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Clinical Spectrum of Cutaneous Adverse Drug Reactions in HIV Patients: A Literature Review

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ABSTRACT

Cutaneous Adverse Drug Reactions (CADRs) represent a significant clinical concern among individuals living with Human Immunodeficiency Virus (HIV), who exhibit heightened susceptibility due to profound immune dysregulation, declining CD4+ cell counts, and frequent exposure to multiple pharmacologic agents. This literature review synthesizes current evidence regarding the spectrum, risk factors, and drug triggers of CADRs in HIV-infected populations. CADRs in this group encompass a wide range of manifestations, from mild exanthematous eruptions to life-threatening reactions such as Stevens–Johnson Syndrome (SJS), Toxic Epidermal Necrolysis (TEN), Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS), and Acute Generalized Exanthematous Pustulosis (AGEP). Multiple risk determinants, including polypharmacy, co-infection with tuberculosis (TB), impaired immunological status marked by low CD4+ cell counts, and host-related factors such as age, sex, and genetic predisposition, contribute to the increased incidence and severity of CADRs in this population. Antituberculosis agents, antiretroviral therapy (particularly nevirapine-based regimens), and cotrimoxazole frequently emerge as primary drug triggers, with several studies reporting high rates of hypersensitivity reactions linked to these medications. Overall, the evidence underscores the importance of vigilant monitoring, early recognition of cutaneous reactions, and appropriate therapeutic adjustments to prevent adverse outcomes in HIV-infected patients receiving complex multidrug regimens.

Keywords: HIV; cutaneous adverse drug reaction; drug hypersensitivity; literature review

INTRODUCTION

Human Immunodeficiency Virus (HIV) is a retrovirus that invades and destroys its primary target cells, namely $CD4^+$ T lymphocytes, as well as other immune cells expressing the CD4+ receptor [1]. The progressive decline in $CD4^+$ cell counts and subsequent immunosuppression contribute to the high prevalence of cutaneous manifestations in individuals living with HIV, affecting approximately 90% of patients. Impairment of the immune system further predisposes patients with HIV, particularly those with advanced immunodeficiency, to an increased risk of developing CADR [2]. Moreover, the use of multiple pharmacological agents, including antifungals, antiparasitics, antibiotics, and antivirals administered for the prevention or treatment of opportunistic infections, as well as antiretroviral therapy (ART) employed in HIV management, can further heighten the likelihood of hypersensitivity reactions to medications [3].

Cutaneous Adverse Drug Reactions (CADRs) are defined as undesirable alterations in the structure or function of the skin, its appendages, or the mucous membranes, and encompass all drug-related cutaneous eruptions regardless of their underlying etiology [4]. The spectrum of CADR manifestations is broad and can mimic nearly any dermatologic condition, whether inflammatory or non-inflammatory in nature [5]. In individuals living with HIV, the clinical presentations may include exanthematous drug eruptions, urticaria and angioedema, erythroderma, erythema multiforme, Toxic Epidermal Necrolysis (TEN) or Stevens–Johnson Syndrome (SJS), Acute Generalized Exanthematous Pustulosis (AGEP), Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS), and vasculitis [6]. While some of these manifestations are mild and self-limiting, others may progress to severe, life-threatening complications.

A study conducted at UPIPI, Dr. Soetomo General Hospital Surabaya, reported that the incidence of Cutaneous Adverse Drug Reactions (CADRs) among patients with HIV/AIDS reached 2.35% in 2017 and 1.75% during the 2016–2017 period [7]. Another investigation from the Government General Hospital affiliated with the Government Medical College in Suryapet, Telangana, India, documented that 16.08% of patients developed CADRs between November 2017 and October 2020. These findings underscore that CADR represents a clinically significant issue, particularly among individuals with HIV/AIDS who are more susceptible to such adverse reactions [8].

