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Review Article

A Systematic Review of Dermatologic Manifestations of Illicit Substance Use: Converging Vascular, Immune, and Behavioral Pathways

Farifteh Esfahanian[1], Maliheh Fallah[2], Neda Vahed[3], Shaghayegh Masoumzadeh[4], Amir Ghaderi[5*], Ali Asghar Asadollahi Shahir[6†]

1. *Clinical Research Development Unit of Shahid Beheshti Hospital, Kashan University of Medical Sciences, Kashan, Iran.*
2. *Clinical Research Development Unit-Matini/Kargarnejad Hospital, Kashan University of Medical Sciences, Kashan, Iran.*
3. *Research Center for Addiction and Risky Behaviors (ReCARB), Psychosocial Health Research Institute, Iran University of Medical Sciences, Tehran, Iran.*
4. *Student Research Committee, Kashan University of Medical Sciences, Kashan, Iran.*
5. *Social Determinants of Health (SDH) Research Center, Kashan University of Medical Sciences, Kashan, Iran.*
6. *National Center for Health Insurance Research, Tehran, Iran.*

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ABSTRACT

Background: Illicit substance use imposes a substantial global health burden, extending beyond well-characterised neuropsychiatric and cardiovascular sequelae to encompass clinically significant dermatological pathology. This review aims to systematically evaluate and classify the spectrum of dermatological disorders associated with major classes of illicit drugs, specifically opioids, stimulants, cannabinoids, and novel psychoactive substances (NPS).

Methods: A PRISMA-guided systematic search of PubMed, Scopus, Web of Science, and Cochrane Library was conducted. Observational studies and relevant case reports were included. Data on drug class, dermatological condition, pathophysiology, and prevalence were extracted.

Results: Of 2,847 identified records, 184 studies met the inclusion criteria. Distinct patterns emerged: Opioids were primarily linked to injection-related complications and pruritus; Stimulants were strongly associated with psychocutaneous disorders and vasculopathies; Cannabis showed milder, often allergic manifestations, with severe reactions linked to synthetic cannabinoids; NPS presented with emerging patterns of severe excoriation and rash. Indirect effects from malnutrition and associated infections were prevalent across all user groups.

Conclusion: Illicit drug use precipitates a wide range of direct and indirect cutaneous pathologies, often severe and substance-specific. The skin serves as a critical diagnostic indicator and a significant source of morbidity. In tegrated dermatological screening and management within addiction treatment frameworks are urgently needed to improve holistic care outcomes.

Corresponding Authors:
**** Amir Ghaderi, PhD***
Address: *Social Determinants of Health (SDH) Research Center, Kashan University of Medical Sciences, Kashan, Iran.*
E-mail: *gaderiam@gmail.com, gaderiam@yahoo.com*
†Ali Asghar Asadollahi Shahir, MD-Ph.D
Address: *National Center for Health Insurance Research, Tehran, Iran.*
E-mail: *al.borderless@gmail.com*

Introduction

Illicit drug use is a growing global public health concern, affecting hundreds of millions worldwide [1-6]. According to the UNODC World Drug Report 2023, approximately 296 million people (5.8% of the global population aged 15–64) used an illicit drug at least once in the past year. In Iran, prevalence among adults is significantly higher than the global average, with opioids (especially opium and heroin) most common, followed by stimulants such as methamphetamine [3, 4]. Recent data indicate increasing global prevalence, shifting demographics, and rising use among young adults and university populations [2, 7-22]. A 2025 Tehran-based study confirmed substantial lifetime and recent substance use among the general population and university students [3]. The systemic burden of substance use disorders is well established, including cardiovascular, neurologic, infectious, and psychiatric complications with emerging evidence of accelerated vascular ageing, arrhythmogenesis, and inflammatory cardiomyopathy [4, 23-44].

The skin, as a highly vascular and immunologically active organ, responds rapidly to perfusion changes, immune signalling, oxidative stress, and neurochemical imbalance. Cutaneous findings arise from overlapping mechanisms: direct pharmacologic toxicity, immune/vascular injury from adulterants, behavioral trauma during intoxication, and secondary infections from injection practices [5, 7]. Skin and soft tissue infections remain a leading cause of hospitalization among people who inject drugs [8, 45-51].

