

REVIEWS

EPIDEMIOLOGY OF DRUG-INDUCED DERMATOSES: PREVALENCE, RISK FACTORS, AND CLINICAL OUTCOMES

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ABSTRACT

INTRODUCTION: Drug-induced dermatoses are adverse skin reactions triggered by medications, ranging from mild rashes to severe conditions like Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN). Understanding their prevalence, risk factors, and clinical presentations is essential for effective prevention and management. This study investigates the epidemiology, types, and risk factors associated with drug-induced dermatoses, providing insights for healthcare providers.

MATERIALS AND METHODS: A comprehensive review of 125 studies (2000–2024) was conducted using databases such as MEDLINE, Embase, and the Cochrane Central Register of Controlled Trials. Data from literature reviews, observational studies, clinical trials, and case reports were analyzed to determine the prevalence, implicated drugs, and demographic or clinical factors influencing these reactions.

RESULTS: Drug-induced dermatoses include a spectrum of reactions from mild to life-threatening. Commonly implicated drugs are antibiotics, NSAIDs, and antiepileptics, with prevalence rates of 0.5% to 5% among high-risk drug users. Severe reactions include SJS/TEN, drug reactions with eosinophilia and systemic symptoms (DRESS), and acute generalized exanthematous pustulosis (AGEP). Significant risk factors include genetic predispositions (e.g., HLA-B*1502 for carbamazepine), age extremes, pre-existing conditions such as autoimmune diseases or HIV, and factors related to the type, dose, and duration of drug exposure.

CONCLUSION: Healthcare providers should identify high-risk drugs and implement measures such as genetic screening to minimize adverse reactions. Improving diagnostics, clinicians' education, and antibiotic stewardship is vital. Further research into genetic biomarkers and personalized medicine could reduce the clinical and societal impact of these reactions, enhancing patient care and outcomes.

Keywords: *drug-induced dermatoses, epidemiology, adverse drug reactions, Stevens-Johnson syndrome, toxic epidermal necrolysis, drug reaction with eosinophilia and systemic symptoms, antibiotics*

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INTRODUCTION

The epidemiology of drug-induced dermatoses represents an important aspect of contemporary medical science. This article aims to examine current trends in the prevalence and frequency of dermatological reactions induced by drugs. Through analysis of available literature and epidemiological data, the article will summarize the main classes of drugs as-



sociated with such problems, as well as the factors influencing the risk and severity of the reactions. The provided information aims to emphasize the importance of observation, registration, and early intervention management in maintaining the safety and effectiveness of drug therapy.

As the largest organ of the human body, the skin is subject to various reactions to medical substances. The frequency of these reactions can reach up to 10% among hospitalized patients and can be observed in 1-3% of those taking a combination of different medications (1). Although the majority of drug-induced dermatoses are mild and self-resolving, between 2

Table 1. Classification of drug-induced dermatoses with their description.

Classification of Drug-Induced Dermatoses	Description
Common Drug Eruptions	
Morbilloform (Exanthematous) Drug Eruption	Widespread red rash resembling measles
Urticaria and/or angioedema	Swelling and hives; rarely progresses to anaphylaxis
Severe Cutaneous Adverse Reactions	
Drug hypersensitivity syndrome	Severe immune response involving skin and other organs
Drug reactions with eosinophilia and systemic symptoms	Involves eosinophilia with fever, skin rash, and multi-organ involvement
Stevens-Johnsons syndrome-/toxic epidermal necrolysis	Life-threatening conditions with skin and mucosal detachment
Erythroderma	Widespread red scaling of the skin, often accompanied by systemic symptoms and high risk of infection and fluid loss
Other Cutaneous Adverse Reactions	
Acute generalized exanthematous pustulosis	Sudden widespread pustular rash
Generalized pustular psoriasis	Psoriasis flare with widespread pustules
Serum sickness	Fever, hives, joint pain, and lymph node swelling
Hypersensitivity vasculitis	Immune-mediated small vessel inflammation, causing palpable purpura
Fixed drug eruption	Recurring blisters and plaques at the same site
Lichenoid drug eruption	Lichen planus-like lesions
Drug-Induced photosensitivity	Reactions include phototoxicity (sunburn) or photoallergy (eczema on sun-exposed areas)
Bullous drug eruptions	Immunobullous reactions that resolve upon drug withdrawal
Drug-induced lupus erythematosus	Lupus-like autoimmune response triggered by drugs
Additional Drug-Induced Skin Conditions	
Drug-induced skin pigmentation	Changes in skin color due to medication
Triggered skin conditions	Exacerbation of conditions like psoriasis (e.g., by lithium) or eczema (e.g., by retinoids)
Systemic contact dermatitis	Widespread rash from a sensitizing drug
Allergic/photocontact dermatitis	Allergic skin reactions or sun-induced reactions
Hair changes	Increase (hypertrichosis) or loss (alopecia) of hair
Nail dystrophy or pigmentation	Abnormal nail growth or color changes

and 6.7% of cases can progress to potentially dangerous and life-threatening conditions (2).

