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FDG-avid pulmonary mucous gland adenoma mimicking lung cancer on 18 F-FDG PET/CT: a rare case report and literature review

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Abstract

Background Pulmonary mucous gland adenoma (MGA) is an exceptionally rare benign tumor. Even with the assistance of 18 F-FDG PET/CT, the accurate diagnosis of MGA as lung cancer remains challenging. Only one case of fluorodeoxyglucose(FDG)-avid pulmonary mucous gland adenoma and two case of low FDG uptake pulmonary mucous gland adenoma have been reported in English literature, while a single case of moderately increased FDG uptake pulmonary mucous gland adenoma has been documented in French literature. To minimize misdiagnosis and select appropriate treatment strategies, it is crucial to comprehensively analyze its 18 F-FDG PET/CT manifestations in conjunction with clinical symptoms and pathological findings.

Case presentation In our study, we present a case involving a 70-year-old woman with clinical manifestations of persistent cough and sputum with an FDG-avid mucous gland adenoma mimicking lung cancer on 18 F-FDG PET/CT imaging. Ultimately, the patient underwent a potentially unnecessary video-assisted thoracoscopic lobectomy, and the pathological diagnosis was determined to be MGA. The patient was discharged and remained clinically well without any complaints for a period of 6 months.

Conclusions The use of 18 F-FDG PET/CT lacks specificity in detecting MGA and may lead to misdiagnosis as a lung malignancy. A comprehensive analysis combining clinical manifestations, bronchoscopy findings, imaging results, and pathological findings is essential for accurate identification of pulmonary mucus gland adenoma.

Keywords Pulmonary mucous gland adenoma, 18F-FDG PET/CT, Lung cancer

Introduction

Pulmonary mucous gland adenoma (MGA) is an exceedingly rare benign tumor of the lung epithelium [1], originating from the larger airway mucosa's glands that secrete mucus [2]. The majority of cases are centrally located and arise from the bronchus, with only a few occurring in peripheral airways. The accurate differentiation between pulmonary mucous gland adenoma and lung cancer is challenging due to their similarities in imaging features and clinical presentations, necessitating pathological diagnosis for definitive distinction. The 18 F-FDG PET/CT findings of MGA have not

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been extensively characterized thus far. Only one case of fluorodeoxyglucose(FDG)-avid pulmonary mucous gland adenoma and two case of low FDG uptake pulmonary mucous gland adenoma have been reported in English literature, while a single case of moderately increased FDG uptake pulmonary mucous gland adenoma has been documented in French literature. The 18 F-FDG PET/CT findings of MGA have not been extensively characterized thus far. The use of 18 F-FDG PET/CT lacks specificity in detecting MGA and may lead to misdiagnosis as a lung malignancy.

Case presentation

The patient, a 70-year-old woman, was admitted to the hospital due to a persistent cough lasting over 10 months. Initially, the cough was dry but later progressed to include white sticky sputum. No other symptoms such as fever, chills, hemoptysis or chest pain were observed and physical examination did not reveal any abnormalities. The medical, family, and psycho-social history including relevant genetic information of this patient were normal. There were no hypertension, diabetes and other basic diseases of this patient who was non-smoker. The laboratory studies also showed results within normal ranges.

A chest computed tomography(CT) scan conducted at the fourth month revealed an irregular patchy shadow measuring 5.0 × 2.6 cm in the right lower lobe of the lung with obstruction of the bronchus in the superior segment (Fig. 1A). Fiberoptic bronchoscopy confirmed that jelly-like material obstructed the bronchus in question (Fig. 1B). Biopsy results from bronchoscopy slides did not indicate malignant cells. The patient opted for clinical observation and underwent another chest computed tomography after six months which revealed an irregular mass measuring 5.2 × 2.9 cm in size in the right lower lung (Fig. 1C/1D), indicating tumor growth compared to half a year ago and suggesting malignancy based on clinical assessment. Further evaluation using 18 F-FDG PET/CT demonstrated high uptake of FDG in the proximal area of the mass with the maximum standardized uptake value(SUVmax) of 5.1 (Fig. 2), while no increased FDG uptake was observed distally from this region within the mass itself. Consequently, a clinical diagnosis of pulmonary malignancy was made and subsequently treated