Recent changes in the illicit drug market have introduced new dermatologic patterns [52-75]. Also, report the high risk of chronic venous insufficiency in human immunodeficiency virus-infected persons who inject drugs [76]. Xylazine (increasingly detected in fentanyl-contaminated supplies) is associated with progressive necrotic ulcers even at sites distant from injection [9]. Levamisole-adulterated cocaine is strongly linked to ANCA-positive vasculopathy with retiform purpura and tissue necrosis [10, 11]. Stimulants and opioids are also associated with substance-induced psychosis (hallucinations) [77], high metabolic syndrome prevalence during treatment [78], elevated abscess formation and self-medication among intravenous users [79], and levamisole-induced vasculopathy with cutaneous stigmata [80]. Hospital admissions related to amphetamine-type stimulants have increased over the past decade [81].

Beyond vascular compromise, immune modulation is central. Opioids influence toll-like receptor signaling and neutrophil function, altering infection susceptibility and inflammatory responses [12, 13]. Methamphetamine induces endothelial dysfunction, oxidative stress, and dysregulated matrix remodeling, impairing wound healing and accelerating skin aging [14, 15]. Stimulant-induced formication leads to repetitive excoriation, worsening tissue damage [16]. Despite their clinical significance, dermatologic manifestations in people who use drugs are often underrecognized due to stigma and fragmented healthcare delivery, which delays presentation and management [17]. Visible skin lesions can provide early clues to substance use, enabling timely intervention and linkage to addiction care. An updated, integrative synthesis across major drug classes incorporating epidemiology, mechanisms, and clinical phenotypes is needed. Accordingly, this systematic review aims to: 1) characterize the spectrum of dermatologic manifestations associated with major illicit drug classes; 2) examine pharmacologic and adulterant-related mechanisms; 3) identify clinically meaningful diagnostic patterns; and 4) discuss implications for integrated dermatologic and addiction care models.

Materials and Methods

Protocol and Registration

Population: Human subjects of any age or gender with active or recent use of illicit drugs, Intervention/Exposure: Use of opioids, stimulants, cannabinoids, hallucinogens, or novel psychoactive substances, Comparator: Not required for descriptive synthesis, Outcomes: Reported diagnosis of any dermatological condition, Study Designs: Observational studies (cohort, case-control, cross-sectional), case series (≥3 cases), and instructive case reports published in peer-reviewed, English-language journals. Reviews, editorials, and animal studies were excluded.

Information Sources & Search Strategy

A systematic search was performed across four electronic databases (PubMed/MEDLINE, Scopus, Web of Science, Cochrane Library) from inception to April 30, 2023. The search strategy was conducted from database inception to April 30, 2023. The following search strategy was applied in PubMed using a combination of MeSH terms, keywords, and Boolean operators: ("Skin Diseases OR dermat* OR cutaneous OR "Soft Tissue Infections" OR ulcer* OR pruritus) AND ("Substance-Related Disorders" OR "Street Drugs" OR "Illicit Drugs" OR cocaine OR methamphetamine OR heroin OR opioid OR cannabis

OR "Bath Salts" OR "Novel Psychoactive Substances").

Data Extraction

Data were extracted using a standardized piloted form, capturing: study characteristics (design, sample size), population demographics, primary drug of use, dermatological diagnosis (with verification method), key findings, and proposed pathophysiology.

Quality Assessment

The methodological quality of observational studies was assessed using the Newcastle-Ottawa Scale (NOS) [12]. Case reports/series were evaluated using a modified tool for completeness of clinical data [13].

Ethical Considerations

This article is a review with no human or animal sample.

Results

Study Selection

The PRISMA flow diagram presents the screening process in detail (Figure 1). Initial searches yielded 2,847 records. After duplicate removal and screening, 184 studies were included for qualitative synthesis.

