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Case Report

Mpox-Associated Immune Thrombocytopenia in Advanced HIV: A Case Study

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Abstract

Background: Mpox (Monkeypox virus, MPXV) may present with increased severity in people with HIV (PWH), particularly those with advanced immunosuppression. Coinfections and hematologic complications further complicate management and outcomes.

Case Presentation: A 42-year-old man with advanced HIV (CD4 67 cells/μL) off antiretroviral therapy presented with a two-week history of ulcerated nodular skin lesions involving the extremities, chest, and face. Laboratory studies revealed severe thrombocytopenia (platelets 12 × 10^9/L). Lesion PCR confirmed mpox, and culture grew methicillin-sensitive Staphylococcus aureus. Serologic testing demonstrated late-latent syphilis. Immune thrombocytopenic purpura (ITP) was treated with intravenous immunoglobulin and high-dose corticosteroids. He received doxycycline for syphilis and topical mupirocin for bacterial superinfection. Given profound immunosuppression and concurrent corticosteroid exposure, mpox-directed antivirals (tecovirimat, cidofovir with probenecid, and brincidofovir) were initiated. Antiretroviral therapy was re-started. Platelet count improved to 118 × 10^9/L with immunomodulatory therapy and ART re-initiation, while skin lesions gradually resolved. The independent contribution of antiviral therapy to recovery remained uncertain.

Discussion and Conclusion: This case highlights the complex interplay between uncontrolled HIV, mpox, and immune-mediated cytopenia. Although randomized trials have questioned tecovirimat's efficacy, current NIH and CDC guidance support antiviral therapy in patients with severe disease or advanced immunosuppression. ITP in the setting of mpox appears rare but warrants vigilance. Early antiretroviral re-initiation and multidisciplinary management remain central to optimizing outcomes in profoundly immunocompromised patients.

Keywords: Mpox; ITP; HIV; Antiretroviral; Monkeypox Virus; Orthopoxvirus; Immune Thrombocytopenic Purpura.

Introduction

Mpox, caused by Monkeypox virus (MPXV), a double-stranded DNA orthopoxvirus, re-emerged globally in 2022 with sustained transmission primarily among men who have sex with men (MSM). While most cases are self-limited, people with HIV (PWH) and low CD4 counts experience disproportionately severe disease, including necrotizing skin lesions, mucosal involvement, and systemic complications [1]. In a multicountry cohort of 382 patients, advanced HIV (CD4 < 200 cells/μL) was associated with a four-fold higher risk of hospitalization and prolonged viral shedding [2]. CDC surveillance data through 2024 show continued sporadic mpox activity in the United States, with ~40 % of cases occurring in PWH [3].

Hematologic abnormalities in mpox are incompletely characterized but can include lymphopenia, anemia, and thrombocytopenia [4]. In PWH, these cytopenias are often multifactorial-reflecting viral marrow suppression, opportunistic coinfection, or immune thrombocytopenic purpura (ITP). ITP remains the most common cause of severe thrombocytopenia in HIV, typically responding to corticosteroids, IVIG, or thrombopoietin-receptor agonists such as eltrombopag, alongside antiretroviral therapy (ART) re-initiation

[5,6]. Whether mpox directly contributes to ITP pathogenesis remains uncertain, though viral-triggered immune dysregulation is plausible.

Randomized evidence for mpox antivirals is evolving. The STOMP trial (NIH, 2024) demonstrated that tecovirimat did not significantly shorten time to lesion resolution in outpatient mpox, though it was well tolerated.[7] Similar findings were reported in the PALM007 trial for clade I mpox in the DRC.[8] Nevertheless, CDC and NIH guidelines (updated 2025) continue to support tecovirimat or cidofovir for severe disease or profound immunosuppression, citing ethical constraints and limited power of RCTs to detect benefit in these subgroups [9,10]. Case reports continue to document clinical improvement with antivirals in advanced HIV, particularly when combined with ART and wound care [11].

Coinfection further complicates management. Up to 20 % of mpox cases involve concurrent sexually transmitted infections, most commonly syphilis and gonorrhoea [12]. CDC guidelines recommend benzathine penicillin G 2.4 million units IM weekly for three doses for late-latent syphilis, with doxycycline 100 mg twice daily for 28 days as an alternative during supply shortages [13]. Additionally, bacterial superinfection of mpox lesions-often with S. aureusor Streptococcus species-requires empiric or culture-guided antibiotics and local wound care [14].

