Diagnostic value of serum Aspergillus IgG antibody for pulmonary aspergillosis in non-agranulocytic patients

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Abstract

Background: At present, serum Aspergillus IgG and IgM antibodies are mainly used in the diagnosis of chronic pulmonary aspergillosis (CPA), but the diagnostic value in invasive pulmonary aspergillosis (IPA) in non-agranulocytic patients is still unclear. The aim of this study was to investigate the diagnostic value of serum Aspergillus IgG and IgM antibody detection for pulmonary aspergillosis in non-agranulocytic patients.

Methods: 58 cases of pulmonary aspergillosis (37 IPA and 21 CPA cases), 15 cases of bacterial pneumonia and 50 cases of healthy control group were collected. Serum G test and GM test were performed, and Aspergillus IgG and IgM antibody were detected in all patients. The sensitivity and specificity, cut off value and AUC of Aspergillus IgG and IgM antibodies were further obtained by the ROC curves.

Results: The positive rates of G test and Aspergillus IgG antibody in pulmonary aspergillosis group were significantly higher compared with bacterial pneumonia group or healthy group ($P = 0.015$ and $P < 0.0001$, respectively). Aspergillus IgG antibody can preferably distinguish CPA from bacterial pneumonia and healthy controls (sensitivity = 0.952, specificity = 0.692, cut off value = 75.46, AUC = 0.873). However, the performance of it was poor in distinguishing IPA from CPA, healthy group and pneumonia group (AUC < 0.7).

Conclusions: Serum Aspergillus IgG has certain clinical value in the diagnosis of pulmonary aspergillosis in non-agranulocytic patients.

Background

Pulmonary aspergillosis is a group of lung diseases caused by *Aspergillus* infection or inhalation of Aspergillus antigen. Pulmonary aspergillosis is uncommon in non-agranulocytic patients, and only a small of data are available. Nevertheless, in recent years, the incidence of pulmonary aspergillosis in non-granulocytic patients has increased with the aging, the increase of chronic diseases, the use of broad-spectrum antibiotics, hormones, immunosuppressive drugs and invasive operations [1, 2]. Moreover, the clinical manifestations of these patients lack specificity, often the diagnosis is difficult. As a result, the patient’s treatment is delayed, which affects the prognosis of patients. Pulmonary
Aspergillosis can be divided into different types in the light of different classification criteria. According to the onset time, course and symptoms, it can be divided into acute pulmonary aspergillosis (APA), subacute pulmonary aspergillosis and chronic pulmonary aspergillosis (CPA). Furthermore, CPA is usually seen in immunocompetent individuals with underlying respiratory disorders, and the prevalence of CPA worldwide is about 3 million [3]. Unfortunately, respiratory physicians may not detect CPA until the disease progresses to an advanced stage, owing to the lack of specific clinical manifestations. More seriously, without timely diagnosis and long-term antifungal treatment, the 5-year mortality rate is nearly 80% [4]. Based on the forms and causes of aspergillosis, pulmonary aspergillosis can be divided into three types: invasive, allergic and parasitic, which reflect different immune statuses of the host. Among them, invasive pulmonary aspergillosis (IPA) has become a common type of severe pneumonia with the highest mortality, and one of the important reasons is that it is difficult to be diagnosed [5]. In addition, patients with agranulocytosis are considered to be the predominant group of IPA, and relevant international guidelines for diagnosis and treatment also focus on this group [6]. It is well known that serum Aspergillus IgG and IgM antibodies are mainly used in the clinical diagnosis of CPA [7]. Synchronously, serum G test and GM test, as non-invasive diagnostic methods, are mainly used for the diagnosis of IPA in agranulocytic patients, but they are not sensitive to non-agranulocytic patients. In this study, we explored the diagnostic value of G test, GM test, serum Aspergillus IgG and IgM antibodies detection for IPA and CPA in non-agranulocytic patients.

