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Contents

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REVIEW

- 10746 Management of acute kidney injury in gastrointestinal tumor: An overview

Su YQ, Yu YY, Shen B, Yang F, Nie YX

- 10765 Application of vascular endothelial cells in stem cell medicine

Liang QQ, Liu L

MINIREVIEWS

- 10781 Application of traditional Chinese medicine in treatment of *Helicobacter pylori* infection

Li RJ, Dai YY, Qin C, Huang GR, Qin YC, Huang YY, Huang ZS, Luo XK, Huang YQ

ORIGINAL ARTICLE

Case Control Study

- 10792 Impact of cytomegalovirus infection on biliary disease after liver transplantation - maybe an essential factor

Liu JY, Zhang JR, Sun LY, Zhu ZJ, Wei L, Qu W, Zeng ZG, Liu Y, Zhao XY

- 10805 Blood tests for prediction of deep endometriosis: A case-control study

Chen ZY, Zhang LF, Zhang YQ, Zhou Y, Li XY, Huang XF

Retrospective Cohort Study

- 10816 Association between neutrophil-to-lymphocyte ratio and major postoperative complications after carotid endarterectomy: A retrospective cohort study

Yu Y, Cui WH, Cheng C, Lu Y, Zhang Q, Han RQ

- 10828 Application of MAGnetic resonance imaging compilation in acute ischemic stroke

Wang Q, Wang G, Sun Q, Sun DH

Retrospective Study

- 10838 Ninety-four thousand-case retrospective study on antibacterial drug resistance of *Helicobacter pylori*

Zhang Y, Meng F, Jin J, Wang J, Gu BB, Peng JB, Ye LP

- 10850 Adjacent segment disease following Dynesys stabilization for lumbar disorders: A case series of mid- and long-term follow-ups

Chen KJ, Lai CY, Chiu LT, Huang WS, Hsiao PH, Chang CC, Lin CJ, Lo YS, Chen YJ, Chen HT

- 10861 Identification of independent risk factors for intraoperative gastroesophageal reflux in adult patients undergoing general anesthesia

Zhao X, Li ST, Chen LH, Liu K, Lian M, Wang HJ, Fang YJ

- 10871** Value of the controlling nutritional status score and psoas muscle thickness per height in predicting prognosis in liver transplantation

Dai X, Gao B, Zhang XX, Li J, Jiang WT

- 10884** Development of a lipid metabolism-related gene model to predict prognosis in patients with pancreatic cancer

Xu H, Sun J, Zhou L, Du QC, Zhu HY, Chen Y, Wang XY

- 10899** Serum magnesium level as a predictor of acute kidney injury in patients with acute pancreatitis

Yu XQ, Deng HB, Liu Y, Qu C, Duan ZH, Tong ZH, Liu YX, Li WQ

- 10909** Pedicle complex tissue flap transfer for reconstruction of duplicated thumbs with unequal size

Wang DH, Zhang GP, Wang ZT, Wang M, Han QY, Liu FX

- 10919** Minimally invasive surgery *vs* laparotomy in patients with colon cancer residing in high-altitude areas

Suo Lang DJ, Ci Ren YZ, Bian Ba ZX

Observational Study

- 10927** Surgery for chronic pancreatitis in Finland is rare but seems to produce good long-term results

Parhiala M, Sand J, Laukkanen J

- 10937** Association of overtime work and obesity with needle stick and sharp injuries in medical practice

Chen YH, Yeh CJ, Jong GP

- 10948** Serum gastrin-17 concentration for prediction of upper gastrointestinal tract bleeding risk among peptic ulcer patients

Wang JX, Cao YP, Su P, He W, Li XP, Zhu YM

- 10956** Predictive risk scales for development of pressure ulcers in pediatric patients admitted to general ward and intensive care unit

Luo WJ, Zhou XZ, Lei JY, Xu Y, Huang RH

META-ANALYSIS

- 10969** Clinical significance of signet ring cells in surgical esophageal and esophagogastric junction adenocarcinoma: A systematic review and meta-analysis

Wang YF, Xu SY, Wang Y, Che GW, Ma HT

- 10979** Percutaneous biliary stent combined with brachytherapy using ¹²⁵I seeds for treatment of unresectable malignant obstructive jaundice: A meta-analysis