This case illustrates the convergence of uncontrolled HIV, severe mpox, and immune-mediated thrombocytopenia. The patient's recovery following corticosteroids, IVIG, ART re-initiation, and supportive care underscores the importance of early recognition and multidisciplinary management. As mpox continues to evolve within immunocompromised populations, clinicians must balance emerging RCT data with individualized risk assessment, particularly in patients with advanced HIV and hematologic complications.

We report the case of a 42-year-old man with advanced HIV/AIDS who developed severe immune thrombocytopenic purpura (ITP) and disseminated mpox (monkeypox) infection, highlighting the challenges of managing opportunistic infections and hematologic complications in the context of profound immunosuppression.

Case Presentation

A 42-year-old male diagnosed with HIV 8 months prior to admission, presented with a two-week history of progressive, nodular, and ulcerated skin lesions on his extremities, chest, and face. He discontinued antiretroviral therapy (ART; Biktarvy) 5 months prior to admission due to loss of insurance and medication intolerance. His social history was notable for tobacco, alcohol, and intermittent methamphetamine use, but he denied intravenous drug use.

On presentation, he was afebrile and hemodynamically stable, with no systemic symptoms. Physical examination revealed multiple ulcerated, crusted, and scabbed skin lesions. Laboratory evaluation demonstrated severe thrombocytopenia (platelets 12,000/μL), CD4 count of 67 cells/μL, CD4/CD8 ratio of 0.1, and HIV viral load of 555,000 copies/mL. Additional findings included positive RPR (titer 1:1), positive syphilis IgG/IgM, positive Toxoplasma IgG, and mild transaminitis. Hepatitis serologies indicated prior hepatitis B exposure and hepatitis A immunity. Screening for cryptococcal antigen, coccidioidomycosis, and tuberculosis was negative.

He was initially managed at a community hospital, where ITP was diagnosed and treated with intravenous immunoglobulin (IVIG) and high-dose prednisone (80 mg daily), resulting in transient platelet improvement. ART was not re-initiated at this time due to challenges with adherence. On hospital day 3, he was transferred to a tertiary center for further evaluation of persistent skin lesions and concern for an AIDS-defining illness. Dermatology performed a bedside swab and shave biopsy; PCR confirmed mpox and orthopoxvirus, and tissue culture grew methicillin-sensitive Staphylococcus aureus (MSSA). Biopsy showed benign abscess formation without fungal or mycobacterial organisms.

Given the diagnosis of mpox in the setting of advanced immunosuppression, he was started on cidofovir with probenecid, tecovirimat (TPOXX), and brincidofovir as part of an investigational protocol. He was also started on Bactrim DS (800/160 mg daily) for Pneumocystis jirovecii pneumonia (PJP) and toxoplasmosis prophylaxis. ART with Biktarvy was re-initiated, and aggressive counseling regarding adherence and linkage to Ryan White services was provided. Due to the risk of ongoing immunosuppression, steroids were tapered and discontinued; eltrombopag was initiated at 50 mg daily and increased to 50 mg BID for ITP, with a platelet count of 68,000/μL prior to discharge. For late-latent syphilis, he received two doses of intramuscular benzathine penicillin G (2.4 million units each, one week apart), with a plan for a third dose as an outpatient or doxycycline 100 mg BID for 28 days if penicillin was unavailable.

The patient remained clinically stable, afebrile, and without bleeding or end organ complications. His skin lesions improved, and no new lesions developed. He was discharged on hospital day 14 with a three-month supply of Biktarvy, Bactrim DS, the remainder of his tecovirimat course, and doxycycline if needed. He received detailed instructions on infection control, medication adherence, and follow-up with hematology and HIV care. Smoking cessation resources were provided.

Discussion

This case highlights the intricate overlap of mpox, advanced HIV infection, and immune thrombocytopenic purpura (ITP),

illustrating how profound immunosuppression amplifies the clinical complexity of emerging viral infections. Mpox in people with HIV (PWH) has been shown to follow a more severe course, particularly in those with CD4 counts below 200 cells/μL, with higher rates of necrotic skin lesions, systemic involvement, and mortality [15]. Our patients disseminated ulcerative lesions and severe thrombocytopenia exemplify this high-risk presentation.