Methods

Patients and Data Collection

58 pulmonary aspergillosis cases in non-agranulocytic patients were enrolled which were admitted to Tianjin Chest Hospital from July 2017 to July 2018, and possible IPA cases and cases of allergic bronchopulmonary aspergillosis were excluded. The diagnostic criteria referred to the consensus of experts in diagnosis and treatment of pulmonary mycosis and the criteria of the European Organization for Research and Treatment of Cancer (EORTC) [8, 9]. During the same period, 15 cases of bacterial pneumonia and 50 cases of health examination were treated as control groups. The sex
and age of control groups had no significant difference compared with pulmonary aspergillosis group. Moreover, the following data were collected: demographic data (age, gender, weight), serum indexes, imaging features, biochemical indicators, bacterial and fungal culture results, bronchoscopic findings, and the treatment outcomes. In addition, all participants has signed the informed consent voluntarily, and the study has been approved by the ethics committee of Tianjin Chest Hospital (2018KY-009-01).

**Serological Testing**

5 ml venous blood were drawn before using any antibiotics. Serum was separated from the blood for tests directly or stored frozen at −80°C. The serum (1,3)-β-D-glucan test (G test) was conducted with a chromogenic method [10]. In brief, 5 μl serum samples were firstly pretreated for 10 min at 37°C with 20 μl of a solution containing 0.6 M KCl and 0.125 M KOH, and assayed with the Glucatell reagent in a kinetic, chromogenic format for 30 min at 37°C. Subsequently, the optical densities at 405 nm (OD405) were read. Finally, the concentration of G in each sample was calculated by using a calibration curve with standard solutions of 6.25 to 100 pg/ml. Cases were judged positive if the level of G was ≥120 pg/ml in at least one serum sample. The serum galactomannan test (GM test), serum Aspergillus IgG and IgM antibodies were carried out with commercial enzyme-linked immunosorbent assay (ELISA) kits (Dyna Biotechnology Co., Ltd. Tianjin, China) according to the manufacturer’s instructions. Results were expressed as the ratio of the OD obtained from the case serum sample and the control. It was considered a true positive when 2 consecutive samples for an case tested positive (undetermined: 1.0 ~ 1.5, negative: < 1.0).

**Statistical Analysis**

SPSS 21.0 software was used for statistical analysis. Comparison between two groups were performed by chi-square test. Fisher’s test results were used when the sample size was small and the theoretical number was small. Mann-Whitney U test was used in the course of disease, age and serum indicators except lymphocyte count indicators. Independent sample t test was used for lymphocyte count indicators. The sensitivity, specificity and optimal threshold were determined by using the receiver operating characteristic curve (ROC curve). The standard of this study was $P < 0.05$ with statistical
Results

Patient Characteristics

Characteristics of the 58 pulmonary aspergillosis patients were shown in Table 1. There were 36 males and 22 females, aged from 46 to 75 (60.7 ± 14.6), and 37 IPA (63.8%) and 21 CPA cases (36.2%) were included. Among them, 7 cases (12.1%) were no underlying other diseases, 26 cases (44.8%) combined with chronic respiratory disease, and 15 cases (25.9%) with diabetes (Table 1).

Characteristics Comparison between IPA and CPA Cases

Clinical features between IPA and CPA cases were compared and exhibited in Table 2, including microbiological findings, clinical symptoms, thoracic CT signs, involving lobes of lung, and serum indexes. It was obvious from Table 2 that the course of disease was longer in CPA cases than IPA cases. Some clinical symptoms, such as fever, dyspnoea and haemoptysis, were very different between IPA and CPA cases ($P < 0.05$). Ulteriorly, there were observable differences between above two groups in thoracic CT signs of infiltrates, air crescent sign and ground-glass attenuation, lung lobes of right middle, right lower and left upper, serum indexes of LDH, albumin, PCT levels and lymphocyte count ($P < 0.05$, Table 2).

