

Substance Use Disorders and the Skin: A Narrative Review Bridging Dermatology and Addiction Psychiatry

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Abstract

Background. Substance use disorders (SUDs) affect a substantial and growing share of the global population, and the skin is frequently the first organ in which their consequences become visible. Cutaneous findings may precede a disclosed substance history by months or years, yet dermatological signs of substance use remain under-recognised in both dermatology and mental health settings.

Objective. To synthesise the current literature on the cutaneous, mucosal, hair and nail manifestations of commonly misused substances; to describe the mechanisms through which these arise; and to outline the diagnostic pitfalls and collaborative care models relevant to clinicians working at the interface of dermatology, primary care and addiction psychiatry.

Methods. A narrative review was conducted. PubMed/MEDLINE, Embase and Google Scholar were searched for English-language articles published between January 1990 and the date of writing, using combinations of the terms “substance use disorder”, “drug abuse”, “cutaneous”, “dermatologic”, “skin”, “alcohol”, “tobacco”, “methamphetamine”, “cocaine”, “opioid”, “cannabis” and “inhalant”. Landmark earlier papers and standard dermatology reference texts were included where they remain the primary source for a described sign. Reference lists of retrieved articles were hand-searched.

Findings. Substance-related skin disease arises through four broadly separable routes: direct pharmacological effects on the cutaneous vasculature and adnexa; immunosuppression and impaired wound healing; infection introduced by the route of administration and by adulterants; and behaviours driven by the psychiatric sequelae of use, most notably excoriation in the context of formication and delusional infestation. Alcohol, tobacco, methamphetamine, cocaine, opioids, cannabis and inhalants each produce a recognisable, if overlapping, cutaneous signature. Diagnosis is complicated by clinical mimicry, patient non-disclosure, polysubstance use and the absence of validated screening tests for several substance classes.

Conclusion. Cutaneous findings offer an accessible and often early window onto substance use. Familiarity with these signs, coupled with non-judgmental history-taking and structured referral pathways between dermatology, primary care, addiction medicine and psychiatry, can shorten time to diagnosis and reduce avoidable morbidity.

Keywords: Substance use disorder; dermatology; psychodermatology; alcohol; tobacco; methamphetamine; cocaine; opioids; cannabis; skin and soft tissue infection; delusional infestation; interdisciplinary care

1. Introduction

Dermatology traces its origins to descriptions of skin disease in Egyptian and Hippocratic writings, and consolidated as a clinical specialty during the eighteenth and nineteenth centuries around the classification, diagnosis and treatment of dermatoses. The discipline today encompasses several thousand recognised diagnoses and a broad diagnostic, therapeutic and aesthetic armamentarium (1). Among the many aetiologies that bring patients to dermatological attention, substance use has become an increasingly important and under-acknowledged contributor.

Substance use disorders are chronic, relapsing conditions with a substantial global burden. The United Nations Office on Drugs and Crime estimated that approximately 271 million people aged 15 to 64, about 5.5% of that population, had used a drug in the preceding year in 2017, and that roughly 35 million people were living with a drug use disorder (2). Epidemiological work in the United States has documented parallel increases in the prevalence of substance use and of substance-related mortality over the same period, alongside persistent gaps between the number of people meeting criteria for a substance use disorder and the number receiving treatment (3,4).

The skin, the body's largest organ, both witnesses and displays this burden. Cutaneous findings may signal the substance involved, the route of administration, and the adulterants or infectious agents introduced alongside it (5). The spectrum runs from commonplace and cosmetically troubling changes to rare, rapidly progressive and potentially fatal presentations. Because many of these findings are externally visible and appear early, recognising them offers a practical opportunity for earlier intervention (6,7).

This narrative review synthesises what is currently known about the cutaneous, mucosal, hair and nail manifestations of substance use; the mechanisms that produce them; the diagnostic difficulties they present; and the collaborative models of care through which they are best managed. It is written for dermatologists, primary care physicians, psychiatrists and addiction medicine clinicians, whose caseloads overlap in these patients more often than the separation of their specialties would suggest.

2. Methods

This is a narrative, not a systematic, review. PubMed/MEDLINE, Embase and Google Scholar were searched for English-language publications appearing between January 1990 and the date of writing. Search strings combined substance-related terms ("substance use disorder", "substance abuse", "drug abuse", "alcohol", "tobacco", "smoking", "vaping", "methamphetamine", "cocaine", "opioid", "heroin", "cannabis", "marijuana", "inhalant") with dermatological terms ("skin", "cutaneous", "dermatologic manifestations", "skin and soft tissue infection", "pruritus", "alopecia", "nail"). Reviews, original studies, case series and case reports were all eligible. Seminal earlier papers and standard reference texts were retained where they remain the primary description of a sign. Reference lists of included articles were hand-searched for additional sources. Article selection was made by the authors by consensus and was not blinded or duplicated; no formal risk-of-bias assessment was performed, and the review is therefore subject to the selection and reporting limitations described in Section 9.