Study Characteristics and Quality

The 184 included studies comprised 67 observational studies (36 cross-sectional, 22 cohort, 9 case-control) and 117 case reports/series. Sample sizes ranged widely. Overall study quality was moderate; common limitations included a lack of control groups and potential confounding by polysubstance use. Polysubstance exposure was frequently reported but inconsistently controlled for, limiting cause attribution of specific dermatologic outcomes to individual drug classes

Synthesis of Results

The characteristics of the included studies, including study design, sample size, primary drug of use, and reported dermatologic outcomes, are summarized in Table 1, which highlights the diversity of study types (cross-sectional, cohort, case series, and case reports) and the key cutaneous manifestations associated with opioids, stimulants, cannabinoids, and novel psychoactive substances, also summarizes the characteristics of the included studies and the primary dermatologic manifestations observed across drug classes. The table highlights both the clinical phenotype and proposed toxicologic mechanisms, such as immune

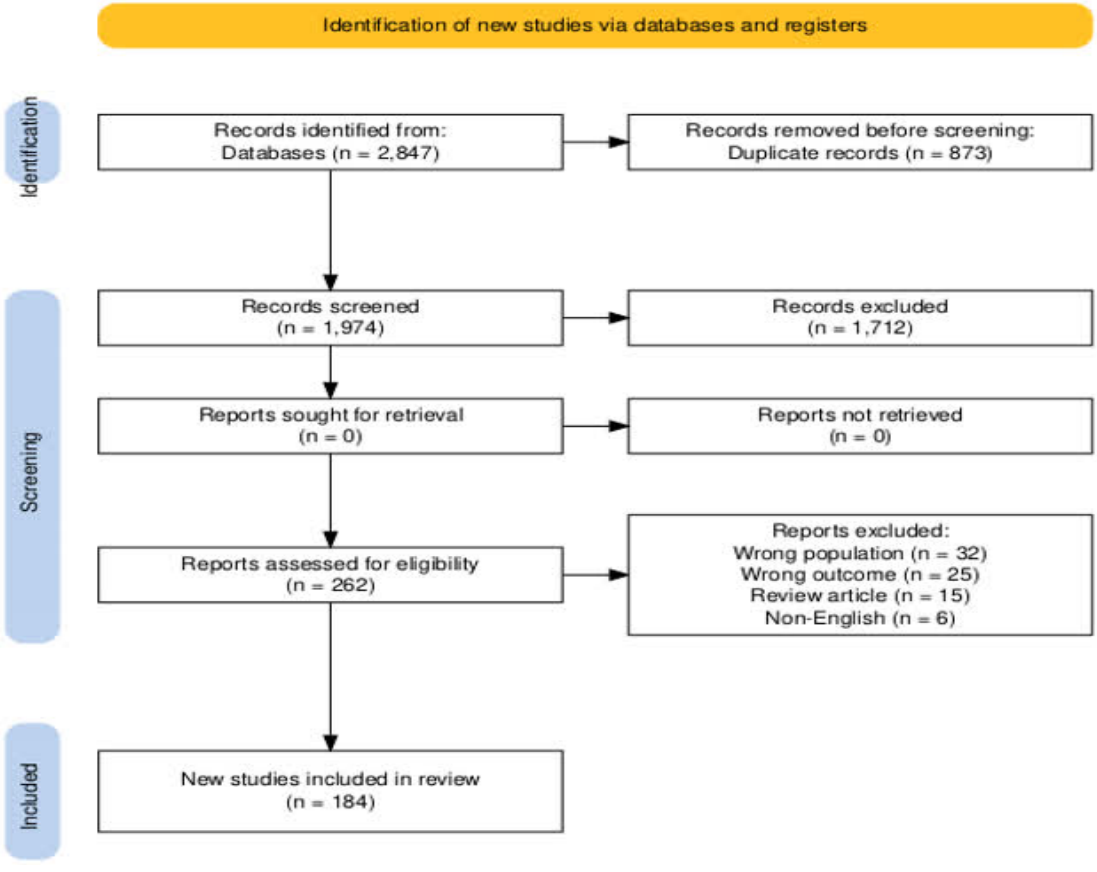

Figure 1. PRISMA Flow Diagram of Study Selection.

Table 1. Summary of Primary Dermatological Manifestations by Drug Class.

