Original Article

Computed tomography characteristics of chronic bronchitis and its association with disease severity and clinical outcomes in viral pneumonia: a retrospective cohort study

Xiaofei Wang[1,2], Zhentao Zhou[1,3], Yuting Peng[1,4], Yinying Tang[1,2], Qi Dai[1], Jianjun Zheng[1], Jingfeng Zhang[1]

[1]Department of Radiology, Ningbo No. 2 Hospital, Ningbo, China; [2]Health Science Center, Ningbo University, Ningbo, China; [3]Department of Radiology, Xiangshan First People's Hospital Medical and Health Group, Ningbo, China; [4]School of Public Health, Hangzhou Medical College, Hangzhou, China

Contributions: (I) Conception and design: X Wang, Q Dai; (II) Administrative support: J Zheng, J Zhang; (III) Provision of study materials or patients: X Wang, Q Dai; (IV) Collection and assembly of data: X Wang, Z Zhou, Y Tang; (V) Data analysis and interpretation: X Wang, Z Zhou, Y Peng; (VI) Manuscript writing: All authors; (VII) Final approval of manuscript: All authors.

Correspondence to: Jingfeng Zhang, MD. Department of Radiology, Ningbo No. 2 Hospital, No. 41 Northwest Street, Haishu District, Ningbo 315000, China. Email: jingfengzhang73@163.com.

Background: Chronic bronchitis (CB) patients' excessive mucus and airway changes may worsen viral pneumonia severity, but there is a lack of objective clinical/imaging assessment criteria. This study used quantitative computed tomography (CT) to link CB pathology with pneumonia severity/prognosis, guiding early interventions for high-risk groups.

Methods: This retrospective cohort study included 42 patients with CB diagnosed with viral pneumonia and 208 non-CB viral pneumonia controls. Baseline demographic, clinical, and laboratory parameters were collected alongside thoracic CT-derived metrics, including mucus plugging score (a bronchopulmonary segment-based scoring system quantifying mucus obstruction severity), CT severity score (range, 0–25 per lobe), and emphysema quantification. Follow-up CT imaging was performed at 3 months post-diagnosis to assess pulmonary structural remodeling, with longitudinal documentation of CT severity scores. Participants were stratified into two cohorts by mucus plugging score: high mucus burden (≥4, group 1) and low mucus burden (<4, group 2). Intergroup comparisons utilized Fisher's exact test for categorical variables, with continuous variables analyzed via independent *t*-tests (normally distributed) or Mann-Whitney *U* tests (non-parametric distributions). Multivariate logistic regression modeling identified independent predictors of disease progression.

Results: This study enrolled 260 patients who were categorized into group 1 (n=42; CB prevalence: 35.70%) and group 2 (n=218; CB prevalence:19.70%). Comparative analysis demonstrated that CB patients in group 2 were significantly older [70, interquartile range (IQR) (62, 77) *vs.* 79, IQR (74.5, 86.5) years; P<0.001] and exhibited a higher female predominance (74.4% *vs.* 25.6%; P=0.03), alongside lower red blood cell count (RBC) [3.73, IQR (3.39, 4.29)×10^{12}/L *vs.* 4.05, IQR (3.81, 4.53)×10^{12}/L; P=0.02] and hemoglobin levels [119, IQR (105.5, 131) *vs.* 124, IQR (115.5, 136) g/L; P=0.04] compared to non-CB counterparts. Imaging analysis revealed that non-CB patients had greater thoracic reticular patterns [6.02, IQR (1.42, 10.08) *vs.* 2.50, IQR (0.57, 6.19) cm^3; P=0.02] and emphysema severity [0.21, IQR (0.04, 0.73) *vs.* 0.05, IQR (0.01, 0.19) cm^3; P<0.001], whereas CB patients across both groups showed marked bronchial wall thickening [group 1: 21.60, IQR (10.30, 37.69) *vs.* 6.28, IQR (1.54, 15.71) cm^3, P=0.03; group 2: 35.08, IQR (13.38, 51.59) *vs.* 18.90, IQR (3.43, 45.53) cm^3, P=0.01]. Notably, CB patients in group 2 displayed larger bronchial lumen volumes [13.63, IQR (5.19, 25.29) *vs.* 5.00, IQR (0.67, 13.46) cm^3; P<0.001]. Multivariate analysis of the low mucus burden group identified female sex [odds ratio (OR) =3.39], age (OR =1.06), and emphysema (OR =1.56) as independent risk factors for disease progression (all P<0.05). Longitudinal CT follow-up indicated stable severity scores in non-CB patients, whereas high mucus-secreting CB patients (group 1) demonstrated

significant reductions in mucus plugging scores over time.

Conclusions: CB features—mucus hypersecretion, emphysema, and airway thickening—worsen viral pneumonia severity and prognosis. Key predictors include advanced age (OR =1.06), female sex (OR =3.39), and emphysema (OR =1.56). High-risk CB patients, especially with emphysema, need enhanced CT monitoring and therapies improving mucociliary clearance and airway repair.

Keywords: Viral infection; mucus hypersecretion; chronic bronchitis (CB)

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Introduction

Viral respiratory infections are the primary cause of acute respiratory diseases in healthy individuals across all age groups. Common viruses associated with these infections include influenza, respiratory syncytial virus, parainfluenza, and severe acute respiratory syndrome coronavirus (SARS-CoV). Upon invading the respiratory tract, these viruses promptly activate the innate defense response of the airway epithelium by stimulating macrophages, epithelial cells, and dendritic cells (1). However, the specific pathogenesis leading to abnormal mucus secretion in the airways due to viral infections remains unclear. Some research suggests that severe coronavirus infection may trigger a cytokine release syndrome, initiating a proinflammatory cascade that results in excessive mucus production by the infected respiratory epithelial cells. This disrupts the mucociliary clearance function, further causing airway obstruction and dyspnea (2). Airway mucus hypersecretion is a pathophysiological process in which various pathogenic factors induce excessive mucus production in the airway mucosa. This condition is caused by multiple pathogenic factors, leading to abundant secretions from cells within the airway, which stimulate the hyperplasia and hypertrophy of goblet cells and submucosal glands in the mucosa, ultimately resulting in mucus overproduction. A previous study (3) has shown that excessive airway mucus secretion also occurs in acute respiratory infections, such as those involving coronaviruses, and that it is a significant risk factor for the onset, progression, and prognosis of airway inflammation.