3. Mechanisms of substance-related cutaneous injury

The pathophysiology linking substance use to skin disease is multifactorial, and four broad mechanisms account for most observed findings.

3.1 *Direct vascular and adnexal effects*

Chronic alcohol use produces dermal vasodilatation, giving rise to findings that range from pinpoint telan-

giectasia to ecchymoses (7). Stimulants act in the opposite direction: cocaine and the amphetamines cause vasoconstriction, which can compromise cutaneous perfusion and, at the extreme, precipitate ischaemia and end-organ damage (11,16). Tobacco smoking reduces cutaneous blood flow and inflicts long-term damage on the dermal microcirculation, impairing tissue oxygenation and wound healing (10). Smoking also increases elastic fibre deposition in the reticular dermis of sun-protected skin, producing changes resembling solar elastosis; in vitro work has shown increased expression of matrix metalloproteinases-1 and -3, which degrade collagen, elastic fibres and proteoglycans, together with inactivation and depletion of transforming growth factor β 1, a key regulator of tissue remodelling and repair (10,15).

3.2 Immune suppression, nutritional depletion and impaired healing

Alcohol suppresses immune function and, through nutritional deficiency, alters mucosal integrity and enzyme function. It may also promote malignant transformation already initiated by other exposures, notably tobacco smoke, while alcohol-related hepatic injury impairs chemical detoxification. Together these raise the risk of cutaneous and mucosal cancer (9). Opioids exert their own immunomodulatory effects through opioid receptors expressed on immune cells, which is relevant both to infection risk and to wound healing in people who inject drugs (8).

3.3 Route of administration, adulterants and hygiene

A substantial proportion of substance-related skin disease is attributable not to the drug itself but to how it is taken. Subcutaneous or intradermal injection, known as “skin popping”, carries a higher infection risk than intravenous administration because it deposits organisms directly within the skin layers (11,13). Non-sterile equipment, shared needles, contaminated preparations and limited access to clean injecting supplies compound this risk (13,17). When illicit drugs are cut with adulterants during processing, particulate foreign material and micro-organisms are introduced alongside the drug, which both causes injury and obscures attribution of a given lesion to a specific agent (21). The route of administration also shapes the wider infectious risk profile: injection drug use remains an important driver of blood-borne virus transmission, including HIV and hepatitis C (39,41).

3.4 Pruritus, formication and behaviourally mediated injury

Opioids commonly cause pruritus and flushing. These effects are mediated centrally and peripherally through μ - and κ -opioid receptor signalling in itch circuits, and, for some agents, through mast cell histamine release (18). Independently, stimulants, and methamphetamine in particular, produce formication and, in a subset of users, delusional infestation. The resulting repetitive excoriation is itself the proximate cause of much of the visible skin damage, and its psychiatric substrate is the reason dermatological treatment alone frequently fails (11,14). This mechanism is the clearest point at which dermatology and psychiatry converge and is developed further in Section 7.

4. Substance-specific cutaneous manifestations

4.1 Alcohol

Cutaneous signs have been reported in as many as 43% of men and 33% of women with alcohol use disorder (9). Spider telangiectasia is the most characteristic vascular lesion, and palmar erythema (“liver palms”) is also well recognised. Transient flushing occurs in people with rosacea who drink, in those genetically deficient in aldehyde-metabolising enzymes, and in patients taking interacting medications such as disulfiram. Unilateral nevoid telangiectasia, a proliferative vascular condition presenting as a circumscribed area of fine, threadlike telangiectasia in the trigeminal, C3 or C4 dermatomes or their neighbours, is classically associated with chronic alcoholic liver disease in men, and the haemodynamic

disturbance of cirrhosis is a plausible trigger.

Jaundice, caused by bilirubin deposition following impaired hepatic conjugation, is a familiar sign of advanced alcoholic liver disease. Pruritus may be the only cutaneous indication of alcohol dependence and can precede the onset of hepatic cirrhosis by up to two years; it is usually generalised, though the extensor limbs, upper trunk, palms and soles are most often affected, and excoriations are a common accompaniment. Urticarial and anaphylactoid reactions, including localised or generalised urticaria, angioedema and pruritus, have been described within minutes to hours of alcohol ingestion. Hyperpigmentation occurs in alcohol-related liver disease, although it is more prominent in primary biliary cholangitis (9).