Results of Serum G test, GM test, Aspergillus IgG and IgM Antibodies in Each Group

Results of serum G test, Aspergillus IgG antibody, Aspergillus IgM antibody and GM test were listed in Table 3A-C among different groups. Primitively, positive rates of above serum indexes were counted among pulmonary aspergillosis, bacterial pneumonia and healthy groups, and Table 3A was the statistical result. It was illustrated that the positive rates of serum G test and Aspergillus IgG antibody were notable higher in pulmonary aspergillosis group than bacterial pneumonia and healthy groups ($P = 0.015$ and $< 0.0001$, respectively). Afterwards, in order to study whether different types of
pulmonary aspergillosis could be distinguished, the pulmonary aspergillosis group was divided into IPA and CPA groups according to the disease type. Table 3B was the comparison result among IPA, CPA, bacterial pneumonia and healthy groups, and Table 3C shown comparison between IPA and CPA groups. Besides G test and Aspergillus IgG antibody, the positive rate of GM test also showed notable differences among IPA, CPA, bacterial pneumonia and healthy groups (P = 0.022) (Table 3B). Nevertheless, G test and Aspergillus IgG antibody were no markedly statistical difference between IPA and CPA groups (P ≥ 0.5), and the positive rate of GM test statistically evident difference (P = 0.04) (Table 3C).

ROC Curves of Serum Aspergillus IgG Antibody in Different Groups

The ROC curves of Aspergillus IgG antibody in different groups were drawn. Fig. 1 A-F displayed ROC curves of Aspergillus IgG antibody with remarkable significance (P < 0.05), and the cutoff value (sensitivity, specificity) and the area under curve (AUC) were also shown. It was revealed that Aspergillus IgG antibody with the highest specificity (0.952) when IPA group compared with CPA groups (Fig. 1B), with the highest sensitivity (0.952) when CPA group compared with IPA, bacterial pneumonia and healthy groups (Fig. 1F), and with both the highest AUC (0.873) and the highest sensitivity (0.952) when CPA group compared with bacterial pneumonia and healthy groups (Fig. 1D). Furthermore, the AUC value was bigger in Fig. 1D than that of Fig. 1C, so as to in Fig. 1F than that of Fig. 1E. That was, serum Aspergillus IgG antibody had a better performance for distinguish CPA than IPA.

Discussion

Although pulmonary aspergillosis in non-agranulocytic patients has increased with the development of society, the frequency remains low relative to that in agranulocytic patients. So far, few data are available in non-agranulocytic cases, and most of them are case reports [11–14]. Consequently, more cases and more studies are urgently needed to understand non-agranulocytic pulmonary aspergillosis, so as to provide more references or clues for the diagnosis and treatment of the disease. In this article, 58 cases were reported, and the sample size was rare and higher. IPA is a life-
threatening infection in patients mainly with prolonged neutropenia. One clinical challenge of non-agranulocytic IPA cases is the frequently lack of specific clinical features, especially in those without underlying disease [15]. In our study, we roundly compared clinical features between IPA and CPA cases with relevant diagnostic methods commonly used in clinic (Table 2), including microbial cultivation, thoracic CT and serum detection. Some special characteristics for IPA were spotted, such as shorter disease course, frequent infiltrates, special lobe of lung, lower serum albumin level, which might be used for differential diagnosis or auxiliary diagnosis.