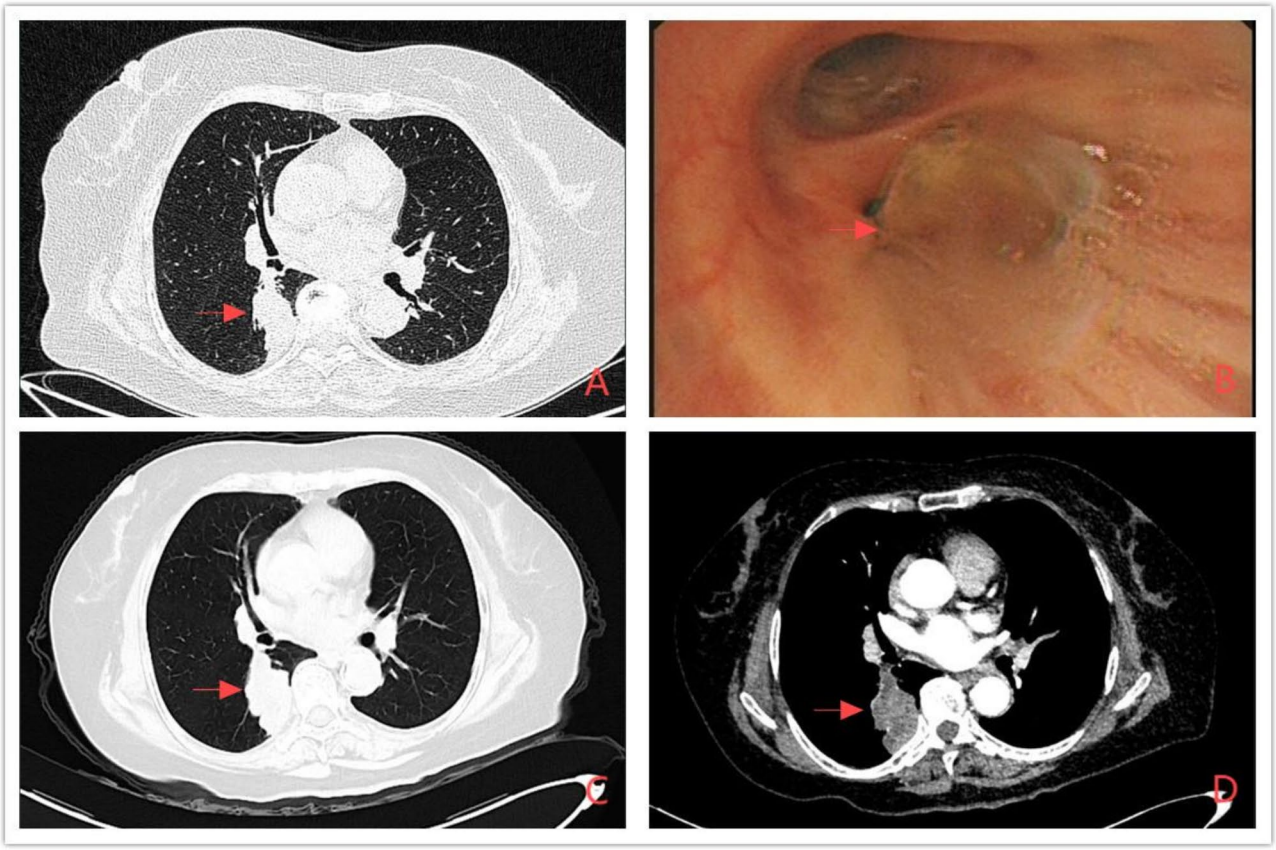

Fig. 1 **A**: Chest computed tomographic scan revealed a 5.0 × 2.6 cm irregular patchy shadow in the right lower lung (arrow) six months prior to surgery. **B**: Fiberoptic bronchoscopy demonstrated obstruction of the bronchus in the superior segment of the right lower lung by a jelly-like material (arrow) six months before surgery. **C/D**: Subsequent chest computed tomographic scans exhibited an irregular mass measuring 5.2 × 2.9 cm in the right lower lung (arrow), representing an increase compared to Fig. 1A, observed after six months

