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CASE REPORT

Fatal Mpox Coinfection in Advanced AIDS: A Case Report

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Abstract: The mpox virus (MPXV) is a zoonotic pathogen that has been causing a worldwide pandemic outbreak since 2022, posing a significant threat to global public health security. During this outbreak, monkeypox has been more prevalent among immunocompromised individuals, particularly those infected with HIV (Human Immunodeficiency Virus). We report a case of a 21-year-old male patient with advanced AIDS in China who developed severe complications, including skin tissue infection, proctitis, and conjunctivitis, following monkeypox infection. Despite early aggressive treatment, the patient ultimately succumbed to infectious shock and multiple organ failure. This case strongly underscores that such patients experience severe skin infections and rapid disease progression, necessitating urgent early recognition and treatment.

Keywords: AIDS, mpox virus, clinical manifestations, differential diagnosis

Introduction

Mpox is a viral disease caused by the mpox virus, a member of the orthopoxvirus genus. It was discovered in humans in 1970 in the Democratic Republic of Congo (formerly Zaire), and sporadic outbreaks of the virus have been recorded throughout Africa.[1] Infections are often transmitted to humans through contact with animal hosts.[2] The major clinical characteristics of typical mpox infection, such as fever, lymphadenopathy, and headache, are followed by a skin rash 2–4 days later, which proceeds from macules and papules to blisters, pustules, and crusts.[3] Due to similar transmission routes, in the current epidemic, monkeypox mainly occurs among people with AIDS. Both are mainly transmitted through sexual contact, especially among male homosexual patients. WHO epidemiologic statistics reveal that up to 52% of mpox patients worldwide are HIV-positive.[4] Most HIV-infected individuals with CD4 counts exceeding 500 cells per cubic millimeter exhibit a self-limiting course similar to that of uninfected individuals. However, patients with advanced HIV infection often present with atypical clinical symptoms, potentially including prolonged disease progression lasting several months, persistent ulceration and necrosis of skin lesions, susceptibility to secondary skin infections, and disseminated systemic lesions.[5] These symptoms are frequently linked to immune system failure. According to research, patients with advanced AIDS who have CD4+ T lymphocyte counts less than 200 cells/cm^3 are at risk for severe secondary bacterial infections of the skin and circulation as well as significant skin necrosis.[6] These individuals may also have a variety of negative effects on public health, including as increased risks of transmission, a burden on healthcare systems, and difficulties with prevention and surveillance. Therefore, in order to effectively address the issues provided by co-infection with HIV and monkeypox, comprehensive efforts are needed to improve screening and monitoring, encourage vaccination, optimize treatment procedures, and raise public awareness. Although most cases of monkeypox infection are in HIV-positive individuals, deaths are relatively rare, with a mortality rate ranging from 1% to 11%.[7] This article reports a case of a patient with advanced AIDS who died after being infected with disseminated Mpox. In contrast to normal situations, the patient's rash continued to worsen while receiving treatment, resulting in extensive body lesions

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and side effects such conjunctivitis and proctitis. Rapid illness progression and insufficient infection management finally resulted in mortality. By describing the clinical characteristics, diagnosis, and treatment of this patient, the aim is to provide a reference for clinicians in the diagnosis and treatment of monkeypox infection in patients with advanced AIDS.