The diagnosis gold standard of pulmonary aspergillosis mainly relies on chest imaging, microbial culture and histopathological examination. However, the imaging manifestations are poor in specificity for non-agranulocytic patients, and the phenomena of “the same disease with different image, and the different disease with same image” exist [16, 17]. As for microbiological and histopathological examination, it is difficult to obtain pathological specimens, positive rate of culture is low, and possibly contaminated and colonized. Therefore, the clinical diagnosis of non-agranulocytic pulmonary aspergillosis is difficult, and it is not always feasible to obtain histo- or cytopathological demonstration of the fungus in order to meet the gold standard [18]. As a non-invasive diagnostic method of pulmonary mycosis, the detection of serum antigens and antibodies has attracted more and more attention. G test and GM test are mainly used for the clinical diagnosis of IPA in agranulocytic patients, but the positive rate of IPA in non-agranulocytic is too low to meet clinical needs [19, 20]. For patients with agranulocytosis or severe immunosuppression, it is difficult for the body to produce an immune response. Accordingly, the detection of specific antibodies against Aspergillus is of little significance. With the increase of non-agranulocytosis and non-immunocompromised host, the diagnostic significance of antibody detection for pulmonary aspergillosis needs to be reevaluated. Serum Aspergillus antibody detection is mainly used in the diagnosis of CPA [21, 22]. Meanwhile, the diagnostic value of Aspergillus antibody is not clear for IPA in non-agranulocytic patients because of varying results [18]. Additionally, diagnosing chronic pulmonary aspergillosis (CPA) is complicated, and there are limited data available [23]. Here, we compared the performances of G test, GM test, Aspergillus IgG antibody by using serum samples from
non-agranulocytic patients with underlying pulmonary aspergillosis diseases, and further subdivided IPA and CPA (Table 3A-C). There are few studies on serum Aspergillus IgM antibody, and its significance in the diagnosis of pulmonary aspergillosis is not clear. This study showed that there was no significant difference in serum Aspergillus IgM antibodies between pulmonary aspergillosis, bacterial pneumonia and healthy people. The reasons may include: 1. IgM is the earliest immunoglobulin produced after infection or immunization. It has strong bactericidal and regulatory effects, but its content in blood is low, half-life is short, and it is susceptible to interference factors. 2. Non-granulocyte-deficient hosts may undergo a period of Aspergillus colonization and slow invasion before infection due to their relatively sound immune function. IgM often occurs in the early stage of infection. Therefore, Aspergillus IgG antibody detection is more significant than Aspergillus IgM antibody detection. Our results revealed that Aspergillus IgG antibody reflected the greatest differences among pulmonary aspergillosis (even IPA and CPA subdivision), bacterial pneumonia and healthy group ($P < 0.0001$) (Table 3 A, B). It was indicated that Aspergillus IgG antibody might a potential diagnostic index for pulmonary aspergillosis in non-agranulocytic patients, and it was further evaluated the performance through ROC curves.

As exhibited in Fig. 1, Aspergillus IgG had notable different in pulmonary aspergillosis (even IPA and CPA subdivision), bacterial pneumonia and healthy group ($P < 0.05$), and both the specificity and sensitivity were 40.5–95.2% and 58.8–95.2%, and the highest AUC 0.873. Previous studies have shown that the sensitivity and specificity of Aspergillus IgG antibody detection for CPA diagnosis are 75–96% and 97–99% [24]. The specificity and sensitivity were lower than the previous report, it might because that the underlying condition of the research population and the experimental methods are different. Our study further certified that serum Aspergillus IgG antibody had a better performance for distinguish CPA than IPA. From acute invasive infection to chronic consumptive diseases, different types of pulmonary aspergillosis can overlap with each other. Generally, IPA occurs in patients with impaired immune function in varying degrees, while CPA occurs in patients without or with impaired immune function in a lower degree. Therefore, serum Aspergillus antibody levels differ in different types of pulmonary aspergillosis, which is of greater significance to patients with CPA. Above all, we
suspected that serum Aspergillus IgG has certain clinical value in the diagnosis of pulmonary aspergillosis in non-agranulocytic patients, especially for non-agranulocytic CPA. Howbeit, it was believed that serum Aspergillus IgG could not replace the traditional isolation and culture of fungi, and should be combined with other diagnostic methods and clinical practice. In addition, further studies were needed to determine the role of Aspergillus specific antibodies in the pathogenesis, diagnosis and treatment of aspergillosis.