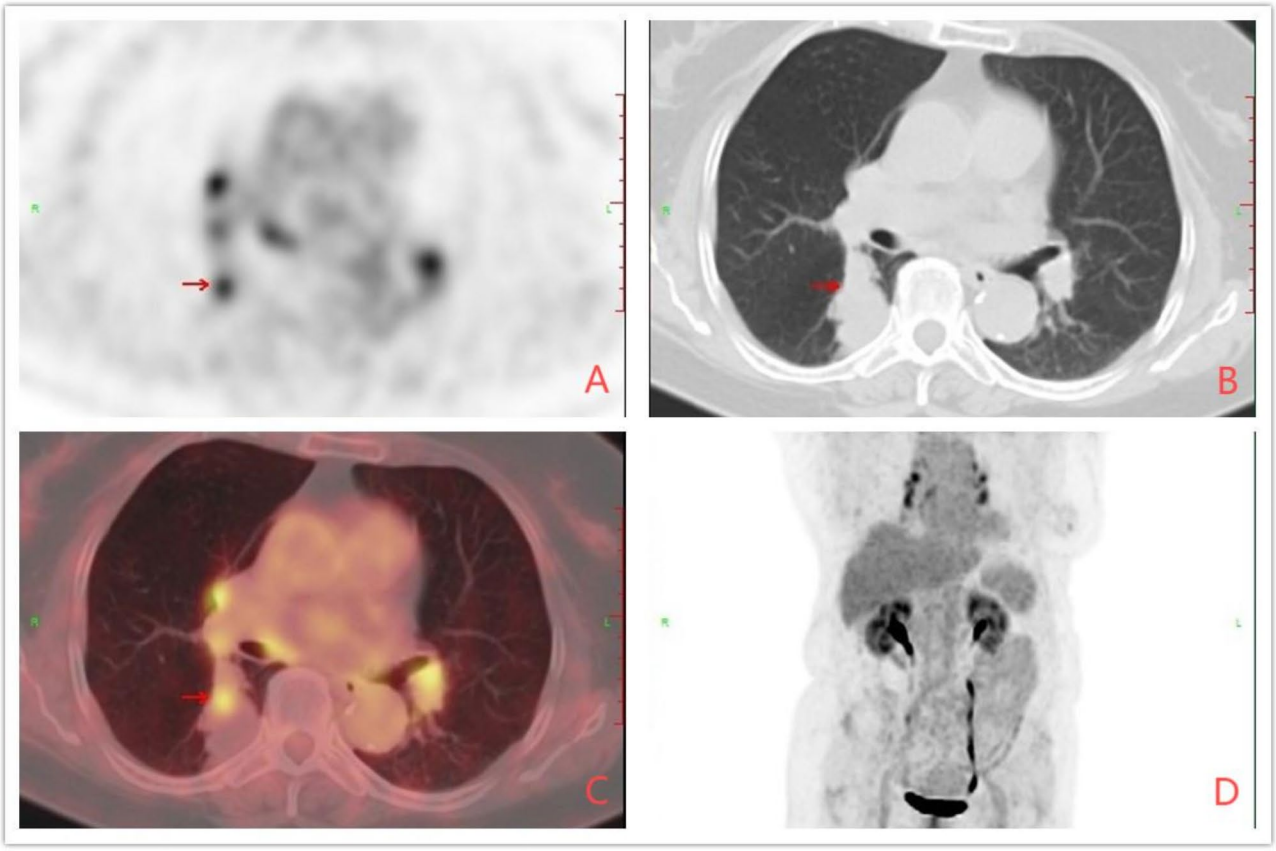

Fig. 2 18 F-FDG PET/CT demonstrated elevated FDG uptake in the proximal region of the mass, with a maximum standardized uptake value (SUVmax) of 5.1, while no increased FDG uptake was observed in the distal area

through right lower lobectomy surgery which intraoperative frozen section analysis indicated presence of benign tumor. The patient's persistent cough disappeared after surgery with no complications.

Histologically, a jelly-like lesion measuring 5.5 cm in maximum diameter was observed in the right lower lobe. Within the lesion (Fig. 3A), a nodule measuring $0.7 \times 0.5 \times 0.3$ cm was identified and diagnosed as mucinous adenoma. There was no infiltration between the tumor and the surrounding normal alveolar tissue, and a distinct pushing margin was formed as a result of the expansive growth of the tumor. It predominantly consisted of irregular glands, acini, and tubules lined by single-layered columnar or cuboidal cells exhibiting clear cytoplasm, basally situated nuclei with fine chromatin distribution. Papillary growth was evident within the tumor and low-power view revealed the presence of a vascular axis (Fig. 3B). At high magnification, papillae were characterized by cuboidal or columnar epithelium displaying basophilic cytoplasm, small nuclei with uniform chromatin distribution devoid of obvious nucleoli or mitotic figures (Fig. 3C). The bronchial margin showed no evidence of involvement. Immunohistochemistry demonstrated positive staining for CK7 (Fig. 4A) and P53 (Fig. 4B), while negative staining was observed for TTF-1 (Fig. 5A), NapsinA (Fig. 5B), PD-L1 (Fig. 5C), P40 (Fig. 5D), as well as CK5/6 and ALK(D5F3). The Ki-67 proliferation index was determined to be 2% (Fig. 3D). The ultimate pathological diagnosis was determined to be MGA. Following surgery, the patient was discharged and remained clinically well without any complaints for a period of 6 months.