DRUG-INDUCED DERMATOSES

The complexity of adverse drug reactions arises from their diverse nature, which can mimic various dermatological conditions. Currently, there are 30 different patterns of skin reactions to drugs documented, ranging from mild dermatitis to serious, life-threatening conditions. In (Table 1), a detailed classification of the types of drug-induced dermatoses can be found. Among the most commonly encountered drug-induced dermatoses are maculopapular eruptions, urticaria, angioedema, fixed drug eruptions, and erythema multiforme. Meanwhile, rarer but more severe patterns include erythroderma, drug reactions with eosinophilia, as well as SJS and TEN (1).

According to data from the Al-Rashed Allergy Center in Kuwait, following a conducted observational, prospective study based on patient data registry for a period of 12 years (from 2007 to 2019), it has been found that anti-inflammatory and analge-

sic drugs account for 39.22% of all confirmed drug allergies, with antibiotics following closely at 38.1% (where β -lactam antibiotics constitute 73.98% of the total antibiotics and 28.21% of all drugs). Anesthetics contribute 1.8%, while radiographic contrast agents contribute 0.31%. The most common symptoms among confirmed patients include urticaria (57.80%), angioedema (42.50%), respiratory symptoms (47.60%), and erythema (33.60%). Symptoms of anaphylaxis or anaphylactic shock are reported by 284 patients (44.00%) among confirmed cases (3).

Practically every pharmacological agent can lead to the development of drug-induced dermatoses, with antibiotics, nonsteroidal anti-inflammatory drugs, and antiepileptic drugs being among the most common pharmacological agents associated with such reactions (1). Table 2 provides a comprehensive overview of various types of medications and the drug-induced dermatoses they can cause.

Antibiotics

Antibiotics are a significant risk factor for the development of SJS and TEN, which are the most severe forms of drug hypersensitivity with mortality

Table 2. Drug classes with examples of associated dermatologic reactions.

Drug Class	Examples of Associated Dermatologic Reactions
Antibiotics	Morbilloform rash, urticaria, photosensitivity, fixed drug eruption, SJS, TEN
Non-steroidal anti-inflammatory drugs (NSAIDs)	Urticaria, angioedema, photosensitivity, fixed drug eruption, SJS, TEN
Antiepileptics	DRESS, SJS, TEN, photosensitivity, morbilliform rash
Psychotropic drugs (e.g., lithium)	Exacerbation of psoriasis, acne, hair changes
Anticoagulants	Skin necrosis, urticaria, purpura
ACE inhibitors	Angioedema, maculopapular rash, photosensitivity
Calcineurin inhibitors	Skin cancer risk, photosensitivity, drug-induced acne
Antifungal agents	Urticaria, fixed drug eruption, photosensitivity, SJS
Anti-tubercular drugs	Morbilloform rash, fixed drug eruption, photosensitivity, drug-induced lupus
Antiretroviral drugs (HIV therapy)	Photosensitivity, hyperpigmentation, morbilliform rash, SJS, TEN
Xanthine oxidase inhibitors (e.g., allopurinol)	SJS, TEN, DRESS, maculopapular rash, erythema, pruritus
Chemotherapy agents	Acral erythema, alopecia, photosensitivity, lichenoid reactions, nail changes
Biologics	Psoriasiform reactions, eczema, injection site reactions, systemic drug reactions

rates up to 50%. According to data from MEDLINE and Embase databases in 2022, experimental and observational studies have been conducted describing the risk of SJS/TEN. Among antibiotics associated with SJS/TEN, sulfonamides are associated with 32% of cases, followed by penicillins at 22%, cephalosporins at 11%, fluoroquinolones at 4%, and macrolides at 2% (4). Ampicillin and amoxicillin, in conjunction with clavulanic acid, are assuming an increasingly significant role in adverse drug reactions associated with beta-lactam antibiotics. Fluoroquinolones are primarily associated with anaphylactic and photosensitivity reactions (5). Glycopeptides are commonly associated with DRESS. Acute generalized exanthematous pustulosis is primarily attributed to antibiotics like pristinamycin and aminopenicillins, with quinolones, (hydroxy-)chloroquine, and sulfonamides emerging as notable causative agents (6). These results underscore the importance of antibiotic stewardship, clinician education and awareness, as well as risk-benefit assessment for the choice and duration of antibiotics (4).

