Clinical Application of High-Resolution Computed Tomographic Imaging Features of Community-Acquired Pneumonia

Yunqiang Nie
Cuiyun Li
Jingling Zhang
Hui Wang
Xin Lv
Xinyi Xu
Miao Guo

Corresponding Authors: Yunqiang Nie, e-mail: nieyunqiang1978@163.com; Miao Guo, e-mail: 779841608@qq.com

Background: This article discusses the value of high-resolution computed tomography (HRCT) in the diagnosis and treatment of pulmonary infections. Lung infection caused by pathogens is an important cause of death. Traditional methods to treat lung infection involved empirical antibiotic therapy. Thin-slice CT scanning is widely used in the clinical setting, and HRCT scan can very clearly show alveolar and bronchiolar involvement of infection.

Material/Methods: In total, 178 patients with community-acquired pneumonia (CAP) were enrolled. All the patients underwent CT scan, qualified sputum, and blood samples for culture or immunological biochemical tests. CT imaging features, pathogenic bacteria, and treatment results were used for statistical analysis.

Results: In 77 patients with lobar consolidation, the rate of detection was 43.26% (77/178), and in 101 patients with lobular pneumonia it was 56.74% (101/178). In 51 patients, pathogenic bacteria were detected (28.65%, 51/178). Sixteen of 33 patients detected with bacteria had cavities (48.5%, 16/33) and 35 of 145 patients detected with bacteria had no cavities (24.1%, 35/145). The difference between the 2 groups was statistically significant ($\chi^2=7.795$, $P=0.005$). According to the pathogenic bacteria, 38 patients were cured (74.51%, 38/51), and according to the CT imaging features 81 patients were cured (71.05%, 81/114). No statistically significant difference was found between them ($\chi^2=0.209$, $P=0.647$).

Conclusions: Treatment effect of CAP based on HRCT findings is not inferior to treatment effect guided by microbial characterization.

MeSH Keywords: Lung • Pleural Cavity • Tomography, Emission-Computed

Full-text PDF: http://www.medscimonit.com/abstract/index/idArt/895638
**Background**

Lung infection is a common respiratory disease caused by pathogens such as *Streptococcus pneumoniae, Haemophilus influenzae*, mycoplasma, fungi, *Klebsiella pneumoniae*, and viruses, and is an important cause of death [1]. With an increase in the number of immunologically compromised patients, the number of patients who lack clinical symptoms has increased tremendously [2]. Thus, the key to solving this problem is timely and effective antibiotic therapy [3]. Traditional methods first involved empirical antibiotic therapy, and later, antibiotic therapy was adjusted based on the antibiotic sensitivity tests. The results of diagnostic significance of culturing the bacteria was obtained in 3–5 days; however, the positive rate of culturing was only 40% [4]. Thin-slice computed tomography (CT) scanning is widely used in the clinical setting. The imaging feature of high-resolution CT (HRCT) scanning can very clearly identify secondary pulmonary lobules and can find alveolar and bronchiolar involvement of the infection. Due to different pathological characteristics of different pathogens, lobar pneumonia exudes edema fluid and neutrophils fill the alveolar space. The histological features of bronchial pneumonia include inflammation around the bronchioles. HRCT imaging has certain characteristics that are relevant in detecting pathogenic bacteria. Thus, in this study, early and progressed HRCT imaging features were used combined with laboratory findings. Pathogenic bacteria were evaluated promptly to guide the treatment. The aim of this study was to show how high-resolution CT imaging characteristics can guide anti-microbial treatment.

**Material and Methods**

**General characteristics**

In total, 178 patients (133 men and 45 women; mean age, 52.329±19.132 years) with community-acquired pneumonia (CAP) from Linyi City People’s Hospital, Shandong, China, between January 1, 2012, and December 31, 2014, were enrolled in this study. The inclusion criteria were based on the United States Infectious Disease Society/United States Thoracic Society “Guide to the Diagnosis and Treatment of Community-Acquired Pneumonia in Adults (2007)” as follows: (1) the advent of cough, sputum, or existing respiratory symptoms, and purulent sputum; (2) fever; (3) pulmonary consolidation syndrome and/or moist rales; and (4) white blood cell count $>$10×10^9/L or <4×10^9/L, with or without an accompanying shift to the left. With 1 of the aforementioned conditions, the clinical diagnosis of CAP can be established based on (5) an X-ray scan of the chest showing patchy infiltrate shadows or interstitial change, with or without pleural effusion, and excluding tuberculosis (TB), lung tumor, noninfectious interstitial lung disease, pulmonary edema, atelectasis, pulmonary embolism, pulmonary eosinophilia, and pulmonary vasculitis. Exclusion criteria include the following: (1) patients aged <18 years; (2) patients who did not undergo chest X-ray scan or CT; (3) evidence to suggest the chest shadow of a tumor or other noninfectious diseases; (4) patients with a medical history of hospital-acquired infections 1 week before hospitalization; (5) patients allergic to fluoroquinolone, beta-lactam, or macrolide drugs; (6) pregnant and breastfeeding women; and (7) patients with epilepsy and unstable mental status.