Discussion and conclusions

The first report of MGA dates back to 1882 when Muller identified it as a distinct pathological entity separate from lung carcinoma, initially naming it bronchial adenoma arising from mucous gland [3]. Previous literature on MGA primarily consists of case reports, with limited available information. Gender and age distribution of MGA cases showed no significant differences, affecting both men and women equally across all age groups (mean age: 52), including children [2]. Patients with MGA may either be asymptomatic or present with nonspecific symptoms such as cough, wheezing, and obstructive pneumonitis resulting from the endobronchial mass [4, 5]. Some patients may also be misdiagnosed with pneumonia or chronic obstructive pulmonary

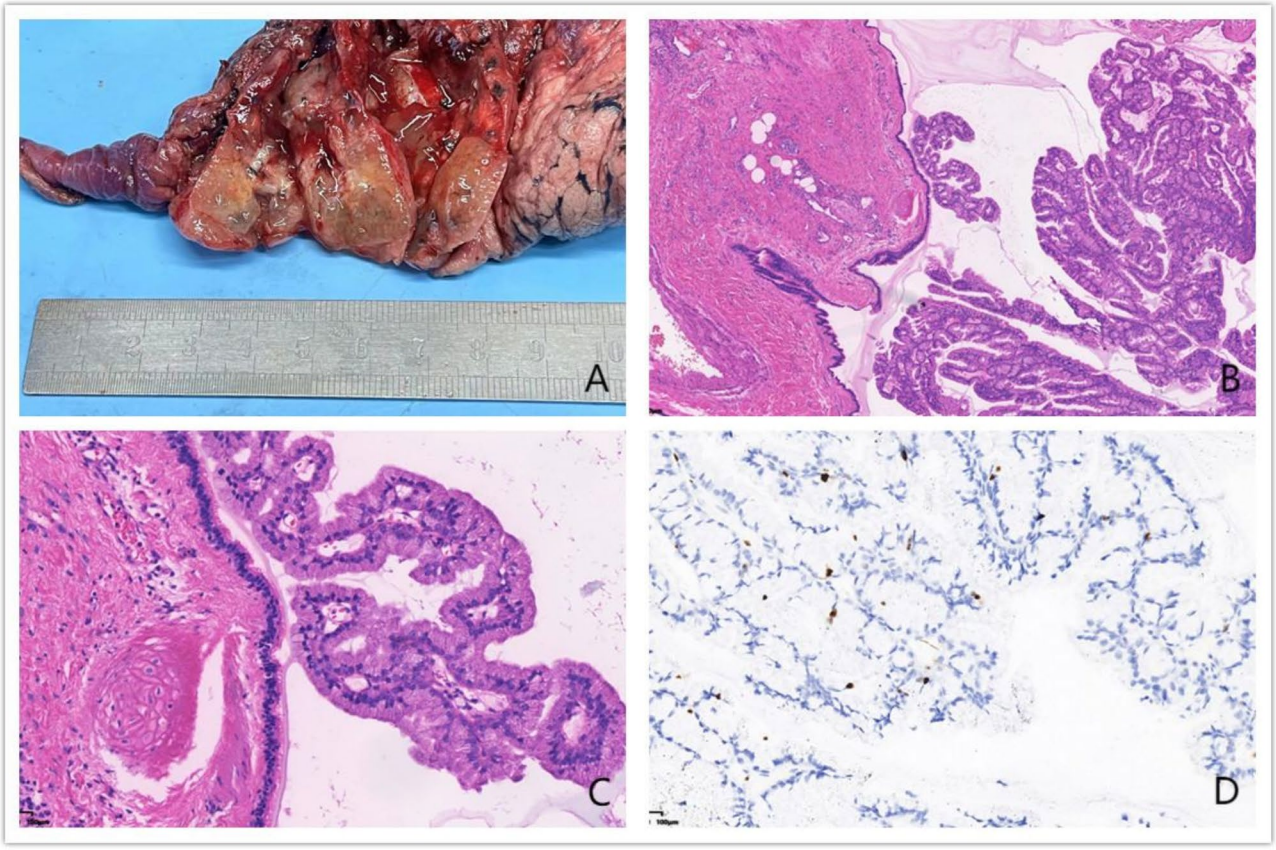

Fig. 3 **A**: In the right lower lobe, a jelly-like lesion measuring 5.5 cm in maximum diameter was observed, with a central nodule measuring 0.7 × 0.5 × 0.3 cm. **B**: The tumor exhibited papillary growth and displayed a vascular axis when examined at low power view (H-E×4). **C**: At high magnification (H-E×20), the papillae were lined with cuboidal or columnar epithelium characterized by basophilic cytoplasm, small nuclei with uniform chromatin, absence of obvious nucleoli, and lack of mitotic figures. **D**: Immunohistochemistry revealed a Ki-67 proliferation index of 2%

disease. Additionally, there have been rare instances where hemoptysis occurs due to tumor necrosis in patients with MGA [6].

