

## CASE REPORT

# A Case of IgA Nephropathy Complicated with Pulmonary Infection by Mycobacterium Abscess

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### SUMMARY

**Background:** In August 2023, our hospital confirmed a case of IgA nephropathy complicated with pulmonary infection by Mycobacterium abscess. The patient sought medical attention at our hospital due to "gross hematuria for 10 years, recurrence for 10 days, coughing and sputum production". The patient had pulmonary tuberculosis 15 years ago and had been cured. He had bronchiectasis for 10 years.

**Methods:** Chest CT, fiberoptic bronchoscopy examination, urine routine (urine analysis + sediment quantification), urine trace protein measurement//urine creatinine (random urine), urine protein quantification (24-hour urine), antinuclear antibody measurement (ANA), sputum culture, alveolar lavage fluid bacterial culture, alveolar lavage fluid acid fast staining, and alveolar lavage fluid mNGS.

**Results:** Chest CT: Cystic dilation of bronchi in both lungs, mainly in the lower lungs, with visible phlegm clots inside. Fibrobronchoscopy: A large amount of white foam like secretions can be seen in the lumens of the middle lobe of the right lung and the lower lobes of both lungs. Urinary routine (urine analysis + sediment quantification): protein+↑, occult blood+++ . Urine Microprotein Determination//Urine Creatinine (Random Urine): Microalbumin 156.00 mg/L, Urine mALB/Urine Creatinine 132.73 mg/g; Quantitative determination of urine protein (24-hour urine): total protein 0.93 g/24-hour urine; Antinuclear antibody assay (ANA): weakly positive; Sputum bacterial culture: negative; Bacterial culture of bronchoalveolar lavage fluid: Mycobacterium abscess++, NGS in bronchoalveolar lavage fluid: Mycobacterium abscess. Clinical treatment plan: 0.25 g of azithromycin qd po+ 0.4 g of amikacin sulfate qd ivgtt+ 1 g cefmetazole sodium q12hours ivgtt. After 10 days of treatment, the patient improved and was discharged.

**Conclusions:** This article reports a case of IgA nephropathy complicated with pulmonary abscess mycobacterial infection. Mycobacterium abscess was quickly and accurately identified by mNGS. Reasonable treatment measures were adopted clinically. The patient improved and was discharged. This study has important reference significance for the clinical diagnosis and treatment of Mycobacterium abscess infection. In addition, mNGS, as a novel detection method, has considerable prospects for rapid diagnosis of pathogens.