Chronic bronchitis (CB) is characterized by a persistent productive cough lasting more than 3 months and recurring over 2 consecutive years (4). The pathogenic factors contributing to CB encompass inhalation of respiratory irritants, recurrent bacterial and/or viral infections, preexisting chronic respiratory diseases, and continual exposure to environmental pollutants. The pathophysiology of CB involves an excessive production and secretion of mucus by goblet cells, resulting in a distinctive cough. This condition aggravates airflow obstruction and deteriorates lung function. An epidemiological study (5) has demonstrated that individuals with CB are at a significantly increased risk of developing new-onset chronic obstructive pulmonary disease and exhibit a higher mortality rate. The characteristic pathological alterations in CB—including goblet cell hyperplasia, mucus hypersecretion, and subsequent mechanical obstruction of small airways, airway

Highlight box

Key findings

- The pathological characteristics of chronic bronchitis (CB), including mucus hypersecretion, emphysema, and wall thickening, were found to be key factors that were associated with greater severity of viral pneumonia and worse clinical outcomes.

What is known and what is new?

- The pathological characteristics of CB include mucus hypersecretion, emphysema, and wall thickening.
- In this study, mucus hypersecretion in patients with CB was identified as a factor correlated with a greater severity of viral pneumonia and worse clinical outcomes.

What is the implication, and what should change now?

- This study examined the characteristics of CB in the context of mucus hypersecretion, emphysema, and bronchial wall thickening, with the aim of characterizing the manifestations of CB within the framework of viral lung infections. Moreover, we identified critical factors that aggravate viral pneumonia and potentially influence its clinical outcomes, thereby offering novel insights and evidence for the diagnosis and management of CB. The special pathological characteristics of CB and their implications suggest that clinicians should confirm the diagnosis as early as possible. After diagnosis, measures such as controlling symptoms, preventing infections, and administering appropriate medication should be applied.

structural remodeling, and abnormal surface tension—would lead to adverse clinical outcomes such as accelerated decline in pulmonary function and heightened susceptibility to lower respiratory tract infections (6).

However, the mechanisms underlying viral infection susceptibility in CB patients, as well as the associations between computed tomography (CT)-derived phenotypes and clinical prognosis, remain poorly defined. Crucially, there is a lack of validated CT-based quantitative parameters for stratifying post-viral pneumonia severity in this population.

The objective of this study was to investigate the association of CB and its related CT imaging characteristics with the severity and clinical outcomes of viral pneumonia. The purpose of this study was to provide a foundation for personalized clinical treatment plans following viral infection, with the ultimate goal of reducing the adverse effects of acute respiratory infections. We hypothesized that CB and its characteristics increase the severity of viral pneumonia, leading to more severe clinical manifestations and poorer clinical outcomes. We present this article in accordance with the STARD reporting checklist (available at https://jtd.amegroups.com/article/view/10.21037/jtd-2025-638/rc).

Methods

Study design and participants

The study was conducted in accordance with the Declaration of Helsinki and its subsequent amendments. This study received approval from the Ethics Committee for Human Research at Ningbo No. 2 Hospital (Ningbo, Zhejiang, China; approval No. YJ-NBEY-KY-2024-046-01). Informed consent was taken from all the patients. We retrospectively collected data from consecutively admitted patients meeting inclusion criteria between December 2022 and December 2024 at Ningbo No. 2 Hospital. Random sampling was not employed given the observational cohort design. Participants were stratified into two cohorts by mucus plugging score: high mucus burden (mucus plugging score ≥4, group 1) and low mucus burden (mucus plugging score <4, group 2). The patients were further stratified into CB and non-CB groups based on whether they met the diagnostic criteria for CB [post-bronchodilator forced expiratory volume in 1 second/forced vital capacity (FEV1/FVC) <0.7 with chronic cough and sputum production for ≥3 months per year over ≥2 consecutive years]. The inclusion criteria were as follows: age 18 years or older, confirmed etiological diagnosis of viral pneumonia, availability of complete clinical data and initial and follow-up chest CT images, and willingness to provide informed consent. Meanwhile, the exclusion criteria were as follows: a history of lung cancer or related treatment, diagnosis of infection with other pathogens or noninfectious diseases (e.g., drug-induced lung inflammation), and incomplete data or poor CT image quality (*Figure 1*). Baseline covariates encompassed demographic characteristics (age, sex, smoking history) and laboratory parameters [red blood cell count (RBC), white blood cell count (WBC), hemoglobin (Hb), and C-reactive protein (CRP)]. CB severity was systematically evaluated using CT severity scores and quantitative imaging biomarkers, including consolidation, ground-glass opacities (GGOs), reticular patterns, emphysema, Pi10 (the square root of the wall area of a hypothetical airway with an internal perimeter of 10 mm, standardized metrics reflecting airway wall thickness), and bronchial-related metrics (e.g., lumen diameter, wall area%). Primary outcomes focused on radiographic prognosis, defined by longitudinal changes in CT severity scores and the temporal evolution of specific imaging features (e.g., resolution of consolidation, progression of GGO).

CT image acquisition and interpretation

All participants underwent an initial CT scan upon admission. A follow-up CT was conducted within a 3-month period with a 96-slice, dual-source, dual-detector scanner (SOMATOM Force, Siemens Healthineers, Erlangen, Germany). The selection of this 3-month interval for follow-up was grounded on comprehensive clinical experience, as this duration offers an adequate observation window to evaluate the trajectory of a patient's recovery.

During the CT procedure, patients were directed to assume a relaxed bodily posture and maintain breath-holding at the peak of deep inhalation. The scanning coverage encompassed the area from the apex of the thorax to the plane of the bilateral diaphragmatic surfaces. The specific scanning parameters were as follows: tube voltage, 100–130 kV; an automated tube current; pitch setting, 1.2; the gantry rotation time, 2.88 seconds; collimation, 0.6 mm × 192 mm; slice thickness, 5 mm; and image-reconstruction slice thickness, 1 mm.