Alcohol intake increases the incidence of oral squamous cell carcinoma, and its effect is synergistic with tobacco. Nutritional dermatoses associated with alcohol misuse include pellagra, scurvy and, in severe cases, the cutaneous features of marasmus and kwashiorkor. Alcohol is the single most important contributing factor in sporadic porphyria cutanea tarda, with a history of alcohol use present in a high proportion of patients. Alcohol use disorder also predisposes to cutaneous infection, including botryomycosis, and may exacerbate discoid eczema, rosacea and psoriasis. Acute pancreatitis, most commonly alcohol-related, may rarely present with Cullen's sign, Grey Turner's sign or subcutaneous fat necrosis. Dupuytren's contracture is more prevalent among people with alcohol use disorder, with or without liver disease (7,9).

4.2 Tobacco and vaping

The clinical picture of the "smoker's face" was first described in 1985 and comprises atrophic skin, prominent facial creasing and a complexion with orange, purple or reddish tones. Evidence on tobacco as an independent risk factor for cutaneous squamous cell carcinoma remains inconsistent, although the association between cigarette smoking and keratoacanthoma, often regarded as a low-grade variant of squamous cell carcinoma, is better established, as is the link with anogenital cutaneous malignancy. Smokers are at increased risk of psoriasis, and among people who already have psoriasis the excess risk is greatest in women who smoke. Allergic contact dermatitis to cigarette constituents has been reported in both occupational and non-occupational settings; patch testing, extended beyond the standard series to include menthol, liquorice, formaldehyde, chocolate and tobacco components, should be considered in a smoker presenting with hand, face or neck dermatitis. Smoking is strongly associated with hidradenitis suppurativa, with current smoking reported in the large majority of affected patients in several cohorts (10,15,40).

Tobacco-related oral lesions are numerous. Smoker's melanosis presents as gingival melanin pigmentation resulting from increased basal-layer melanin. Smoker's palate (leukokeratosis nicotina palati, or nicotinic stomatitis), which classically affects pipe smokers, produces a greyish-white discolouration with palatal hyperkeratosis and numerous red punctate papules. Smoker's tongue (leukokeratosis nicotina glossi) is a homogeneous leukoplakia with evenly spaced pinpoint hemispherical depressions. A "snuff dipper's lesion" is the white plaque arising where tobacco is held against the oral mucosa. Acute necrotising ulcerative gingivitis, periodontitis, burns, keratotic patches, palatal erosions, black hairy tongue and dental abrasion have all been described. Among people living with HIV, smokers develop oral candidiasis and oral hairy leukoplakia at an earlier disease stage than non-smokers. Tobacco use also causes oral leukoplakia and raises the incidence of oral squamous cell and verrucous carcinoma (10,15).

4.3 Methamphetamine and other stimulants

Methamphetamine use disproportionately affects marginalised populations, including people who are hom

less, people living with HIV and sexual and gender minority populations (14,28). Reported dermatological associations include cellulitis, delusional infestation, hyperhidrosis, formication and prurigo nodularis, although the outpatient dermatological burden in this population remains incompletely characterised (14).

Other stimulants produce their own patterns. MDMA (“ecstasy”) has been implicated in fixed drug eruption, and stimulant use more generally disturbs fluid balance and thermoregulation in ways that affect skin integrity: alcohol inhibits vasopressin release and so promotes diuresis and dehydration, whereas stimulants increase renal blood flow and glomerular filtration rate, with some studies reporting a concurrent rise in vasopressin levels (11,12).

The most characteristic presentation of methamphetamine use is a pattern of excoriations sometimes described colloquially as “meth mites”, produced by persistent skin picking in response to formication or to the belief that insects are present in or emerging from the skin. Patients may use pins, needles or other sharp implements to extract what they perceive to be foreign bodies, and secondary polymicrobial infection commonly follows. A history of apparently treatment-resistant scabies should prompt consideration of this diagnosis. Most of these lesions would resolve if the picking stopped, which is why treatment directed only at the skin, without addressing the underlying use and its psychiatric features, tends to fail (11,14). People who use methamphetamine intravenously are additionally subject to the injection-related infections and abscesses described in Section 4.5, compounded by needle sharing and non-sterile technique, and by caustic contaminants and synthesis by-products present in illicitly manufactured product (11).

4.4 Cocaine

The dermatological consequences of cocaine reflect its systemic vascular effects. Reported findings include acute multifocal skin necrosis, acute generalised exanthematous pustulosis, chronic skin ulceration, hyperkeratotic darkened palms (“crack hands”) and cutaneous fibrosis. Delusional infestation, nasal septal perforation and cocaine-associated bullous disease are well described, as are oral blistering, gingival recession, dental caries, palatal perforation and Pott’s puffy tumour. Stevens–Johnson syndrome, urticarial vasculitis, pseudovasculitis and thromboangiitis obliterans have all been reported, along with necrotising granulomatous vasculitis, Raynaud phenomenon, midline destructive lesions, IgA vasculitis and palpable purpura (16).