Case Report

A 23-year-old male patient reported the onset of a facial rash one week ago and presented to the hospital with a progressive spread of the rash (Figure 1A) and the development of cough, low-grade fever, and anal pain. He had been infected with HIV for seven years. He had been treated with irregular Eckuo protection antiretroviral therapy for two years, with a poor viral immune response (8 CD4+ lymphocytes/μL on admission and an HIV-1 viral load measurement of 51,673 Copies/mL). On admission, T37.5°C, blood pressure, respiration, and heart rate were average. Physical examination showed a central sunken rashon the face, trunk, and extremities, with the face being the most severe, and enlarged superficial lymph nodes in the neck, axilla, and groin. The patient was diagnosed with Penicillium marneffei infection three years ago, presenting with a typical umbilicated rash (Figure 1B). After three months of itraconazole treatment, the patient did not return for follow-up. Therefore, on admission, Marneffei reinfection was considered, and the patient was given oral antifungal treatment with voriconazole. Laboratory results indicated an ultrasensitive C-reactive protein of 28.73 mg/L, an interleukin 6 test of 57.8 pg/mL, blood sedimentation of 28 mm/h, and lactic acid of 2.77 mmol/L, with all other values within normal ranges. The cryptococcal antigen test was negative, and the Fungal (1-3)-β-D glucan assay and Aspergillus galactomannan assay were normal. Chest CT scan revealed several solid nodules in both lungs and infectious lesions were evaluated. On the second day of hospitalization, the CDC throat swab test returned positive. Nucleic acid testing of skin lesions and blood samples confirmed mpox virus infection. Immediate isolation and treatment were initiated.

The patient's blood and rash secretions had negative fungal cultures, and the rash continued to spread after antifungal treatment, tentatively ruling out Marneffei infection. Upon admission, the patient received cefazolin sodium for infection control. Three days later, culture of the rash exudate revealed Pseudomonas aeruginosa and Staphylococcus aureus infections. Consequently, the antimicrobial regimen was adjusted to meropenem combined with vancomycin, while continuing antiretroviral therapy (ART) (Tivicay + tenofovir alafenamide, later supplemented with Eltrombopag). Symptomatic supportive treatments such as skin care, rectal care, immunity enhancement, electrolyte supplementation, and Chinese medicine were also administered. Despite aggressive treatment and care, the patient's symptoms did not

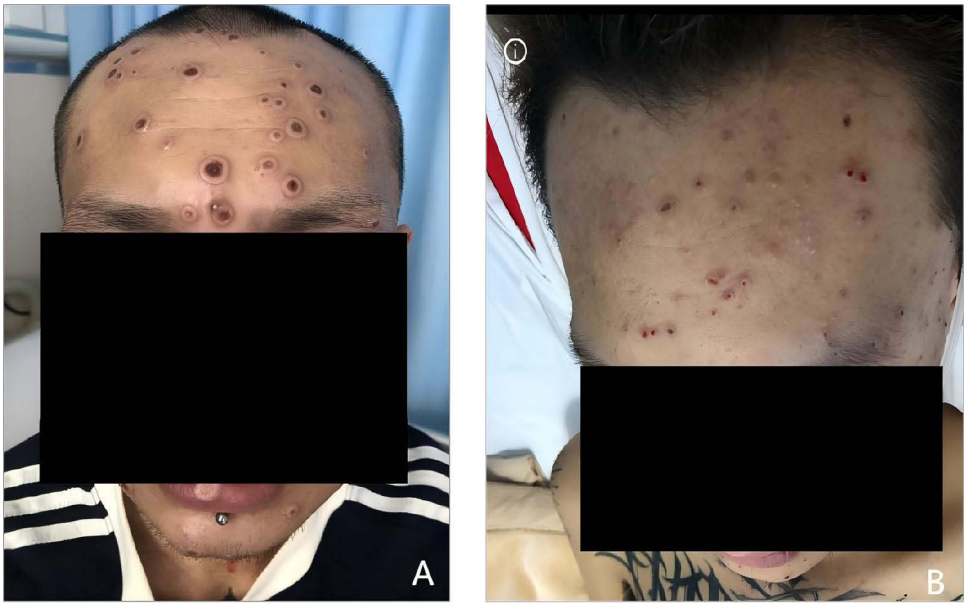

Figure 1 Clinical presentation. (**A**) The condition of the rash at admission. (**B**) Facial rash (Basket Bacteria marneffei rash) on November 21, 2020.