Chen WY, Kong CL, Meng MM, Chen WQ, Zheng LY, Mao JT, Fang SJ, Chen L, Shu GF, Yang Y, Weng QY, Chen MJ, Xu M, Ji JS

CASE REPORT

- 10994** Prenatal ultrasonographic findings in Klippel-Trenaunay syndrome: A case report

Pang HQ, Gao QQ

- 10999** Immunoglobulin G4-related lymph node disease with an orbital mass mimicking Castleman disease: A case report
Hao FY, Yang FX, Bian HY, Zhao X
- 11007** Treatment for subtrochanteric fracture and subsequent nonunion in an adult patient with osteopetrosis: A case report and review of the literature
Yang H, Shao GX, Du ZW, Li ZW
- 11016** Early surgical intervention in culture-negative endocarditis of the aortic valve complicated by abscess in an infant: A case report
Yang YF, Si FF, Chen TT, Fan LX, Lu YH, Jin M
- 11024** Severe absence of intra-orbital fat in a patient with orbital venous malformation: A case report
Yang LD, Xu SQ, Wang YF, Jia RB
- 11029** Pulmonary Langerhans cell histiocytosis and multiple system involvement: A case report
Luo L, Li YX
- 11036** Complete androgen insensitivity syndrome caused by the c.2678C>T mutation in the androgen receptor gene: A case report
Wang KN, Chen QQ, Zhu YL, Wang CL
- 11043** Ultrasound guiding the rapid diagnosis and treatment of perioperative pneumothorax: A case report
Zhang G, Huang XY, Zhang L
- 11050** Chronic colchicine poisoning with neuromyopathy, gastric ulcers and myelosuppression in a gout patient: A case report
Li MM, Teng J, Wang Y
- 11056** Treatment of a giant low-grade appendiceal mucinous neoplasm: A case report
Xu R, Yang ZL
- 11061** Thoracoscopic resection of a large lower esophageal schwannoma: A case report and review of the literature
Wang TY, Wang BL, Wang FR, Jing MY, Zhang LD, Zhang DK
- 11071** Signet ring cell carcinoma hidden beneath large pedunculated colorectal polyp: A case report
Yan JN, Shao YF, Ye GL, Ding Y
- 11078** Double-mutant invasive mucinous adenocarcinoma of the lung in a 32-year-old male patient: A case report
Wang T
- 11085** Acute myocarditis presenting as accelerated junctional rhythm in Graves' disease: A case report
Li MM, Liu WS, Shan RC, Teng J, Wang Y
- 11095** Lingual nerve injury caused by laryngeal mask airway during percutaneous nephrolithotomy: A case report
Wang ZY, Liu WZ, Wang FQ, Chen YZ, Huang T, Yuan HS, Cheng Y

- 11102** Ventricular fibrillation and sudden cardiac arrest in apical hypertrophic cardiomyopathy: Two case reports
Park YM, Jang AY, Chung WJ, Han SH, Semsarian C, Choi IS
- 11108** *Rhizopus microsporus* lung infection in an immunocompetent patient successfully treated with amphotericin B: A case report
Chen L, Su Y, Xiong XZ
- 11115** Spermatocytic tumor: A rare case report
Hao ML, Li CH

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Double-mutant invasive mucinous adenocarcinoma of the lung in a 32-year-old male patient: A case report

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Abstract

BACKGROUND

Invasive mucinous adenocarcinoma of the lung, formerly known as mucinous bronchioloalveolar carcinoma, is a rare category of lung tumors and radiologically characterized by dense pneumonic consolidation, ground-glass opacity, crazy paving, and nodules. However, early pleural effusion is uncommon in this malignancy.

CASE SUMMARY

The case of a 32-year-old male patient who visited our facility with symptoms of cough and gradually aggravated shortness of breath was reported. X-ray examination revealed a massive left hydrothorax. The patient underwent thoracentesis, and pleural fluid tumor markers, including carcinoembryonic antigen, carbohydrate antigen 19-9, neuron-specific enolase, and cytokeratin 21-1 fragment, were significantly elevated. A similar tendency was observed among the serum tumor markers. After draining the pleural effusion, the patient underwent chest computed tomography, and no obvious mass was found in the lung. Thoracoscopy revealed that the left visceral pleura was covered with nodular, cauliflower-like protrusions of various sizes. These histopathological results suggested cancerous cells, and the immunohistochemical findings were consistent with mucinous adenocarcinoma of pulmonary origin. It tested positive for cytokeratin, cytokeratin 5/6, carcinoembryonic antigen, and thyroid transcription factor-1.