**Methods**

A retrospective analysis of 178 patients with CAP was done. We conducted a retrospective analysis to demonstrate the relationship between HRCT imaging characteristics and the etiology of lung lesions and the value of HRCT in treating community-acquired pneumonia.

The following examinations were performed: complete blood count, erythrocyte sedimentation rate, C-reactive protein, biochemical tests, sputum cultures, blood culture, and serum antigen test. CURB-65 criteria and 64-row volume reconstruction CT (Lightspeed VCT 64, GE Company, Fairfield, USA) scan were arranged during hospitalization. Chest CT scans were performed on arrival for patients who required hospitalization. CT scans were repeated after 72 hours if the empirical treatment did not show improvement, especially if pathogens were not detected or the disease deteriorated. Two radiologists, both associate professors or higher, reviewed the photographs simultaneously, eliminating the need for a double-blind approach. Empirical antimicrobial and symptomatic treatment was provided within 8 hours after diagnosis. Empirical anti-microbial treatment was given, with prevalence of stable vital signs and no etiology. We chose antibiotics that were effective against common pathogens, including streptococcus pneumonia, haemophilus influenza, and mycoplasma pneumonia. Beta-lactam or fluoroquinolone antibiotics were administered separately or in combination during empirical anti-microbial treatment.

**Standards of CT evaluation**

The 64-row CT had a window width of 1500, window level of −700, and thickness of layers of 5 mm. Sensitive areas were detected using a 0.625-mm reconstruction. Focus imaging features were lobar consolidation; lobular central lesions; cavity; ground-glass opacity; fast occurrence of cavity or pleural effusion; bronchiectasis or bronchial wall thickening; distribution of the subpleura; distribution in the confined segment or lobe; distribution of cross-segment, entire segment, or involvement of the whole leaf; or occurrence of interlobular septal thickening. The assessment standard for CT scan after 3–10 days included: (1) improved CT inflammation absorption $>$50% and
(2) progressive lesions expansion over 50%, or a cavity containing pleural fluid.

We adjusted the choice of antibiotics based on the scan results. If the first empirical antibiotics treatment was not effective within 72 hours, the antibiotics treatment was adjusted based on high-resolution CT imaging characteristics such as deterioration in lesions, expansion of consolidated lesion area, appearance of cavities, and pleural effusion or bronchiectasis inflammation-like changes, combined with possible pathogens (e.g., cocci infection, bacillus infection, and fungal infection) prompted by high-resolution CT imaging characteristics, antibiotics treatment may be adjusted. We used beta-lactam antibiotics with enzyme inhibitors or carbapenem antibiotics against gram-negative bacilli; vancomycin, teicoplanin, or linezolid against positive cocci, and voriconazole, itraconazole, or caspofungin against fungus.

**Assessment standards of treatment response**

The following conditions were considered as treatment failures or ineffective treatment: clinical conditions deteriorated after empirical therapy for 24 hours; clinical conditions at 72 hours remained precarious; and the clinical symptoms did not show improvement after 7 days and occurrence of fever.

Cure criteria were: (1) patients could maintain normal body temperature for more than 24 hours; (2) heart rate was ≤100 times/min at rest; (3) breath was ≤24 times/min at rest; (4) systolic blood pressure was ≥90 mmHg; and (5) arterial oxygen saturation was normal without oxygen uptake.

**Detection of pathogen**

**Detection of bacteria**

Respiratory secretion real specimens (i.e., phlegm, pharyngeal swab, or nasopharyngeal aspirate) were obtained from qualified patients before vaccination and were cultivated in blood plate, chocolate agar plate, and MacConkey plate at 35°C for 16–24 hours using a general method for separation and identification of bacteria. The K-B method was used to separate the cultivated bacteria in the activity assay. *In vitro* susceptibility testing by dilution method was used to determine the minimum inhibitory concentration (MIC) using the National Committee for Clinical Laboratory Standards 2001, for separating different bacteria based on the MIC value and judged for drug susceptibility (S), intermediate (I), and resistance (R).

