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Primary Bronchial Adenoid Cystic Carcinoma with Locally Invasive Mediastinal Progression: A Rare Case Report and Radiological Dilemma

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Abstract | **Case Report**

Primary adenoid cystic carcinoma (ACC) of the tracheobronchial arbor is an exceptionally rare malignancy originating from submucosal seromucinous glands, accounting for less than 0.2% of primary lung tumors. While typically centered in the trachea, primary involvement of the mainstem bronchus is exceedingly unusual. It displays slow volumetric progression coupled with relentless transmural, perineural, and mediastinal infiltration, frequently confounding initial diagnosis. Primary bronchial ACC is an insidious entity where luminal tumor downsizing does not ensure biological resectability. High-resolution multiplanar MDCT remains indispensable to map transmural mediastinal and vascular encasement, detect therapy-related lung injury, and screen for osteoblastic metastases.

Keywords: Adenoid cystic carcinoma; Left main bronchus; Mediastinal invasion; Aortic encasement; Multidetector CT; Restaging.

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INTRODUCTION

Primary salivary gland-type tumors of the lower respiratory tract represent fewer than 0.1% to 0.2% of all pulmonary malignancies [1]. Among these, adenoid cystic carcinoma (ACC) arises from the intercalated duct elements of submucosal seromucous glands. Although ACC represents up to 30%–50% of primary tracheal malignancies, its primary origin within the lobar and mainstem bronchi is extraordinarily rare, accounting for only a small subset of central airway lesions [2,3].

Unlike conventional non-small cell lung carcinomas (NSCLC), tracheobronchial ACC classically lacks a causal association with tobacco smoking and affects younger patients across both sexes [3,4]. However, when diagnosed in a male chronic smoker, it creates a diagnostic trap, as clinical and radiologic manifestations are routinely attributed to smoking-induced bronchogenic carcinomas. Biologically, ACC is characterized by an "iceberg" phenomenon: despite an indolent mitotic index, it exhibits aggressive longitudinal submucosal tracking and perineural spread extending far beyond the endoluminal tumor margin [4,5].

On multidetector computed tomography (MDCT), it typically appears as a sessile polypoid mass or circumferential mural thickening with extraluminal extension [5]. Because complete surgical resection (R0) is curative but frequently prevented by silent mediastinal extension, multiplanar CT characterization of tumor interfaces with vital structures is paramount. We describe the MDCT features of locally invasive left mainstem bronchial ACC in a 47-year-old male smoker under systemic therapy.

CASE PRESENTATION

A 47-year-old male chronic smoker was managed in the oncology department for a primary left mainstem bronchial neoplasm, initially discovered during the evaluation of chronic cough and progressive exertional dyspnea. Flexible bronchoscopy with endobronchial biopsy confirmed adenoid cystic carcinoma displaying a predominant cribriform/tubular architectural pattern. Baseline contrast-enhanced CT staged the disease as locally advanced cT4N2bM0 owing to primary lesion volume, direct mediastino-vascular involvement, and regional nodal disease. The multidisciplinary tumor board initiated systemic platinum-based doublet chemotherapy.

A restaging thoraco-abdominopelvic CT examination was performed with volumetric millimetric collimation before and after intravenous iodinated contrast injection during the portal venous phase a tissue-

attenuation mass centered on the left mainstem bronchus causing subtotal luminal narrowing with an anterior intraluminal protrusion and homogeneous enhancement. partial primary tumor regression (18 × 37 × 42 mm vs. 38 × 49 × 60 mm) with clearance of downstream mucoid impaction.

- *Medially:* Transmural infiltration of the adjacent thoracic esophagus with loss of intervening fat planes.
- *Laterally:* Direct abutment of the left pulmonary artery trunk with obliteration of the fat cleavage line.
- *Posteriorly:* Semicircular (180°) circumferential encasement of the descending thoracic aorta.

Parenchymal and Interstitial Findings:

Progression of bilateral apical pleural capping and fibrotic retractile consolidation, with new retractile bands in the right upper lobe accompanied by architectural distortion and traction bronchiectasis. Bilateral branching centrilobular "tree-in-bud" micronodules persisted, alongside ground-glass opacities, septal thickening, trace left pleural fluid, and a minimal pericardial effusion.

Subdiaphragmatic and Skeletal Findings:

Abdominal and pelvic viscera were normal, and deep lymph nodes remained strictly subcentimetric. On the bone window, dense focal sclerotic lesions were identified in the posterior elements of the S2 and S3 sacral segments, suspicious for secondary deposits and warranting skeletal scintigraphic correlation.