Non-Steroidal Anti-Inflammatory Drugs (NSAIDs)

Adverse drug reactions are a major source of morbidity and frequent medical consultations. NSAIDs rank as the second most common class, after antibiotics—particularly beta-lactam antibiotics such as penicillins and cephalosporins—linked to hypersensitivity reactions. A wide array of cutaneous manifestations, including morbilliform rashes, urticaria, and angioedema, are commonly associated with NSAID use, especially with agents like ibuprofen. Specifically in cases of allergic reactions to NSAIDs, it is essential to consider pseudoallergic reactions in the diagnostic process (pseudoallergy is a reaction that mimics an immune-mediated response by stimulating immune cells such as mast cells or immunoglobulin (IgE). This group also contains anaphylactoid reactions that mimic IgE-mediated anaphylaxis reaction (5). Other dermatologic conditions, such as vasculitis, SJS and TEN, photosensitivity, fixed drug eruptions, livedo-like dermatitis, linear drug eruptions, lichenoid drug eruptions, acute generalized exanthematous pustulosis, eczematous eruptions, contact dermatitis, and drug-induced pemphigoid, are also well-documented (7). A recent review of 206 NSAIDs linked to suspected

cutaneous adverse reactions found ibuprofen to be the most frequently implicated, responsible for 25.7% of cases. The primary reactions observed were angioedema and urticaria, constituting 54.4% of cases, with ibuprofen as the leading suspect. The second most common reaction type was maculopapular eruption (26.2%), primarily associated with celecoxib. Fixed drug eruptions were predominantly linked to NSAIDs in the enolic acid group. Additionally, five cases of drug hypersensitivity syndrome were identified, along with five cases of SJS or TEN (8).

Antiepileptic drugs (AEDs)

Based on data compiled from reputable sources, including the Cochrane Central Register of Controlled Trials, MEDLINE, ISI Web of Knowledge, and the Korea Institute of Drug Safety and Risk Management, it has been established that antiepileptic drugs (AEDs) represent a significant subset of medications implicated in the onset of severe drug-induced dermatoses. These dermatoses, which encompass conditions such as SJS, TEN, DRESS, AGEP, and DIHS, pose significant clinical challenges (9). Notable AEDs implicated in these adverse reactions include phenytoin, carbamazepine, valproic acid, oxcarbazepine, levetiracetam, phenobarbital, primidone, zonisamide, rufinamide (10), and lamotrigine. In individuals experiencing hypersensitivity or allergic responses to antiepileptic drugs, a substantial level of cross-reactivity is observed, ranging between 40% to 80% (9). Furthermore, a comparative analysis between aromatic and non-aromatic AEDs reveals that aromatic AEDs are more strongly associated with severe drug eruptions. Among these eruptions, DRESS stands out as the most commonly reported adverse event, with lamotrigine emerging as the predominant causative AED (11).

Antipsychotic Medications

Antipsychotic medications can cause skin reactions in about 5% of patients, with most cases being mild and manageable, such as rashes, changes in skin pigmentation, and photosensitivity. These reactions, while usually not serious, can impact patient adherence to treatment. Rarely, severe skin reactions such as SJS and TEN may occur, requiring immediate discontinuation of the medication. Olanzapine, for example, is known for its metabolic side effects but can also rarely cause reversible skin lesions that typically

resolve upon stopping the drug. Careful monitoring, early recognition of serious symptoms, and switching medications when necessary are key to effectively managing these side effects (12).

Anticoagulant Medications

Skin reactions can occur during anticoagulant therapy with agents such as coumarin derivatives, unfractionated and low molecular weight heparins, heparinoids, danaparoid, and hirudins. These reactions range from localized allergic responses to skin necrosis. Diagnostic tests, including intracutaneous testing, can confirm allergies and potential cross-reactions. Although rare, necrosis induced by coumarin and heparin has serious implications due to its high morbidity and occasional mortality; therefore, cutaneous tests are not advised for these patients. Future anticoagulant options may involve synthetic thrombin inhibitors and Factor Xa inhibitors, like fondaparinux (13).