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#### KEYWORDS

Mycobacterium abscess, mNGS of alveolar lavage fluid, IgA nephropathy

## CASE PRESENTATION

### Case

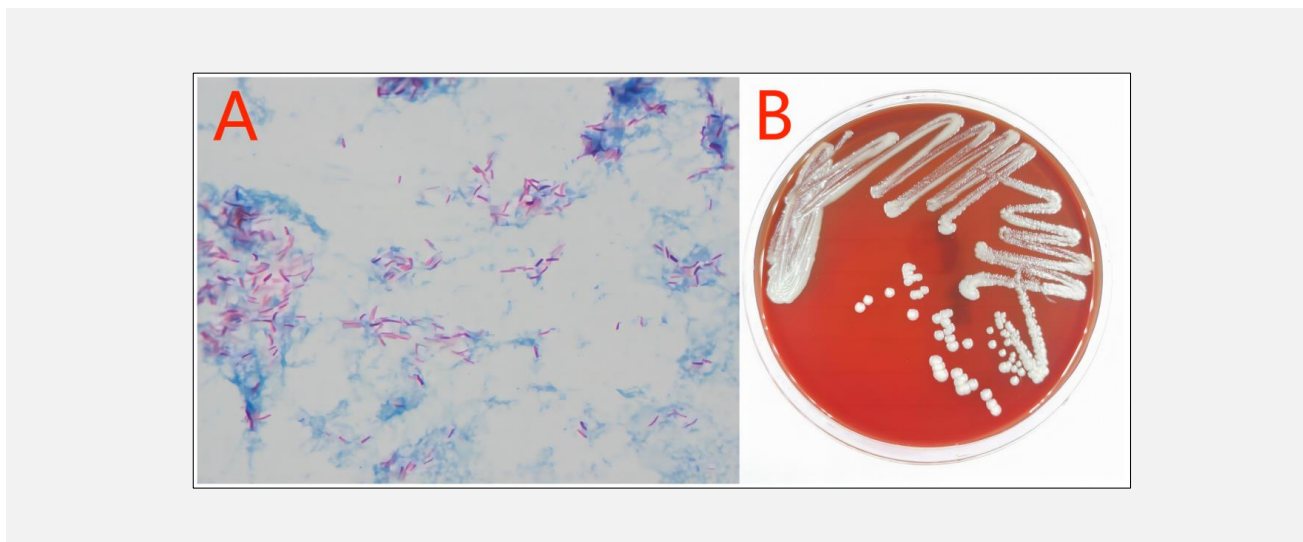
The patient, a 24-year-old female, visited our hospital in August 2023 due to "gross hematuria for 10 years, recurrence for 10 days, coughing and sputum production.". Case characteristics: The patient was a young female with a long course of disease. Ten years ago, the patient had no obvious cause of gross hematuria, which was a complete hematuria. The patient sought medical attention at another hospital. The 24-hour urine protein quantification was 0.1 g/24-hour urine, and a renal biopsy showed IgA nephropathy. After oral administration of nephritis rehabilitation tablets, Huangkui capsules, and Zhengqing Fengtongning sustained-release tablets, the gross hematuria disappeared, and no follow-up was conducted for 10 years. Ten days ago, the patient experienced a recurrence of gross hematuria, which was complete hematuria without blood clots. Today, the patient sought further treatment at our hospital. The patient had pulmonary tuberculosis 15 years ago and was cured. Bronchiectasis has been present for 10 years, with cough and a small amount of yellow green phlegm. After admission, clinical examinations such as chest CT, fiberoptic bronchoscopy, urine routine (urine analysis + sediment quantification), urine trace protein determination//urine creatinine (random urine), urine protein quantification (24-hour urine), antinuclear antibody assay (ANA), alveolar lavage fluid bacterial culture, acid fast staining of alveolar lavage fluid, and alveolar lavage fluid mNGS were conducted. Chest CT: Cystic dilation of bronchi in both lungs, mainly in the lower lungs, with visible phlegm clots inside. Fibrobronchoscopy: A large amount of white foam like secretions can be seen in the lumens of the middle lobe of the right lung and the lower lobes of both lungs. Quantitative determination of urine protein (24-hour urine): total protein 0.93 g/24-hour urine, urine routine (urine analysis + sediment quantification): protein+, occult blood++, red blood cell count 818.50/ $\mu\text{L}$ , microscopic RBC quantification 668.00/ $\mu\text{L}$ , microscopic WBC quantification 12.00/ $\mu\text{L}$ , urine trace protein determination//urine creatinine (random urine): microalbumin 156.00 mg/L  $\uparrow$ , urine mALB/urine creatinine 132.73 mg/g Cr, antinuclear antibody assay (ANA): weakly positive, Lymphocyte subpopulation analysis: NK cell percentage 5.32%, NK cell count  $0.057 \times 10^9/\text{L}$ , renal function test: creatinine 86.90  $\mu\text{mol}/\text{L}$ , glomerular filtration rate evaluation 80.71 mL/minute/1.73  $\text{m}^2$ , Liver function test: globulin 41.1 g/L, albumin to globulin ratio 1.00, AST/ALT: 2.04, blood lipids: high-density lipoprotein cholesterol 0.83 mmol/L, immunoglobulin light chain test: immunoglobulin $\lambda$ Light chain 9.20 g/L, immunoglobulin: immunoglobulin IgA (immunoassay) 5.30 g/L, coagulation function: fibrinogen detection: 4.45 g/L, erythrocyte sedimentation rate measurement (ESR): erythrocyte sedimentation rate: 64.0 mm/hour, inflammatory marker: interleukin6: 13.02 pg/mL, Blood routine + hypersensitive CRP (whole blood): lymphocyte percent-