Iconography

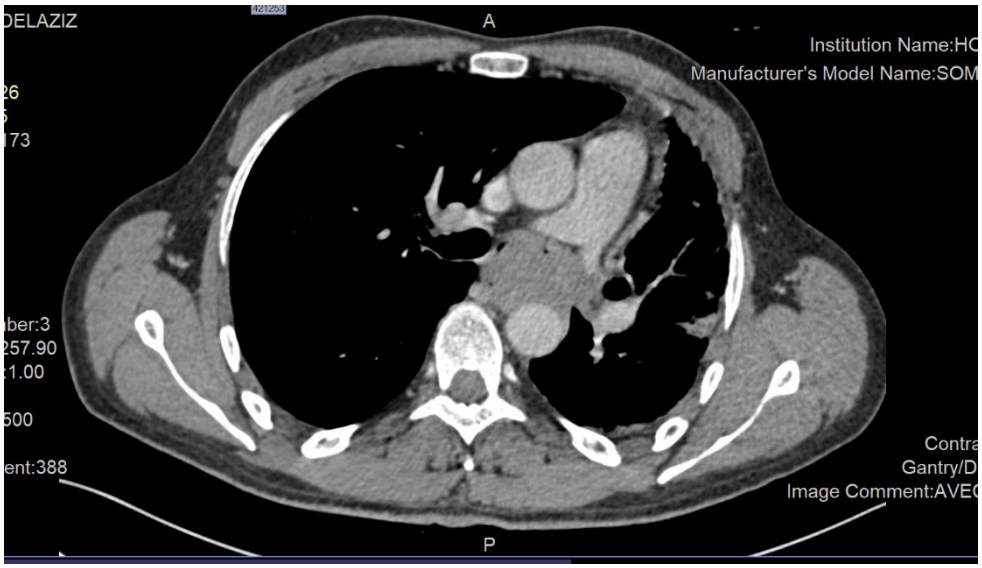


Figure 1: Contrast-enhanced axial thoracic CT scan (mediastinal window)

Demonstrates the soft-tissue tumor centered on the left mainstem bronchus, resulting in subtotal luminal obstruction, loss of fat planes with the left pulmonary artery, direct esophageal infiltration medially, and 180-degree circumferential encasement of the descending aorta posteriorly.

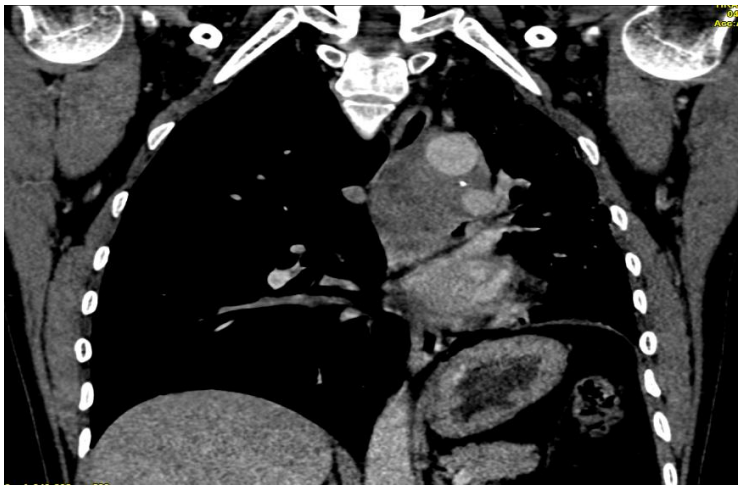


Figure 2: Coronal multiplanar CT reconstruction (mediastinal window)

Illustrates the craniocaudal extent of the infiltrating left mediastinohilar mass along the primary bronchial bifurcations.