**Detection of atypical pathogen**

The following tests were performed: (a) using an enzyme-linked immunoassay, *Legionella pneumophila* urinary antigen test was used to assay the urine specimen using kits purchased from the CORTEZ Company (CA, USA); and (b) 2 serum samples were collected from the patients with acute disease and convaleseing patients (2–4 weeks) using the particle agglutination detection of 2 *Mycoplasma pneumoniae* antibodies in the serum, using testing kits purchased from Fujirebio, Tokyo, Japan. A microimmunofluorescent method was used to detect *Chlamydia pneumoniae* immunoglobulin M (IgM) antibodies in double serum specimen, and the testing kit was purchased from the CORTEZ Company. An indirect immunofluorescence assay testing was used to determine *L. pneumophila* IgG in the 2 serum antibodies, and the kits for this were purchased from the CORTEZ Company.

**Determining the standards of testing positive for pathogens**

The following observations were made: (a) Eligible sputum specimens produced 1 or more strains of bacteria and showed moderate growth; (b) pathogens were detected in blood cultures; and (c) 2 serum samples were collected at 2- to 4-week intervals. The antibody titer increased or decreased by 4 times or more.

This study was approved by the Medical Ethics Committee of Linyi City People’s Hospital, Shandong, China. The nature, purpose, and potential risks of this study were explained to each subject prior to enrollment. Written informed consent was obtained from all enrolled patients.

**Statistical methods**

The SPSS 16.0 software package was used for statistical analysis. The nonpaired chi-square test was used. *P*<0.05 was considered as statistically significant.

**Results**

**Positive results of the pathogen and CT characteristics**

In total, 178 patients with a CURB-65 score of 2 points [5] (the score enables stratification of patients for 3 different management options) were included in this study. Those with low risk of death (scores 0 and 1, mortality <2%) were suitable for management as outpatients. Patients with a score of 2 (mortality 9%) were considered for hospital-supervised treatment. Patients with a score of ≥3 were at high risk of death (>19%) and hence were treated in hospitals, sometimes in the intensive care unit. Overall, pathogenic bacteria were detected in 51 of 178 (28.65%) patients: *S. pneumoniae* in 14 patients (7.87%, 14/178); *H. influenzae* in 2 patients (1.12%, 2/178); *Klebsiella* in 5 patients (2.8%, 5/178); *Aspergillus* in 12 patients (6.74%, 12/178); *Mycoplasma* in 3 patients (1.69%, 3/178);
pulmonary TB in 12 patients (6.74%, 12/178); and *Escherichia coli* in 3 patients (1.69%, 3/178). Further, 13 patients had non-infectious diseases: 5 patients with pulmonary infarction like pneumonia, 2 patients with pulmonary vasculitis, 3 patients with pneumonia adenocarcinoma, and 3 patients with interstitial lung disease. In 77 patients with lobar consolidation, pathogenic bacteria were detected in 17 patients, including *Streptococcus pneumoniae* in 12 patients, *Klebsiella pneumonia* in 3 patients, and *Aspergillus* in 1 patient. In 101 patients with lobular consolidation, pathogenic bacteria were detected in 34 patients. In 65 patients with only lobular consolidation, the detected bacteria were as follows: TB in 5 patients, *Aspergillus* in 3 patients, *H. influenza* in 2 patients, *S. pneumoniae* in 3 patients, and *E. coli* in 2 patients. In 20 patients with centrilobular consolidation with cavity lesions, the detected bacteria were as follows: *Aspergillus* in 8 patients, TB in 6 patients, and *Klebsiella pneumoniae* in 1 patient. In 8 patients with lobular consolidation and pleural effusion, the detected bacteria were only *E. coli* in 1 patient. In 5 patients with lobular consolidation and tree-in-bud, the detected bacteria were TB in 1 patient and mycoplasma in 1 patient. In 3 patients with ground-glass opacity and centrilobular consolidation, mycoplasma was detected in 1 patient. CT features and pathogenic bacteria are presented in Table 1.

