

Clinical features and risk-factors analysis of coexistence of tuberculosis and tumour in the same lesion: a case-control study

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Abstract

Background: The coexistence of tuberculosis and lung cancer in the same single-lung lesion is a rare and clinically challenging special disease entity, which often leads to misdiagnosis or delayed treatment. This study aims to systematically analyse the clinical, imaging and serological characteristics of patients with tuberculosis complicated by lung cancer within the same lesion, so as to provide evidence-based references for the early differential diagnosis and improved prognosis of this disease.

Methods: A retrospective case-control study was conducted on 150 subjects at Guangzhou Chest Hospital between 2020 and 2024. The patients were divided into the isolated pulmonary tuberculosis group (62 cases), isolated lung cancer group (58 cases), and tuberculosis-lung cancer coexistence group in the same lesion (30 cases). Baseline clinical data, imaging distribution features of lesions, tumour pathological types, serum inflammatory and tumour marker data of the three groups were collected, and statistical comparative analysis was performed.

Results: Patients in the coexistence group were significantly older than those in the tuberculosis-only group ($P=0.001$). Lesions in the coexistence group were predominantly distributed in the upper (56.7%) and lower (33.3%) lobes of the lungs, while lesions in the tuberculosis-only group (75.8%) presented with multi-lobar and bilateral pulmonary distribution. In terms of pathological types, squamous lung cancer predominated in the coexistence group (46.7%), whereas adenocarcinoma absolutely predominated in the tumour-only group (75.9%). Serological tests showed that the levels of carcinoembryonic antigen (CEA) and cytokeratin 19 fragment (CYFRA21-1) in the coexistence group were significantly higher than those in the tuberculosis-only group ($P=0.05$). There was no statistically significant difference in the levels of C-reactive protein (CRP) and procalcitonin (PCT) among the three groups ($P=0.05$).

Conclusions: The main pathological types of tuberculosis complicated with lung cancer within the same lesion are squamous cell carcinoma and adenocarcinoma. Patients are predominantly elderly individuals, with lesions mostly distributed in the upper and lower lobes of the lungs, accompanied by elevated CEA and CYFRA21-1 levels. Multidisciplinary joint diagnosis and treatment combined with tumour biomarker screening are of significant clinical value in optimising treatment regimens and enhancing diagnostic accuracy.

1. Introduction

Tuberculosis (TB) is one of the infectious diseases with the highest mortality worldwide[1], while malignant neoplasms represent the second leading cause of death after cardiovascular disease among TB patients[2], and lung cancer ranks first in mortality among malignant tumours in China[3]. Epidemiological studies have confirmed that tuberculosis infection can significantly increase the risk of lung cancer[4], with a combined incidence of 0.22%-27%[5], which has shown an upward trend year by year in recent years. The elevated risk of lung cancer persists for more than 20 years after tuberculosis cure, and the 5-year window following tuberculosis confirmation is a high-risk period for malignant transformation.

Tuberculosis and lung cancer share substantial overlaps in clinical symptoms and imaging manifestations. Chronic cough, haemoptysis, chest pain and pulmonary nodular shadow are their common typical manifestations, posing great challenges for differential diagnosis[6], and the overall clinical misdiagnosis rate exceeds 40%[7]. Most current domestic and international studies focus on the combined pattern of tuberculosis and lung cancer with separate lesions at different sites, while studies on synchronous coexistence within a single lesion (both tuberculous lesions and malignant tumour tissues pathologically identified in the identical lesion) remain scarce[8]. This special subtype is highly concealed and prone to clinical missed diagnosis, thereby delaying anti-tumour therapy, deteriorating patient prognosis and further increasing disability and mortality rates[9, 10].

Existing studies have verified that chronic pulmonary inflammation, fibrous scar hyperplasia, systemic immune disturbance and local accumulation of carcinogens induced by tuberculosis are core triggers for malignant transformation of pulmonary epithelial cells[11]. Meanwhile, long-term anti-tuberculosis treatment can further suppress systemic immune function and facilitate immune escape of tumour cells.

At present, studies focusing on specific clinical features of tuberculosis complicated with lung cancer within a single lesion are still limited[5]. This study aimed to investigate the clinical, imaging, pathological and serological characteristics of patients with tuberculosis coexisting with tumours within the same lung lesion, to provide evidence for the early diagnosis of pulmonary tuberculosis (PTB) combined with lung cancer, reduce the rate of underdiagnosis, improve prognosis, and further explore the potential pathogenesis.