All 260 patients underwent follow-up CT scans with the same scanner employed in their initial examination. Two senior cardiothoracic radiologists who were blinded to clinical or laboratory findings and patient outcomes

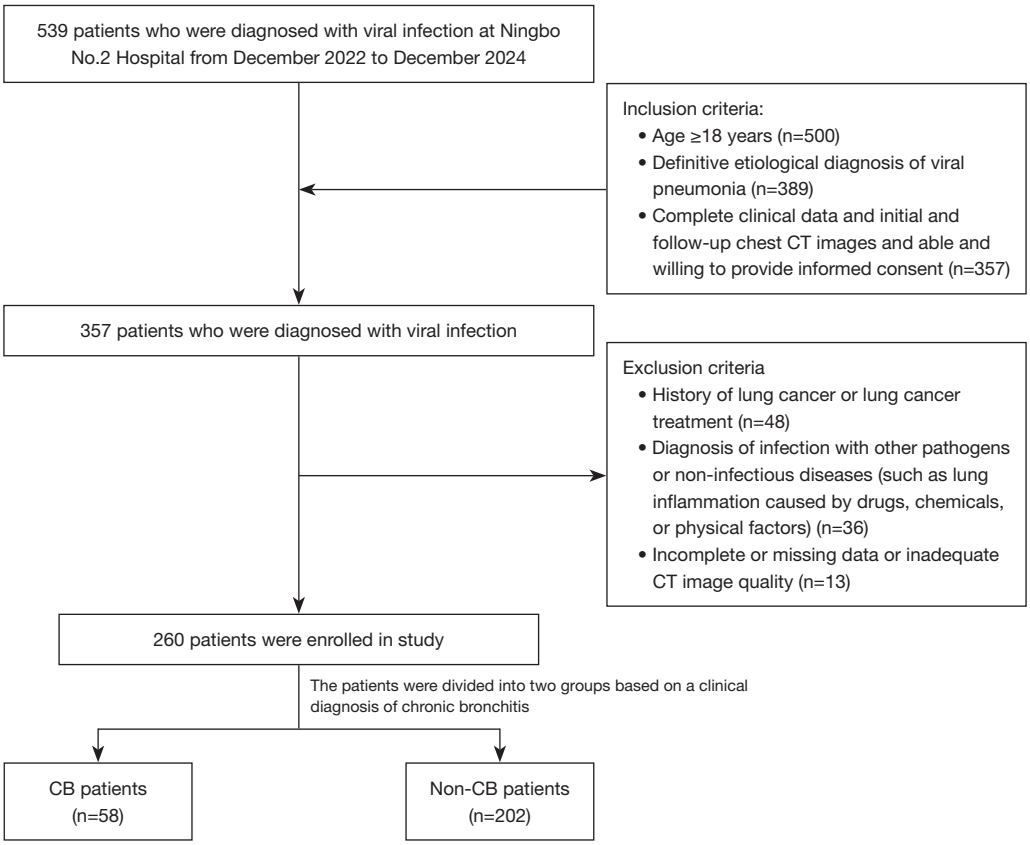

Figure 1 Flowchart of participant inclusion. CB, chronic bronchitis; CT, computed tomography.

reviewed all CT images in a random order. Each lung segment was systematically and independently examined by the two radiologists for the presence of mucous plugs, with a score of 1 or 0 assigned accordingly. The scores were then summed, with the 18 lung segments in both lungs being considered, resulting in a total score ranging from 0 to 18. Mucus plugs were defined as foci of heterogeneous density that completely obstructed the airway lumen that had no correlation with airway size or generation, with lesions within 2 cm of the parietal or diaphragmatic pleura being excluded. The final scores from both raters were averaged to generate a CT mucus score for each participant (7) (*Figure 2*). The mean values of the two radiologists' assessments were calculated. Patients were divided into the following two groups according to a mucus score: the high mucus burden group (score ≥4; group 1) and the low mucus burden group (score <4; group 2).

For each patient, the primary CT patterns (8) were identified as interleukin-6 (IL-6), GGO, consolidation, reticulation, and emphysema, in accordance with the Fleischner Society glossary. The bronchial wall and lumen volumes for all patients were evaluated. To assess the severity of pulmonary lesions evident in the imaging results, two radiologists semi-quantitatively scored all CT images based on the affected area within each of the lung's five lobes. The scoring scheme is as follows: 0, no involvement; 1, less than 5% involvement; 2, 5–25% involvement; 3, 26–49% involvement; 4, 50–75% involvement; and 5, more than 75% involvement. The overall CT score was derived by adding the scores from each individual lobe, resulting in a range of 0 to 25 (9) (*Figure 3*).

Statistical analysis

Statistical analyses were conducted using SPSS version 27.0 (IBM Corporation, Armonk, NY, USA). Missing values were imputed via the median. Data that were normally distributed were presented as mean and standard deviation (SD), while nonnormally distributed data were presented as the median and interquartile range (IQR). Categorical data

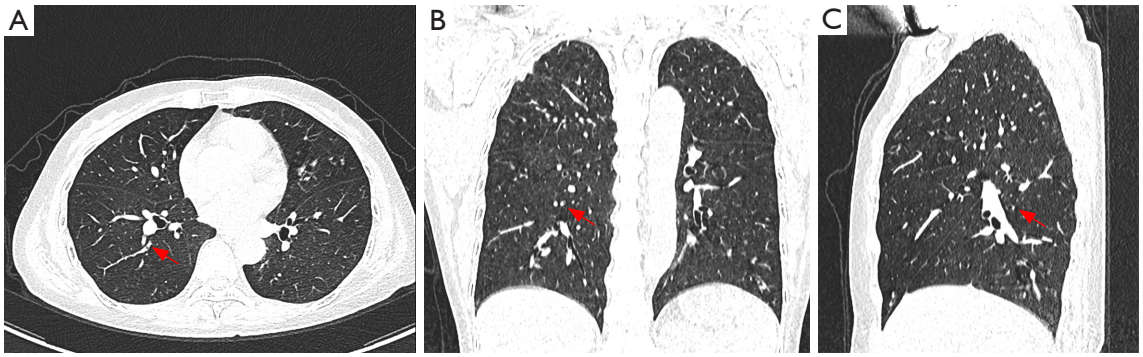

Figure 2 The CT imaging of a 63-year-old patient. A nodular high-density shadow within the middle segmental bronchus of the right upper lung lobe (the location is indicated by arrows) with a mucus score of 1. (A) Axial image. (B) Coronal image. (C) Sagittal image. CT, computed tomography.