CONCLUSION

The patient was diagnosed with a rare case of lung mucinous adenocarcinoma. Subsequent genetic testing was positive for epidermal growth factor receptor-21 mutations and echinoderm microtubule-associated protein-like 4-lymphoma anaplastic kinase fusion. This prompted treatment with afatinib and crizotinib.

Key Words: Lung mucinous adenocarcinoma; Hydrothorax; Double mutant; Case report

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Core tip: Invasive mucinous adenocarcinoma of the lung is a rare category of lung tumors, radiologically characterized by dense pneumonic consolidation, ground-glass opacity, crazy paving, and nodules. Early pleural effusion is uncommon in this malignancy. Molecularly, invasive mucinous adenocarcinoma has frequent Kirsten rat sarcoma viral oncogene mutations and a lower prevalence of epidermal growth factor receptor and anaplastic lymphoma kinase rearrangements. Here, a rare case of double-mutant invasive mucinous lung adenocarcinoma presenting as a massive malignant pleural effusion in a 32-year-old male patient was reported.

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INTRODUCTION

According to the global cancer statistics in 2018, lung cancer is the leading cause of cancer-related mortality, killing approximately 1761000 people worldwide[1]. Histologically, lung cancer is divided into non-small cell lung cancer and small cell lung cancer, with the former pathological type accounting for 80% of cases[2]. Invasive mucinous adenocarcinoma (IMA) of the lung was introduced as a new category in the 2015 World Health Organization classification of lung tumors. It accounts for 2%-5% of lung adenocarcinomas, the most common histologic subtype of non-small cell lung cancer[3,4]. IMA is diagnosed based on tall columnar cell morphology with abundant intracellular/extracellular mucus and invasive adenocarcinoma patterns, such as lepidic, acinar, papillary, and predominant patterns. Due to its pathological and growth characteristics, the typical computed tomography (CT) findings of IMA include consolidation, ground-glass opacity, and nodules.

In contrast, pleural IMAs are rare[5-7]. A case of IMA presenting as a massive pleural effusion in the left thoracic cavity in a 32-year-old male patient was reported. After drainage, multiple nodules on the left visceral pleura, instead of apparent pulmonary masses, were found on a chest CT scan. Gene testing showed that the biopsy tissue had a double mutation, namely positive epidermal growth factor receptor-21 (EGFR-21) mutations and echinoderm microtubule-associated protein-like 4-anaplastic lymphoma kinase (ALK) fusion. To the best of my knowledge, this is the first published report on dual-mutation IMA with early pleural invasion in a young patient.

CASE PRESENTATION

Chief complaints

A 32-year-old male chronic smoker (ten cigarettes a day for more than 10 years) was referred to our hospital's respiratory department on June 25, 2020. He complained of shortness of breath and a cough.

History of present illness

The patient had a history of progressive shortness of breath and a cough for 20 d before consult.

History of past illness

He had no prior chronic diseases but recently lost 4 kg of his body weight.

Personal and family history

The patient denied having a family history of lung cancer, hypertension, and coronary heart disease.

Physical examination

Physical examination showed that respiratory sounds were absent in the patient's left lung field.

Laboratory examinations

He was admitted to our department where he underwent thoracentesis that resulted in the extraction of 700 mL of pleural effusion. Related laboratory results of the yellow turbid pleural fluid revealed an exudate with dramatically high tumor biomarkers and normal adenosine deaminase, which was unexpected. Detailed data are as follows: total cell count = $8015 \times 10^6/L$, leukocyte count = $440 \times 10^6/L$, positive Rivalta test, lactate dehydrogenase = 676 U/L, total protein 51 g/L, adenosine deaminase = 13 U/L, carcinoembryonic antigen = 34.6 ng/mL (normal range: 0-5), carbohydrate antigen 19-9 > 1000 U/mL (normal range: 0-37), neuron-specific enolase = 22.65 ng/mL (normal range: 0-18), and cytokeratin 21-1 fragment > 500 ng/mL (normal range: 0-3.3). The patient's serum tumor biomarkers exhibited a less obvious upward trend than their counterparts in the pleural effusion, with carcinoembryonic antigen 7.96 ng/mL, carbohydrate antigen 19-9 204.2 U/mL, and cytokeratin 21-1 fragment 6.33 ng/mL. Cytological tests revealed allotype tumor cells in the hydrothorax. We then performed closed thoracic drainage because of the massive pleural effusion and apparent symptoms of this patient.