2. Methods

2.1 Study subjects

The patients in the case and control groups of this study were all selected from inpatients admitted and treated at Guangzhou Chest Hospital between January 2020 and December 2024. All enrolled patients were aged ≥ 18 years with complete clinical, pathological, imaging and follow-up data. According to the results of pathological and microbiological examinations, the research subjects were divided into three groups.

2.1.1 Inclusion criteria

Intrapulmonary Tuberculosis-Lung Cancer Coexistence Group:

1. The simultaneous presence of tumour cells and *Mycobacterium tuberculosis* within a single identical lung lesion was confirmed by pathological examination, with positive acid-fast staining or tuberculosis polymerase chain reaction (PCR) test results.
2. Chest imaging supported the diagnosis of tuberculosis combined with a tumour.

3. No anti-tumour therapy was administered before enrolment.

Simple Lung Cancer Group:

1. Primary lung cancer was confirmed through pathological testing, with no imaging manifestations or aetiological evidence of pulmonary tuberculosis infection.

Tuberculosis alone group:

1. Active or stable pulmonary tuberculosis was confirmed by aetiological examinations including acid-fast staining, bacterial culture or PCR test, with no evidence of any pulmonary malignant tumour.

2.1.2 Exclusion criteria

Patients with the following conditions are excluded from enrolment:

1. Patients with other previous or concurrent active lung infections (e.g., pneumonia, sepsis, viral infections) that could affect the study outcomes.
2. Patients with comorbid severe immune system disorders (e.g., human immunodeficiency virus (HIV) infection, active autoimmune diseases).
3. Patients previously received systemic immunotherapy (e.g., programmed cell death protein 1/programmed death-ligand 1 (PD-1/PD-L1) inhibitors, cytotoxic T-lymphocyte-associated protein 4 (CTLA-4) inhibitors).
4. Patients with severe hepatic or renal failure (e.g., Child-Pugh class C cirrhosis, estimated glomerular filtration rate (eGFR) < 30 ml/min).
5. Patients who were lost to follow-up or had incomplete follow-up data.

2.2 Data collection

Basic demographic information of the patients was collected, including age, gender, smoking history, TB treatment history, TB regimen, survival time, and prognosis. Tumour type, stage, and pathological classification were confirmed through imaging (e.g., computed tomography (CT), magnetic resonance imaging (MRI), positron emission tomography-computed tomography (PET-CT) and pathology.

2.3 Laboratory detection indicators

Serum tumour markers including carcinoembryonic antigen (CEA), carbohydrate antigen 125 (CA125), carbohydrate antigen 19-9 (CA19-9), neuron-specific enolase (NSE), cytokeratin 19 fragment (CYFRA21-1) were detected using the chemiluminescence method or enzyme-linked immunosorbent assay (ELISA). Immunoturbidimetric assay was adopted to measure C-reactive protein (CRP) and procalcitonin (PCT), so as to evaluate the level of systemic inflammation.

2.4 Statistical analysis

Data were analysed using Statistical Package for the Social Sciences (SPSS) statistical software. All measurements were tested for normality. Those that conformed to a normal distribution were expressed as mean $\pm$ standard deviation (Mean $\pm$ SD) and an independent samples t-test was used for between-group comparisons. Measurements that did not conform to a normal distribution were expressed as median and interquartile range (Median, IQR), and the Mann-Whitney U-test was applied. Categorical variables were expressed as frequencies and percentages, and differences between groups were assessed using the chi-square test (χ^2 test).

2.5 Ethical statement

The Ethics Committee of Guangzhou Chest Hospital approved this study. All participants signed an informed consent form before the start of the study, and the study process strictly adhered to the principles of medical ethics.

3. Results

3.1 Comparison of general information

A total of 150 patients were enrolled in this study, including 62 patients in the tuberculosis alone group, 58 patients in the simple lung cancer group, and 30 patients in the lesion coexistence group. The differences in age among the three groups—namely, the simple tumour group, the simple tuberculosis group, and the tuberculosis and tumour coexistence group—were statistically significant [41(29.0,58.5) vs. 62(52.0,67.0) vs. 63(56.8,68.3), $H=32.711$, $P<0.001$]; The age of the tuberculosis and tumour coexistence group was significantly older than that of the simple tuberculosis group. The differences in gender and smoking status among the three groups were not statistically significant ($\chi^2=3.688$, 5.157, both $P>0.05$)(Table 1).

3.2 Imaging lesion distribution characteristics

The difference in the site of disease among the three groups of simple tumour group, simple tuberculosis group and tuberculosis and tumour coexistence group was statistically significant ($\chi^2=73.404$, $P<0.001$), in which the site of disease in the tuberculosis and tumour coexistence group was found in the upper (56.7%) and lower (33.3%) lobes of the lungs with a highly localized distribution compared to the simple tuberculosis group, whereas in the simple tuberculosis group the site of disease was found in more than one lobe of the lungs or both lobes of the lungs, which represented the typical imaging features of active pulmonary tuberculosis, and the difference was statistically significant ($P<0.001$). (Table 2).