Conclusions
In conclusion, serum Aspergillus IgG has certain clinical value in the diagnosis of pulmonary aspergillosis in non-agranulocytic patients.

Abbreviations
CPA: chronic pulmonary aspergillosis; IPA: invasive pulmonary aspergillosis; AUC: area under curve; APA: acute pulmonary aspergillosis; GM test: galactomannan test; G test: (1,3)-β-D-glucan test; ELISA: enzyme-linked immunosorbent assay; ROC: receiver operating characteristic curve.

Declarations
Ethics approval and consent to participate
All participants has signed the informed consent voluntarily, and the study has been approved by the ethics committee of Tianjin Chest Hospital (2018KY–009–01).

Consent for publication
Not applicable.

Availability of data and material
All data used during the study are available on reasonable request.

Competing interests
The authors declare that they have no competing interests.

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Authors’ contributions
HP participated in the conceived of the study, design of the study and modify of manuscript; QY participated in the conceived of the study, carried out the studies, performed the statistical analysis, and draft the manuscript, JH, BX, XL, HQ, HZ and MZ participated in its design, coordination, perform the statistical analysis and modified the manuscript. All authors read and approved the final manuscript.

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Table 1 Characteristics of 58 pulmonary aspergillosis cases in non-agranulocytic patients

| Characteristics          | No. of patients (n=58) | %    |
|--------------------------|------------------------|------|
| Gender                   |                        |      |
| Male                     | 36                     | 62.07|
| Female                   | 22                     | 37.93|
| Age                      |                        |      |
| ≥60                      | 32                     | 55.17|
| <60                      | 26                     | 44.83|
| Case classification      |                        |      |
| IPA                      | 37                     | 63.79|
| CPA                      | 21                     | 36.21|
| Underlying disease       |                        |      |
| chronic respiratory disease | 26                 | 44.83|
| Organ failure            | 1                      | 1.72 |
| Chronic cardiovascular disease | 5               | 8.62 |
| pulmonary tuberculosis   | 4                      | 6.90 |
| chronic liver disease    | 1                      | 1.72 |
| Diabetes                 | 15                     | 25.86|
| Autoimmune disease       | 1                      | 1.72 |
| Others                   | 8                      | 13.79|
| No underlying disease    | 7                      | 12.07|

IPA, invasive pulmonary aspergillosis; CPA, chronic pulmonary aspergillosis.

Table 2 Comparison of clinical features between IPA and CPA cases

| Characteristics               | IPA (n=37) | CPA (n=21) | Overall (n=58) |
|-------------------------------|------------|------------|----------------|
| Ratio of male to female patients | 23:14      | 13:8       | 36:22          |
| Age, median years             | 64 (54.513, 72) | 60 (46.524, 66) | 64 (53.753, 70.248) |
| Course of disease (median, day) | 17 (10, 30) | 75 (23.251, 187.532) | 20 (13, 60) |

Microbiological findings

|                      | IPA (n=37) | CPA (n=21) | Overall (n=58) |
|----------------------|------------|------------|----------------|
| Staphylococcus aureus | 1 (2.703)  | 0          | 1 (1.724)      |
| Pseudomonas aeruginosa| 1 (2.703)  | 1 (4.762)  | 2 (3.448)      |
| Candida albicans     | 1 (2.703)  | 1 (4.762)  | 2 (3.448)      |
| Pathogen                        | Count (Percentage) |
|--------------------------------|--------------------|
| Acinetobacter baumannii        | 1 (2.703)          |
| Klebsiella pneumoniae          | 2 (5.405)          |
| Aspergillus                    | 8 (21.622)         |
| Others                         | 2 (5.405)          |
| **Klebsiella pneumoniae**      |                    |
|                                | 2                  |
|                                | 0                  |
|                                | 11 (18.966)        |
|                                |                    |
| **Aspergillus**                |                    |
|                                | 3 (14.286)         |
|                                |                    |
| **Others**                     |                    |
|                                | 3 (5.172)          |