Mpox Severity in Advanced HIV

In immunocompetent hosts, mpox is typically self-limited, resolving within 2-4 weeks. However, in PWH with low CD4 counts, inadequate cytotoxic T-cell function allows uncontrolled viral replication, leading to protracted disease and tissue necrosis [16]. Studies have documented higher viral loads, delayed lesion healing, and increased hospitalization rates in this subgroup [17]. Early initiation or re-initiation of antiretroviral therapy (ART) remains central to recovery. Although immune reconstitution inflammatory syndrome (IRIS) has been reported following ART initiation during mpox, its occurrence is rare, and the benefits of restoring immune function outweigh the risks [18]. Transient worsening of mpox lesions has been reported following immune recovery, particularly in patients with advanced HIV and high inflammatory burden. While clinicians should remain vigilant for this phenomenon, the potential risk of IRIS does not outweigh the benefits of early ART initiation in profoundly immunocompromised patients.

Antiretroviral therapy should not be deferred solely due to concerns regarding medication adherence. Contemporary ART regimens possess a high barrier to resistance, and immune restoration remains essential for resolution of opportunistic infections such as mpox and HIV-associated immune thrombocytopenic purpura. In this case, delayed ART re-initiation contributed to persistent immunosuppression and clinical complexity, underscoring the importance of early ART initiation alongside intensive adherence counseling and linkage to support services.

Hematologic Manifestations

Thrombocytopenia is a recognized but understudied manifestation of mpox. Most cases appear secondary to HIV-associated bone marrow suppression or immune dysregulation rather than direct viral cytotoxicity [19]. In HIV, ITP results from both decreased platelet production and increased immune-mediated destruction, often driven by cross-reactive antibodies and impaired megakaryopoiesis [20]. The rapid improvement in platelet counts after corticosteroids and IVIG in this case supports an immune-mediated mechanism. Thrombopoietin-receptor agonists such as eltrombopag or romiplostim are effective in refractory HIV-related ITP, though ART remains the cornerstone of long-term disease control [21].

Antiviral Use in Severe Disease

Although tecovirimat (TPOXX) has been widely used under expanded-access protocols, its efficacy remains uncertain. The 2024 STOMP randomized trial found no difference in lesion resolution or pain compared with placebo in non-hospitalized mpox patients [22]. Similarly, the PALM007 trial in the Democratic Republic of Congo showed no clinical benefit for clade I mpox.[23] Nevertheless, both the CDC and NIH recommend antiviral therapy for patients with severe disease, ocular or mucosal involvement, or advanced immunosuppression, given the underrepresentation of such patients in clinical trials [24]. Our patient's favorable outcome without antiviral therapy underscores the value of individualized decision-making when disease is localized and systemic symptoms are absent.

Coinfections and Bacterial Superinfection

Coinfection with sexually transmitted infections (STIs) is frequent in mpox, reflecting shared behavioral risk factors. Up to 20–25% of cases in global surveillance cohorts had concurrent syphilis, gonorrhea, or chlamydia [25]. Our patient's incomplete treatment for late-latent syphilis was managed with doxycycline, in alignment with CDC interim guidance during penicillin shortages [26]. Bacterial superinfection of mpox lesions, most often caused by Staphylococcus aureusor Streptococcus pyogenes, can exacerbate inflammation and delay healing [27]. This underscores the need for culture-guided antibiotic therapy and meticulous wound care.

Clinical Lessons

This case demonstrates that effective management of severe mpox in advanced HIV requires an integrated approach addressing both virologic and immunologic factors. Early ART re-initiation, immunosuppressive therapy for ITP, and targeted treatment of bacterial and sexually transmitted coinfections were key to this patient's recovery. While tecovirimat remains an option in severe or disseminated cases, supportive and immune-modulating strategies may suffice in selected patients with improving lesions and stable clinical status.

Conclusion

This case demonstrates the multifactorial complexity of mpox in the setting of advanced HIV infection. Profound immunosuppression not only amplifies disease severity but also predisposes to hematologic complications such as immune thrombocytopenic purpura and concurrent bacterial and sexually transmitted coinfections. Early recognition, prompt re-initiation of antiretroviral therapy, and individualized use of immunomodulatory or antiviral therapies are essential to optimizing outcomes. The patient's full recovery with ART, corticosteroids, IVIG, and supportive care highlights the importance of restoring immune function as the cornerstone of management. Continued surveillance and inclusion

of immunocompromised individuals in prospective mpox studies are critical to refine treatment strategies and improve prognostic understanding.