3.3 Distribution of tumour pathological types

There was a significant difference in the distribution of pathological subtypes between the coexistence group and the simple lung cancer group ($\chi^2=9.247$, $P=0.01$). Compared with the simple tumour group, squamous carcinoma was predominant in the tuberculosis and tumour coexistence group, whereas adenocarcinoma predominated in the simple tumour group (Table 3).

3.4 Comparison of inflammatory markers

The differences in inflammatory markers PCT and CRP among the three groups of people in the tumour-only group, TB-only group and TB and tumour coexistence group were not statistically significant ($\chi^2=3.480$, $H=2.975$; all $P>0.05$). (Table 4).

3.5 Comparison of tumour markers

The three groups, tumour alone, TB alone and TB and tumour coexisting group, were found to have the highest levels of the tumour markers CEA

[2.02 (0.83,3.42) vs 3.00 (1.79,6.22) vs 2.42 (2.10,4.98), $H=12.977$], CA19-9 [8.47 (4.99,19.46) vs 13.24 (7.75,23.12) vs 17.49 (7.02,29.59), $H=7.084$], and CYFRA21-1 [1.72(1.48,2.20) vs 2.61(1.92,5.57) vs 2.36(1.87,3.85), $H=31.014$] were statistically significant (all $P<0.05$). Among them, CEA and CYFRA21-1 were significantly higher in the TB and tumour coexistence group compared to the TB alone group, there was no significant difference compared with the simple lung cancer group. And the differences among the three groups in CA125 and NSE were not statistically significant ($H=4.662$, 0.833 ; all $P>0.05$). (Table 5).

4. Discussion

The coexistence of tuberculosis and malignancy within the same pulmonary lesion is clinically rare. Previous studies both domestically and internationally have primarily focused on ectopic and multifocal tuberculosis combined with lung cancer. This study concentrates on the clinically rare and extremely challenging-to-differentiate subtype of synchronous coexistence of tuberculosis-lung cancer within the same lesion. This study is the first rigorously designed case-control study to reveal the unique clinical characteristics and risk factors of such patients by comparing clinical data from a group with pure tumor, a group with pure tuberculosis, and a group with coexistence of tuberculosis and tumor within the same lesion.

First, patients in the coexisting group of our study were significantly older than those in the TB-only group (63 vs. 41 years, $P < 0.001$), which aligns with previous reports indicating that the risk of developing TB-associated malignancies increases with age[12]. This age-related trend may be attributed to the cumulative effects of environmental and genetic factors and the immune senescence process observed in older individuals. It has been shown that a >95% decline in thymic output with age directly leads to the atrophy of the initial T-cell pool, which weakens the recognition of Mycobacterium

tuberculosis or tumour antigens[13], thereby promoting latent TB reactivation and tumour immune escape[14].

Imaging lesion distribution serves as an important point for the differential diagnosis of this disease. Our analysis showed that lesions in the tuberculosis-only group were characterised by multilobar diffuse lesions as the core feature, whereas lesions in the coexistence group were mostly confined to a single pulmonary lobe and concentrated in the upper and lower lobes of the lungs (total 90.0%), consistent with the classical manifestation that pulmonary tuberculosis tends to occur in the upper lobes[15]; this finding carries clinical reference value. The coexistence of tuberculosis and malignancy in the same lesion may be attributed to tuberculosis-induced chronic inflammation, fibrous tissue hyperplasia and scar formation. Persistent tissue injury and disturbed tissue repair promote cellular transformation and induce tumorigenesis, especially in areas of active infection[16,17]. This dual pathological change highlights the complex interplay between tuberculosis-induced fibrosis and tumour growth, and the underlying molecular mechanisms warrant further investigation. Nevertheless, this characteristic supports the scar carcinoma mechanism underlying malignant transformation of tuberculous lesions.

Our study found that squamous lung cancer (46.7%) and adenocarcinoma (43.3%) were the predominant cancer types in lesions with both tuberculosis and malignancy. This distribution contrasts with the tumour-only group, where adenocarcinoma was more prevalent (75.9%), and squamous carcinoma accounted for only 11%. These findings are noteworthy because adenocarcinoma is typically the most common subtype of tuberculosis-associated lung cancer, a trend highlighted in previous studies[18]. However, combined with previous literature, it can be confirmed that long-term inflammatory stimulation of tuberculous cavities and bronchial walls induces squamous metaplasia of airway epithelium. Persistent disorders of cellular injury, repair and proliferation substantially increase the risk of squamous cell carcinogenesis[19]. This pathological characteristic further corroborates the causal association between the tuberculous microenvironment and pulmonary malignant transformation.