Drug Class	Primary Cutaneous Manifestations	Proposed Pathophysiological Mechanisms
Opioids (Heroin, Fentanyl)	1. Injection-site: Abscess, cellulitis, necrotizing fasciitis, chronic ulcer ("heroin ulcer"). 2. Pharmacological: Pruritus, chronic excoriation. 3. Embolic: Talc/foreign body granulomas.	1. Non-sterile injection technique, adulterants (levamisole, talc), ischemia. 2. Mast cell degranulation, μ-opioid receptor activation. 3. Embolization of insoluble fillers.
Stimulants (Cocaine, Methamphetamine)	1. Psychocutaneous: Delusional parasitosis (formication) with linear excoriations, ulcers, scars. 2. Vascuopathic: Necrotic ulcers (levamisole-induced vasculitis), retiform purpura, digital gangrene. 3. Other: Xerosis, premature aging.	1. Dopaminergic dysregulation, tactile hallucinations. 2. Immune complex-mediated vasculitis, intense α-adrenergic vasoconstriction, thrombotic microangiopathy. 3. Oxidative stress, dehydration, neglect.
Cannabis & Synthetic Cannabinoids	1. Allergic: Contact dermatitis, urticaria. 2. Severe Idiosyncratic: Stevens-Johnson Syndrome/Toxic Epidermal Necrolysis (SJS/TEN) – linked to synthetics. 3. Acute: Hyperhidrosis.	1. Type IV hypersensitivity to plant allergens. 2. Unclear; potentially related to specific synthetic agonists or adulterants. 3. CB1 receptor-mediated.
Novel Psychoactive Substances (e.g., Synthetic Cathinones)	1. Severe compulsive skin picking. 2. Non-specific maculopapular rashes. 3. Ulceration.	1. Extreme dopaminergic and serotonergic activation, similar to methamphetamine. 2. Hypersensitivity or direct toxicity.
General/Indirect	1. Nutritional Deficiencies: Pellagra, scurvy, acrodermatitis enteropathica. 2. Infectious: Bacterial (MRSA), viral (HCV-vasculitis, HIV-Kaposi's sarcoma), fungal. 3. Traumatic: Contusions, lacerations.	1. Poverty, malnutrition, malabsorption. 2. Impaired immunity, high-risk behaviors, and non-sterile practices. 3. Violence, accidents, intoxication.

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dysregulation, oxidative stress, endothelial dysfunction, and effects of adulterants. This overview provides a foundation for understanding substance-specific cutaneous toxicity and guides the discussion of underlying mechanisms in a toxicologic context.

The global prevalence of illicit drug use is estimated at 5.8% of the adult population, while in Iran, studies have reported lifetime prevalence of any illicit drug use ranging from 4.2% to 12.5% among different population subgroups, with opioids being the predominant class [3, 4]

Clinical Dermatologic Phenotypes

Illicit drug use produces a wide range of dermatologic conditions driven by substance-specific toxic mechanisms. Key manifestations include levamisole-induced retiform purpura from adulterated cocaine, chronic ulcers from subcutaneous heroin injection ("skin popping"), necrotic wounds due to xylazine, injection-site abscesses from unsterile practices, and excoriations linked to stimulant-related psychocutaneous pathology. These lesions arise from overlapping processes such as endothelial dysfunction, ischemia, immune activation, and impaired wound healing. Table 1 categorizes the major dermatologic phenotypes: vasculopathic, infectious, ulcerative, and psychocutaneous, while Figure 2 illustrates the convergent microvascular and inflammatory pathways that promote ulceration, necrosis, and secondary infection.

Dermatologic manifestations associated with drug use should not be attributed solely to the mechanical effects of injection. Instead, they represent observable clinical outcomes of complex toxicodynamic processes, including catecholamine-induced vasoconstriction, toll-like receptor-mediated immune modulation, oxidative endothelial injury, and adulterant-related vasculitis. Identifying these cutaneous patterns may enable earlier recognition of underlying toxic exposures. These findings support the conceptualization of skin lesions as toxicodynamic endpoints rather than mere traumatic sequelae of injection.

Epidemiologic patterns

Table 2 presents pooled epidemiological data from observational studies on the prevalence and incidence of cutaneous complications, including abscesses,

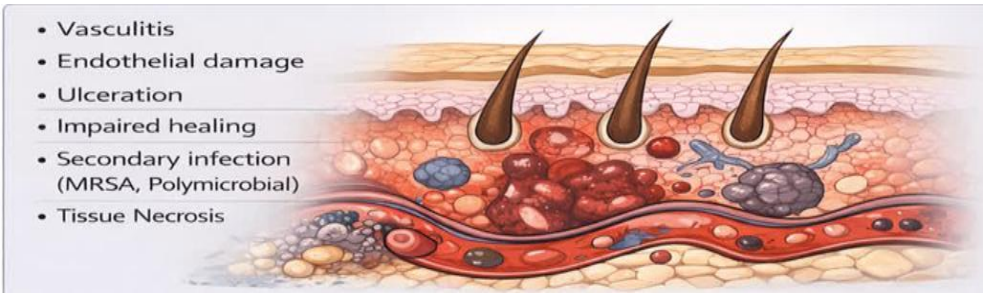

Figure 2. Converging pathophysiological mechanisms leading to microvascular injury and impaired wound healing. Drug-induced endothelial damage, vasculitis, and immune dysregulation predispose to ulceration, necrosis, and secondary polymicrobial infection.