age: 12.8%, neutrophil percentage: 79.8%, eosinophil percentage: 0.1%, total lymphocyte count:  $1.08 \times 10^9/\text{L}$ , total neutrophil count:  $6.74 \times 10^9/\text{L}$ , total number of eosinophils:  $0.01 \times 10^9/\text{L}$ , red blood cell distribution width: 37.6 fL, whole blood hypersensitive C-reactive protein: 20.28 mg/L. Sputum bacterial culture: negative; Acid fast staining of alveolar lavage fluid: positive (Figure A), Bacterial culture of bronchoalveolar lavage fluid: Mycobacterium abscess++ (Figure B), NGS in bronchoalveolar lavage fluid: Mycobacterium abscess. Clinical treatment plan: 0.25 g azithromycin qd po+ 0.4 g amikacin sulfate qd ivgtt+ 1 g cefmetazole sodium q12 hours ivgtt. After 10 days of treatment, the patient improved and was discharged. Continued treatment after discharge: Bailing tablets 1.76 g tid po; Kidney inflammation rehabilitation tablets 2.4 g tid po; Acid hydroxychloroquine tablets 0.2 g bid po.

## DISCUSSION

IgA nephropathy is one of the common primary glomerular diseases, characterized by IgA deposition as the main immunopathological feature [1]. Clinical manifestations include lower back pain, hematuria, proteinuria, renal function damage, etc. It presents with recurrence, diversity, and chronic progression [2]. Up to 30% of patients can progress to renal failure within 20 years [3], threatening their life safety. At present, the treatment of IgA nephropathy includes supportive therapy and immunosuppressive therapy [4]. Mycobacterium abscess is a specialized aerobic Gram-positive bacterium, belonging to one of the common nontuberculous mycobacteria (NTM) [5]. Pulmonary infection by Mycobacterium abscess is usually caused by the patient's weakened body resistance, which is mistakenly inhaled into the lungs from the oral or nasal cavity [6]. Patients may experience symptoms such as fever, cough, and sputum, and the sputum is usually purulent, accompanied by symptoms such as chest pain and difficulty breathing [7]. If an individual suffers from certain diseases or receives immunosuppressive treatment, which leads to damage to their immune system, Mycobacterium abscess is more likely to cause infection [8]. This article reports a case of IgA nephropathy complicated with Pulmonary infection by Mycobacterium abscess. The patient was diagnosed with IgA nephropathy 10 years ago and was treated with immunosuppressive agents such as Zhengqing Fengtongning sustained-release tablets. Although the condition has improved, the patient's immune defense is greatly challenged by the invasion of foreign pathogens.

IgA nephropathy patients usually experience gross hematuria, proteinuria, or deterioration of renal function after upper respiratory tract infection [9]. Qian H [10] reported that 15% of adult IgA nephropathy patients seek medical attention following the first symptom of gross hematuria after upper respiratory tract infection. The patient in this case was diagnosed with IgA ne-



**Figure A:** *Mycobacterium abscess* Acid fast staining x 1,000.

**Figure B:** Growth of *Mycobacterium abscess* in Blood agar medium at 35°C, 5% CO<sub>2</sub>, 4 days.

phropathy with bronchiectasis 10 years ago. During treatment, the patient experienced persistent hematuria accompanied by cough and sputum symptoms, most likely due to upper respiratory tract infection and the use of immunosuppressants exacerbating kidney damage, further worsening renal function. IgA nephropathy patients should use nephrotoxic drugs with caution. Common nephrotoxic drugs include nonsteroidal anti-inflammatory drugs, immunosuppressants, contrast agents, etc. [11]. The treatment of IgA nephropathy should be based on non-immunosuppressive agents [12], also known as supportive therapy, to reduce the incidence of disease progression.