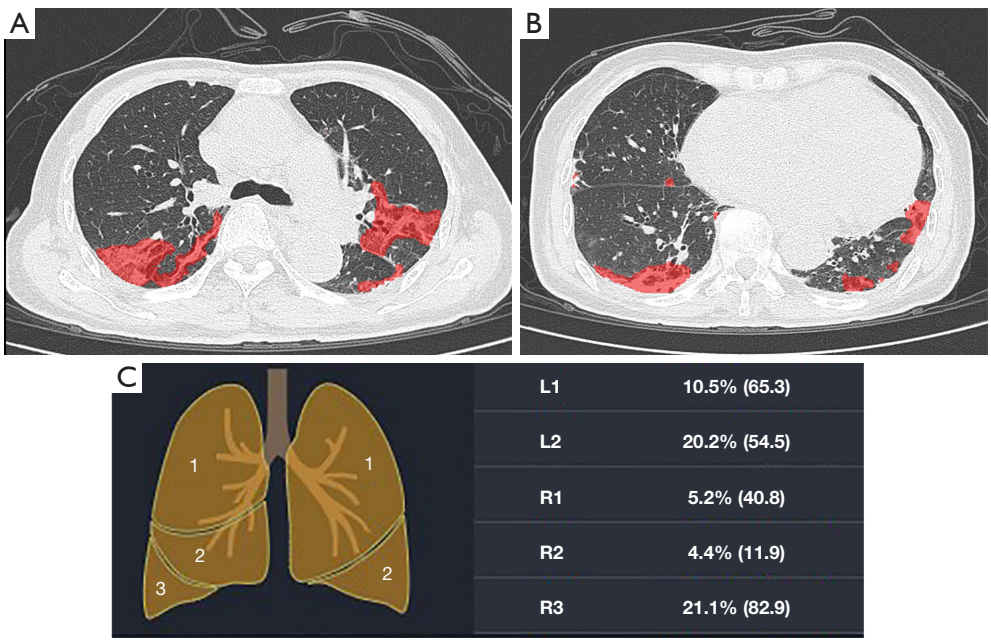

Figure 3 The CT imaging of lung infection in a 76-year-old female. (A-C) The aortic arch level, tracheal bifurcation level, and four-chamber heart level, respectively. The areas covered in red are identified as regions of lung infection. Based on the quantification of the severity of lung infection inflammation, the CT score is 9. CT, computed tomography; L1, left upper lung; L2, left lower lung; R1, right upper lung; R2, right middle lung; R3, right lower lung.

were presented as the number and percentage. Continuous variables that met the criteria of normality and homogeneity of variance tests were expressed as the mean ± SD, and the *t*-test was employed for intergroup comparisons. Conversely, data not adhering to a normal distribution were presented as the median and IQR, with the Mann-Whitney test being used for intergroup comparisons. Categorical variables underwent descriptive analysis using frequency and percentage [n (%)]. The choice of nonparametric tests was determined based on factors such as theoretical frequency and other relevant criteria. Logistic regression analysis was conducted to identify independent risk factors for CB patient classification and evaluate the diagnostic performance of the predictive model. All statistical tests

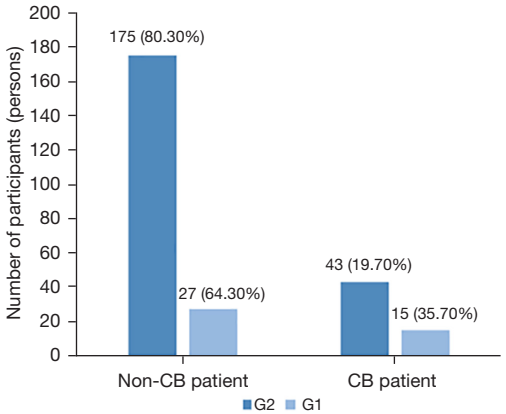

Figure 4 Number and proportion of the two groups. The figure shows the distribution of patients with or without CB. Patients without CB constituted the vast majority (80.30%) of patients, while patients with CB accounted for 19.70% in group 2, while in group 1, 35.70% of patients had CB, indicating a difference in the distribution of patients with CB between the two groups. Group 1, high mucus group; score ≥4. Group 2, low mucus group; score <4. CB, chronic bronchitis.

were two-sided, with a P value <0.05 considered statistically significant.

Results

Demographic information and patient characteristics

This study enrolled 260 patients who were categorized into group 1 (n=42; CB prevalence: 35.7%) and group 2 (n=218; CB prevalence: 19.7%) (*Figure 4*). Patients diagnosed with CB were significantly older (P<0.001) and more likely to be female (P=0.03) compared to those without CB. Patients with CB had a lower RBC count (P=0.02) and Hb levels (P=0.04), but no significant difference was found in white blood cell count. No significant differences were observed in mucus score, IL-6 level, or other indicators between the two groups. Although there were no significant differences in sex, white blood cell count, RBC count, Hb level, or mucus score (all P values >0.05), age was significantly different in patients in group 1 (*Table 1*).

Comparison of initial CT findings in groups 1 and 2

In group 2 (*Table 2*), patients with CB had significantly higher emphysema volumes (P=0.002) after pulmonary viral infections, as well as significantly lower luminal volume (P<0.001). In addition, more severe reticulation changes were observed on chest CT images in patients with CB (P=0.02), while no significant differences were found between the two groups in terms of low attenuation area (LAA; used to quantify emphysema severity) or Pi10 indices. Although there were no significant differences in LAA, LAA percentage (LAA%), reticulation, GGO, consolidation, or Pi10 indices between the two groups, statistically significant differences in emphysema in group 2 and wall volume among patients were observed in both groups.

Univariate analysis of the two groups after lung viral infection

Age was found to be a significant factor affecting the probability of CB (P=0.05) in group 1, and the risk of CB increased with age [odds ratio (OR) =1.10], but the actual impact range was small [95% confidence interval (CI): 1.00–1.22]. In group 2, univariate analysis showed that sex (P=0.02) and age (P<0.001) were significantly associated with the classification of patients with CB. Among hematological indicators, RBC count (P=0.02) and Hb level (P=0.04) were lower in patients with CB. In terms of chest CT imaging features, emphysema (P=0.005) was a significant factor in patients with CB. The decrease in wall volume (P=0.02) and the significant decrease in luminal volume (P=0.002) were significantly associated with the classification of patients with CB. The negative effect of luminal volume (OR <1) was more pronounced, indicating that the decrease in luminal volume was a strong predictor of CB (*Table 3*).