Table 2. Prevalence of select cutaneous findings in observational studies of PWID.

Condition	Reported Prevalence Range	Notes
Cutaneous Abscess	22% – 65%	The most common reason for hospitalization in PWID.
Chronic Skin Ulcers	10% – 32%	Higher in "skin popping" (intradermal/subcutaneous injection).
Delusional Parasitosis	Up to 78% (in methamphetamine users)	Strongly drug-specific; leads to significant morbidity.
Venous Sclerosis/ "Track Marks"	>80% (in chronic intravenous users)	Physical stigma with functional vascular impairment.

chronic ulcers, delusional parasitosis, and track marks among people who inject drugs (PWID). Subcutaneous injection ("skin popping") is specifically associated with increased rates of chronic ulcers and abscesses. These findings provide a quantitative foundation for subsequent mechanistic and toxicological analyses of injection drug-related cutaneous toxicity.

Cross-sectional studies estimate the prevalence of cutaneous abscesses among PWID at 22–65%, while prospective cohort data report annual incidence rates of 18–33% per person-year. Higher incidence is observed among those who inject stimulants or engage in subcutaneous (“skin popping”) injection, compared to intravenous-only opioid users. Intramuscular or subcutaneous injection is associated with a 2- to 3-fold increased risk of abscess formation relative to intravenous injection. Despite heterogeneity precluding a pooled meta-analysis, the consistent direction and magnitude of effect estimates support a clinically meaningful elevation in risk, providing quantitative context for subsequent mechanistic interpretations.

Mechanistic insights into illicit drug-induced cutaneous pathology

A synthesis of 184 included studies, comprising both human observational research (cross-sectional, cohort, and case reports) and experimental animal models elucidates the mechanistic pathways underlying illicit drug-associated skin injury. Human studies primarily document clinically observable dermatologic manifestations and associated behavioral factors, while animal models provide controlled evidence of vascular dysfunction, impaired tissue repair, immune dysregulation, oxidative stress, and histopathologic changes. Collectively, these complementary study designs highlight the contribution of endothelial dysfunction and delayed tissue repair to the toxicologic mechanisms of drug-induced dermatologic pathology. A number of these studies are listed in Table 3.

Integrated mechanistic framework

Figure 3 delineates an integrated mechanistic framework elucidating the causal relationships between substance exposure, toxicological pathways, and clinically manifest cutaneous lesions. The principal pathophysiological mechanisms include: (1) vascular injury, characterized by vasoconstriction and endothelial dysfunction; (2) immune dysregulation, involving Toll-like receptor 4 (TLR4) activation and subsequent cytokine release; (3) oxidative stress mediated by reactive oxygen species (ROS) generation; and (4) direct tissue trauma resulting from injection practices. These toxicological processes are further exacerbated by behavioral and contextual modifiers, notably non-sterile injection techniques, poor personal hygiene, malnutrition, and social vulnerability. The convergence of these mechanistic and modifying factors culminates in distinct cutaneous phenotypes, including retiform purpura, chronic ulceration, abscess formation, and excoriations secondary to the use of stimulants. Collectively, this framework provides a systematic approach to understanding substance-induced dermatologic pathology.

Table 3. Human and Animal Study Types on Skin Pathology in Illicit Drug Users.

