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Multiple infections in a patient with anti-interferon gamma antibody-associated adult onset immunodeficiency: A case report

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Abstract

We present a rare case of anti-interferon-γ autoantibody-associated Adult Immunodeficiency Syndrome (AID) successfully managed despite complicated by multiple opportunistic infections. This report underscores the critical need for clinical vigilance in recognizing immunodeficiency states, maintaining a high index of suspicion for co-infections, and implementing timely therapeutic interventions to achieve optimal patient outcomes.

Keywords: AIGAs; Nontuberculous mycobacterial (NTM); Novel coronavirus (2019-nCOV); Multiple infections; Case report.

Abbreviations: AID: Anti-Interferon-γ Autoantibody-Associated Adult Immunodeficiency Syndrome; NTM: Nontuberculous Mycobacterial; 2019-nCOV: Novel Coronavirus; AIGAs: Anti-Interferon-γ Autoantibodies; MF: *Mycobacterium Fortuitum*; mNGS: Metagenomic Next-Generation Sequencing.

Introduction

Anti-interferon-γ autoantibody-associated adult immunodeficiency syndrome is a rare disorder caused by pathogenic Anti-Interferon-γ Autoantibodies (AIGAs), which potently increase susceptibility to disseminated opportunistic infections [1], particularly Nontuberculous Mycobacterial (NTM) diseases, *Talaromyces marneffei* infection, and *Salmonella* spp. Bacteremia [2].

Herein, we describe an adult patient with anti-interferon-γ autoantibody-associated immunodeficiency complicated by concurrent infections with *Mycobacterium Fortuitum* (MF), *Salmonella* enterica, SARS-CoV-2, and *Mycoplasma pneumoniae*. The clinical presentation featured a sweet syndrome-like neutrophilic dermatosis, generalized lymphadenopathy, and multisystem involvement. This case underscores the critical importance of early recognition of suspected immunodeficiency states and prompt

identification of infectious pathogens through comprehensive microbiological workup to optimize clinical outcomes.

Case Presentation

A 55-year-old female farmer, long-term resident of southern China, received tramadol for knee and ankle pain in late March 2024. She subsequently developed erythema, papules, and pustules on the lower limbs, which were diagnosed as drug-induced dermatitis in April. Clinical improvement occurred after discontinuing tramadol and initiating antihistamine therapy.

In June 2024, new edematous erythema, papules, pustules, and pseudobullets emerged on limbs, along with scattered lower - limb pigmentation, upper - limb mild and lower - limb moderate pitting edema, and palpable enlarged lymph nodes in neck, axilla, and groin. She had a history of lumbar surgery for disc herniation and spinal stenosis a year ago and long - term use of enteric - coated aspirin and clopidogrel due to cerebral infarction. Upon admission, blood tests showed elevated inflammatory indicators, moderate anemia, and hypokalemia. Considering poor appetite and nutrition, hypoproteinemia and hypokalemia were thought to be from low potassium intake, but infectious, blood system, or tumor diseases were not ruled out. Further examinations, like abdominal ultrasound (left kidney cyst, hepatomegaly), superficial lymph node ultrasound (enlarged lymph nodes), chest CT (pneumonia, serous cavity effusion), bone marrow cell morphology (reactive images), and anti-γ-interferon autoantibody test (indicating immunodeficiency), were carried out. Lymph node and skin biopsies were done. She was treated with methylprednisolone for anti - inflammation, drugs for cerebral infarction, and received potassium and protein supplements. After that, symptoms were improved (Figure 1).

On July 5, 2024, she had a high fever. Inflammation marker reexaminations was elevated. After blood culture, levofloxacin was given for suspected infection, and methylprednisolone was stopped. Skin biopsy showed interface dermatitis, and lymph node puncture showed reactive hyperplasia (Figure 2A, B, C & D). Metagenomic Next-Generation Sequencing (mNGS) of lymph node tissue detected MF, EB virus, and cytomegalovirus. The subsequent cultivation results also indicated NTM which was identified as MF through species identification (Figure 2E). Blood culture later found *Salmonella*. The anti - infection regimen was adjusted to moxifloxacin, clarithromycin, rifampicin against NTM and ceftriaxone against *Salmonella*, and the fever abated. Jaundice and significant decrease in platelets on July 8 led to discontinuation of clopidogrel and rifampicin, with addition of hepatoprotective agents. On July 9, cough, expectoration, dyspnea, and recurrent fever occurred; diagnostic testing revealed SARS-CoV-2 RNA positivity and *Mycoplasma pneumoniae* culture confirmation. Appropriate antiviral (nirmatrelvir-ritonavir) and antibacterial (azithromycin) therapies were initiated, leading to clinical improvement.