Multivariate analysis and receiver operating characteristic (ROC) curve analysis for all patients in group 2 after lung viral infection

Multivariate analysis revealed significant associations between CB patient classification and sex, age, and emphysema in group 2. Specifically, sex (P=0.009) was significantly associated with CB patient classification, with female patients at higher risk (OR =3.39). Age (P=0.004) was also a significant risk factor, with the risk of CB increasing with age (OR =1.06). Emphysema (P=0.005) remained significant in multivariate analysis, suggesting its status as an independent risk factor for CB. Notably, although luminal volume approached significance in the multivariate model (P=0.06), its effect was relatively small (OR =0.96) and the lower limit of the CI was close to 1, suggesting that

Table 1 Demographic information and patient characteristics

Characteristics	Patients without CB	Patients with CB	*t*/Z value	P value
Sex				
G1			0.05	0.82
M	12 (44.44)	8 (53.33)		
F	15 (55.56)	7 (46.47)		
G2			4.96	0.03*
M	80 (45.71)	11 (25.58)		
F	95 (54.29)	32 (74.42)		
Age (years)				
G1	77 [72.5, 82]	83 [77.5, 87]	–1.79	0.08
G2	70 [62, 77]	79 [74.5, 86.5]	–4.71	<0.001***
Smoking history				
G1			0.01	0.01*
Never-smoker	5 (18.52)	2 (13.33)		
Ex-smoker	8 (29.63)	3 (20.00)		
Current smoker	14 (51.85)	10 (66.67)		
G2			9.47	0.002**
Never-smoker	20 (11.43)	5 (11.63)		
Ex-smoker	45 (25.71)	8 (18.60)		
Current smoker	110 (62.86)	30 (69.77)		
WBC				
G1	7.80 [6.00, 9.10]	6.20 [5.35, 8.80]	0.96	0.34
G2	6.50 [4.80, 7.70]	6.50 [5.15, 9.50]	–0.97	0.33
RBC				
G1	4.03 [3.51, 4.38]	3.78 [3.39, 4.16]	0.84	0.41
G2	4.05 [3.81, 4.53]	3.73 [3.39, 4.29]	2.43	0.02*
Hb				
G1	122 [107, 132.5]	121 [102, 124]	0.91	0.37
G2	124 [115.5, 136]	119 [105.5, 131]	2.04	0.04*
Mucus score				
G1	6.00 [4.75, 7.00]	6.00 [4.75, 7.00]	–0.09	0.94
G2	1.50 [0.50, 2.00]	2.00 [0.50, 2.50]	–0.92	0.36
CT score				
G1	10.00 [6.50, 12.50]	8.00 [7.00, 9.500]	1.10	0.27
G2	5.00 [0.00, 8.00]	6.00 [1.50, 10.00]	–1.67	0.09

Data are presented as n (%) or median [interquartile range]. G1: group 1, high mucus group; score ≥4. G2: group 2, low mucus group; score <4. *, P<0.05; **, P<0.01; ***, P<0.001. CB, chronic bronchitis; CT, computed tomography; F, female; Hb, hemoglobin; M, male; RBC, red blood cell count; WBC, white blood cell count.

Table 2 Comparison of initial CT findings in groups 1 and 2

Characteristics	Patients with CB	Patients without CB	*t*/Z value	P value
LAA%				
G1	0.59 [0.20, 1.78]	0.80 [0.19, 1.35]	−0.03	0.99
G2	1.46 [0.26, 6.64]	1.85 [0.15, 6.02]	0.13	0.99
Reticulation				
G1	7.07 [3.50, 12.02]	6.28 [3.69, 11.23]	−0.07	0.96
G2	2.50 [0.57, 6.19]	6.02 [1.42, 10.08]	−2.33	0.02*
GGO				
G1	13.85 [5.2, 2.17]	8.53 [4.22, 15.48]	1.011	0.32
G2	2.18 [0.67, 6.93]	2.73 [0.99, 12.37]	−0.99	0.32
Consolidation				
G1	0.25 [0.07, 0.54]	0.18 [0.12, 0.29]	0.709	0.49
G2	0.09 [0.02, 0.27]	0.09 [0.03, 0.24]	−0.30	0.76
Emphysema				
G1	0.06 [0.02, 0.12]	0.29 [0.06, 0.90]	−1.89	0.06
G2	0.05 [0.01, 0.19]	0.21 [0.04, 0.73]	−3.12	0.002**
Pi10				
G1	4.77 [3.98, 5.32]	4.90 [1.64, 5.42]	0.38	0.71
G2	4.25 [3.44, 5.00]	4.19 [2.61, 5.09]	0.66	0.51
Wall volume				
G1	21.60 [10.30, 37.69]	6.28 [1.54, 15.71]	2.24	0.03*
G2	35.08 [13.38, 51.59]	18.90 [3.43, 45.53]	2.50	0.01*
Lumen volume				
G1	5.29 [2.69, 12.30]	4.57 [1.02, 12.26]	0.55	0.59
G2	13.63 [5.19, 25.29]	5.00 [0.67, 13.46]	3.95	<0.001***

Data are presented as median [interquartile range]. G1: group 1, high mucus group; score ≥4. G2: group 2, low mucus group; score <4. *, P<0.05; **, P<0.01; ***, P<0.001. CB, chronic bronchitis; CT, computed tomography; GGO, ground-glass opacity; LAA, low attenuation area.

although luminal volume may exert a degree of influence on the classification of patients with CB, its effect is limited (*Table 4*). ROC curve analysis showed that when the optimal cutoff value for emphysema was 0.15, the sensitivity of emphysema was 0.88 and the specificity was 0.66 (*Table 5*). The mean area under the curve (AUC) for all variables was 0.83. According to the established diagnostic discrimination benchmarks (e.g., Hosmer-Lemeshow criteria), AUC values of 0.7–0.9 indicate moderate to strong predictive utility, aligning with the observed performance of the included variables (*Figure 5*).