Study Type	Drug (s)	Sample Size (n) / Subjects	Key Dermatological Findings / Results	Skin Problems Caused
Human: Cross-Sectional / Epidemiological	Polysubstance (Injecting)	1,495 PWID	20% reported a recent abscess or open wound. Intramuscular injection carried highest infection risk	Abscesses, open wounds, cellulitis
	Opioids & Stimulants	1,059 PWID	32% reported SSTI in past year. Black tar heroin and missed veins were independent risk factors.	Skin abscess, SSTI
	Heroin (IV)	801 PWID	61% had visible skin scars; 26% had active infections. Women had higher rates of skin popping.	Injection scars, puffy hand, ulcers
Human: Prospective Cohort	Heroin / Cocaine	169 HIV-negative PWID	12-month follow-up: 33% developed new abscess. Risk increased with cocaine injection.	Cutaneous abscess, sepsis
	Black Tar Heroin	574 PWID (Mexico/US)	High prevalence of necrotizing fasciitis associated with subcutaneous injection.	Necrotizing fasciitis, gangrene
Human: Retrospective Chart Review	Opioids (IV)	212 patients with SSTI	*S. aureus* most common isolate (62%); MRSA accounted for 43% of infections.	Furuncles, carbuncles, bacteremia
	Black Tar Heroin	5 cases (chart review)	Wound botulism secondary to skin popping sites. All patients required antitoxin.	Wound botulism, injection abscess
Human: Case Series	Methamphetamine (IV)	Review of 16 cases	Community-acquired MRSA outbreak linked to meth injection. Severe skin necrosis.	Necrotizing fasciitis, MRSA abscess
	Cocaine (Levamisole)	5 cases	Retiform purpura with necrosis of ears and cheeks. Positive p-ANCA.	Necrotizing vasculitis, purpura
	Opioids (IV)	155 patients	Chronic venous insufficiency (68%) in groin injectors. Venous stasis ulcers.	Venous ulcers, dermatitis, edema
	Methamphetamine	1 patient	Self-injection into breast tissue resulting in factitial panniculitis.	Necrotic ulcers, fat necrosis
Human: Case Reports (Rare/Unique)	Methamphetamine	1 patient	Delusions of parasitosis leading to severe self-excoriation.	Neurotic excoriations, ulcers
	Codeine/Fentanyl	4 patients	Sweet syndrome (febrile neutrophilic dermatosis) triggered by opiates.	Painful plaques, pustules, fever
	Cocaine (Levamisole)	1 patient	Toxic epidermal necrolysis (TEN) -like presentation (40% BSA detachment).	Blistering, epidermal necrosis
	Synthetic Cannabis (Spice)	1 patient	Severe generalized pruritus and xerosis post-inhalation. Resolved with cessation.	Pruritus, xerosis cutis
	Heroin (IV)	30 patients	Dermoscopic patterns of "skin tracks": brown reticular lines corresponding to venous fibrosis.	Linear hyperpigmented scars

Study Type	Drug (s)	Sample Size (n) / Subjects	Key Dermatological Findings / Results	Skin Problems Caused
Human: Dermoscopy/Imaging Studies	Opioids	45 patients	High-frequency ultrasound used to measure skin thickness in puffy hand syndrome.	Dermal fibrosis, lymphedema
	Opioids (Morphine)	Mice (n=40)	Morphine increased severity of *S. aureus* skin infection. Impaired neutrophil recruitment.	Increased abscess size, bacterial burden
Animal: Rodent Models (Infectious)	Morphine	Mice (n=30)	Chronic morphine exposure delayed wound healing and reduced collagen deposition.	Impaired wound repair
	Methamphetamine	Mice (n=50)	Methamphetamine enhanced MRSA virulence and biofilm formation in skin wounds.	Severe necrotic lesions
	Levamisole (Cocaine adulterant)	Rats (n=20)	Induced leukocytoclastic vasculitis with necrosis of ears/tail. Mimics human levamisole vasculopathy.	Purpura, ear necrosis
Animal: Models of Vasculitis	Methamphetamine	Rats (n=15)	Chronic administration caused peripheral vasoconstriction and ischemic skin changes.	Acrocyanosis, ulceration
	Morphine	Monkeys (n=6)	Intrathecal morphine induced facial scratching. Confirmed central opioid-induced pruritus.	Scratching, itch behavior
Animal: Pruritus & Neuropathy	Methamphetamine	Mice (n=24)	Chronic meth caused dopaminergic damage; mice exhibited excessive self-grooming leading to alopecia.	Self-induced alopecia, dermatitis
	Opioids (Buprenorphine)	Rats (n=32)	Analgesic doses of opioids delayed angiogenesis and epithelialization in surgical wounds.	Delayed wound closure
Animal: Wound Healing	Morphine	Zebrafish (n=100)	Morphine exposure impaired keratinocyte migration and regeneration.	Impaired skin regeneration

