]

Most tumors arise in the central airways, typically the trachea or main bronchi, where they cause progressive airway obstruction and nonspecific symptoms such as persistent cough, wheezing, dyspnea, or recurrent bronchitis. [18] Because these manifestations often mimic asthma or chronic obstructive airway disease, diagnosis is frequently delayed. Imaging may reveal smooth, well-defined endobronchial mass, but definitive diagnosis requires bronchoscopy with biopsy and histopathologic evaluation. [3] [4] Microscopically, PACC exhibits characteristic cribriform, tubular, and solid patterns composed of epithelial and myoepithelial cells within a hyaline stroma. [5]

Molecular studies frequently identify MYB gene rearrangements, most notably the MYB–NFIB fusion, which serve as diagnostic markers and may offer future therapeutic potential. [11] [16] Immunohistochemical stains such as CK7, CD117 (c-Kit), SOX10, p63, and S100 aid in confirming tumor lineage and excluding histologic mimics. Complete surgical resection remains the cornerstone of curative-intent treatment, though achieving negative margins can be challenging due to submucosal spread. [6] Adjuvant radiotherapy is often recommended in cases of positive or close margins. [3] Although long-term survival is common, late recurrences and distant metastases can occur many years after initial therapy. [5]

2. Case Presentation

2.1. Clinical Presentation

A 51-year-old woman with a 14-month history of intermittent dry cough and progressive exertional dyspnea presented to her pulmonologist after two courses of antibiotic therapy for presumed recurrent bronchitis had failed to provide lasting relief. She experienced episodic wheezing for over three months, which her doctor initially diagnosed as late-onset asthma and treated with inhaled bronchodilators that provided only partial relief. She denied hemoptysis, fever, or significant weight loss. A chest radiograph obtained at that visit demonstrated subtle right paratracheal soft-tissue fullness, prompting referral to the pulmonary service for further evaluation. The chronicity of symptoms, lack of response to conventional therapies, and the radiographic abnormality raised concern for an underlying endobronchial or central airway lesion.

2.2. Physical Examination, History, Imaging, and Radiographic Differential Diagnosis

Physical examination revealed a well-nourished woman in no acute distress. Auscultation demonstrated faint expiratory monophonic wheezing over the central airways bilaterally. No cervical or supraclavicular lymphadenopathy was palpated. Personal history was notable for a 10 pack-year smoking history (remote, cessation 15 years prior) and well-controlled hypothyroidism. Family history was non-contributory for malignancy. Routine laboratory studies, including a complete blood count and comprehensive metabolic panel, were within normal limits. Serum tumor markers were not significantly elevated.

Computed tomography (CT) of the chest with contrast demonstrated a well-defined, homogeneous, lobulated intraluminal mass within the distal trachea and proximal right mainstem bronchus, measuring 2.8 cm in greatest dimension. The mass had smooth borders infiltrating the mainstem bronchus, without mediastinal invasion. Positron emission tomography (PET-CT) revealed mild-to-moderate FDG avidity of the lesion without regional nodal or distant metabolic activity. Pulmonary function testing demonstrated a fixed obstructive pattern.

The radiographic differential diagnosis included adenoid cystic carcinoma, carcinoid tumor, mucoepidermoid carcinoma, squamous cell carcinoma arising from the central airway, and, less likely, a solitary fibrous tumor or inflammatory myofibroblastic tumor.

2.3. Multidisciplinary Tumor Board Meeting

The case was presented to the multidisciplinary thoracic tumor board, which included thoracic surgery, pulmonology, medical oncology, radiation oncology, radiology, and pathology. Discussion centered on the mass's central location, its relationship to the carina, and the anticipated surgical complexity given its proximity to the right mainstem bronchus. The board acknowledged the characteristic indolent behavior of low-grade salivary-type tumors of the airway and debated the adequacy of bronchoscopic sampling versus upfront surgical resection.

The consensus recommendation was to proceed with rigid bronchoscopy for tissue diagnosis and airway assessment, followed by surgical planning for sleeve resection or tracheal resection with reconstruction, aiming to achieve clear

surgical margins. Preoperative anesthesia and airway management planning were emphasized, given the degree of luminal compromise.