### Table 1. Chest CT imaging features and microbiological tests.

| High-resolution CT features                  | Number of patients | Detection of pathogenic bacteria and number | Positive rate of pathogenic bacteria (%) |
|---------------------------------------------|--------------------|--------------------------------------------|-----------------------------------------|
| Lobar consolidation                        | 58                 | *Streptococcus pneumoniae* 10              | 7.87                                    |
|                                            |                    | *Klebsiella pneumonia* 3                 |                                         |
|                                            |                    | *Aspergillus* 1                          |                                         |
| Lobar consolidation with cavity            | 13                 | *Klebsiella pneumonia* 1                  | 0.06                                    |
| Lobar consolidation with hydrothorax       | 6                  | *Streptococcus pneumoniae* 2             | 1.12                                    |
| Total                                      | 77                 |                                           |                                         |
| Centrilobular consolidation                | 65                 | *TB* 5                                    | 7.87                                   |
|                                            |                    | *Aspergillusfumigatus* 3                |                                         |
|                                            |                    | *Haemophilus influenzae* 2              |                                         |
|                                            |                    | *Streptococcus pneumoniae* 2          |                                         |
|                                            |                    | *Escherichia coli* 2                    |                                         |
| Centrilobular consolidation with cavity    | 20                 | *Aspergillus* 8                          | 8.43                                    |
|                                            |                    | *TB* 6                                    |                                         |
|                                            |                    | *Klebsiella pneumoniae* 1              |                                         |
| Lobular consolidation with hydrothorax     | 8                  | *Escherichia coli* 1                     | 0.06                                    |
| Lobular consolidation with tree in bud      | 5                  | *Mycoplasma* 2                           | 1.69                                    |
|                                            |                    | *TB* 1                                    |                                         |
| Lobular consolidation with ground-glass opacity | 3                | *Mycoplasma* 1                           | 0.06                                    |
| Total                                      | 101                |                                           |                                         |
| **Total**                                  | **77**             | **17**                                    | **7.87**                                |

CT – computed tomography, TB – tuberculosis.

### Table 2. CT features affect bacteria detection.

| CT features                  | Number of positive bacteria | Rate of detection | Chi-square test |
|------------------------------|----------------------------|-------------------|-----------------|
| Lobar consolidation         | 17                         | 22.1% (17/77)     |                |
| Lobular consolidation       | 34                         | 33.7% (34/101)    |                |
| Cavity                      | 16                         | 48.5% (16/33)     |                |
| Noncavity                   | 35                         | 24.1% (35/145)    |                |

CT – computed tomography.
Pathogenic bacteria were detected in 51 patients (28.65%, 51/178), of which 17 patients had lobar consolidation and 34 patients had lobular consolidation ($P > 0.05$). Sixteen cases of pathogenic bacteria were detected in the patients with pulmonary cavity lesions and 35 cases in patients with no cavity lesions ($P < 0.05$). CT features and pathogenic bacteria are presented in Table 2.

Among pathogenic bacterial infections with basic diseases, the complication rate in *Aspergillus* infection was up to 91.7% ($P < 0.05$), for which low albumin was the main factor. The complication rate in TB infection was 69.2% ($P < 0.05$), for which high blood sugar was the main factor. Clinical complications and pathogenic bacteria are presented in Table 3.

**Table 3. Pathogenic bacteria and clinical complications.**

| Bacterium                  | Number of patients | Hypoalbuminemia | Diabetes mellitus | COPD | Uncomplicated cases |
|----------------------------|--------------------|-----------------|-------------------|------|-------------------|
| *Streptococcus pneumoniae* | 14                 | 2               | 1                 | 0    | 11                |
| *Aspergillus*              | 12                 | 7               | 3                 | 1    | 1                 |
| *Escherichia coli*         | 3                  | 1               | 1                 | 0    | 1                 |
| *Klebsiella pneumoniae*    | 5                  | 1               | 0                 | 0    | 4                 |
| *Haemophilus influenzae*   | 2                  | 0               | 0                 | 0    | 2                 |
| *Mycobacterium tuberculosis*| 12                 | 0               | 7                 | 2    | 4                 |
| *Mycoplasma*               | 3                  | 0               | 0                 | 0    | 3                 |

Fasting plasma glucose level $> 6.10$ mmol/L; 2-h blood glucose level after the meal $> 11.2$ mmol/L, low albumin $< 25$ g/L. COPD – chronic obstructive pulmonary disease.

**Figure 1. Streptococcus pneumoniae.** A 23-year-old male patient. The CT density of consolidation of the left pulmonary artery level was low, and after 2 days significantly higher density area was expanding. The patient was previously healthy, had fever, cough, and sputum for 5 days, and was hospitalized. Empirical beta-lactam antibiotics combined with fluoroquinolone antibiotics were provided. The symptom was progressive and body temperature was 40°C; cough, cough with phlegm, no hemoptysis, and no chills were found. The result of the sputum culture showed *Streptococcus pneumoniae*. After 2 days, the CT scan showed the progression of lesions in the left pulmonary artery level. Vancomycin was selected for anti-infection treatment; however, even after 1 week, there was still no improvement. The CT scan showed the progression of lesions and it was decided to switch to linezolid for anti-infection treatment. After 3 days, the patient had hypothermia and the symptoms improved.