Imaging examinations

Chest X-ray showed a severe left hydrothorax (Figure 1), and the patient was hospitalized with a suspected diagnosis of tuberculous exudative pleurisy. After pleural effusion drainage, the patient underwent chest CT, demonstrating no evident pulmonary masses or nodules; however, multiple nodules were found in the left visceral pleura (Figure 2). Subsequent thoracoscopy revealed that the visceral pleura was filled with nodular, cauliflower-like protrusions of various sizes (Figure 3).

FINAL DIAGNOSIS

The initial pathological diagnosis was allotypic epithelioid cells, and immunohistochemical analysis showed tumor cell positivity for cytokeratin, cytokeratin 5/6, carcinoembryonic antigen, thyroid transcription factor-1, and Ki-67 (20%). The tumor tested negative for calretinin, cytokeratin 20, and p53, which were compatible with lung IMA (Figure 4). Genetic testing revealed positive EGFR-21 mutations and echinoderm microtubule-associated protein-like 4-ALK fusion. This prompted treatment with alfatiniib and crizotinib.

TREATMENT

Considering the side effects of the two targeted drugs, we initially prescribed alfatiniib alone with a daily dose of 40 mg. After 1 mo, re-examination showed decreased pleural nodules, no pleural effusion recurrence, and significantly reduced serum lung cancer biomarkers. Meanwhile, the patient had a long-standing unresolved left lateral chest pain. After 2 mo, the second follow-up showed no changes in serum lung cancer biomarkers. However, the number of pleural nodules increased, and small metastases were observed in the contralateral lung. We adjusted the therapeutic plan based on these findings. Both alfatiniib and crizotinib were administered.

OUTCOME AND FOLLOW-UP

The patient's left lateral chest pain resolved within 1 wk. Thus far, the patient has taken alfatiniib 40 mg per day and crizotinib 250 mg twice per day for more than 10 mo, and side effects, such as mild diarrhea and skin rashes, have occurred. After 12 mo, chest CT scan re-examination showed apparent reductions in the pleural nodules and no recurrence of pleural effusion.

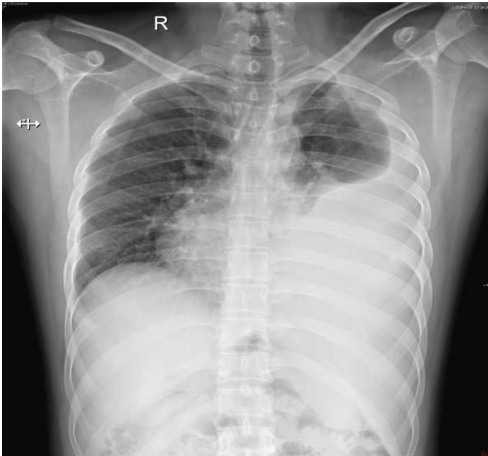


Figure 1 A chest radiograph showed severe left hydrothorax.