Two months later, an occipital ulcerative lesion (following trauma) yielded Salmonella on culture, which resolved after 18 days of imipenem-cilastatin sodium therapy. Maintenance treatment with moxifloxacin and clarithromycin for nontuberculous mycobacterial infection continued. Serial assessments demonstrated normalization of chest CT findings, platelet counts, and absence of new cutaneous lesions at follow-up.

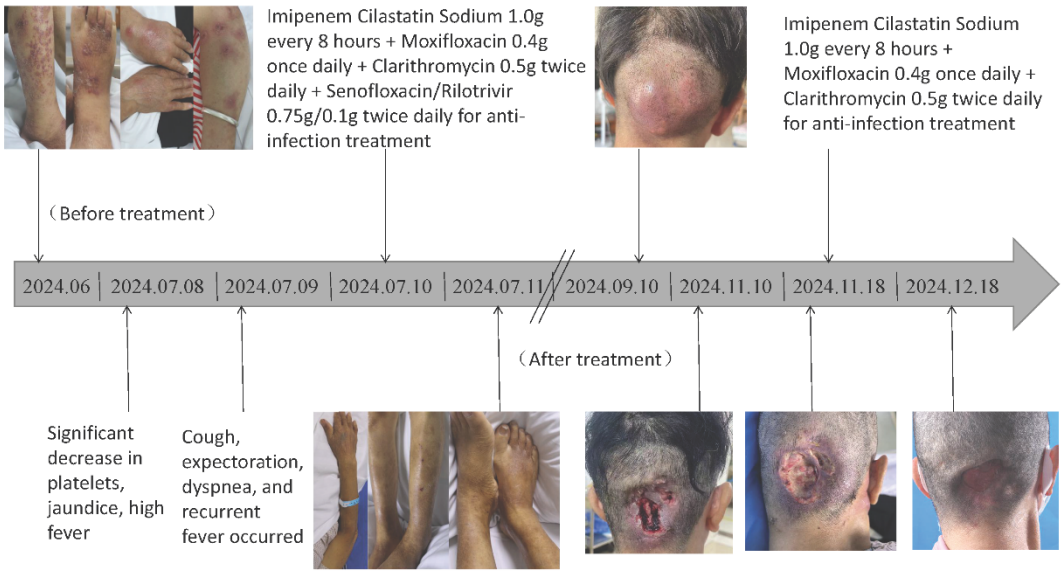

Figure 1: Timeline of patient's clinical manifestations and treatment.