2.4. Management, Pathology, Immunohistochemistry, Molecular Studies, and Final Diagnosis

The patient underwent rigid bronchoscopy, which revealed a smooth, firm, submucosal mass arising from the posterior wall of the distal trachea and extending into the proximal right mainstem bronchus. Endobronchial biopsies were obtained, and histological examination was highly consistent with the diagnosis of PACC. Following a multidisciplinary review recommendation, the patient underwent lung-sparing surgery with carinal resection and reconstruction. Microscopic examination of the tumor demonstrated nests and tubules of small, basaloid epithelial and myoepithelial cells displaying mixed architectural patterns, including solid, tubular, and cribriform, with pseudo-cystic spaces containing pale basophilic material. Perineural invasion was identified. Histomorphology confirmed the biopsy diagnosis of PACC (Figure 1 A, B, C, D).

IHC studies demonstrated strong diffuse positivity for CK7, p63, and SOX10. S100 protein highlighted the outer myoepithelial cell layer. CD117 (c-Kit) showed strong membranous positivity. The final surgical pathology specimen confirmed PACC, with the bronchial resection margins focally positive for tumor microscopically. MYB fluorescence in situ hybridization (FISH) revealed MYB-NFIB gene fusion, a hallmark molecular alteration in adenoid cystic carcinoma, confirming the diagnosis.

Pathologic differential diagnosis had included mucoepidermoid carcinoma, basaloid squamous cell carcinoma, and small cell carcinoma, all of which were excluded by morphology and IHC profile. Metastatic disease, especially from salivary glands, was ruled out by PET-CT.

2.5. Outcome, Follow-Up, Complications, and Surveillance, Two Years After Initial Treatment

Given the focally positive bronchial margin identified on final pathology, the case was referred to the tumor board, which recommended adjuvant radiation therapy to the surgical bed. The patient completed a course of external beam radiation therapy (60 Gy in 30 fractions) without significant acute toxicity. She tolerated the postoperative period reasonably well, though she experienced prolonged air leak requiring extended chest tube drainage for nine days. She was discharged on postoperative day 14.

Surveillance imaging with CT of the chest, abdomen, and pelvis were performed at six-month intervals. At her two-year follow-up, the patient was clinically well, with no radiographic evidence of local recurrence or distant metastasis. She reported mild exertional dyspnea for which she was enrolled in a pulmonary rehabilitation program. Quality of life was reported as satisfactory. Oncologic surveillance was continued on a biannual basis, given the well-documented capacity of adenoid cystic carcinoma for late recurrence.

2.6. Late Metastatic Disease, Tumor Board Discussing, Treatment, and Outcome

Approximately seven years after initial surgical resection, routine surveillance CT imaging revealed multiple bilateral pulmonary nodules, the largest measuring 1.8 cm, and a lytic lesion involving the right iliac bone. PET-CT confirmed metabolic activity in both the pulmonary nodules and the pelvic osseous lesion. CT-guided biopsy of the largest pulmonary nodule was performed; histology demonstrated recurrent adenoid cystic carcinoma with MYB-NFIB fusion confirmed on molecular testing, consistent with metastatic disease from the primary pulmonary tumor.

The case was brought before the multidisciplinary tumor board. Given the indolent pace of the disease, low tumor burden, and preserved performance status, the board elected to initiate active surveillance with close-interval imaging. When modest radiologic progression was documented at six months, systemic therapy was initiated with lenvatinib, a multikinase inhibitor with reported activity in adenoid cystic carcinoma. Stereotactic body radiation therapy (SBRT) was delivered to the symptomatic pelvic bone lesion for local control and pain palliation.

At 18-month follow-up from the diagnosis of metastatic disease, the patient maintained stable disease by RECIST (Response Evaluation Criteria in Solid Tumors) criteria, reported well-controlled pain, and continued to perform her daily activities independently. Ongoing enrollment in a clinical trial evaluating TRK inhibitors in *MYB*-rearranged tumors was discussed.