Table 1 Changes in Inflammatory Indicators

Parameter	Aug 25	Sep 1	Sep 6	Sep 11
WBC (10^9/L)	8.3	11.9	19.5	32.7
NEUT (10^9/L)	6.66	9.74	17.22	30.18
CRP (mg /L)	28.78	76.93	>200.00	>200.00
IL6 (pg/mL)	57.8	107.93	182.9	185.4
SAA (mg/L)	66	226.3	210.2	/
PCT (ng /mL)	<0.05	0.06	0.24	0.56

Abbreviations: WBC, white blood cell count; NEU, neutrophils; CRP, c-reactive p-rotein; IL6, interleukin-6; SAA, serum amyloid A; PCT, procalcitonin.

improve. During hospitalization, inflammatory indicators continue to rise (Table 1), infection proved difficult to control, and infectious lesions in the rash tissue progressively worsened (Figure 2). The patient developed severe colitis, conjunctivitis, and respiratory infection, ultimately succumbing to septic shock and multiple organ failure.

Discussion

In line with the clinical manifestations of late-stage HIV-associated monkeypox infection as reported in recent investigations, the patient's primary presentation consisted of a widespread rash, low-grade fever, and enlarged inguinal lymph nodes.[8] Despite the original diagnosis of Marneffei infection, we had a strong suspicion of monkeypox infection. In order to confirm monkeypox infection, we immediately performed blood mNGS analysis and monkeypox virus nucleic acid testing. This prevented a misdiagnosed patient from receiving ineffective treatment. It has been found to cause significant skin necrosis and serious secondary bacterial skin and blood infections in people with advanced AIDS who had CD4+ T-lymphocyte counts of less than 200 cells per cubic millimeter.[6] These individuals have a prolonged presence of skin ulcers and a constant new rash. This characteristic allows for the simultaneous existence of many phases of the rash. Skin ulcers are prone to subsequent tissue infection, which can progress to sepsis, one of the leading causes of mortality in this outbreak. In previous reports, monkeypox was primarily distinguished from other febrile rash diseases such as chickenpox, herpes zoster, measles, and dengue fever;[9] however, in tropical Asia, where Malnifaux infections are common, we emphasize the importance of distinguishing between co-morbid Talaromyces marneffei infections. Advanced AIDS patients with fever and rash who suspect monkeypox should have monkeypox CDC throat swabs and monkeypox virus nucleic acid testing as soon as feasible.

A case report of a 33-year-old HIV-infected patient (CD4+ t-cell count, 35 per cubic millimeter) who received two courses of oral tekovirim was reported to have eventually developed infectious shock and multi-organ failure leading to death.[10] As in this reported case, the same extensive skin lesions, infectious shock, and multi-organ failure were

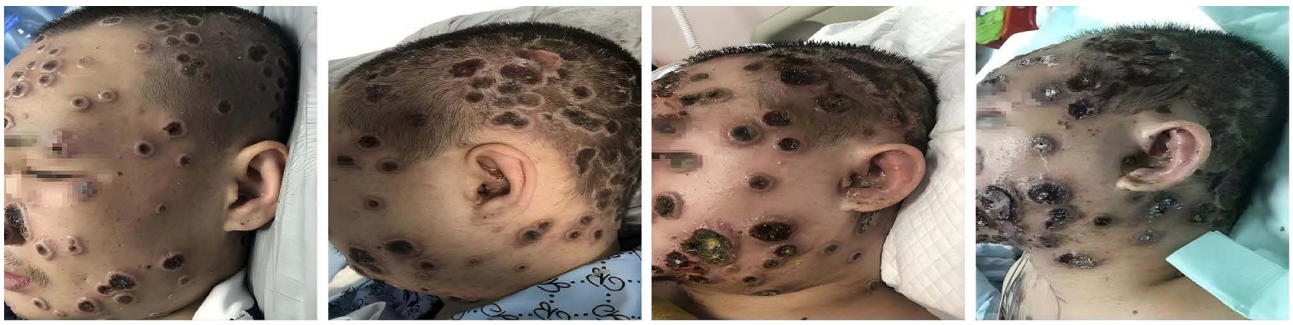

Figure 2 The scalp was progressing during the hospital stay.