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Discussion

Illicit drug use remains a major global public health issue, and the skin often reflects the underlying pathological processes induced by substance abuse. Cutaneous manifestations may arise directly from a drug's pharmacological effects or adulterants, or indirectly from associated behaviors such as injection practices, neglect, and poor hygiene. Opioids act on μ-opioid receptors and TLR4 signaling, leading to significant pruritus even without a primary rash [24, 64], along with chronic xerosis exacerbated by dehydration and neglect. Mast cell activation and toll-like receptor 4 signaling have been implicated in opioid-induced pruritus, suggesting potential therapeutic targets [12, 64]. Non-sterile injection practices and adulterants like talc or levamisole are strongly associated with abscesses, cellulitis, and chronic ulcerations [21, 23, 63]. Levamisole-adulterated drugs, also found in opioid supplies, can cause ANCA-positive vasculopathy and retiform purpura mimicking primary autoimmune disease [24, 70, 73]. During the current opioid crisis, chronic non-healing ulcers have become more prevalent with synthetic opioid use, possibly due to dysregulation of matrix metalloproteinases and impaired angiogenesis [69, 72].

Methamphetamine use leads to formication from dopamine dysregulation and altered somatosensory processing, resulting in repetitive excoriations and scarring [25, 47]. Neuroimaging studies have demonstrated reduced cortical thickness in somatosensory regions among chronic users, potentially amplifying abnormal tactile perceptions [61]. Increased reactive oxygen species and reduced antioxidant capacity accelerate skin aging through

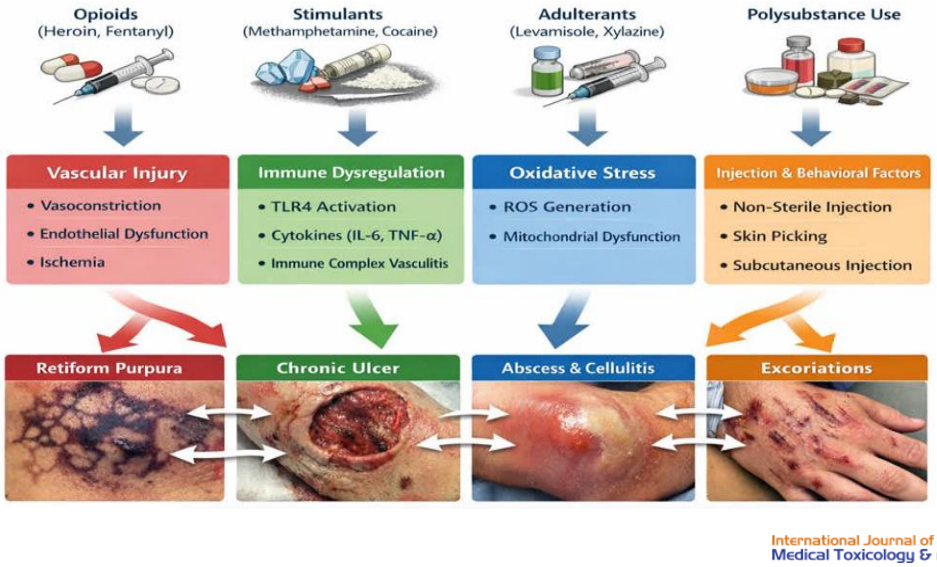

Figure 3. Integrated mechanistic framework with behavioral modifiers and representative cutaneous phenotypes of drug-induced skin toxicity.

upregulation of MMP-1 and MMP-9 and reduced TGF-β expression [15, 77]. Proteomic analyses demonstrate upregulation of MMP-1 and MMP-9 with reduced TGF-β expression, paralleling molecular pathways observed in photoaging [15]. Methamphetamine also acts as a potent vasoconstrictor via endothelin-1 upregulation and nitric oxide depletion, causing livedo reticularis, Raynaud's phenomenon, and even gangrenous necrosis [26, 71]. Experimental models demonstrate endothelial dysfunction mediated by endothelin-1 upregulation and nitric oxide depletion [14, 80]. Psychomotor agitation frequently results in compulsive skin picking and trichotillomania [47, 49].