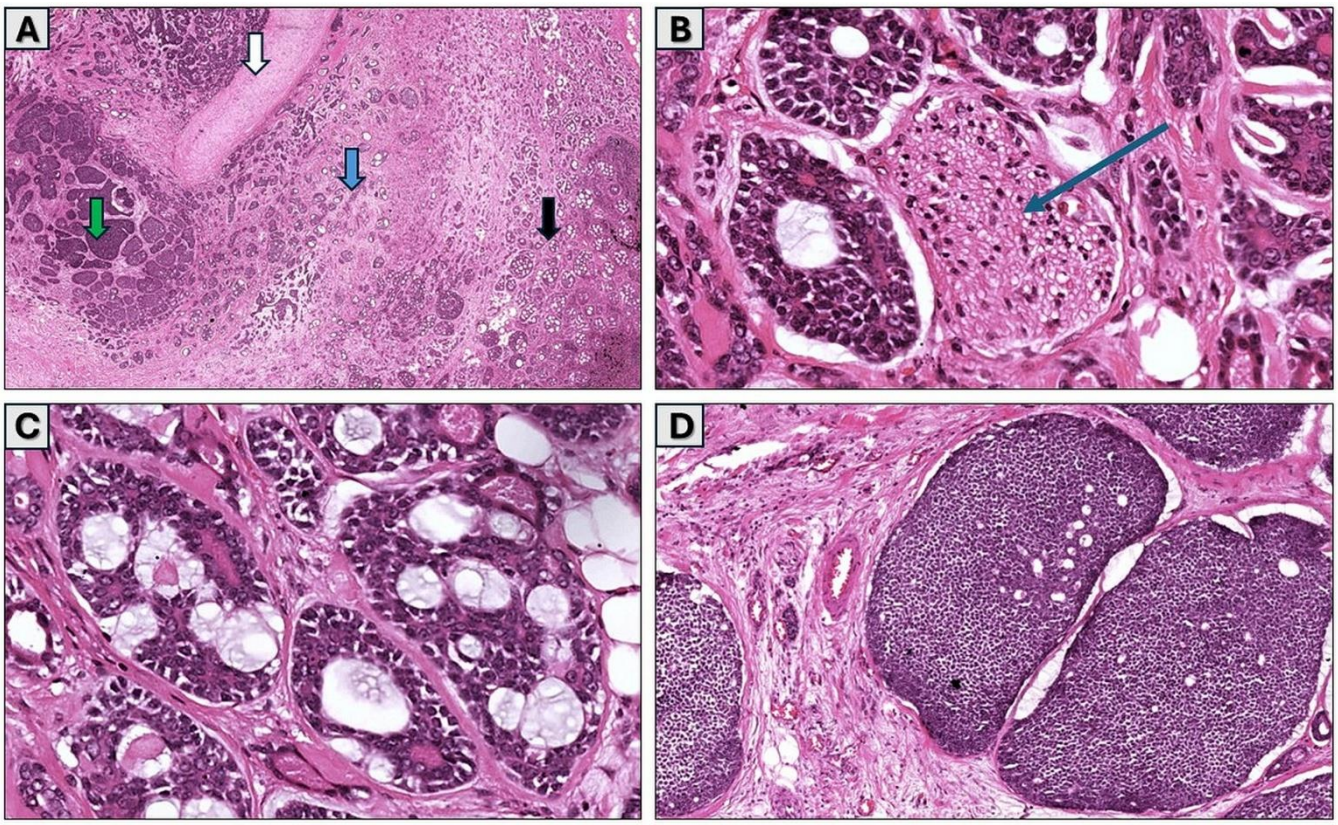

Figure 1 Microscopic examination of pulmonary adenoid cystic carcinoma

1A: Low-power view showing tumor infiltrating the bronchial tissue. The tumor displays mixed architectural patterns, including solid (green arrow), tubular (blue arrow), and cribriform (black arrow). The white arrow indicates the bronchial cartilage (H&E Stain X20).

- 1B: High power view showing tubular pattern with prominent perineural invasion (blue arrow) (H&E Stain X40).
- 1C: High power view showing cribriform pattern (H&E Stain X40)
- 1D: High power view showing solid pattern (H&E Stain X40)

3. Discussion

3.1. Background (History, epidemiology, risk factors, and WHO classification)

Pulmonary adenoid cystic carcinoma (PACC) is an uncommon malignant neoplasm of the respiratory tract that arises from the secretory glands of the tracheobronchial tree. [6] This lung tumor is classified as a salivary gland–type and exhibits similar morphological and molecular features to adenoid cystic carcinoma found in salivary glands. [4] Although rare, it represents one of the more frequently encountered primary tracheal malignancies and accounts for only a small fraction of all primary pulmonary tumors. [6,7]

Unlike the majority of primary lung cancers, PACC is not strongly associated with tobacco exposure and may occur across a wide age range, often affecting younger individuals compared with typical bronchogenic carcinomas. [4] The tumor most commonly originates in the central airways, particularly the trachea and main bronchi, where its slow but infiltrative growth can progressively compromise the airway lumen. [7] As a result, patients often present with nonspecific respiratory complaints such as persistent cough, wheezing, dyspnea, or hemoptysis, symptoms that may initially be mistaken for more common obstructive airway diseases. [8]