This literature review seeks to delineate the spectrum of CADR in individuals living with HIV, identify the classes of medications most frequently implicated in these reactions, and examine the risk factors that contribute to their development, including immunological indicators such as $CD4^+$ cell count. Furthermore, this review aims to outline the clinical implications of CADR in HIV-infected patients, thereby providing a comprehensive framework to support early recognition, prevention strategies, and more effective clinical management.

REVIEW CONTENT

A. Pathophysiology

Cutaneous adverse drug reaction represents an undesirable or harmful reaction to a medication, which may manifest on the skin or its appendages, including the nails, hair, and glands. The skin is the organ most frequently affected by drug reactions, with an incidence of approximately 45%. To date, the precise mechanisms underlying drug-induced hypersensitivity reactions in the skin have not been fully elucidated. These reactions typically occur in individuals with a genetic predisposition, as exemplified by abacavir hypersensitivity, which is strongly associated with the HLA-B*5701 allele [9]. Two principal mechanisms have been proposed to explain drug presentation within the body: the hapten-dependent and hapten-independent pathways [10]. In patients with HIV, the frequency of drug allergies increases due to altered drug metabolism, immune dysregulation, oxidative stress, genetic susceptibility, and viral-related factors. A decline in CD4+ cell count further exacerbates the severity of these reactions [9]. Drug allergy in HIV-infected individuals is primarily driven by complex immune system dysfunction and an increased propensity for hypersensitivity reactions. Immune dysregulation characterized by heightened immune activation, a shift toward Th2-dominant responses, elevated IgE levels, and a reduction in CD4+ T lymphocytes contributes to the loss of immune tolerance toward drug antigens. These abnormalities cause medications to be recognized as foreign substances, triggering exaggerated immune responses that lead to various clinical manifestations of hypersensitivity, particularly cutaneous reactions [11]. This constellation of disturbances reflects impaired host immune regulation against antigens, ultimately contributing to the emergence of hypersensitivity reactions.

B. Clinical Manifestations

(1) Urticaria Dan Angioedema

Urticaria is a dermatologic condition characterized by edema and erythema, typically accompanied by pruritus and transient in nature, whereas angioedema refers to deeper, localized swelling of the skin or mucosal tissues, usually non-pruritic but often associated with pain or a burning sensation. The characteristic lesions of urticaria consist of rapidly appearing and disappearing circumscribed or elevated edematous areas, known as wheals, which may appear erythematous or pale depending on the degree of edema. Angioedema, in contrast, often presents with pain, is infrequently pruritic, and may persist for several days, while urticarial lesions tend to develop abruptly, rarely lasting more than 24–36 hours, and may occasionally worsen unpredictably [1]. In cases of chronic urticaria, the condition is frequently associated with autoimmune diseases, infections, and immune dysregulation, indicating that urticaria is not solely a cutaneous disorder but may also reflect underlying systemic abnormalities [12]. Angioedema involving the face, lips, or oral mucosa is generally not life-threatening; however, laryngeal angioedema constitutes a medical emergency requiring immediate airway intervention due to the potential for upper airway obstruction [13].

(2) Exanthematous Drug Eruption

Exanthematous eruptions, also commonly referred to as morbilliform or maculopapular eruptions, represent the most frequently encountered form of cutaneous drug reaction, accounting for approximately 95% of all CADR cases [1]. These eruptions are typically pruritic and often begin as macules that evolve into papules, which may subsequently coalesce into plaques; in some instances, they may progress to erythroderma. Drug-induced exanthematous eruptions characteristically present with pruritus, initially appearing on the trunk and spreading symmetrically to the extremities. Most of these reactions are classified as delayed-type hypersensitivity responses, developing several days after exposure to the offending medication [14].

(3) Fixed Drug Eruption

Fixed Drug Eruption (FDE) is characterized by the recurrence of lesions at the same anatomical site upon re-exposure to the causative medication. Clinically, FDE typically presents as well-demarcated, oval erythematous patches that may arise on various areas of the body, including the face, tongue, trunk, extremities, and genital region [15]. Some patients may report a burning sensation or pain, whereas others may experience systemic symptoms such as fever, malaise, and gastrointestinal disturbances [1].