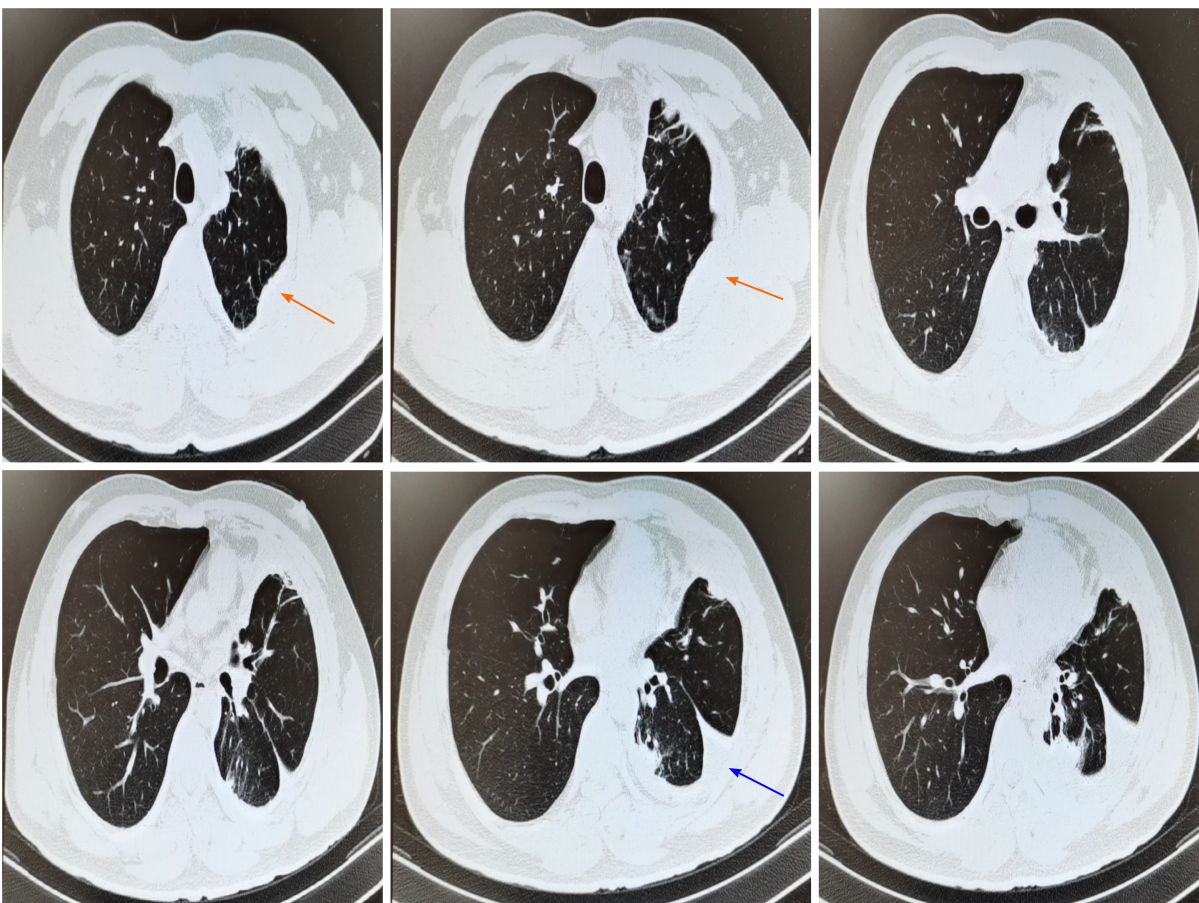


Figure 2 Chest computed tomography demonstrated multiple nodules on left visceral pleura (indicated by orange arrows) and part of atelectasis in the left lower lung (showed by the blue arrow).

DISCUSSION

Lung cancer, which is associated with high morbidity and mortality among all types of tumors, frequently occurs in the sixth to eighth decades of life. It is uncommon among people around 30 years of age[8]. A new and rare type of adenocarcinoma, IMA, was reclassified as a variant of invasive adenocarcinoma by the International Association for the Study of Lung Cancer/American Thoracic Society/European Respiratory Society lung adenocarcinoma classification system because of its unique clinical, pathological, and radiological features as well as its unique genetic characteristics (lower prevalence of EGFR mutations)[9,10].