Under bronchoscopy, MGA can be observed as a well-defined intraluminal mass that is firm, smooth, shiny, and occasionally pedunculated with a pink or white appearance. It partially or completely obstructs the bronchus of the lobe or segment. The tumor typically exhibits sessile fixation but may also present with a pedicle occasionally [3].

On imaging, mucous gland adenoma (MGA) was observed as a well-defined nodule with an air crescent sign in the bronchial region [7], or as a pulmonary mass [8] or peripheral pulmonary nodule [9, 10, 11] on chest computed tomography scan. Although 18 F-FDG PET/CT is considered the most accurate imaging tool for differentiating malignant and benign pulmonary nodules, there is still ongoing discussion regarding the optimal cutoff value of standardized uptake value (SUV). A maximum SUV (SUVmax) > 2.5 has commonly been used as a diagnostic threshold strongly suggesting malignancy [12, 13]. To our knowledge, only three cases of MGA detected by 18 F-FDG PET/CT have been reported in English literature and one case in French literature. Jaehyuk et al. [14] reported a case of FDG-avid MGA mimicking lung cancer on 18 F-FDG PET/CT with an SUVmax of 4.9. Based on the findings from 18 F-FDG PET/CT indicating possible malignancy, video-assisted thoracoscopic surgery was performed to obtain a biopsy sample. Ahmet et al. [10] reported a case of low FDG uptake MGA with an SUVmax of 2.0. Despite the low uptake observed in PET imaging, they decided to perform resection to confirm the diagnosis based on information suggesting that certain lung cancers such as carcinoid tumors can exhibit low SUV levels despite their malignant nature. Mini-thoracotomy and wedge resection were performed during the same session. Komatsu et al. [11] also reported a case of low FDG uptake MGA with an SUVmax of 2.08. The case involved a patient with a peripheral lung nodule containing a cavity who underwent wedge resection of the lung. Intraoperative frozen section pathology raised suspicion for adenocarcinoma, leading to a right lower lobectomy and mediastinal lymphadenectomy. However, the final postoperative pathological diagnosis confirmed