(4) Erythema Multiforme

Erythema multiforme is an acute-onset mucocutaneous syndrome. First described by von Hebra in 1860 as *erythema exudativum multiforme*, the term refers to the characteristic pattern of lesions that develop on the skin and mucous membranes. Based on the extent of mucosal involvement, erythema multiforme is classified into erythema multiforme minor (EMm), in

which only the skin and lips are affected, and erythema multiforme major (EMM), in which one or more mucous membranes are involved [16]. The lesions of erythema multiforme typically present as erythematous papules or plaques with a well-defined, concentric pattern, and they may persist for a week or longer. Their size varies from a few millimeters up to approximately 3 cm, and they may expand over the course of 24–48 hours. Some patients report a burning sensation or pruritus in the affected areas [1].

(5) Vasculitis

Vasculitis refers to inflammation of the blood vessel walls, a process that can give rise to a wide range of clinical manifestations. Cutaneous involvement is frequently the initial presentation of vasculitis [17]. The dermatologic signs observed in patients with vasculitis vary depending on the size of the affected vessels. The most common clinical manifestation of small-vessel vasculitis is palpable purpura. These purpuric lesions typically appear symmetrically on the lower extremities or the dorsal aspects of the feet due to increased hydrostatic pressure. Purpura often resolves with residual hyperpigmentation, although scarring is uncommon. The lesions may be asymptomatic, pruritic, or associated with a burning sensation [18].

(6) Drug Rash with Eosinophilia and Systemic Symptoms (DRESS)

Drug Rash with Eosinophilia and Systemic Symptoms (DRESS) is a severe drug-induced reaction that carries a significant risk of mortality. DRESS is clinically distinct from other forms of drug hypersensitivity. In affected patients, symptoms typically emerge several weeks to several months after exposure to the offending agent. The most common clinical manifestations include widespread cutaneous eruptions, fever, and involvement of internal organs such as the liver, kidneys, or lungs [19]. The rash most frequently presents as a symmetric maculopapular eruption distributed across the trunk and extremities, often covering more than 50% of the body surface area. The lesions tend to exhibit a darker hue compared with standard morbilliform eruptions. Symptomatically, patients often report pruritus or pain accompanied by a burning sensation [20].

(7) Stevens-Johnson Syndrome (SJS)

Stevens–Johnson Syndrome (SJS) is a life-threatening acute mucocutaneous reaction characterized by extensive necrosis and detachment of the epidermis and mucosal epithelium. The precise etiology of SJS remains incompletely understood; however, drug exposure is considered the most significant contributing factor. More than 100 medications have been reported as potential triggers of SJS [21]. The cutaneous lesions typically present as widespread erythematous macules with central necrosis, purulent blistering, and subsequent epidermal detachment. These lesions frequently coalesce and often exhibit a positive Nikolsky sign, in which gentle lateral pressure results in further epidermal sloughing. Targetoid lesions are commonly observed and arise from central epidermal necrosis. Over time, the affected skin undergoes widespread desquamation, leading to extensive superficial ulceration and loss of the epidermal barrier [22].

(8) Toxic Epidermal Necrolysis (TEN)

Toxic Epidermal Necrolysis (TEN) is a life-threatening acute mucocutaneous reaction characterized by extensive necrosis and detachment of the epidermis and mucosal epithelium. In 1956, Lyell described patients who experienced epidermal loss due to necrosis and subsequently introduced the term TEN [1]. The earliest lesions typically manifest as atypical target lesions or purulent macules on the face, upper trunk, and extremities. As the disease progresses, these lesions enlarge and coalesce, rapidly evolving into vesicles or bullae. The epidermis may detach, and gentle lateral pressure can induce further separation of the epidermis from the dermis, known as a positive Nikolsky sign. In TEN, the skin barrier is profoundly compromised, predisposing patients to infections and sepsis, which can ultimately lead to multiorgan failure. This represents the most common cause of mortality in affected individuals [23].