From a histopathologic perspective, pulmonary adenoid cystic carcinoma demonstrates distinctive architectural patterns including cribriform, tubular, and solid arrangements composed of epithelial and myoepithelial cell populations embedded within a hyalinized stromal matrix. [4] Perineural invasion and submucosal extension are

frequently observed and contribute to the tumor's local spread along the airway wall. [8] These features can make complete surgical excision difficult, even when the tumor appears well localized on imaging studies. [7]

Management of PACC primarily involves surgical resection, when possible, as this approach offers the best opportunity for long-term disease control. [6] However, the tumor's tendency for microscopic extension beyond visible margins often also requires radiotherapy to improve local control. [8] Despite its relatively slow course compared with other pulmonary malignancies, late recurrence and distant metastases have been reported, undermining the importance of prolonged surveillance following treatment. [7,9]

3.2. Pathogenesis, Pathophysiology

Primary pulmonary adenoid cystic carcinoma (PACC) is understood to arise from a dual population of myoepithelial and ductal cells within the submucosal glands of the tracheobronchial tree. [10] Its pathogenesis is most driven by a characteristic chromosomal translocation, t(6;9), which creates a MYB-NFIB gene fusion. This fusion oncogene, present in approximately 40–50% of PACCs, leads to the overexpression of the MYB oncoprotein. [3,12] When MYB is activated, it promotes tumor formation by increasing the expression of genes that control the cell cycle, cell growth, and survival. This includes upregulating BCL2, a protein that prevents apoptosis. [11] This molecular underpinning correlates with the tumor's clinical behavior: an indolent but persistent growth pattern characterized by a high propensity for local infiltration. A key pathophysiologic feature is perineural invasion, a mechanism facilitated by the tumor's interaction with the nerve microenvironment, in part through the expression of neurotrophic factors such as brain-derived neurotrophic factor (BDNF). [13] This relentless, infiltrative nature along nerve sheaths and submucosal planes explains the high rates of local recurrence and the challenge in achieving negative surgical margins, as seen in this case.

3.3. Comparative Analysis of Our Case with Existing Literature. (Clinical, radiology, pathology, lab, diagnosis, management, and outcome)

This case of pulmonary adenoid cystic carcinoma (PACC) mirrors several of the patterns reported in the literature but also reflects many of the diagnostic and management difficulties associated with this tumor, making early detection difficult. The patient in this case had a long history of cough, shortness of breath, and intermittent wheezing that had initially been treated as chronic bronchitis and, later, as late-onset asthma. This is a typical central airway ACC presentation. Due to the slow growth of these tumors and often the lack of systemic symptoms, many patients will be treated for asthma or recurrent infections, only to find that imaging studies will ultimately reveal an airway lesion. [8,14] Reported cases commonly involve an adult in their 40s to 60s who has similar nonspecific respiratory complaints and limited constitutional symptoms, as was the case with our patient. [6]

The findings from the imaging study and diagnostic testing closely correlate with those previously reported. Imaging of the PACC typically shows it as an intraluminal or circumferential mass (or both) in the tracheobronchial tree. They have smooth edges and some degree of mediastinal invasion. [8] PET scans show that PACC displays mild to moderate FDG uptake. The amount of FDG that the tumors take up is consistent with a relatively lower aggressiveness when compared to other bronchogenic carcinomas. [14] Both the CT and PET scan images from our patient were compatible with these findings. They matched the tumors' characteristic features as defined in the patient's histologic findings. The tumors showed cribriform, tubular, and solid growth patterns, with basaloid and myoepithelial cells, and often exhibited perineural invasion. IHC studies show PACC tumors typically stain positive for CK7, p63, SOX10, S100, and CD117 [15], and over 70% have MYB-NFIB chromosomal translocation. [16] Based upon the above descriptions of our patient's characteristics, we were able to distinguish between a variety of different tumor types, including carcinoids, mucoepidermoid carcinomas, and basaloid squamous cell carcinomas, which have all been well-described in literature as a considerable differential diagnosis. [14]

The management of the patient that aligns with currently recommended treatment courses of action continues to involve surgical resection and reconstruction of the airway in patients with localized PACC. Clear margins in localized PACC are hard to achieve due to its submucosal airway infiltration. [17] Our patient underwent carina resection and reconstruction, followed by adjuvant radiation therapy due to microscopic margin involvement. This treatment strategy is supported by small case series demonstrating improved local control after carinal resection with adjuvant treatments. [2] The treatment of patients with PACC, who are struggling to make decisions regarding management because of the complexity and coordination of both the surgical treatment and the perioperative management of these multidisciplinary tumors, is supported by tumor board discussions that help guide the treatment of these patients. [8] In the case of metastatic disease, the use of traditional chemotherapy has been of limited value. However, there is some evidence from small clinical trials that targeted therapies such as lenvatinib and other multikinase inhibitors have produced some positive clinical results. [5] The use of lenvatinib in our patient reflects these emerging approaches.