The complication rate in TB infection was 69.2% ($P < 0.05$), for which high blood sugar was the main factor. Clinical complications and pathogenic bacteria are presented in Table 3.

**HRCT image features and pathogenic bacteria (Figures 1–4)**

**Effect of treatment**

Initial empirical beta-lactam antibiotic and/or quinolone antibiotics were used. When a patient’s condition did not get better or if it worsened, the sensitive antibiotics were adjusted
Figure 2. *Klebsiella pneumoniae*. A 71-year-old male patient had cough and hemoptysis for >20 days, with fever for 5 days. His body temperature was 38°C. A CT scan of the chest performed on June 20, 2014, showed that the left lung had lobar consolidation and fission-shaped leaves. Anti-infection treatment as an outpatient did not lead to improvement. To exclude the possibility of lung cancer, he was hospitalized. A CT scan performed on July 19, 2014 showed consolidation of the density shadow in the apical posterior segment of the left lung. The lesion density was not uniform, with a thick wall cavity and smooth wall, and adjacent pleural thickening. Results of the lung biopsy showed chronic inflammation. Results of the sputum culture showed *Klebsiella pneumoniae* ESBL (+), and then sensitive antibiotic was chosen and the patient was cured.

Figure 3. *Aspergillus*. A 56-year-old female patient experienced cough and asthma for >15 days, and was hospitalized. She had a medical history of diabetes, fasting blood glucose level of 11.9 mmol/L, and albumin level of 22.2 g/L. A CT scan showed changes in the centrilobular nodules, formation of cavity, localized bronchiectasis, bronchial wall thickening, and surrounding consolidation. Sputum culture detected *Aspergillus*, and the patient received oral voriconazole 200 mg twice a day, which was doubled on the first day. After 1 week of treatment, the CT scan showed significant absorbing.

Figure 4. *Mycobacterium tuberculosis*. A 45-year-old male patient experienced fever for >1 month, had slight fever, cough without expectoration, with night sweats. A local CT scan showed right lower lobe cavity, tree-in-bud, and a nodule around the cavity. He was diagnosed with tuberculosis using bronchoscopy and was referred to a tuberculosis hospital.
Table 4. Treatment outcome by the pathogenic bacteria or CT features (excluding noninfectious diseases).

| Guide to therapy        | Cured | Rate of cure   | Chi-square test |
|-------------------------|-------|----------------|-----------------|
| Pathogenic bacteria     | 38    | 74.5% (38/51)  | $\chi^2 = 0.209, P = 0.647$ |
| CT features             | 81    | 71.1% (81/114) |                 |

CT – computed tomography.

to the CT imaging features or the results of antibiotic susceptibility. After treatment, 119 patients showed improvement and 26 patients showed aggravated conditions.

Based on the results of drug sensitivity and the patients’ conditions, the sensitive antibiotics were adjusted; 38 patients were cured (74.51%, 38/51); condition of 8 patients deteriorated (15.69%, 8/51), and 5 patients did not show any response (9.80%, 5/51).

The results of bacterial culture were not obtained from 114 patients. When the treatment did not show improvement or even worsened after 72 hours, a CT examination of the chest was performed again immediately, and the CT features and the patient’s changing condition were evaluated, adjusting the use of antibiotics. Lobar consolidation only progressed in 29 patients, pleural effusion occurred in 13 patients, and the pathogenic bacteria identified were S. pneumoniae. Cavity lesions occurred in 7 patients and the possible pathogenic bacteria involved were K. pneumoniae or Staphylococcus aureus. Lobular consolidation progress fusion occurred in 27 patients, pleural effusion occurred in 17 patients. Possible pathogenic bacteria involved were Staph. aureus, pneumococcus, or K. pneumoniae; cavity lesions occurred in 10 patients and possible pathogenic bacteria involved were Staph. aureus and E. coli, which occurred with bronchial wall thickening and small cavities in 6 patients and prompt Aspergillus tree-in-bud appearance in 5 patients, suggesting TB or mycoplasma. Based on patients’ conditions and CT imaging features, high levels of antibiotics were used, even in the absence of bacterial drug resistance and deteriorating patient conditions. We used beta-lactam antibiotics with enzyme inhibitors or carbapenem antibiotics against gram-negative bacilli; vancomycin, teicoplanin, or linezolid against positive cocci; and voriconazole, itraconazole, or caspofungin against fungus. Eighty-one patients were cured (71.05%, 81/114) and in 18 patients the condition worsened (15.79%, 18/114), including 1 patient in whom the disease progressed to respiratory distress syndrome and who died due to multiple organ failure; and 15 patients did not respond to the treatment (13.16%, 15/114). In the pathogenic bacteria group, 51 patients showed a positive rate of 28.65% (51/178); in the no pathogenic bacteria group, 114 patients were identified. Therapeutic effect between the 2 groups did not show any significant statistical difference ($\chi^2 = 0.209, P = 0.647$). Using the CT features of treatment can provide the same effect as the pathogenic bacteria group. The results are presented in Table 4.