(9) Acute Generalized Exanthematous Pustulosis (AGEP)

Acute Generalized Exanthematous Pustulosis (AGEP) typically emerges within 24–48 hours following the administration of certain medications, although some agents may induce reactions as late as 10–22 days afterward. Prodromal symptoms include fever (>38°C), malaise, and leukocytosis, predominantly neutrophilia with eosinophilia present in approximately 30% of cases. The cutaneous lesions are characterized by edematous erythema accompanied by numerous sterile, non-follicular, pruritic pustules, most commonly distributed on the trunk and intertriginous areas [1,24]. Systemic involvement may also occur, manifesting as elevated liver enzymes, steatosis or hepatomegaly, impaired renal function, and pleural effusion with associated hypoxemia. In AGEP, multisystem involvement is possible, with clinical features such as hypotension, tachycardia, tachypnea, and fever. Laboratory findings typically reveal marked neutrophilic leukocytosis, mild eosinophilia, and increases in aspartate transaminase and alanine aminotransferase, suggestive of acute kidney injury and varying degrees of hepatic dysfunction [25].

C. Risk Factors

(1) Polypharmacy

Polypharmacy refers to the condition in which an individual is concurrently taking multiple medications [26]. Polypharmacy can precipitate adverse drug reactions (ADR), including cutaneous adverse drug reactions (CADR). The theoretical mechanisms underlying this phenomenon include pharmacodynamic and pharmacokinetic interactions among medications, increased metabolic burden, and the limited capacity of organs such as the liver and kidneys to process multiple drugs simultaneously [27]. A study conducted in Uganda reported that approximately 59.5% of patients receiving HAART were also taking additional medications, thereby

increasing the likelihood of clinically significant drug interactions [28].

(2) Co-Infection

Co-infection, particularly HIV and tuberculosis (TB), has been identified as a significant risk factor for the development of CADR. A cohort study from Thailand demonstrated that HIV–TB co-infected patients receiving anti-tuberculosis therapy experienced a notably high incidence of CADRs, underscoring how therapeutic complexity, multiple drug exposures, and altered physiological states contribute to increased susceptibility to cutaneous reactions. Moreover, the profound immune dysregulation characteristic of HIV infection may further potentiate hypersensitivity responses to anti-TB medications, thereby amplifying the overall risk of dermatologic adverse events. Additional evidence from the same retrospective cohort showed that CADRs occurred in approximately 15–16% of HIV–TB co-infected individuals undergoing standard TB treatment [29].

(3) $CD4^+$ Count

Previous studies have shown that cutaneous drug allergies are relatively common among individuals living with HIV, with a reported prevalence of 16.9%. Most cases were observed in male patients and were predominantly associated with low $CD4^+$ counts [30]. Low $CD4^+$ cell count has been found to be significantly associated with the occurrence of adverse drug reactions (ADR), as the majority of patients who developed ADRs had $CD4^+$ levels below 200 cells/mm^3[28]. $CD4^+$ T lymphocytes act as central regulators of the immune system and comprise two major subsets, Th1 and Th2, which are distinguished by the cytokines they produce. Th1 cells secrete IFN-γ and IL-2 to support cellular immune responses, whereas Th2 cells release IL-4, IL-5, IL-6, and IL-10, which promote humoral immunity and stimulate B-cell activation. In individuals infected with HIV, declining $CD4^+$ levels not only reflect the degree of immunodeficiency but are also associated with alterations in adaptive immune response patterns [31].

As HIV infection progresses, a shift from Th1- to Th2-dominant immunity occurs, characterized by reduced Th1 cytokines such as IFN-γ and IL-2, along with increased Th2 cytokines such as IL-4 [32]. This immunologic shift enhances B-cell production of IgE, thereby increasing susceptibility to allergic reactions, including drug hypersensitivity [33]. Collectively, these findings highlight that low $CD4^+$ count and dysregulated immune balance in HIV infection play crucial roles in predisposing patients to cutaneous adverse drug reactions.