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present. Consistent with the results reported in the latest study, the leading cause of death was infectious shock and multi-organ failure in 20 out of 27 cases (74%).[6] The primary focus is on symptomatic assistance, therapy, and management of consequences; no particular anti-MPXV drugs are available. Although the US Food and Drug Administration (FDA) has approved tecovirimab, brincidofovir, and cidofovir for the treatment of smallpox.[11] However, there is currently a lack of pharmacokinetic data for TPOXX in patients with extremely low CD4 counts, and once extensive skin necrosis has been established, it is difficult to reverse.[12] Therefore, more research is needed to determine whether Tecovirimat is effective in patients with very low CD4 counts. Furthermore, patients with high levels of inflammation may be more susceptible to the nephrotoxicity risk associated with brincidofovir or cidofovir, therefore its usage should be careful. The prognosis for monkeypox infection in immunocompromised persons may be severe, as studies show that CD4+ < 50/μL is an independent mortality risk factor in advanced AIDS patients.[13] For these patients, ART regimens based on quickly acting integrase inhibitors should be used, closely monitored for IRIS. Aiming to keep CD4+ counts over 200 cells/μL, ART management must be improved at the same time through case management and educational outreach. Future studies should concentrate on methods to successfully restore immune function and lower infection risk in people with concurrent HIV and monkeypox infection, as immune reconstitution is frequently difficult in these patients.

The most severe form of transmission is currently thought to be among gay or bisexual men and other men who have sex with men,[14] which suggests that the spread through the sexual network is gradually increasing. This further establishes mpox-related stigmatization, especially its effects on the LGBTQ+ population, as a significant social and public health concern. In the fight against mpox, the important lessons from the HIV/AIDS epidemic must be strictly avoided. It is important to note that Mpox is a zoonotic virus that can spread to people through intimate contact. It is damaging and inaccurate to link the disease to particular gender identities or sexual orientations. Therefore, in order to stop misinformation at its source, researchers and healthcare providers have an obligation to communicate outbreak information using neutral, correct language that emphasizes behavioral hazards rather than group identities. This case report is a single case report based on a single patient, which limits the universality of the results. In addition, the patient in this case had limited treatment options, lacked specific anti-MPXV treatments, and lacked effective means in disease care.

Conclusion

This case highlights the following points. When AIDS patients present with central depressed rashes or skin ulcers, monkeypox must be considered. Clinical manifestations and outcomes of monkeypox in HIV-positive patients may vary significantly depending on CD4 cell counts. Individuals with advanced AIDS and low CD4 counts face high mortality rates from monkeypox infection. Therefore, it is essential to assess HIV viral load and CD4 counts in HIV patients at risk for severe monkeypox. Treating monkeypox in advanced AIDS patients presents multiple challenges, necessitating a comprehensive approach that considers immune function, treatment efficacy, and psychosocial support to enhance therapeutic outcomes and improve prognosis.

Abbreviations

MPXV, mpox virus; HIV, human immunodeficiency virus; AIDS, acquired immune deficiency syndrome; T, body temperature; CT, Computed tomography; CDC, Centers for Disease Control; ELISA, enzyme-linked immunosorbent assay.

Data Sharing Statement

No datasets were generated or analysed during the current study.

Ethics Approval and Consent to Participate

This case was reviewed and approved by the Medical Ethics Committee of Baise People's Hospital.

Consent for Publication Statement

The publication of this case report and any accompanying pictures has obtained the written informed consent of the the patient's mother and her consent for publication.

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Disclosure

The authors report no conflicts of interest in this work.

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