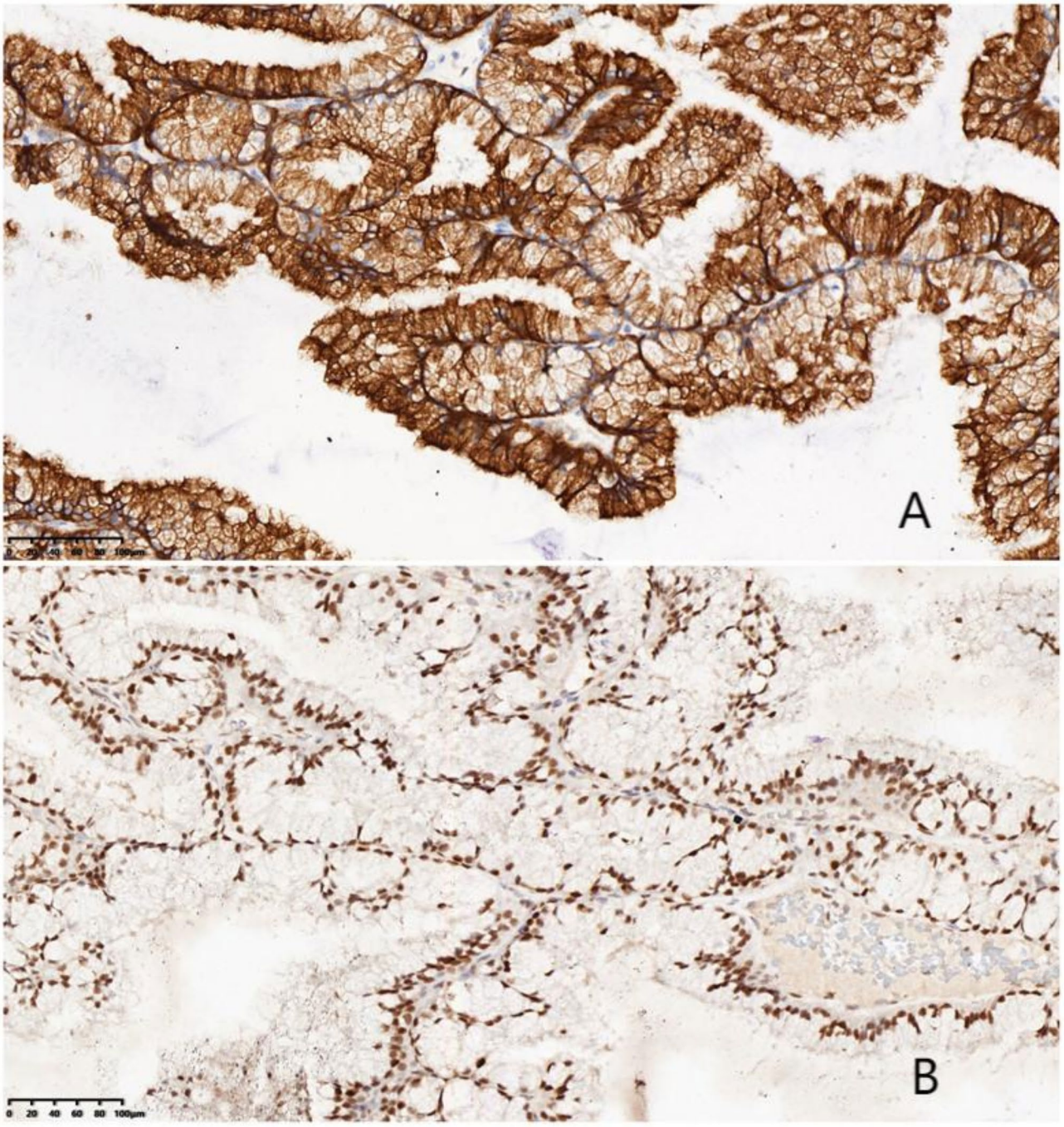

Fig. 4 The immunohistochemical analysis revealed positive staining for CK7 (**A**) and P53 (**B**)

MGA. Vergnenègre et al. [15] reported a case of pulmonary mucous gland adenoma with moderately increased FDG uptake, exhibiting an SUVmax of 2.6. In their case, the MGA tumor was successfully resected using an endoscopic procedure. However, the characterization of MGA on 18 F-FDG PET/CT findings remains limited in the literature. Previous studies have shown that MGA can present with either high or low FDG uptake on 18 F-FDG PET/CT scans, lacking specificity and often leading to misdiagnosis as a pulmonary malignant tumor. Similarly, our clinical case was initially misdiagnosed as lung cancer due to its high FDG uptake (SUVmax 5.1) on 18 F-FDG PET/CT scan and subsequent enlargement observed on chest computed tomography follow-up; ultimately resulting in right lower lobectomy.

The MGA case we presented demonstrated high FDG uptake (SUVmax 5.1) but a low Ki-67 index (2%), which appears paradoxical. Similarly, the case reported