(4) Gender

The distribution of sex among HIV patients experiencing cutaneous drug allergies has shown no significant difference between males and females [34]. However, a study conducted by Vellaisamy et al. in 2020 reported that individuals with HIV/AIDS who developed drug-induced allergic reactions were predominantly male. In contrast, findings from Kouotou et al. (2017) demonstrated a different pattern, in which the majority of patients presenting with drug allergies were female, accounting for 34 out of 41 cases (82.9%). These discrepancies suggest that sex-related variations may influence the occurrence of cutaneous adverse drug reactions through factors such as body fat percentage, hormonal influences, and differences in enzymatic activity [35].

Hormonal factors play a critical role in modulating immune responses. In women, estrogen enhances adaptive immune activity, including increased antibody production, whereas androgens in men tend to suppress immune activation by reducing pro-inflammatory cytokine production and promoting anti-inflammatory pathways [36]. Additionally, the higher body fat percentage typically observed in women may affect the distribution of lipophilic drugs, prolonging exposure of immunogenic metabolites to the immune system and potentially increasing the risk of hypersensitivity reactions. Conversely, men, who generally have lower body fat, may distribute and eliminate drugs more rapidly, resulting in shorter exposure to immunoreactive metabolites. These differences are further reinforced by genetic factors, as the X chromosome carries numerous genes involved in immune regulation; thus, women, who possess two X chromosomes, often exhibit stronger immune responses than men [36].

(5) Age

Previous research conducted by Li et al. in 2016 demonstrated that HIV/AIDS patients experiencing drug-induced allergic reactions were more frequently found in the ≤50-year age group [34]. Similar findings were reported by Kim et al. in a study conducted between 2003 and 2006, which showed that drug hypersensitivity reactions among individuals with HIV/AIDS occurred predominantly in those aged 30–50 years [37]. In contrast, a study by Armeinesya et al. in 2018 found that hypersensitivity reactions to cotrimoxazole were distributed almost evenly across all age groups, indicating that age does not play a major role in determining the risk of developing allergic reactions to this medication [9].

These variations suggest that age is not a singular determinant in the development of CADR. Other factors such as immunological status, comorbidities, polypharmacy, and genetic variations influencing drug metabolism likely contribute more substantially to the susceptibility of HIV-infected individuals to drug hypersensitivity.

D. Drug Triggers

(1) Antituberculosis

The study conducted by Widhani et al. provides a comprehensive overview of CADR among 454 HIV infected patients undergoing antituberculosis therapy. Within this cohort, 10.6% of patients developed CADR, a significant proportion given the therapeutic complexity encountered in TB–HIV co-infected individuals. The majority of patients were

also receiving concomitant medications, including cotrimoxazole (79.2%), antiretroviral therapy (14.6%), and toxoplasmosis treatment (18.8%), which increases the potential for drug interactions that may precipitate CADR. The most frequently observed manifestation was maculopapular rash (66.7%), followed by more severe dermatologic reactions such as erythema multiforme, Stevens–Johnson Syndrome (SJS), and toxic epidermal necrolysis (TEN). These findings underscore the heightened susceptibility of HIV patients on multiple concurrent therapies to developing cutaneous reactions, and demonstrate that antiretrovirals and opportunistic infection treatments alongside antituberculosis play a substantial role in triggering CADR [38].

Meanwhile, the study by Katran et al. examined hypersensitivity profiles in 148 patients and revealed that 63.5% experienced type I hypersensitivity reactions, whereas 36.7% exhibited type IV reactions, with some individuals presenting features of both. Urticaria was identified as the most common type I manifestation (38.5%), whereas maculopapular eruptions represented the predominant type IV presentation (24.3%). Among all cases, the causative drug was successfully identified in 35.2% of patients, with pyrazinamide being the most frequently implicated agent (48.1%), followed by rifampicin, isoniazid, and ethambutol. These results highlight the considerable potential of anti-tuberculosis medications to induce various forms of hypersensitivity reactions, both immediate and delayed. Consequently, recognizing reaction patterns and accurately identifying the offending agent are crucial steps in implementing safe and effective management strategies [39].