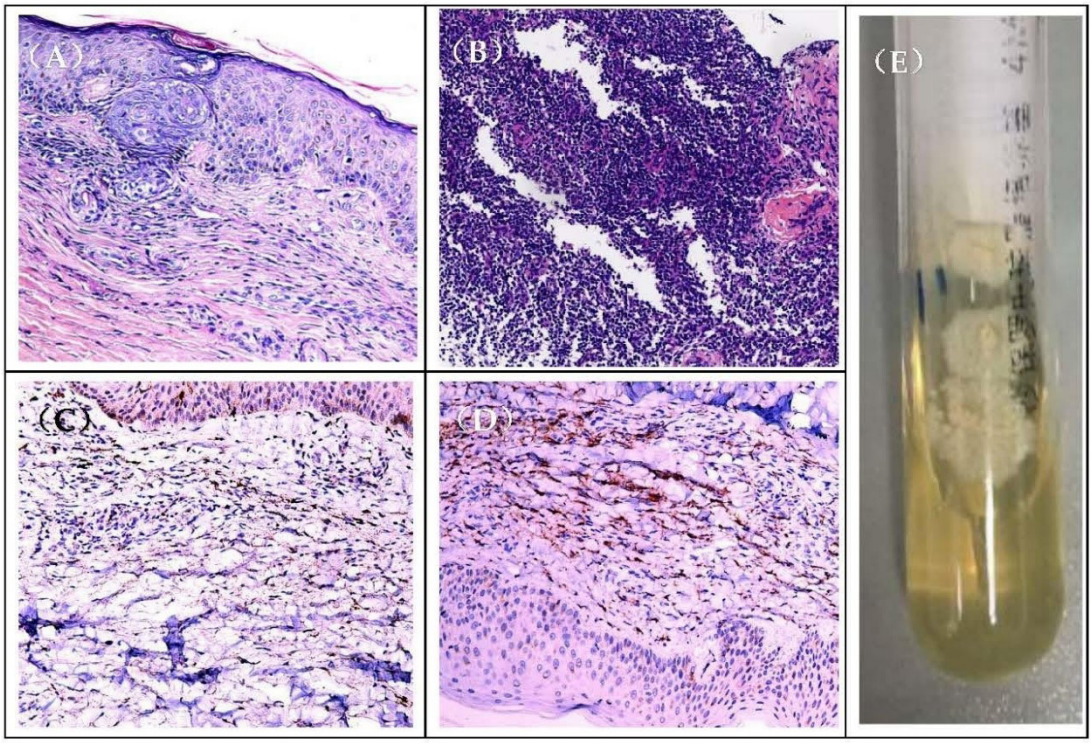

Figure 2: **(A)** A skin biopsy from the patient's lesional site. The epidermis appears essentially normal, with pathological changes primarily localized to the dermis. No typical sweet syndrome-like papillary dermal edema is observed, and there is no significant neutrophilic infiltration; instead, the infiltrate consists mainly of lymphocytes and histiocytes (hematoxylin and eosin staining; ×40). **(B)** An enlarged inguinal lymph node shows reactive lymphoid hyperplasia (hematoxylin and eosin staining; ×40). **(C)** A skin biopsy from the lesional site demonstrates positive expression in dermal infiltrating cells (CD68 immunohistochemical staining; ×40). **(D)** A skin biopsy from the lesional site shows positive expression (Myeloperoxidase (MPO) immunohistochemical staining; ×40). **(E)** Colonies of MF grown on Sabouraud dextrose agar from the enlarged inguinal lymph node after 20 days of incubation at room temperature, as identified by bacterial species identification.

Discussion

Our case underscores that patients with AIGAs exhibit profound immune compromise, necessitating heightened clinical vigilance for concomitant infections during diagnostic and therapeutic management. First, comprehensive pathogen screening, including molecular and culture-based methods, should be prioritized in the early clinical course. Second, timely intervention is critical to improving outcomes in these patients. Notably, during SARS-CoV-2 infection, this patient developed not only typical respiratory symptoms (cough, expectoration) but also abrupt thrombocytopenia and fulminant jaundice, raising questions about whether these manifestations could serve as early indicators of severe COVID-19 in AID patients—a hypothesis warranting further investigation.

Previous literature summarizes AIGAs-associated clinical features as lymphadenitis and reactive skin diseases, often accompanied by nonspecific systemic symptoms (fever, cough, bone pain, weight loss) [1]. Reactive dermatoses typically include sweet syndrome, pyoderma gangrenosum, and pustular psoriasis. Infections such as NTM primarily involve lymph nodes, bone marrow, bones, lungs, and skin soft tissues, with rare blood culture positivity [3]. Clinicians should suspect AIGAs in patients presenting with unexplained lymphadenopathy, reactive skin lesions, and negative HIV serology, particularly when rare opportunistic infections emerge. A recent review highlighted NTM as the most common pathogen (followed by *Talaromyces marneffei*), with over 50% of patients experiencing coinfections and a 56% recurrence rate [4].

This case demonstrated MF detection via both skin lesion culture and mNGS. As an opportunistic pathogen, MF primarily infects individuals with cell-mediated immunity deficits or glucocorticoid exposure [5], often entering through cutaneous wounds to cause localized infections. MF infections have been associated with surgical procedures, including total hip/knee arthroplasty and post-laparoscopic surgery infections [6]. Clinically, they typically present as single-site cutaneous lesions, though multiple skin lesions have been reported in settings of repeated penetrating trauma (e.g., local injections, acupuncture, or hair removal) [7]. While systemic dissemination is rare, immunocompromised patients, including those with AIGAs, may develop disseminated disease, manifesting as lymphadenopathy without cutaneous involvement [8]. In our patient, reactive skin lesions and lymphadenopathy occurred without single-site localization, consistent with immune-mediated pathogenesis: negative skin tissue culture and absence of mycobacterial pathological features contrasted with positive lymph node culture, mNGS, and lymphadenitis histology. Bronchoscopy was not tolerated, precluding evaluation of pulmonary involvement.

Thrombocytopenia observed during the disease course was likely SARS-CoV-2-associated, aligning with literature linking thrombocytopenia in COVID-19 to increased mortality [9]. Early recognition of platelet decline prompted rapid viral testing and intervention, including antiviral therapy and platelet transfusion, which correlated with clinical improvement. This reinforces the prognostic value of platelet monitoring in SARS-CoV-2-infected patients, particularly those with underlying immunodeficiency [10].

Conclusion

We present a successfully managed case of AIGAs complicated by multiple infections. Clinical manifestations, including reactive cutaneous eruptions, lymphadenopathy, and fever, warrant heightened clinician awareness to prioritize immune function screening, such as HIV serology, anti-interferon-γ autoantibody testing, and other relevant biomarkers, to identify underlying immunodeficiency and concomitant opportunistic infections at an early stage. For patients with confirmed immunodeficiency, meticulous monitoring of evolving clinical symptoms is essential to detect new or overlapping infections promptly, as these often necessitate prolonged antimicrobial therapy and tailored management strategies. Additionally, long-term follow-up to evaluate treatment response and infection recurrence provides critical clinical insights for refining diagnostic algorithms and therapeutic approaches in similar cases.

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