The findings of this case also demonstrated that patients with PACC should have close long-term follow-up for several reasons. Although these tumors generally behave indolently, late recurrences and distant metastases to the lung and bone may occur years after initial treatment. [6,14] The bilateral pulmonary and osseous metastases that developed in this patient approximately 7 years after the initial surgery are consistent with this pattern and emphasize that long-term follow-up is necessary. Most patients experience extended survival after recurrence because the tumor grows slowly, as shown in this case. [8]

Overall, this case further emphasizes findings in the literature that pulmonary adenoid cystic carcinoma often has vague respiratory symptoms, distinct central airway imaging and histologic findings, complex surgical planning, and a high rate of late recurrence despite initial control. Identifying these patterns will help clinicians to consider unusual airway tumors earlier in patients who have chronic "asthma-like" symptoms that do not respond to treatment.

4. What Have We Learned from This Case?

This case provides several critical clinical insights. First, it underscores a key diagnostic lesson: PACC can masquerade as common respiratory conditions such as asthma or bronchitis, leading to significant diagnostic delays. The presence of a fixed obstructive pattern on pulmonary function tests and subtle radiographic abnormalities in a patient with refractory symptoms should serve as a red flag, prompting early bronchoscopic evaluation for a central airway lesion. Second, this case powerfully reinforces the natural history of PACC, defined by an indolent course but a high propensity for late distant recurrence.

The development of metastatic disease seven years after definitive primary treatment highlights the absolute necessity of lifelong oncologic surveillance. Clinicians must remain vigilant, as a patient is never truly considered cured.

Finally, the management of this patient's metastatic disease illustrates an evolving, effective paradigm. It demonstrates that for low-burden, slow-growing recurrences, active surveillance is a viable initial strategy. Upon progression, the use of targeted systemic therapy, such as the multikinase inhibitor lenvatinib, combined with local ablative treatments, such as SBRT, at symptomatic sites can achieve durable disease control while preserving quality of life, challenging the historically nihilistic view of metastatic PACC.

Abbreviations

- Primary pulmonary adenoid cystic carcinoma (PACC);
- Immunohistochemistry IHC);
- Stereotactic body radiation therapy (SBRT).

5. Conclusion

This case provides several critical clinical insights. First, it underscores a key diagnostic lesson: PACC can masquerade as common respiratory conditions such as asthma or bronchitis, leading to significant diagnostic delays. The presence of a fixed obstructive pattern on pulmonary function tests and subtle radiographic abnormalities in a patient with refractory symptoms should serve as a red flag, prompting early bronchoscopic evaluation for a central airway lesion. Second, this case powerfully reinforces the natural history of PACC, defined by an indolent course but a high propensity for late distant recurrence. The development of metastatic disease seven years after definitive primary treatment highlights the absolute necessity of lifelong oncologic surveillance. Clinicians must remain vigilant, as a patient is never truly considered cured. Finally, the management of this patient's metastatic disease illustrates an evolving, effective paradigm. It demonstrates that for low-burden, slow-growing recurrences, active surveillance is a viable initial strategy. Upon progression, the use of targeted systemic therapy, such as the multikinase inhibitor lenvatinib, combined with local ablative treatments, such as SBRT, at symptomatic sites can achieve durable disease control while preserving quality of life, challenging the historically nihilistic view of metastatic PACC.

Compliance With Ethical Standards

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Disclosure of conflict of interest

All authors make the following declarations:

- Payment/services information: All authors have declared that they received no financial support from any organization for the submitted work.
- Financial relationships: All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might be interested in the submitted work.

Statement of ethical approval

Ethical review and approval were not required for this study involving human participants. The paper has been sufficiently anonymized to maintain the patient's confidentiality.

Statement of informed consent

This study was conducted as a retrospective review of archival pathology material collected during routine clinical care. All data were fully de-identified prior to analysis. No patient contact or intervention was involved, and informed consent was not required in accordance with institutional policies.

Data access statement

All relevant data are included in the paper.

Author contributions

All authors contributed equally to producing this manuscript.

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