Angiotensin-Converting Enzyme Inhibitors

Angiotensin-converting enzyme (ACE) inhibitors are a class of medications widely prescribed for the management of hypertension and heart failure, working by relaxing blood vessels and thereby reducing the workload on the heart. While highly effective, ACE inhibitors can induce a variety of adverse effects, particularly dermatologic reactions, which warrant careful monitoring. These reactions may include life-threatening angioedema, pruritus, bullous eruptions, urticaria, generalized rashes, photosensitivity, and alopecia. Although skin rashes are commonly associated with captopril, such cutaneous reactions can occur with any ACE inhibitor (14). Up to 15% of patients treated with captopril experience skin rashes within the first few weeks, with reactions that include maculopapular, urticarial, pityriasis rosea-like, psoriasis-form, and lupus erythematosus-like presentations. Patch testing has proven clinically useful in diagnosing adverse cutaneous drug reactions, showing positive results in about one-third of cases in a study of captopril reactions. Patch testing across multiple ACE inhibitors suggests a lack of cross-sensitivity, allowing for substitution within the drug class. Oral provocation tests are sometimes required when patch tests are inconclusive. Captopril's unique sulfhydryl group is linked to positive patch test reactions, while studies confirm that patients

sensitized to captopril may tolerate other ACE inhibitors, such as lisinopril and enalapril. Allergen testing can help identify which specific ACE inhibitors to avoid, rather than prohibiting the entire drug class (15).

Calcineurin Inhibitors

Topical calcineurin inhibitors commonly cause transient local skin irritation, such as burning, pruritus, and erythema, which typically resolve within a month. Other reported side effects include allergic contact dermatitis, folliculitis, and reactivation of infections like herpes. Despite concerns raised from animal studies, there is no conclusive evidence linking these medications to an increased risk of malignancies such as skin cancer. Oral calcineurin inhibitors (tacrolimus and ciclosporin) can lead to gingival hyperplasia, hypertrichosis, pruritus, acne, and an increased risk of cutaneous squamous cell carcinoma, particularly with higher doses (16).

Antifungal Medications

Cutaneous adverse drug reactions (CADRs) to antifungal medications are increasingly observed, although these drugs are generally considered safe. Common antifungals associated with CADRs include terbinafine, itraconazole, griseofulvin, and fluconazole. Reactions vary widely from mild rashes and itching to severe cases like SJS, TEN, and DRESS. Terbinafine is the most frequently implicated, causing reactions such as pustular eruptions, AGEP and subacute cutaneous lupus erythematosus (SCLE). Subacute cutaneous lupus erythematosus cases with terbinafine may involve the formation of antinuclear antibodies, likely due to the drug's keratophilic properties. Itraconazole may cause maculopapular rashes, urticaria, and angioedema, with reactions typically subsiding upon cessation. Griseofulvin has been linked to severe hypersensitivity reactions, including serum sickness-like reactions and DRESS, with symptoms like purpuric rashes, fever, and lymphadenopathy that may require corticosteroid treatment. Notably, CADRs to these drugs seem idiosyncratic rather than dose-dependent, with genetic factors potentially playing a role. Given the varied and sometimes serious nature of CADRs, dermatologists should counsel patients on potential reactions and report cases to monitor trends. There is an observed increase in antifungal CADRs, especially to

terbinafine, highlighting the need for ongoing monitoring and reporting (17).

Anti-Tubercular Agents

Tuberculosis remains a significant global health concern and a leading cause of mortality worldwide. Treatment with anti-tubercular agents can result in a range of adverse effects, including dermatological reactions. The most frequently observed cutaneous manifestation was maculopapular rash, which accounted for 42.5% of cases. Ethambutol emerged as the most commonly implicated drug, responsible for 45% of the reactions, followed by other first-line anti-tubercular medications (rifampicin, isoniazid, pyrazinamide, ethambutol, and streptomycin). Re-challenge with these drugs led to the development of multiple drug hypersensitivity in 25% of the patients, indicating a notable risk of recurrent reactions in such individuals (18).

HIV Medications

Certain HIV medications, such as efavirenz and darunavir, can cause skin rashes. EFV-related rashes usually appear within two weeks of treatment and are characterized by pink or red patches, while DRV rashes tend to develop later and often present as darker, red or purple blisters. These rashes may indicate hypersensitivity, with severe cases involving symptoms like difficulty breathing or dizziness, requiring urgent care. Additionally, SJS, a serious hypersensitivity reaction, has been associated with HIV drugs like nevirapine, causing fever, painful blisters, and flu-like symptoms (19).