(2) Antiretroviral

The study conducted by Wibisono et al. demonstrated substantial variability in antiretroviral (ARV) regimen involvement among 28 patients identified as experiencing CADR. For cases presenting with morbilliform drug eruptions, the combination of Duviral (Zidovudine + Lamivudine) and Nevirapine was the most frequently implicated regimen, accounting for 7 cases (41.4%). This finding indicates a significant contribution of nevirapine in triggering cutaneous reactions in the HIV population, consistent with its well-recognized profile as a medication associated with a high risk of hypersensitivity. Additionally, the combination of Duviral with Efavirenz and the fixed-dose regimen of Tenofovir + Lamivudine + Efavirenz each contributed to 5 cases (29.4%), underscoring that CADR are not confined to a single class of ARV agents. In severe manifestations such as Stevens–Johnson Syndrome (SJS), the Duviral–Nevirapine regimen again represented the predominant cause, responsible for 6 cases (54.4%), followed by the Tenofovir + Lamivudine + Nevirapine regimen, which accounted for 5 cases (45.5%). The dominance of nevirapine-based regimens in both morbilliform eruptions and SJS reinforces the evidence that nevirapine carries a particularly high risk of inducing significant cutaneous reactions, especially among HIV patients with increased immunological vulnerability [7].

Findings from Armeinesya et al. further support this pattern, demonstrating a markedly high frequency of nevirapine-induced cutaneous reactions among HIV patients. Among 28 individuals who developed CADRs, the majority, representing 104 events or 82.5% of all diagnosed drug eruptions, were attributed to nevirapine. This proportion was substantially higher than that associated with other ARV agents, including zidovudine (0.8%), tenofovir (2.4%), and efavirenz (14.3%). The predominance of nevirapine as the principal cause of CADRs highlights its strong immunologic potential to induce hypersensitivity reactions, consistent with international evidence describing its association with delayed-type reactions. Maculopapular rash constituted the most frequent clinical manifestation (89.7%), aligning with the typical spectrum of CADRs observed in HIV populations. Furthermore, severe reactions such as SJS were also documented, although at a lower frequency (8.7%), indicating that nevirapine may provoke not only mild to moderate eruptions but also life-threatening cutaneous conditions [9].

(3) Cotrimoxazole

Based on research data conducted by Maharani et al, Cotrimoxazole is the drug most commonly associated with CADR incidents. Of all cases analysed, Cotrimoxazole accounted for 25 cases or 27.8%, making it the leading cause of CADR compared to other drugs such as Efavirenz and Nevirapine. These findings confirm that the use of Cotrimoxazole in HIV patients requires special caution, given the high proportion of skin reactions it causes [30].

CONCLUSION

CADR constitute a prevalent and clinically important complication in individuals living with HIV, driven by multifactorial mechanisms involving immune dysregulation, polypharmacy, co-infections, and specific drug exposures. The literature consistently demonstrates that declining $CD4^+$ counts, Th1-to-Th2 immune shift, and heightened IgE-mediated responses significantly increase vulnerability to drug hypersensitivity. Among the pharmacologic agents most frequently implicated, antituberculosis medications, nevirapine-containing antiretroviral regimens, and cotrimoxazole account for the majority of reported cases, ranging from mild eruptions to severe, life-threatening reactions such as SJS and TEN. Although demographic variables such as age and sex show inconsistent associations, host genetic factors and metabolic variations further modulate individual risk. Given the substantial morbidity associated with CADR in HIV infected populations, heightened clinical awareness, early diagnostic evaluation, timely withdrawal of suspected agents, and individualized treatment strategies are essential for reducing complications. Strengthening pharmacovigilance and integrating immunologic risk assessment into therapeutic decision-making may further enhance patient safety and improve outcomes in this high-risk group.

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