Discussion

The HRCT imaging features of pulmonary infections were closely related to the pathogenesis of pneumonia [6]. The histological characteristics of lobar pneumonia included edema, fluid exudation and filling of the alveoli with neutrophils. Pathogenic bacteria reached the alveoli and multiplied, leading to inflammatory exudate. The secretions of the bacteria were mainly along the small alveolar foramina and a small airway spread to the center, thus forming a subpleural nonsegmental lobar consolidation [7]. Pneumococcal pneumonia is a very common CAP, and pathology is a fibrinous inflammation. Results of the CT scan showed that lobar consolidation at first occurred in the visceral pleura of the lung periphery, usually close to the interlobular fissure, and gradually extended to the lung segment boundaries and spread to the center, eventually involving the entire lobe [7,8]. Bronchi were usually kept open, causing bronchogram in the consolidated area. Pleural effusion progressed, but it did not result in cavity formation. K. pneumoniae is a common CAP. Results of the CT scan showed lobar consolidation, large amounts of inflammatory exudate, lung parenchyma expansion changes, neighboring bulging fissures, and gradual formation of single or multiple small cavities in the consolidation area, and pleural effusion is common [9]. Bronchopneumonia is based on the histological features of bronchiolar inflammation, mainly involving the distal bronchioles, and local alveoli may be violated but do not produce a large amount of secretions and can be propagating along the alveolar foramina with a multifocal distribution. Infectious lesions are distributed along the segment and may spread across multiple pulmonary lobes. Staph. aureus bacterial infection is common after influenza. The HRCT showed airway involvement in the centrilobular nodules and branching line shadows, which could develop into patchy and gradual subsegmental and segmental lesions with blurred edges [10]. In about 40% of patients with bilateral lung, necrosis and liquefaction can occur. Further development can occur as change in a cavity, thickened walls, visible air fluid level, pleural effusion, or formation of empyema. H. influenzae pneumonia occurs mainly as bronchopneumonia. The HRCT scan showed the centrilobular nodules, occasional pleural effusion, rare empyema, and fewer cavities [11]. Aspergillus infection is common in immunocompromised patients, such as patients with diabetes and poor blood glycemic control, patients with chronic obstructive pulmonary disease, patients with tumor after...
repeated chemotherapy, or patients with long-term hypoalbuminemia. In this study, the incidence of *Aspergillus* infection merged with other clinical diseases was up to 91.7%; hypoalbuminemia was the relevant factor. At the beginning of the infection or disease progression, the common CT feature showed a specific multiple-nodular density shadow, gradually fused into patchy small cavities or local bronchiectasis, bronchial wall thickening; peribronchial infiltration consolidation; ground-glass opacity around the nodule; and rare pleural effusion [12]. *M. pneumoniae* mainly involves ciliated epithelial cells, causing bronchiolitis, with intraluminal exudate neutrophils. Fine bronchial wall inflammatory infiltrates are characteristic and can be extended to neighboring lung parenchyma, causing peribronchial inflammation, lobular and segmental consolidation, including bronchitis, bronchiolitis, peribronchial inflammation, and lung parenchymal inflammation [13]. Therefore, mycoplasma pneumonia on HRCT showed a variety of changes, such as ground-glass density shadow, grid, nodules, patchy consolidation shadows, segmental consolidation, little effusion, no obvious cavity necrosis, change in bronchovascular bundle thickening, bilateral involvement of the lower lobe, and peribronchovascular distribution [14,15].