Mann-Whitney test for initial and follow-up CT

A significant difference was observed in the initial CRP level between patients with and without CB in group 1 (P=0.002). The median CRP level of patients without CB was significantly higher than that of patients with CB, suggesting a more pronounced inflammatory response in patients without CB. However, this difference was not

Table 3 Univariate analysis of the two groups after lung viral infection

Characteristics	B	Standard error	Wald	Degrees of freedom	P value	OR	95% CI of OR	
							Lower limit	Upper limit
G1								
Age	0.10	0.05	3.99	1	0.046*	1.10	1.00	1.22
RBC	−0.38	0.54	0.47	1	0.49	0.69	0.24	1.98
Mucus score	0.06	0.18	0.09	1	0.76	1.06	0.74	1.51
Emphysema	0.98	0.69	2.04	1	0.15	2.66	0.69	10.17
Pi10	−0.12	0.15	0.61	1	0.44	0.89	0.67	1.12
Wall volume	−0.02	0.02	1.55	1	0.21	0.98	0.95	1.01
Lumen volume	−0.01	0.03	0.01	1	0.92	0.99	0.95	1.05
G2								
Sex	0.90	0.38	5.53	1	0.02*	2.45	1.16	5.17
Age	0.08	0.02	19.20	1	<0.001***	1.08	1.04	1.12
RBC	−0.54	0.23	5.72	1	0.02*	0.58	0.37	0.91
Hb	−0.02	0.01	4.13	1	0.042*	0.98	0.97	0.99
Emphysema	0.49	0.17	7.96	1	<0.001**	1.62	1.16	2.28
Wall volume	−0.02	0.01	5.22	1	0.02*	0.98	0.97	0.99
Lumen volume	−0.05	0.02	9.47	1	0.002**	0.95	0.92	0.98

G1: group 1, high mucus group; score ≥4. G2: group 2, low mucus group; score <4. *, P<0.05; **, P<0.01; ***, P<0.001. CI, confidence interval; Hb, hemoglobin; OR, odds ratio; RBC, red blood cell count.

significant at follow-up. A comparison of re-examination indicators among all patients in group 2 revealed significant differences in RBC count and Hb level, indicating a potential tendency toward anemia in patients with CB. Additionally, although the changes in platelet count did not reach statistical significance, a slight increase in platelets was observed after re-examination (P=0.07), potentially indicating a compensatory response. It is worth noting that the levels of inflammatory markers, including CRP, leukocytes, and IL-6, as well as their levels at follow-up, were not significantly different between the two groups (*Table 6*).

Comparison of CT and mucus scores between initial and follow-up scans

In both group 1 and group 2, the initial and follow-up CT scores were lower in the non-CB group as compared to those in the CB group; however, this difference was not statistically significant. Conversely, the CT scores in the CB group were consistently higher at both initial and follow-up assessments, with no significant difference observed between these two time points.

Comparing the initial mucus scores between patients with CB and the non-CB patients in group 1, the scores ranged from 4.75 to 7.00, and this variability was not statistically significant (P=0.94). However, the follow-up results indicated a decrease in mucus scores for both groups, with a more pronounced reduction observed in patients without CB. The median score decreased to 2.5 in these patients as compared to 4.0 in those with CB. In group 2, the mucus scores of patients without CB remained consistently low before and after follow-up, and statistical tests revealed no significant difference between the two scores (P=0.36–0.20). Conversely, while the mucus scores of patients with CB were slightly higher than those without CB at the initial evaluation, they demonstrated a clear decrease at follow-up, although this reduction was not

Table 4 Multivariate analysis and receiver operating characteristic curve analysis for all patients in group 2 after lung viral infection

Characteristics	B	Standard error	Wald	Degrees of freedom	P value	OR	95% CI of OR	
							Lower limit	Upper limit
Sex	1.22	0.47	6.81	1	0.009**	3.39	1.35	8.50
Age	0.06	0.02	8.38	1	0.004**	1.06	1.02	1.10
RBC	−0.61	0.90	0.45	1	0.50	0.55	0.09	3.19
Hb	0.00	0.03	0.00	1	>0.99	1.00	0.94	1.06
Emphysema	0.44	0.16	7.78	1	0.005**	1.56	1.14	2.13
Wall volume	−0.01	0.01	1.31	1	0.25	0.99	0.97	1.01
Lumen volume	−0.04	0.02	3.48	1	0.06	0.96	0.92	1.01

Group 2, low mucus group; score <4. **, P<0.01. CI, confidence interval; Hb, hemoglobin; OR, odds ratio; RBC, red blood cell count.

Table 5 ROC curve for all patients in group 2

Characteristics	AUC	95% CI		Optimal threshold	Sensitivity	Specificity
		Lower limit	Upper limit			
Emphysema	0.83	0.77	0.89	0.15	0.88	0.66

Group 2, low mucus group; score <4. AUC, area under the curve; CI, confidence interval; ROC, receiver operating characteristic.

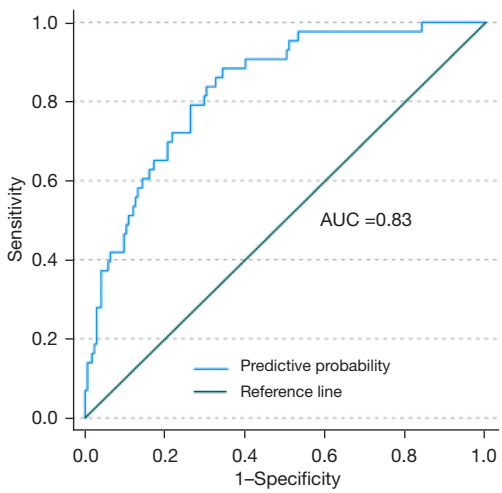

Figure 5 The ROC analysis results show that the average AUC was 0.83. An AUC value within the range of 0.7 to 0.9 suggests a high predictive diagnostic value for the included variables, which is a relatively common scenario. AUC, area under the curve; ROC, receiver operating characteristic.

statistically significant (P=0.20) (*Table 7*).