Differentiating between tuberculosis and lung cancer is diagnostically challenging due to the similarity of symptoms, including cough, hemoptysis, and weight loss[20]. However, traditional inflammatory markers such as CRP and PCT did not show significant differences, suggesting that they may not be sufficient to differentiate between tuberculosis and malignancy when they coexist. This is consistent with other findings that CRP and PCT effectively identify infections but lack specificity when tuberculosis and cancer coexist[21]. The reason is that the inflammatory signals from tuberculosis infection and tumour proliferation overlap and counteract each other, resulting in extremely poor specificity. In contrast, CEA and CYFRA21-1 possess favourable auxiliary diagnostic value: when both markers are abnormally elevated in patients with tuberculosis, it can highly suggest the presence of occult malignant transformation within lesions, serving as core indicators for clinicians to avoid missed diagnosis[22].

The findings of our study carry important guiding significance for clinical diagnosis and treatment. For elderly patients with solitary localized tuberculous pulmonary lesions, if lesions fail to shrink or progress continuously after standard anti-tuberculosis therapy, accompanied by elevated CEA/CYFRA21-1 levels,

pathological biopsy should be performed as early as possible to identify the presence of dual pathological changes within the lesions.

The current study has several limitations. First, this is a single-centre retrospective study with a relatively small sample size in the core coexistence group, leading to certain selection bias. Second, only the clinical and serological phenotypes of patients were analysed, and the specific molecular mechanisms underlying immune escape and malignant transformation within lesions were not explored in depth. In the future, multicentre, large-sample prospective studies are required to supplement prognostic analysis and mechanistic exploration and optimise the diagnosis and treatment system.

5. Conclusion

The coexistence of tuberculosis and lung cancer within the same lesion presents unique clinical characteristics: it tends to occur in elderly individuals, lesions are mostly confined to a single pulmonary lobe, squamous lung cancer and adenocarcinoma represent the main pathological subtypes, accompanied by specific elevations of serum CEA and CYFRA21-1, whereas conventional inflammatory markers have no differential value. Multidisciplinary comprehensive diagnosis relying on imaging localisation combined with specific tumour biomarkers can effectively improve the early diagnostic rate of this disease. Individualised stratified treatment based on the activity of tuberculous lesions is critical to optimising patients' clinical prognosis.

Abbreviations

CA125

carbohydrate antigen 125

CA19-9

carbohydrate antigen 19 – 9

CEA

carcinoembryonic antigen

CRP

C-reactive protein

CT

computed tomography

CTLA-4

cytotoxic T-lymphocyte-associated protein 4

CYFRA21-1

cytokeratin 19 fragment

eGFR

estimated glomerular filtration rate

ELISA

enzyme-linked immunosorbent assay

HIV
human immunodeficiency virus
IQR
interquartile range
MRI
magnetic resonance imaging
NSE
neuron-specific enolase
PCR
polymerase chain reaction
PCT
procalcitonin
PD-1/PD-L1
programmed cell death protein 1 / programmed death-ligand 1
PET-CT
positron emission tomography-computed tomography
PTB
pulmonary tuberculosis
SD
standard deviation
SPSS
Statistical Package for the Social Sciences
TB
tuberculosis

Declarations

Ethics approval and consent to participate

The study was conducted according to relevant guidelines and regulations in the Declaration of Helsinki. The study was approved by the Medical Ethics Committee of Guangzhou Chest Hospital (Affiliated Institution: Guangzhou Chest Hospital), with the approval number KY-2024-041. Written informed consent was waived because the study posed minimal risk to participants and all data were anonymized. Clinical trial number: not applicable.

Consent for publication

Not Applicable.

Availability of data and materials

The datasets used during the current study are available from the corresponding author on reasonable request.

Competing interests

The authors declare no competing interests.

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Authors' contributions

Zou J, Tan L, Luo L conceived and designed the study. Zhang X and Liu L provided administrative support. Luo L, Tan L, Chen J and Yang Z provided study materials and patients. Luo L, Tan L, Zhang X, Liu L, Chen J and Yang Z were responsible for data collection and assembly of data. Luo L, Tan L, Lin A, Chang W and Xie D performed data analysis and interpretation. All authors drafted and critically revised the manuscript. All authors read and approved the final manuscript.

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None.

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Tables

Tables 1 to 5 are available in the supplementary files section

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