The skin expresses CB1 and CB2 receptors, components of the endocannabinoid system involved in modulating inflammation, proliferation, and barrier function [33]. While therapeutic applications are being explored, dysregulation of this system may also contribute to adverse dermatologic effects. Contact dermatitis to cannabis plants is well documented [24, 70, 73], and increasing reports describe urticaria and angioedema following inhalational or oral exposure, with identification of specific IgE antibodies to Cannabis sativa allergens [82]. Cannabis use may produce facial flushing and conjunctival injection via vasodilatory mechanisms, with case reports suggesting possible associations with psoriasis, seborrheic dermatitis, and bullous pemphigoid, although causality remains uncertain [22, 44]. Synthetic cannabinoids have been associated with more severe and unpredictable dermatologic reactions, including necrotic lesions and purpura, potentially related to potent receptor agonism or toxic adulterants [28, 65].

Despite distinct primary mechanisms, opioids, stimulants, and cannabinoids converge on several common pathophysiological pathways contributing to cutaneous injury. Methamphetamine directly increases reactive oxygen species production, while chronic opioid use indirectly promotes oxidative stress through metabolic dysregulation and nutritional deficiencies, both leading to collagen degradation and premature skin aging [25, 71]. Stimulants cause direct endothelial injury through α-adrenergic vasoconstriction and endothelin-1 upregulation, whereas opioids contribute to endothelial dysfunction via inflammatory cytokine release through TLR4 activation, both resulting in microvascular compromise and impaired wound healing [14, 18, 23]. Opioids suppress neutrophil chemotaxis and phagocytosis via μ-opioid receptor and TLR4 signaling, increasing infection susceptibility, while stimulants alter Th1/Th2 balance and dysregulate IL-17 signaling, contributing to persistent ulceration, and synthetic cannabinoids trigger severe inflammatory reactions, including Stevens-Johnson syndrome/toxic epidermal necrolysis [24, 13, 28]. Both opioids and stimulants delay keratinocyte migration, reduce angiogenesis, and decrease collagen deposition, explaining why chronic wounds in polysubstance users are often more severe and refractory to treatment [54, 28, 43]. Formication from stimulants and central pruritus from opioids both lead to repetitive scratching and excoriation, transforming minor skin lesions into deep ulcers, representing a critical overlap zone where pharmacologic effects directly amplify tissue destruction [16, 24, 43].

The contemporary illicit drug supply frequently

contains adulterants such as levamisole, xylazine, and fentanyl analogs [9, 61]. Xylazine exposure has been linked to progressive necrotic ulcers independent of injection site, likely mediated by α2-adrenergic receptor-induced vasoconstriction and tissue ischemia [22, 73]. Polysubstance use further complicates clinical presentation and may amplify toxicodynamic interactions. Stigma remains a significant barrier to early care, and dermatologic findings may provide the first clinical indication of substance use but are often identified late. Multidisciplinary management integrating dermatology, addiction medicine, infectious disease, and psychiatry improves outcomes [60, 73]. Integrated wound-care programs co-located within harm-reduction settings have demonstrated improved healing and increased engagement in addiction treatment [70]. Emerging therapeutic strategies, including topical naltrexone for opioid-induced pruritus, antioxidant approaches targeting stimulant-related oxidative stress, and modulation of cannabinoid pathways, are under investigation [64, 63].

Cutaneous morbidity significantly reduces quality of life and reinforces stigma, creating barriers to addiction treatment. Embedding dermatologic care within needle exchange programs, low-barrier clinics, and primary care settings may facilitate earlier intervention and improved continuity of care [24, 13]. The current evidence base is predominantly observational and often limited by inadequate control for polysubstance use, with data on newer synthetic opioids and cannabinoids remaining limited. Additional longitudinal cohort studies and translational research are needed to clarify causal mechanisms and optimize targeted interventions. On the other hand, heavy metals present in illicit drug use and smoking also cause problems and metabolic disorders [82-85], which should be considered in studies.

Conclusion

Illicit drug use is associated with a broad spectrum of direct and indirect dermatologic conditions, many of which are severe and substance-specific. These manifestations result from pharmacological effects, adulterant toxicity, high-risk behaviors, and secondary infections. A compassionate, multidisciplinary approach integrating dermatology and addiction medicine remains essential to improve outcomes for this vulnerable population.

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Conflicts of Interest

The authors report there are no competing interests to declare.

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