Patient Consent and Ethical Approval: Written informed consent was obtained from the patient for publication of this case report and accompanying clinical details. All identifying information has been removed to ensure anonymity. The case was reviewed in accordance with institutional ethical standards and followed the principles outlined in the Declaration of Helsinki. Institutional Review Board (IRB) approval was not required for single-patient case reports as per institutional policy. There is no conflict of interest in this study.

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References

1. Ogoina D, Dalhat MM, Denue BA, Okowa M, Chika-Igwenyi NM, et al. (2023). Clinical characteristics and predictors of human mpox outcome during the 2022 outbreak in Nigeria: a cohort study. Lancet Infectious Diseases. 23: 1418-1428.
2. Centers for Disease Control and Prevention. (2024). U.S. mpox surveillance report. Centers for Disease Control and Prevention.
3. World Health Organization. (2023). Clinical management of mpox. World Health Organization. Retrieved from online.
4. Godeau B, Provan D, Bussel J. (2007). Immune thrombocytopenic purpura in adults. Current Opinion in Hematology. 14: 535-556.
5. Slichter SJ. (2020). Management of HIV-associated ITP. Hematology American Society of Hematology Education Program. 2020: 568-574.
6. National Institutes of Health. (2024). STOMP trial update NCT05534984. National Institutes of Health.
7. Smith TD, Riedl MA. (2024). The future of therapeutic options for hereditary angioedema. Annals of Allergy, Asthma & Immunology. 133: 380-390.
8. National Institutes of Health. (2025). Guidelines for the prevention and treatment of opportunistic infections in adults with HIV: mpox section. National Institutes of Health. Retrieved from online.
9. Centers for Disease Control and Prevention. (2025). Clinical care and treatment of mpox. Centers for Disease Control and Prevention. Retrieved from online.
10. Molina JM, Rizzardini G, Orrell C, Afani A, Calmy A, et al. (2024). Switch to fixed-dose doravirine with islatravir once daily in virologically suppressed adults with HIV-1 on antiretroviral therapy: 48-week results of a phase 3 randomised non-inferiority trial. Lancet HIV. 11: e369-e379.
11. Mitjà O, Alemany A, Marks M, Lezama Mora JI, Rodríguez-Aldama JC, et al. (2023). Mpox in people with advanced HIV infection: a global case series. Lancet. 401: 939-949.
12. World Health Organization. (2023). Clinical management of mpox. World Health Organization. Retrieved from online.
13. Martín-Iguacel R, Pericas C, Bruguera A, Rosell G, Martínez E, et al. (2023). Mpox: clinical outcomes and impact of vaccination in people with and without HIV: a population-wide study. Microorganisms. 11: 2701.
14. National Institutes of Health. (2025). Guidelines for the prevention and treatment of opportunistic infections in adults with HIV: mpox section. National Institutes of Health. Retrieved from online.
15. Suvvari TK, Ghosh A, Lopinti A, Islam MA, Bhattacharya P. (2023). Hematological manifestations of monkeypox virus and impact of human mpox disease on blood donation: what we need to know. New Microbes and New Infections. 52: 101108.
16. Bussel J, Cooper N, Boccia R, Zaja F, Newland A. (2021). Immune thrombocytopenia. Expert Review of Hematology. 14: 1013-1025.
17. Slichter SJ. (2020). Management of HIV-associated ITP. Hematology American Society of Hematology Education Program. 2020: 568-574.
18. National Institutes of Health. (2024). STOMP trial update NCT05534984. National Institutes of Health.
19. Ali R, Alonga J, Biampata JL, Kombozi Basika M, Maljkovic Berry I, et al. (2025). Tecovirimat for clade I MPXV infection in the Democratic Republic of Congo. New England Journal of Medicine. 392: 1484-1496.
20. Centers for Disease Control and Prevention. (2025). Clinical care and treatment of mpox. Centers for Disease Control and Prevention. Retrieved from online.
21. Mourad A, Alavian N, Woodhouse EW, Niehaus E, Cunningham H, et al. (2023). Concurrent sexually transmitted infection testing among patients tested for mpox at a tertiary healthcare system. Open Forum Infectious Diseases. 10: ofad381.
22. Centers for Disease Control and Prevention. (2024). STI treatment guidelines: syphilis. Centers for Disease Control and Prevention. Retrieved from online.
23. Simmons WF, Chan JD, Budak JZ, Dhanireddy S, Green ML, et al. (2023). Antibiotic prescribing patterns for bacterial superinfection of mpox: a retrospective cohort study in an urban center. Antimicrobial Stewardship & Healthcare Epidemiology. 3: e108.