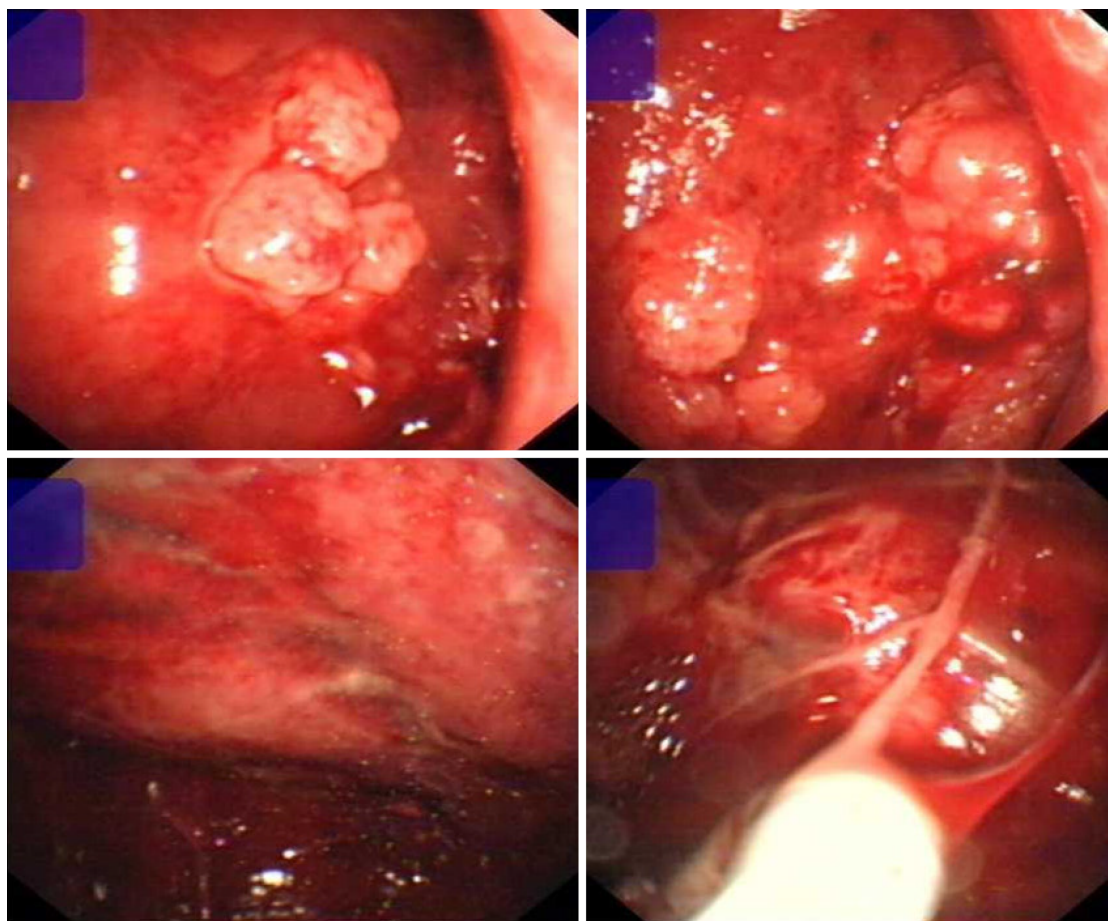


Figure 3 Thoracoscopy revealed visceral pleura almost full of nodular, cauliflower-like protrusions of various sizes.

IMA has a tall columnar morphology with abundant mucus inside or outside the tumor cells. It exhibits adenocarcinoma patterns, including acinar, papillary, micropapillary, and solid predominant patterns. Specifically, IMA is divided into two groups based on the mucinous percentage: pure mucinous (invasive mucinous component > 90%) and mixed mucinous/non-mucinous (non-mucinous invasive pattern > 10%)[11]. IMA, a classification with poor survival outcomes compared with other adenocarcinoma subtypes, frequently spreads aerogenously, forming satellite tumors. Lymph node involvement and distant metastasis are less common[3,12].

On CT, IMA presents as consolidation, nodules, or ground-glass opacity. In a study by Nie *et al*[13], 54 of 68 patients with IMA had solitary-type tumors on their chest CT scan. Nearly 80% of the imaging signs of IMA are solitary nodules or masses. The other 20% of CT findings are the pneumonic type, which is defined as consolidation without a defined shape, distributed along the lung lobe or lung segment, and sometimes with air bronchogram. In contrast, pleural effusion, as an early sign of IMA, is rare. In our case, immunohistochemical analysis showed that the tumor cells were of pulmonary origin. No clear pulmonary signs were observed on chest imaging after pleural effusion drainage. Instead, multiple nodules were observed in the left visceral pleura, which was more suggestive of pleural mesothelioma. Atelectasis was also observed in the left lower lung, which may have negatively influenced assessment and diagnosis.