The CT features can also be valuable references after the deterioration of the disease. The HRCT can obtain meaningful changes in 12 hours. After 72 hours of initial empirical therapy, no significant improvement or worsening of clinical symptoms was observed. The thin-slice CT scan again [16] evaluated the following lesion characteristics: lobar consolidation with effusion was increased, consolidation area was expanded, and no obvious cavitation was found, making possible presence of *S. pneumonia*. The clinical symptoms worsened, possibly due to drug-resistant bacterial infection or progression to severe pneumonia [17]. The antibiotics should be replaced with vancomycin or linezolid. Consolidation and seepage increased with cavitary liquefied necrotic common pathogenic bacteria such as *Staph. aureus, K. pneumonia*, and anaerobic bacteria [18]. The exacerbation of positive coccus pneumonia led to common large thick-walled cavities and visible liquid level. Vancomycin, teicoplanin, or linezolid should be used. Gram-negative bacteria leading to lobar consolidation were very common; with the progression of the disease, small cavities and pleural effusion occurred. After CT evaluation, considering the imaging characteristics of gram-negative bacteria, poor treatment, or disease progression, imipenem cilastatin sodium, meropenem, and other sensitive drugs were used. Bronchopneumonia can produce nodular changes or patchy fusion. New lesions with endobronchial spread sometimes showed tree-in-buds and single or multiple cavities, which suggested TB. Nodular consolidation with halo sign or limitations of peripheral bronchiectasis with bronchial wall thickening, peribronchial infiltration consolidation, and occurrence of small cavities, suggest that *Aspergillus* is a possible pathogenic bacteria [19,20]. In the absence of results of the culture, empirical antifungal therapy should be considered. Drugs used include voriconazole, itraconazole, and caspofungin [21,22]. Different pathogenic bacteria take different amounts of time to induce a cavity: a TB cavity appears after a long time (generally more than 4 weeks); a fungal cavity forms after about 3–7 days; and a bacterial cavity may be faster. However, *S. pneumoniae*, virus, or mycoplasma infection does not produce any cavity. Pleural effusion occurred in community infection bacteria such as *S. pneumoniae*, pulmonary TB, *K. pneumoniae*, *Legionella*, and *E. coli* [23–25].

In this study, only 51 patients showed positive significant pathogenic bacteria, and 38 patients were cured, with a cure rate of 74.5%. There were 114 patients who could not be microbiologically proven and 81 patients were cured, with a cure rate of 71.05% (χ²=0.209, P=0.647). The difference in cure rates between patients with detection of pathogenetic bacteria and patients without a pathogenetic bacteria detected (and therefore treatment guidance with HRCT solely) did not differ significantly. HRCT imaging features can be a differential diagnosis or exclude the diagnosis of possible noninfectious diseases, especially for patients with impaired immune function [26], possible progression to respiratory failure, or multiple organ failure. An earlier adjustment of high levels of antibiotics to targeted anti-infection can guide the clinical treatment [27].

Limitations of this study are as follows: inclusion of patients with CAP only, and failure to include patients with hospital-acquired infection and mixed infection. With distribution of pathogens of mixed pulmonary infections, including fungal, bacteria, atypical pathogens, virus, TB, and other common pathogens, the HRCT image features appear different because of different pathological processes. Whether polymicrobial infection can be clearly identified in imaging needs further study.

**Conclusions**

In this study, patients with CAP in the hospital were the research subjects. Based on the empirical therapy combined with etiological examination and HRCT of the chest, the treatment was adjusted according to the changing conditions with time. In patients with no pathogens, medication was adjusted based on the CT imaging features. To avoid delay, antibiotics were adjusted quickly and effectively. Lung CT image characteristics can effectively guide clinical empirical anti-infection treatment.

**Conflicts of interest and source of funding.**

None.
References:

1. File TM: Community-acquired pneumonia. Lancet, 2003; 362: 1991–2001
2. Sousa D, Justo I, Dominguez A et al: Community-acquired pneumonia in immunocompromised older patients: incidence, causative organisms and outcome. Clin Microbiol Infect, 2013; 19: 187–92
3. Yahav D, Leibovici L, Goldberg E et al: Time to first antibiotic dose for patients hospitalised with community-acquired pneumonia. Int J Antimicrob Agents, 2013; 41: 410–13
4. Huijskens EG, Rossen JW, Kluytmans JA et al: Evaluation of yield of current available diagnostics by sample type to optimize detection of respiratory pathogens in patients with a community-acquired pneumonia. Influenza Other Respir Viruses, 2014; 8: 243–49
5. Chalmers JD, Singanayagam A, Akram AR et al: Safety and efficacy of CURB65-guided antibiotic therapy in community-acquired pneumonia. J Antimicrob Chemother, 2011; 66: 416–23
6. Beigelman-Aubry C, Godet C, Caumes E: Lung infections: the radiologist’s perspective. Diagn Interv Imaging, 2012; 93: 431–40
7. Haroon A, Higa F, Fujita J et al: Pulmonary computed tomography findings in 39 cases of Streptococcus pneumoniae pneumonia. Intern Med, 2012; 51: 3343–49
8. Yagihashi K, Kurihara Y, Fujikawa A et al: Correlations between computed tomography findings and clinical manifestations of Streptococcus pneumoniae pneumonia. Jpn J Radiol, 2011; 29: 423–28
9. Okada F, Ando Y, Honda K et al: Clinical and pulmonary thin-section CT findings in acute Klebsiella pneumoniae pneumonia. Eur Radiol, 2009; 19: 809–15
10. Nguyen ET, Kanne JP, Hoang LM et al: Community-acquired methicillin-resistant Staphylococcus aureus pneumonia: radiographic and computed tomography findings. J Thorac Imaging, 2008; 23: 13–19
11. Okada F, Ando Y, Tanoue S et al: Radiological findings in acute Haemophilus influenzae pulmonary infection. Br J Radiol, 2012; 85: 121–26
12. Horger M, Hebart H, Eisele H et al: Initial CT manifestations of invasive pulmonary aspergillosis in 45 non-HIV immunocompromised patients: association with patient outcome? Eur J Radiol, 2005; 55: 437–44
13. Miyashita N, Kawai Y, Yamaguchi T et al: Clinical potential of diagnostic methods for the rapid diagnosis of Mycoplasma pneumoniae pneumonia in adults. Eur J Clin Microbiol Infect Dis, 2011; 30: 439–46
14. Miyashita N, Sugiu T, Kawai Y et al: Radiographic features of Mycoplasma pneumoniae pneumonia: differential diagnosis and performance timing. BMC Med Imaging, 2009; 9: 7
15. Manns TK, Wagnewitz U, Jamieson FB, Patsios DA: Chest computed tomography predicts microbiological burden and symptoms in pulmonary Mycobacterium xenopi. Respirology, 2013; 18: 92–101
16. Kang M, Deoghuria D, Varma S et al: Role of HRCT in detection and characterization of pulmonary abnormalities in patients with febrile neutropenia. Lung India, 2013; 30: 124–30
17. Garcia-Vidal C, Carratala J: Early and late treatment failure in community-acquired pneumonia. Semin Respir Crit Care Med, 2009; 30: 154–60
18. Seo H, Cha SI, Shin KM et al: Focal necrotizing pneumonia is a distinct entity from lung abscess. Respirology, 2013; 18: 1095–100
19. Guimarães MD, Marchiori E, De Souza Portes Meirelles G et al: Fungal infection mimicking pulmonary malignancy: clinical and radiological characteristics. Lung, 2013; 191: 655–62
20. Franquet T, Serrano F, Gimenez A et al: Necrotizing Aspergillosis of large airways: CT findings in eight patients. J Comput Assist Tomogr, 2002; 26: 342–45
21. Ader F, Bienvenu AL, Rammaert B, Nseir S: Management of invasive aspergillosis in patients with COPD: rational use of voriconazole. Int J Chron Obstruct Pulmon Dis, 2009; 4: 279–87
22. Kontoyiannis DP: Invasive mycoses: strategies for effective management. Am J Med, 2012; 125: 525–38
23. Karadell I, Koc Z, Uluan S et al: Chest radiography and CT findings in patients with the 2009 pandemic (H1N1) influenza. Diagn Interv Radiol, 2011; 17: 216–22
24. Sakai F, Tokuda H, Goto H et al: Computed tomographic features of Legionella pneumophila pneumonia in 38 cases. J Comput Assist Tomogr, 2007; 31: 325–31
25. Okada F, Ando Y, Matsuhashi S et al: Thin-section CT findings of patients with acute Streptococcus pneumoniae pneumonia with and without concurrent infection. Br J Radiol, 2012; 85: e357–64
26. Reynolds JH, Banerjee AK: Imaging pneumonia in immunocompetent and immunocompromised individuals. Curr Opin Pulm Med, 2012; 18: 194–201
27. Thiem U, Heppner HJ, Pientka L: Elderly patients with community-acquired pneumonia: optimal treatment strategies. Drugs Aging, 2011; 28: 519–37