Discussion

Respiratory viral infections trigger a cascade of pathological events initiated by neutrophil infiltration and subsequent epidermal growth factor receptor (EGFR)-mediated mucin biosynthesis, particularly MUC5AC overproduction (10). Concurrently, the expansion of antigen-presenting cells (monocytes, macrophages, and dendritic cells) and their excessive release of proinflammatory cytokines like IL-6 may precipitate systemic cytokine storm syndrome (11). Crucially, diverse respiratory viruses—including coronaviruses, influenza viruses, adenovirus (AdV), cytomegalovirus (CMV), and Epstein-Barr virus (EBV)—share common pathogenic mechanisms involving both disruption of airway epithelial tight junctions and dysregulation of mucin expression profiles (12). These interconnected processes ultimately compromise

Table 6 Mann-Whitney test comparing initial and follow-up CT

Characteristics	Patients without CB	Patients with CB	U value	Z value	P value
CRP					
Initial	37.50 [15.03, 69.63]	63.60 [17.19, 82.84]	3,183.00	−1.56	0.12
Follow-up	9.080 [2.30, 37.34]	14.94 [4.13, 33.66]	3,508.50	−0.69	0.49
WBC					
Initial	6.50 [4.80, 7.70]	6.50 [5.15, 9.50]	3,403.00	−0.97	0.33
Follow-up	7.30 [5.75, 8.70]	7.40 [5.85, 9.40]	3,559.00	−0.55	0.58
RBC					
Initial	4.05 [3.81, 4.53]	3.73 [3.39, 4.29]	4,664.50	2.43	0.02*
Follow-up	3.90 [3.50, 4.38]	3.78 [3.27, 4.02]	4,739.50	2.65	0.008**
Hb					
Initial	124 [115, 136]	119 [105, 131]	4,516.50	2.04	0.04*
Follow-up	119 [108, 133]	112 [101, 125]	4,575.00	2.19	0.03*
PLT					
Initial	200 [148, 248]	179 [133, 247]	4,098.50	0.91	0.37
Follow-up	216 [169, 309]	213 [160, 249]	4,442.00	1.83	0.07
IL-6					
Initial	132.20 [27.77, 132.20]	132.20 [15.13, 132.20]	3,988.50	0.61	0.48
Follow-up	132.20 [132.20, 132.20]	132.20 [42.11, 132.20]	4,208.50	1.20	0.11

Data are presented as median [interquartile range]. *, P<0.05; **, P<0.01. CB, chronic bronchitis; CRP, C-reactive protein; CT, computed tomography; Hb, hemoglobin; IL-6, interleukin-6; PLT, platelet; RBC, red blood cell count; WBC, white blood cell count.

mucociliary clearance capacity and establish a self-perpetuating cycle of mucus hypersecretion, mechanical airway obstruction, progressive ventilation impairment, and exacerbated inflammatory responses. The study (13) found that patients with CB were more likely to exhibit mucus hypersecretion than those without CB, a finding that aligns with the core features of CB pathophysiology. The research by scholars such as Angelis *et al.* has confirmed that the core pathophysiology of CB is chronic airway inflammation, primarily manifested as excessive mucus secretion, narrowing of small airways, and the development of emphysema, which further supports the findings of this study (14). Additionally, the mean age of patients with CB was significantly higher than that of those without CB, which is in line with epidemiological statistics (15,16) and suggests that CB primarily affects individuals over 40 years of age. This further strengthens the reliability and generalizability of the study's conclusions.

Moreover, the study by Rathnayake *et al.* (17) found that smoking alters the cellular composition of the bronchial mucus barrier, reprograms the transcriptome, and increases mucus production. This is reflected in our data, which showed that the proportion of current smokers was significantly higher in the CB group than in the non-CB group. We hypothesize that this phenomenon may be associated with smoking-induced pathophysiological changes, such as activation of the EGFR/IL-13 signaling pathway by tobacco smoke, leading to goblet cell metaplasia and reduced ciliated cells (18). Upregulation of mucin-encoding genes (e.g., MUC5AC, MUC5B) further promotes mucus hypersecretion (19). These alterations collectively increase mucus accumulation in the airways, contributing to a cascade of clinical manifestations. The accumulation of airway mucus enhances susceptibility to viral infections while simultaneously impairing mucociliary clearance, resulting in aggravated infection and prolonged clinical course.

Widysanto *et al.* (5) reported that patients with CB

Table 7 Comparison of CT and mucus scores between initial and follow-up scans

Characteristics	Non-CB patient	CB patient	Z value	P value
CT score				
G1				
Initial	5.0 [0.0, 8.0]	6.0 [1.5, 10.0]	–1.66	0.09
Follow-up	3.0 [0.0, 7.0]	6.0 [0.0, 9.0]	–1.05	0.28
G2				
Initial	10.0 [6.5, 12.5]	8.0 [7.0, 9.5]	1.10	0.27
Follow-up	5.0 [0.0, 13.0]	7.0 [0.0, 9.0]	0.58	0.56
Mucus score				
G1				
Initial	6.00 [4.75, 7.00]	6.00 [4.75, 7.00]	–0.09	0.94
Follow-up	2.50 [1.50, 5.00]	4.00 [2.00, 5.25]	–0.80	0.43
G2				
Initial	1.50 [0.50, 2.00]	2.00 [0.50, 2.50]	–0.92	0.36
Follow-up	1.50 [0.50, 2.00]	1.50 [0.50, 3.00]	–1.27	0.20

Data are presented as median [interquartile range]. G1: group 1, high mucus group; score ≥4. G2: group 2, low mucus group; score <4. CB, chronic bronchitis; CT, computed tomography.

experience long-term airway inflammation, leading to pathological changes such as wall thickening and small airway obstruction. These changes heighten the sensitivity of the airways to external stimuli such as viruses, thereby increasing the susceptibility of these patients to infection. Upon reinfection, these patients are more likely to experience alveolar mucosal damage and intense inflammatory responses. This further exacerbates mucus secretion and impairs gas exchange, ultimately resulting in hypoxia (20). Under hypoxic conditions, the body may have an elevated RBC count and Hb level through compensatory mechanisms, such as negative feedback regulation, to enhance the blood's oxygen-carrying capacity. Consequently, it is hypothesized that RBC count and Hb level in patients with CB are higher than those in patients without CB. However, the findings of this study diverge from this expectation. A potential explanation for this discrepancy could be that prolonged hypoxia and compensatory pathological processes may affect the hematopoietic system or RBC function in patients with CB, resulting in relatively low RBC and Hb levels despite the hypoxic state (20).