**Clinical symptoms, n(%)**

| Symptom          | Count (Percentage) |
|------------------|--------------------|
| Cough            | 34 (91.892)        |
| Fever (>38°C)    | 22 (59.459)        |
| Dyspnoea         | 25 (67.568)        |
| Haemoptysis      | 13 (35.135)        |
| Chest Pain       | 8 (21.622)         |
| Expectoration    | 33 (89.189)        |
| **Thoracic CT signs, n(%)** |                |
| Infiltrates      | 36 (97.297)        |
| Nodules          | 19 (51.351)        |
| segmental areas of consolidation | 4 (10.811) |
| Cavity           | 17 (45.946)        |
| Pleural effusion | 11 (29.730)        |
| Air crescent sign| 2 (5.405)          |
| ground-glass attenuation | 22 (59.459) |
| other            | 1 (2.703)          |
| **Involving lobes of lung, n(%)** |          |
| Right upper lobe | 28 (75.676)        |
| Right middle lobe| 24 (64.865)        |
| Right lower lobe | 30 (81.081)        |
| Left upper lobe  | 28 (75.676)        |
| Left lower lobe  | 27 (72.973)        |

**Serum indexes**

| Index                        | Low (95%CI) | High (95%CI) | Mean (95%CI) |
|------------------------------|-------------|--------------|--------------|
| Erythrocyte sedimentation rate (mm/h) | 46 (26.515, 62) | 30 (19.525, 55) | 42 (25.514, 60.3) |
| White blood cell count (109cells/L) | 8.77 (5.534, 13.495) | 6.97 (5.862, 8.471) | 7.96 (5.788, 12.0) |
| Neutrophil count (109cells/L) | 7.3 (3.555, 11.205) | 4.31 (3.475, 6.685) | 5.31 (3.495, 9.4) |
| Eosinophilia count (109cells/L) | 0.07 (0.005,0.135) | 0.1 (0.035,0.223) | 0.085 (0.012, 0.0) |
| Monocyte count (109cells/L) | 0.45 (0.325,0.57) | 0.57 (0.385,0.695) | 0.49 (0.3475,0.6) |
| Platelet ((109cells/L)) | 273 (199.5,385.5) | 257 (209,294.5) | 263.5 (208.75,33) |
| Serum ALT level (U/L) | 21.8 (15.65,41.55) | 20.2 (13.9,30.4) | 21.65 (15.425,34) |
| Serum creatinine level (μmol/L) | 67 (53.75,78.75) | 67 (60,77.5) | 67 (59,78) |
| Serum albumin level (g/L) | 36.2 (28.725,41) | 42.8 (39.5,44.95) | 40.4 (35.6,43) |
| Serum LDH level (U/L) | 262 (203,373.5) | 225 (187.5,236.5) | 233.5 (198,326) |
Serum CRP level (mg/L) | 4.09 (1.135,11) | 1.7 (0.485,7.335) | 2.85 (0.5375,8.1)  
PCT (ng/ml) | 0.09 (0.05,0.215) | 0.05 (0.05,0.0625) | 0.05 (0.05,0.1)  
Lymphocyte count (10^9 cells/L) | 1.4545±0.63095 | 1.8898±0.57672 | 1.6846±0.638

IPA, invasive pulmonary aspergillosis; CPA, chronic pulmonary aspergillosis; LDH, lactate dehydrogenase; CRP, C-reactive protein; PCT, procalcitonin.