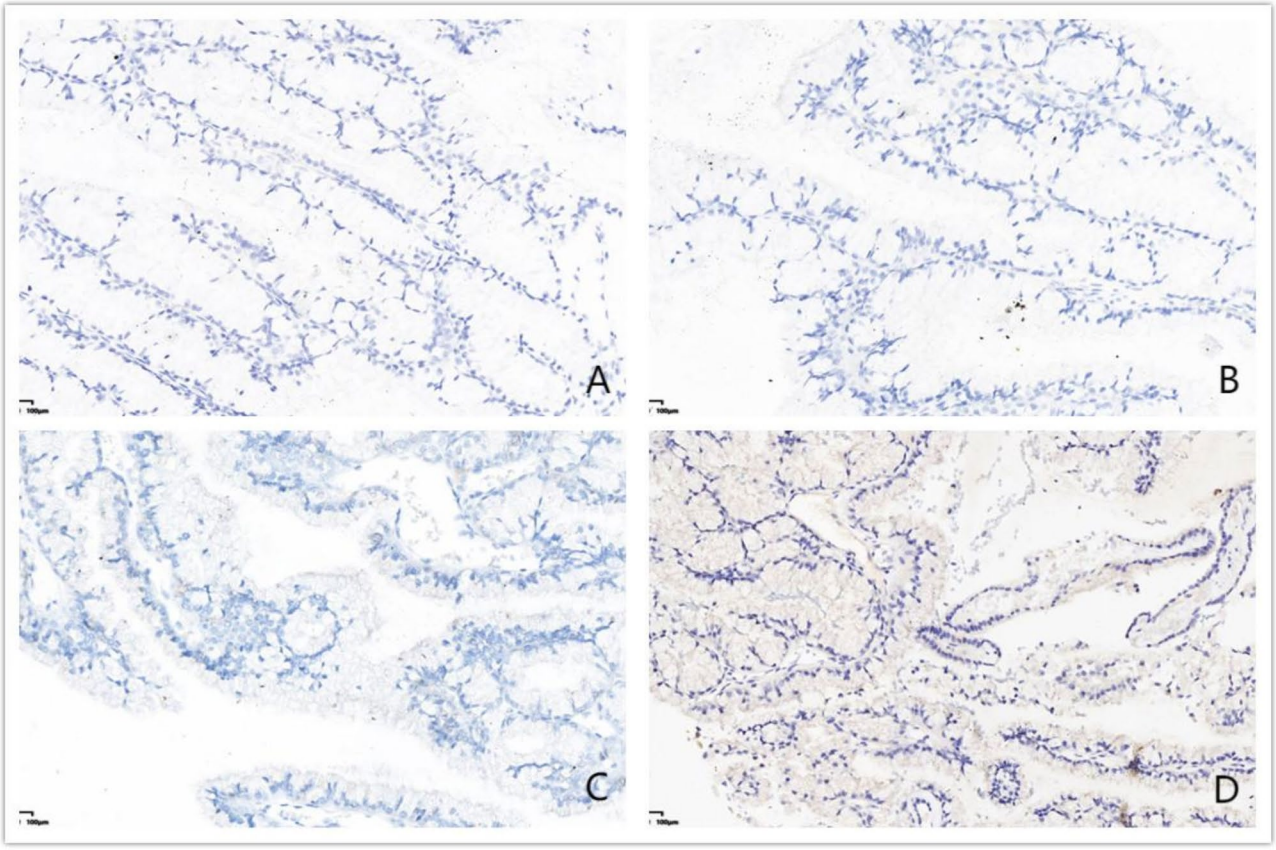

Fig. 5 The immunohistochemistry results demonstrated negative staining for TTF-1 (**A**), Napsin A (**B**), PD-L1 (**C**), and P40 (**D**)

by Ahmet et al. [10] exhibited slightly increased FDG uptake (SUVmax 2.0) with a low Ki-67 index (1%). To date, there is limited literature specifically addressing this phenomenon. However, some studies suggest that although the histological features of MGA are predominantly characterized by glandular hyperplasia, it may also exhibit various interstitial reactions, including hyalinization, inflammatory responses, cholesterol cleft granulomas, and cellular changes [16]. This implies that the elevated FDG uptake in MGA could originate from tumor cell metabolism, hypoxia within the tumor microenvironment, or contributions from inflammatory cells. Typically, a higher Ki-67 index correlates with increased metabolic activity of tumor cells. Tumors with lower Ki-67 indices generally proliferate more slowly but can still grow gradually. As tumors expand, hypoxic regions often develop within the tumor tissue. Hypoxia can activate signaling pathways such as hypoxia-inducible factor (HIF), leading to upregulation of glucose transporters and enhanced glucose uptake by tumor cells [17], thereby increasing FDG uptake. Additionally, the tumor microenvironment is frequently infiltrated by inflammatory cells, such as macrophages and lymphocytes. These cells not only perform immune surveillance but also release cytokines and inflammatory mediators that may influence tumor cell metabolism and promote FDG uptake [18]. Further research and accumulation of cases are necessary to substantiate these hypotheses.

Tumor cells in pulmonary mucinous adenocarcinoma(PMA) demonstrate aberrant fluorodeoxyglucose (FDG) uptake. On PET/CT imaging, this is characterized by localized radiotracer accumulation, with the standardized uptake value (SUV) varying to different extents [19]. However, compared to non-mucinous subtypes of lung cancer, FDG uptake in PMA may be relatively lower due to distinct biological behaviors and metabolic profiles of the tumor cells. In particular, highly differentiated PMA may exhibit only modest increases in FDG uptake, attributed to less aggressive cellular proliferation [20], and in certain instances, SUV values may approximate those of normal tissues [21]. This characteristic closely mirrors the PET/CT findings observed in MGA, contributing to its potential misdiagnosis as lung cancer in clinical settings.

The radiological characteristics of MGA have not been extensively described in the relevant literatures. Based on the analysis of reported case studies [2–10], chest-enhanced CT imaging of MGA typically exhibits well-defined margins, heterogeneous density, mucinous components, and mild enhancement. It predominantly

manifests as a central type, with peripheral presentations being less common. These features provide certain diagnostic clues. When combined with clinical symptoms and patient history, a preliminary diagnosis can be made. However, it is crucial to differentiate MGA from other lung tumors, such as lung cancer, to narrow down the differential diagnosis. Pulmonary mucinous adenocarcinoma exhibits a variety of CT features, which can generally be categorized into two types: nodular and pneumonia-like. According to Watanabe et al. [22], 75% of patients with pulmonary mucinous adenocarcinoma(PMA) present with solitary pulmonary nodules or masses, while the remaining 25% exhibit pneumonia-like patterns. Nodular lesions often appear as small, spiculated, lobulated, vacuolated, or associated with pleural traction signs. Most lesions are peripherally located, with only a few found centrally. Their attenuation values are similar to water but lower than muscle tissue. Pneumonia-like lesions display imaging findings similar to pneumonia, including air bronchogram signs but lacking foliate tree signs. Distinguishing MGA from PMA based solely on chest CT can be challenging. Therefore, while chest CT provides valuable information, it has limitations in differentiating between MGA and PMA. The definitive diagnosis ultimately relies on pathological examination.