Allopurinol

Allopurinol is associated with serious cutaneous adverse reactions, including DRESS syndrome, SJS, and TEN. A study from the Campania regional Center of Pharmacovigilance analyzed 108 cases where allopurinol was a suspected drug. The findings revealed that older adults, with a median age of 71, and women, who accounted for 53.7% of cases, were most commonly affected. Serious skin reactions made up 52% of the reports, including DRESS (5.4%), SJS (14.3%), and TEN (46.4%). Most of these cases (75%) required hospitalization, and 46% resulted in unfavorable outcomes. The study highlights the significant risk of severe skin reactions with allopurinol, particularly in elderly patients, often necessi-

tating medical intervention and frequently leading to poor outcomes (20).

Chemotherapeutic Drugs

Skin, hair, and nails are frequently affected by targeted chemotherapeutic drugs because of rapid cell turnover at these sites. In a clinico-epidemiological study based on National Institutes of Health, 18 different dermatological side effects were observed in patients undergoing chemotherapy. The most common was papulopustular eruptions, affecting 26.25% of patients, with some cases linked to PRIDE complex (papulo-pustular eruptions and/or paronychia, regulatory abnormalities of hair growth, itching, and dryness due to EGFR inhibitors like cetuximab and gefitinib). Other prominent skin effects included hand-foot syndrome in 20% of patients, commonly associated with sorafenib and capecitabine, along with rarer conditions such as lichenoid eruptions and pigmentation changes. Nail abnormalities appeared in 27.5% of patients, predominantly melanonychia, caused by drugs such as taxanes and platinum-based treatments. Additional nail issues included Beau's lines, Muehrcke's lines, and onycholysis. Most of these adverse reactions were classified as probable or possible in causation, underlining the heightened vulnerability of rapidly renewing skin, hair, and nails to targeted chemotherapy drugs (21).

Biologic Drugs

Biologic drugs, particularly TNF inhibitors (TNF-I), are linked to various skin reactions, including psoriasis, vasculitis, lupus-like syndromes, and infections. Some reactions, like injection site irritation, are directly related to drug administration, while others, such as psoriasis and vasculitis, may develop independently. Notably, 80% of these skin conditions are new rather than exacerbations of pre-existing issues, and severe reactions have led to TNF-I therapy withdrawal in about 26% of rheumatoid arthritis patients. TNF-I drugs are not linked to an increased risk of skin cancers. TNF-I drugs can also trigger neutrophilic dermatoses, such as Sweet syndrome and pyoderma gangrenosum, characterized by neutrophilic infiltration without infection. Other drugs linked to ND include granulocyte colony-stimulating factor and certain antibiotics and immunosuppressants (22).

Non-Allergic Hypersensitivity Adverse Drug Reactions

Non-allergic hypersensitivity adverse drug reactions (NAHRs) are drug-induced reactions that mimic allergic responses but do not involve the immune system's specific activation against the drug. Unlike true allergic reactions, NAHRs are not mediated by immunoglobulin E (IgE) antibodies or other immune mechanisms associated with hypersensitivity. In addition to antibiotics and NSAIDs, other commonly utilized drugs or substances that can lead to non-allergic hypersensitivity adverse drug reactions (ADR) include radiocontrast agents (such as gadolinium, iopromide), local anesthetics (like bupivacaine), opioids (such as morphine), curare derivatives (like rocuronium), preservatives (such as benzoate), and coloring agents (like yellow-orange S (23).

RISK FACTORS FOR DRUG-INDUCED DERMATOSES

The development of drug-induced dermatoses is influenced by a variety of host-related and drug-related factors. These factors can either increase the likelihood of an individual developing skin reactions or influence the severity of these reactions. Below is a more detailed breakdown of the major risk factors:

1. Individual Sensitivity and Pre-existing Conditions

Certain individuals are inherently more likely to develop skin reactions to medications due to genetic predispositions, immune system status, and previous allergic reactions.

- ◆ **Genetic sensitivity:** Specific genetic factors, including HLA genotypes (human leukocyte antigen types), are increasingly recognized as key contributors to drug-induced dermatoses. For example, individuals with HLA-B*1502 are at a significantly higher risk for carbamazepine-induced SJS and TEN, particularly in Asian populations (e.g., Han Chinese, Thais, and Indians). Similarly, HLA-B*5801 has been strongly associated with allopurinol-induced SJS/TEN in East Asian and European populations.
- ◆ **Previous allergic reactions and atopy:** Individuals who have had previous allergic reactions to certain drugs, foods, or substances are at increased risk of developing drug-induced

dermatoses. People with a history of drug allergies, food allergies, or hay fever may experience more severe reactions when exposed to the same or similar substances. Patients with a history of atopic dermatitis, asthma, or other allergic conditions are more sensitive to drug reactions

2. Drug-Related Factors

The type, dose, duration, and route of administration of a drug are key contributors to the likelihood of developing drug-induced dermatoses.