### Table 3A Comparisons of serum G test, Aspergillus IgG antibody, Aspergillus IgM antibody and GM test among pulmonary aspergillosis, bacterial pneumonia and healthy groups

| Group                  | G test positive n (%) | Aspergillus IgG antibody positive n (%) | Aspergillus IgM antibody positive n (%) |  
|------------------------|-----------------------|----------------------------------------|----------------------------------------|  
| pulmonary aspergillosis group | 14 (24.138) | 33 (56.897) | 14 (24.138) |  
| bacterial pneumonia group | 1 (6.667) | 3 (20) | 4 (26.667) |  
| healthy group | 3 (6) | 8 (16) | 9 (18) |  
| P | 0.015 | < 0.001 | 0.649 |

G test, (1,3) beta glucan-D test; GM test, galactomaunan test.

### Table 3B Comparisons of serum G test, Aspergillus IgG antibody, Aspergillus IgM antibody and GM test among IPA, CPA, bacterial pneumonia and healthy groups

| Group                  | G test positive n (%) | Aspergillus IgG antibody positive n (%) | Aspergillus IgM antibody positive n (%) | GM I  
|------------------------|-----------------------|----------------------------------------|----------------------------------------|------  
| IPA group | 12 (32.43) | 18 (48.65) | 6 (16.22) | 1 |  
| CPA group | 2 (9.523) | 15 (71.429) | 8 (38.095) | 1 |  
| bacterial pneumonia group | 1 (6.667) | 3 (20.000) | 4 (26.667) | 2 |  
| healthy group | 3 (6.000) | 8 (16.000) | 9 (18.000) | 4 |  
| P | 0.006 | < 0.001 | 0.217 |

G test, (1,3) beta glucan-D test; GM test, galactomaunan test; IPA, invasive pulmonary aspergillosis; 
CPA, chronic pulmonary aspergillosis.

### Table 3C Comparisons of serum G test, Aspergillus IgG antibody, Aspergillus IgM antibody and GM test
between IPA and CPA groups

| Group   | G test positive n (%) | Aspergillus IgG antibody positive n (%) | Aspergillus IgM antibody positive n (%) | GM test positive n (%) |
|---------|-----------------------|----------------------------------------|----------------------------------------|------------------------|
| IPA group | 12 (32.432)           | 18 (48.649)                            | 6 (16.216)                             | 11 (48.649)            |
| CPA group | 2 (9.523)             | 15 (71.429)                            | 8 (38.095)                             | 1 (4.762)              |
| P       | 0.050                 | 0.092                                  | 0.061                                  | 0                      |

G test, (1,3) beta glucan-D test; GM test, galactomaunan test; IPA, invasive pulmonary aspergillosis; CPA, chronic pulmonary aspergillosis.

Figures
Figure 1
ROC curves of Aspergillus IgG antibody in different groups; A: the ROC curve of Aspergillus IgG antibody in pulmonary aspergillosis group compared with healthy group, the cutoff value (sensitivity, specificity) = 71.555 (0.793, 0.677), AUC = 0.780 P < 0.001; B: the ROC curve of Aspergillus IgG antibody in IPA group compared with CPA group, the cutoff value (sensitivity, specificity) = 77.310 (0.405, 0.952), AUC = 0.624 P < 0.001; C: the ROC curve of Aspergillus IgG antibody in IPA group compared with bacterial pneumonia and healthy groups, the cutoff value (sensitivity, specificity) = 134.460 (0.459, 0.923), AUC = 0.727 P < 0.001; D: the ROC curve of Aspergillus IgG antibody in CPA group compared with bacterial pneumonia and healthy groups, the cutoff value (sensitivity, specificity) = 75.460 (0.952, 0.692), AUC = 0.873 P < 0.001; E: the ROC curve of Aspergillus IgG antibody in IPA group compared with CPA, bacterial pneumonia and healthy groups, the cutoff value (sensitivity, specificity) = 71.555 (0.703, 0.588), AUC = 0.641 P = 0.013; F: the ROC curve of Aspergillus IgG antibody in CPA group compared with IPA, bacterial pneumonia and healthy groups, the cutoff value (sensitivity, specificity) = 77.310 (0.952, 0.588), AUC = 0.782 P < 0.001.