Our study identified a noteworthy phenomenon in group 1: non-CB patients demonstrated significantly higher CRP levels compared to CB patients. This observation likely reflects the unique pathophysiology of CB progression, suggesting that CB patients exhibit distinct inflammatory response patterns differing from acute inflammation characteristics (12). Specifically, the long-term chronic airway inflammation in CB patients may lead to altered immunomodulatory mechanisms, manifesting as relative suppression of acute-phase protein release (including CRP) (21). Conversely, elevated CRP levels in non-CB patients may indicate recent acute infections or other unidentified proinflammatory states. This discovery challenges the conventional assumption that CB is invariably associated with more pronounced systemic inflammatory responses, highlighting the need for comprehensive evaluation of inflammatory profiles across patient subtypes. Critical considerations should include disease staging, comorbidities, and treatment history as potential confounding factors. Radiological research (5) has identified the imaging characteristics of patients with CB, such as GGO and bronchial vessel wall thickening. These findings align with the statistically significant features identified in our study. It is well-established that airway epithelial cell dysfunction due to CB can result in obstruction, contraction, and spasms of the small bronchi, which may progress to obstructive emphysema—a condition more common in patients with

CB. Tana *et al.* (22) compared the bronchial lumen volumes between two patient groups and found that there were abnormal epithelial cell proliferation and invasive changes in the airway wall during the pathological CB process. This led to a reduced lumen diameter and obstructed airflow, resulting in a notably smaller lumen volume in patients with CB as compared to those without CB. Moreover, a comprehensive review of multiple studies (23) highlighted that excessive mucus secretion in the airways of patients with CB is a persistent issue, even after viral infection. In our study, we found that while mucus scores were decreased for both patient groups upon re-examination, the decline was more pronounced in patients without CB. The initial mucus scores for patients with CB were marginally higher than those without CB, consistent with the observation of heightened airway mucus secretion in these patients (24). This phenomenon not only worsens airway obstruction but also amplifies local hypoxia, creating an environment conducive to microbial growth and reproduction, thereby elevating infection risk (24). We found that both the initial and follow-up CT scores of CB patients were significantly higher than those without CB. It is speculated that this may have affected pneumonia absorption and daily activity capabilities in patients with CB (25).

Our study involved certain limitations which should be addressed. First, the limited sample size might reduce the representativeness of the sample, thereby making it challenging to generalize the conclusions to a wider patient population. Additionally, relevant confounding factors such as environmental pollution could not be incorporated into the study. Future research should include a larger sample size and an extended follow-up period to produce more robust findings. We have incorporated smoking status data, but the lack of detailed environmental exposure metrics (e.g., air pollution levels, occupational exposures) represents an important study limitation that should be addressed in future research. Second, the existing mucus scoring system only allows for a binary assessment of mucus severity at the lung segment level (26), which does not quantify the amount of mucus partially obstructing the airway lumen. Despite not causing complete airway closure, a partially obstructing mucus could significantly impact gas exchange, airway defense mechanisms, and microbial colonization. Meanwhile, there is a certain degree of unreliability in manually scoring mucus. Therefore, to enhance the accuracy and clinical utility for future studies, new methods or technologies should be explored to quantify mucus in the bronchial and lung segments. Third, consolidation areas resulting from lung infections could obscure the presence of mucus plugs, making their imaging manifestations difficult to identify. Morphological changes in the lung tissue due to consolidation could also alter the natural distribution pattern of mucus in the lungs, leading to inaccurate assessment results from the mucus scoring system. This confounding factor should thus be identified and controlled during data analysis and result interpretation, and experimental designs should aim to minimize interference. Finally, as we employed a single-center, retrospective design, the generalizability and reliability of the conclusions may be limited. To enhance the robustness and generalizability of the conclusions, multicenter, prospective studies should be conducted to further validate and consolidate the existing findings, thereby reducing the susceptibility to bias and errors inherent in single-center studies.

This study examined the variations in mucus secretion and inflammation levels in the lungs of patients with and without CB in the context of viral lung infections. The findings indicated that patients with CB were more susceptible to mucus hypersecretion following a viral infection, aligning with the primary pathological-physiological characteristics of persistent mucus hypersecretion. Furthermore, patients with CB tended to be older, which aligns with the epidemiological data. A comprehensive analysis of the pathophysiological mechanisms revealed that the airways of patients with CB were chronically inflamed, resulting in wall thickening, small airway obstruction, and increased sensitivity to viral stimuli. These factors intensify alveolar mucosal damage, inflammatory responses, and mucus secretion, ultimately impacting gas exchange and hypoxia. Moreover, we systematically analyzed the radiological manifestations in the lungs after viral infection, confirming the presence of specific changes such as emphysema and bronchial wall thickening in patients with CB. Following viral lung infections, the CT scores of patients with CB remained relatively high. Under conditions of high mucus secretion, the reduction in mucus score in patients with CB was less significant than that in patients without CB, suggesting that high mucus secretion adversely affects disease progression and prognosis.

Furthermore, Dal Negro *et al.*'s study (27) has indicated that for patients with CB who have a mucus plugging, the rehydration and restoration of mucous osmotic and viscous/elastic properties should be administered. Only when complementary hydrating and mucolytic agents are applied to clear the mucus accumulated in the lungs will airway

obstruction, inflammation, and indeed, infection broadly improve (28).

In conclusion, this study examined the characteristics of CB in the context of mucus hypersecretion, emphysema, and bronchial wall thickening and clarified the distinct manifestations of CB within the framework of viral lung infections. Moreover, we identified the critical factors that increase the severity of viral pneumonia and shape its clinical outcomes, thereby offering novel insights and evidence for the diagnosis and management of CB.

Conclusions

The pathological features of CB, such as mucus hypersecretion, emphysema, and wall thickening, are key factors exacerbating the severity of viral pneumonia and worsening clinical outcomes. CB-specific pathological changes play a critical role in aggravating viral pneumonia through mechanisms like increased airway obstruction, localized hypoxia, and susceptibility to microbial proliferation. Understanding these associations emphasizes the importance of targeted therapeutic strategies, including improving mucus clearance, reducing inflammation, and mitigating structural damage, thereby enhancing prognosis and clinical outcomes in CB patients.

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Footnote

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Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. The study was conducted in accordance with the Declaration of Helsinki and its subsequent amendments. This study received approval from the Ethics Committee for Human Research at Ningbo No. 2 Hospital (Ningbo, Zhejiang, China; approval No. YJ-NBEY-KY-2024-046-01) and informed consent was taken from all the patients.

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