The incidence rate of NTM disease is increasing year by year and has become one of the important public health problems threatening human health [13]. The patient in this case has symptoms of coughing and a small amount of yellow green phlegm. Upon auscultation, scattered moist rale were heard in both lungs, with obvious moist rale in the bottom of both lungs. Chest CT showed moist rale in both lower lungs. Based on clinical experience, ceftriaxone was used for anti infection treatment, while nebulization was used to assist in sputum excretion. After 3 days of treatment, the patient still coughed a lot of phlegm and had obvious moist rale in both lungs. Combined with other laboratory examination data, this indicates that the patient still has a severe clinical infection. However, multiple sputum cultures yielded negative results. Therefore, with the patient's consent, bronchoscopy, bronchoalveolar lavage fluid culture, acid fast staining of lavage fluid, and mNGS of alveolar lavage fluid were performed. Fibrobronchoscopy results: A large amount of white foam-like secretions can be seen in the lumens of the middle lobe of the right lung and the lower lobes of both lungs. Culture of alveolar lavage fluid: growth of *Mycobacterium ab-*

*abscess*, acid fast staining of lavage fluid: positive, mNGS of alveolar lavage fluid: *Mycobacterium abscess*. The current treatment plan was immediately discontinued and adjusted to 0.25 g of azithromycin qd po + 0.4 g of amikacin sulfate qd ivgtt+ 1 g of cefmetazole sodium q12 hours ivgtt. After 6 days of treatment, the patient's cough and sputum symptoms significantly improved. In terms of detecting pulmonary pathogens, bronchoalveolar lavage fluid is more accurate than sputum, and it is less susceptible to the influence of normal upper respiratory flora. When taking samples, it is also not affected by air ducts or bronchial mucus, which is conducive to searching for pathogenic bacteria [14]. Sputum samples are usually sent for examination through methods such as natural expectoration and tracheal aspiration [15]. Due to the significant influence of external factors on sputum, the judgment process may be affected to some extent. In this case, despite multiple negative sputum cultures, the pathogen was confirmed through bronchoalveolar lavage fluid, confirming the importance of bronchoalveolar lavage in pathogen diagnosis.

It is worth noting that this case is easily confused with pulmonary tuberculosis. Both the symptoms of systemic poisoning and the manifestations of local body damage are similar to those of tuberculosis [16]. The chest CTs of patients with two diseases have similar results and cannot identify *Mycobacterium tuberculosis* and NTM by acid fast staining, which is easily misdiagnosed as tuberculosis [17]. Therefore, when acid fast staining is positive, further differentiation between *Mycobacterium tuberculosis* and NTM can be achieved through mass spectrometry and mNGS detection. In addition, for patients diagnosed with pulmonary tuberculosis, attention should also be paid to tracking the results of sputum or lavage fluid culture to determine the occurrence of pulmonary tuberculosis combined with NTM lung disease.

Mycobacterium abscess is naturally resistant to most first-line anti-tuberculosis drugs [18]. Therefore, when acid fast staining is positive but the anti-tuberculosis treatment effect is not good, one should be alert to the possibility of misdiagnosis or concurrent infection with Mycobacterium abscess or other types of NTM and promptly identify the cause and treat according to drug sensitivity results.

In summary, this article reports a case of IgA nephropathy complicated with pulmonary infection by Mycobacterium abscess. First, we hope to improve doctors' understanding of IgA nephropathy through this case report. Second, we hope to clarify the importance of bronchoalveolar lavage in detecting pulmonary pathogens. Finally, it was confirmed that mNGS technology in bronchoalveolar lavage fluid has considerable application prospects for diagnosing pathogens.

#### Ethics Approval and Consent to Participate:

Ethical review and approval were not required for this study. The patient provided written informed consent to participate in this study.

#### Consent for Publication:

The patient provided written informed consent for study publication.

#### Availability of Data and Materials:

The original data and materials presented in the study are included in the article/supplementary material. Further inquiries can be directed to the corresponding author.

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#### Declaration of Interest:

The authors declare no competing interests.

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