Concerning the molecular features of IMA, several studies have linked IMA with frequent Kirsten rat sarcoma viral oncogene mutations and a lower prevalence of EGFR and ALK rearrangements, indicating a poor prognosis for target-specific drug treatment[14,15]. In our case, the EGFR-21 mutations and echinoderm microtubule-associated protein-like 4-ALK fusion were both positive, which is rare for IMA. This case emphasizes the significance of applying medicine to different targets in lung cancer. We will conduct a continuous follow-up of this young patient in the future.

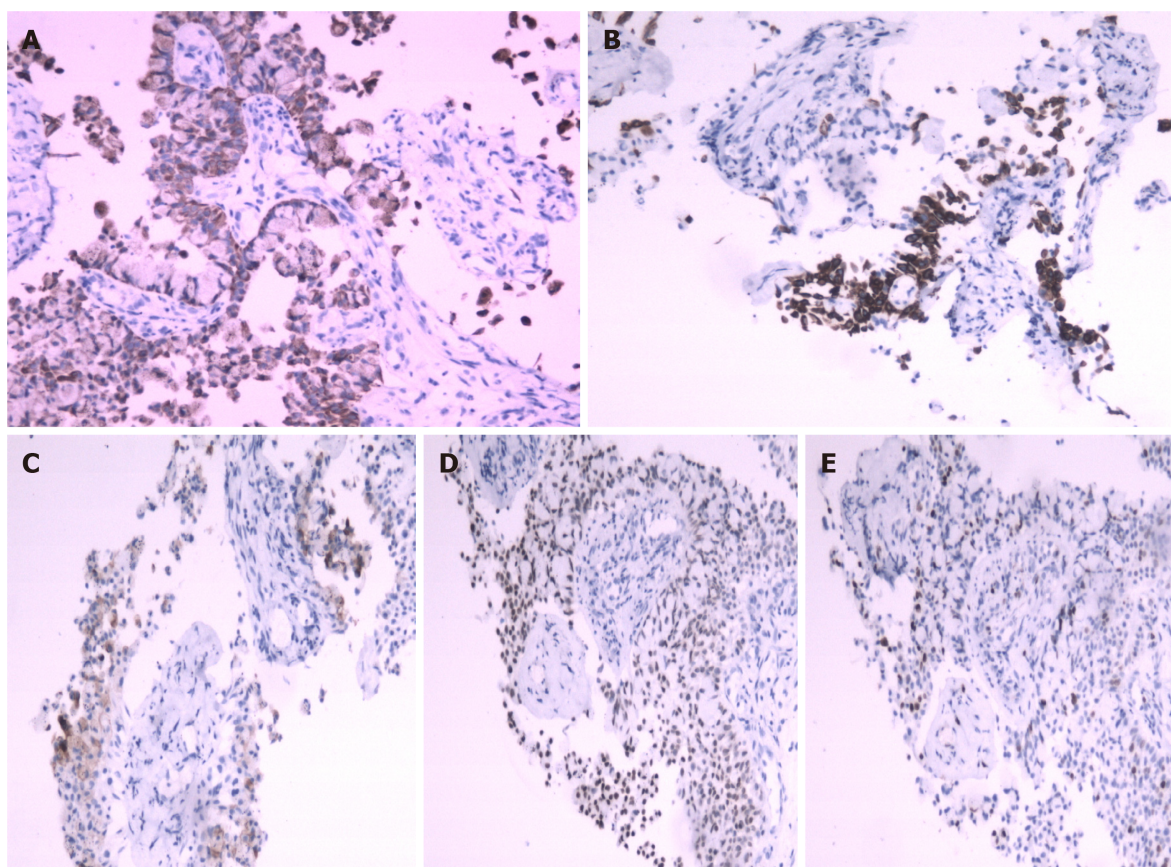


Figure 4 Immunohistochemical analysis showed that the tumor cells were positive for cytokeratin, cytokeratin 5/6, carcinoembryonic antigen, thyroid transcription factor-1 and Ki-67. A: Cytokeratin; B: Cytokeratin 5/6; C: Carcinoembryonic antigen; D: Thyroid transcription factor-1; E: Ki-67.

CONCLUSION

In conclusion, atypical IMA is challenging to diagnose, especially in young patients. It is necessary to consider IMA in patients with unusual laboratory test results and radiological presentations.

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