Caruso D et al. [23] proposed that chest computed tomography texture analysis could potentially serve as a valuable tool in distinguishing between benign and malignant pulmonary nodules. Marin A et al. [24] suggested that an integrated approach combining dynamic 18 F-FDG PET/CT with perfusion CT-derived blood volume may provide assistance in differentiating between benign and malignant pulmonary nodules, especially when dealing with indeterminate cases. The advancements made in the fields of radiology and nuclear medicine are expected to offer significant benefits for the future diagnosis of MGA.

The review conducted by Michael P. Zaleski et al. [16] emphasized that the lack of specific imaging features in MGA located centrally or peripherally necessitates a histopathological basis for definitive diagnosis. Histologically, MGA is characterized by the presence of glandular cavities rich in mucus. Furthermore, the review emphasized the diverse histopathologic features of MGA, noting five distinct growth patterns: pure mucinous, pure cystic, combined cystic-microcystic-papillocystic, combined cystic and acinar, and mucoepidermoid-like. The lack of cellular atypia and mitotic activity in MGA cells is a key histological characteristic that distinguishes these lesions from malignant neoplasms.

The expression patterns of TTF-1 and napsin A serve as valuable markers to differentiate MGA from pulmonary mucinous adenocarcinoma(PMA). In cases of PMA, TTF-1 is typically expressed in the nuclear compartment. Napsin A exhibits high specificity for PMA, making it one of the most sensitive and specific indicators for this type of cancer. Consequently, napsin A plays a crucial role in the accurate diagnosis of PMA [25]. Although there is limited knowledge regarding the immunohistochemical characteristics of mucinous adenomas, reported cases have indicated negative expression for TTF-1 and napsin A [26, 27], while positive expression for keratin, keratin-7, and epithelial membrane antigens like PD-L1 et al. [16]. This immunohistochemical profile, combined with imaging findings suggestive of a small bronchial tumor, should raise suspicion of a MGA. Furthermore, Ryuji Kojima et al. [28] 's study reported the detection of GNAS gene (R201C) mutation in MGA tumor tissues, suggesting that this genetic alteration may play a significant role in tumorigenesis. Genetic testing was not conducted in our presented case due to the prohibitive cost associated with such procedures.

The preferred treatment for MGA is complete surgical resection, which can be achieved through lobectomy or sublobectomy, as well as bronchoplasty [15] or sleeve resection. Excision of MGA via fiberoptic bronchoscopy has been reported in a few cases [29]. The primary objective of tumor surgery is to attain complete resection and achieve a cure. To date, there have been no documented instances of disease metastasis or malignant transformation.

In conclusion, MGA is a rare tumor primarily located in the central lung region, with peripheral occurrences being infrequent. The use of 18 F-FDG PET/CT lacks specificity in detecting MGA and may lead to misdiagnosis as a lung malignancy. The investigation of MGA on 18 F-FDG PET/CT necessitates additional research. Therefore, when employing 18 F-FDG PET/CT for diagnosing lung lesions, it is crucial to consider the possibility of MGA. Hence, a comprehensive analysis combining clinical manifestations, bronchoscopy findings, imaging results, and pathological findings is essential for accurate identification of pulmonary mucus gland adenoma.

Abbreviations

MGA	Mucus gland adenoma
PMA	Pulmonary mucinous adenocarcinoma
CT	Computed tomography
FDG	Fluorodeoxyglucose
SUV	Standardized uptake value
SUVmax	Maximum standardized uptake value

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Not applicable.

Author contributions
CX and ZC was a major contributor in writing the manuscript. CX and ZF analyzed and interpreted the patient data regarding the pulmonary mucus gland adenoma. ZJ performed the histological examination of the tumor. All authors read and approved the final manuscript. CX, ZJ, ZF, and ZC have contributed to the work equally and should be regarded as co-first authors.

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Data availability
Data is provided within the manuscript or supplementary information files.

Declarations

Ethics approval and consent to participate
This case report was reviewed and approved by the Clinical College, Lishui Central Hospital Institutional Review Board.

Consent for publication
The patient signed informed consent for publication of this case report and any associated images.

Competing interests
The authors declare no competing interests.

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