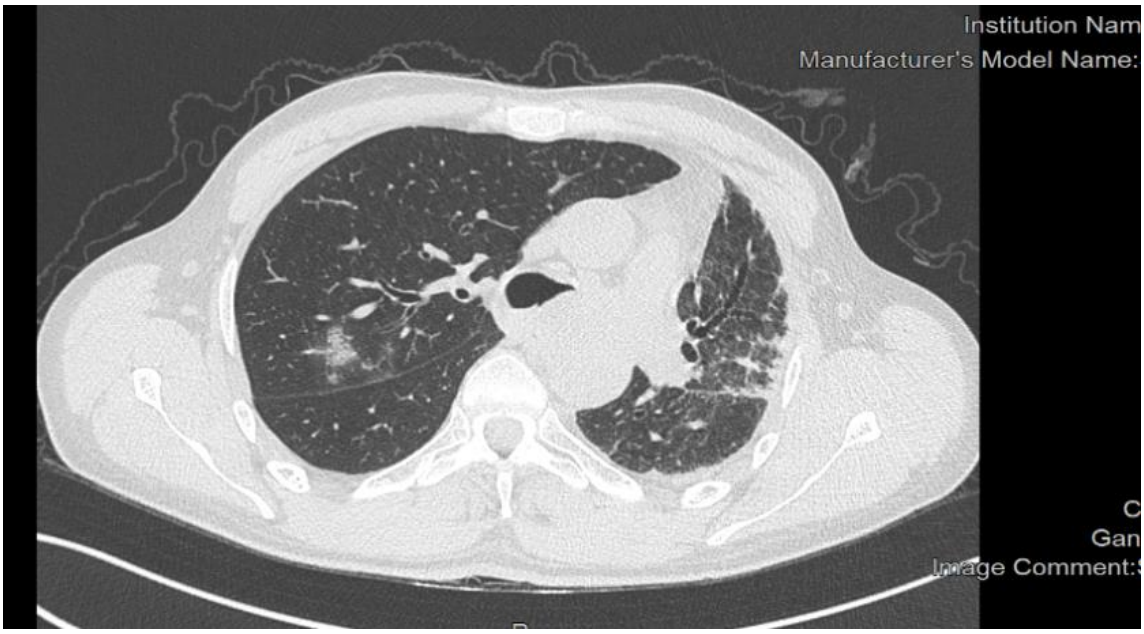


Figure 3: High-resolution CT scan of the lung parenchyma (lung window)

Displays post-therapeutic parenchymal changes, including retractile fibrotic bands, traction bronchiectasis, patchy ground-glass attenuation, and peribronchovascular thickening.

DISCUSSION

Rarity of Bronchial Localization

Primary bronchial ACC remains a clinical rarity, representing <0.2% of primary lung neoplasms [1,2]. The majority of airway ACCs reside within the proximal trachea; presentation in the mainstem bronchus of a middle-aged male chronic smoker routinely delays correct histological identification, as it closely mimics smoking-related NSCLC [2,4].

The definitive radiologic hallmark of bronchial ACC is its disproportionate extraluminal infiltration relative to its endobronchial appearance. The intraluminal component seen on bronchoscopy often represents only the "tip of the iceberg," while tumor cells track microscopically along perineural sheaths and through cartilage rings [4,5]. In this patient, MDCT accurately mapped three criteria of T4 non-resectability:

1. **Aortic Encasement:** 180° encasement of the descending thoracic aorta, a standard sign of adventitial invasion [5].
2. **Esophageal Infiltration :** Obliteration of the para-esophageal fat space with direct mural thickening.
3. **Pulmonary Artery Abutment:** Disappearance of the cleavage plane bordering the main left pulmonary artery.

Although systemic chemotherapy achieved marked luminal downsizing (18 × 37 × 42 mm) and resolved distal obstructive atelectasis, persistent vascular encasement confirms that volumetric shrinkage does not equate to complete surgical resectability in ACC.

Radiological Differential Diagnosis

- **Squamous Cell Carcinoma (SCC):** The most frequent central airway malignancy in male smokers. Unlike the homogeneous enhancement and indolent course of ACC, SCC routinely exhibits central liquefactive necrosis, thick cavitary walls, and early bulky mediastinal lymphadenopathy [3,5].
- **Bronchial Carcinoid:** Characterized by avid, early arterial enhancement and intratumoral punctate calcifications. Typical carcinoids remain well demarcated endoluminally without extensive perivascular encasement [5].
- **Mucoepidermoid Carcinoma (MEC):** Tends to present as a discrete, intraluminal polypoid mass, with peripheral calcifications and far less transmural perineural tracking than ACC [1,3].

Evaluation of Secondary Parenchymal Changes and Skeletal Staging

Beyond the primary site, MDCT identified treatment-related lung injury (traction bronchiectasis, retractile scarring, septal thickening) and tree-in-bud opacities, which raise concern for microaspiration due to esophageal invasion. Furthermore, while early lymphatic spread is uncommon in ACC, late hematogenous dissemination occurs in up to 10%–12% of patients [2,6]. The dense sclerotic lesions at S2–S3 illustrate the propensity of ACC for delayed osteoblastic skeletal dissemination, requiring focused nuclear or MR imaging confirmation.

CONCLUSION

Primary adenoid cystic carcinoma of the left main bronchus is an uncommon airway neoplasm whose indolent histology belies an aggressive, creeping transmural growth pattern. Multiplanar contrast-enhanced MDCT is pivotal to identify critical vascular and esophageal invasion, confirm primary non-resectability, monitor post-chemotherapy downsizing, and detect parenchymal toxicities or late skeletal metastases.

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