- ◆ **Type of drug:** Certain classes of drugs are more likely to cause skin reactions. Antibiotics (especially penicillins, sulfonamides, and cephalosporins), anticonvulsants (like carbamazepine, phenytoin, lamotrigine), and allopurinol are commonly associated with cutaneous drug eruptions, including exanthematous rashes, SJS, and TEN. NSAIDs antidepressants, and antipsychotics can also cause skin reactions, although they tend to be less severe.
 - ◆ **Dose and duration:** The dose and duration of treatment with certain drugs can influence the risk of developing skin reactions. Higher doses and prolonged usage increase the likelihood of sensitization and the onset of adverse cutaneous drug reactions. Drugs taken over long periods (e.g., anticonvulsants or antibiotics) are more likely to cause delayed reactions.
 - ◆ **Route of administration:** Parenteral administration (injections, IV infusions) is more likely to cause skin reactions than oral medications. Parenteral drugs tend to induce faster and more intense immune responses because they bypass the digestive system and enter the bloodstream directly.
 - ◆ **Drug interactions:** Polypharmacy (using multiple drugs simultaneously) increases the risk of drug-drug interactions, which can either amplify skin reactions or alter drug metabolism. For example, certain drugs may inhibit or accelerate the metabolism of others, increasing their toxicity or likelihood of causing a reaction.
- #### 3. Age and Gender
- ◆ **Age:** Children (especially those under 3 years old) and elderly individuals are more prone to drug-induced skin reactions due to their de-

veloping or aging immune systems. Elderly patients may also have multiple underlying health conditions or impaired organ function, which can increase their sensitivity to drug side effects. Children, especially those with chronic diseases, are at a higher risk of antibiotic-induced sensitization.

- ◆ **Gender:** Women are generally more likely to develop drug allergies and skin reactions than men. This is partly due to hormonal differences, as women may have a more reactive immune system, which can enhance the development of allergic reactions. Additionally, women are often prescribed medications at higher rates than men, contributing to the higher incidence of drug-related dermatoses in females.

4. Concomitant Diseases and Health Conditions

Certain pre-existing health conditions can exacerbate the risk of developing drug-induced dermatoses by altering immune function, metabolism, or drug responses:

- ◆ **Autoimmune diseases:** Patients with autoimmune diseases like lupus or rheumatoid arthritis may have a higher risk of developing skin reactions to medications. These conditions can alter the immune response, making it more likely for the body to react negatively to drugs.
- ◆ **Liver and kidney diseases:** Impaired liver or kidney function can affect drug metabolism and clearance, increasing the potential for drug accumulation in the body, which may lead to heightened skin sensitivity.
- ◆ **HIV and immunocompromised states:** Individuals with HIV or those who are immunocompromised due to other reasons (e.g., chemotherapy, organ transplants) have a significantly higher risk of developing severe skin reactions to drugs.

5. Viral Infections

Viral infections (such as herpes virus reactivations, including Epstein-Barr virus, HHV-6, or cytomegalovirus) can increase the risk of drug-induced skin reactions, especially in patients with systemic diseases. These viral infections may interact with the drugs, contributing to the development of serious cutaneous reactions, such as DIHS, SJS, or TEN (24) (25).

Understanding these risk factors is crucial for healthcare providers to identify patients at higher risk and to prevent or minimize the occurrence of serious skin reactions. Genetic screening, especially for high-risk populations, can be a valuable tool in preventing life-threatening drug-induced skin reactions.

CONCLUSION

In conclusion, data from epidemiological studies on drug-induced dermatoses underscore the urgent need for understanding and managing this serious health issue. Analysis of the prevalence, characteristics, and risk factors of drug-induced dermatoses provides valuable information for healthcare practice and guides efforts for prevention and treatment. Optimization of diagnostic methods and prevention of recurrences are key aspects for improving healthcare for patients affected by these adverse reactions. Further research and innovations in this area are essential for developing more effective strategies for managing drug-induced dermatoses and reducing their clinical and social impact.

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