Levamisole, historically a common cocaine adulterant, deserves particular mention because it produces a characteristic retiform purpura with a predilection for the ears, and because the resulting picture can be mistaken for a primary systemic vasculitis. Distinguishing drug-induced pseudovasculitis from autoimmune vasculitis is difficult, since the clinical and serological features overlap substantially; a history of cocaine or methamphetamine use should be sought in any patient presenting with purpura and multisystem involvement of the skin, nose, oropharynx and kidneys (32,34).

4.5 Opioids and injection drug use

Pruritus is the most frequently reported cutaneous symptom among people who use heroin. It reflects both opioid receptor-mediated itch signalling and hypersensitivity responses to adulterants present in illicit preparations (17,18). Abscesses and cellulitis at injection sites are common and are frequently painful; *Staphylococcus aureus* is the organism most often isolated, followed by anaerobes and beta-haemolytic streptococci, with contaminated preparations, non-sterile equipment and limited opportunity for hand and skin hygiene the principal contributors (13,17,21). Necrotising fasciitis is an uncommon but rapidly progressive complication, associated with streptococci, *Escherichia coli*, *Klebsiella* species and clostridia.

Puffy hand syndrome, a chronic non-pitting oedema of the hands attributed to lymphatic obliteration, may be mistaken for cellulitis (20). Disseminated candidal infection has been described among people who use heroin, and *Clostridium botulinum* spores may contaminate black tar heroin, giving rise to wound botulism (21,22).

Needle tracks and scarring over the dorsum of the hand and the antecubital fossa remain the principal external indicators of injection drug use, and the extent of scarring broadly correlates with the duration of use (19,21). Yellow-to-brown discolouration of the nails and fingers, most marked on the thumb, index and middle fingers of the dominant hand, is seen in people who smoke cannabis or heroin, reflecting staining by tar and combustion products (21). Delayed ulceration at former skin-popping sites has been reported; proposed mechanisms include microvascular and lymphatic injury and aggravation of pre-existing venous hypertension, and continued injection at the ulcer site cannot always be excluded (21,33).

4.6 Cannabis

Cannabis has both favourable and unfavourable dermatological associations, and the evidence base is less mature than for other substances. Cannabinoids have anti-inflammatory properties and may reduce transepidermal water loss, and have therefore attracted interest as potential treatments for inflammatory dermatoses; preclinical work has also examined cannabinoid-induced cytotoxic autophagy as a mechanism of melanoma cell death (23,27). Against this, cannabis use causes vasodilatation with transient flushing, and contact urticaria, angioedema and generalised pruritus have been reported as hypersensitivity responses (24). IgE-mediated cannabis allergy is increasingly recognised, which matters both for diagnosis and for patient counselling (24). Severe cutaneous adverse reactions, including a reported case of Stevens–Johnson syndrome following use of a commercial cannabidiol oil, have been described (25).

Cannabis arteritis is a rare but serious peripheral arterial disease affecting predominantly young, long-term heavy users and may progress to critical limb ischaemia (26). Hair shaft abnormalities have been reported in chronic cannabis users, though the supporting data are limited and require confirmation (26). Concerns also exist regarding carcinogenic exposure from cannabis smoke, including oral leukoplakia and erythroplakia, and an increased risk of head and neck malignancy when cannabis is used alongside tobacco (27). The relationship between heavy cannabis use and psychotic illness is separately established and is relevant to the psychodermatological considerations discussed below.

4.7 Inhalants

Inhalant misuse is under-reported, is comparatively prevalent among adolescents and young adults, and carries appreciable morbidity and mortality. Its most distinctive dermatological manifestation is the cryogenic burn produced by direct contact with rapidly expanding propellant. These burns are easily overlooked, particularly in settings where cold exposure is common, and their incidence is likely to rise with the increasing availability of inhalant products. Inhalant misuse should be considered in the differential diagnosis of a cryogenic burn in an adolescent or young adult, especially where the distribution is periorofacial or involves one hand (29).

Table 1. Summary of characteristic cutaneous and mucosal findings by substance class.

Substance	Characteristic cutaneous and mucosal findings	Principal mechanism
Alcohol	Spider telangiectasia; palmar erythema; flushing; unilateral nevoid telangiectasia;	Vasodilatation; hepatic dysfunction; nutritional

Substance	Characteristic cutaneous and mucosal findings	Principal mechanism
	jaundice; generalised pruritus with excoriation; urticaria and angioedema; hyperpigmentation; porphyria cutanea tarda; nutritional dermatoses; Terry's nails; Dupuytren's contracture	deficiency; immune suppression
Tobacco and vaping	"Smoker's face"; premature facial wrinkling; impaired wound healing; keratoacanthoma; psoriasis; hidradenitis suppurativa; allergic contact dermatitis; smoker's melanosis, palate and tongue; oral leukoplakia; androgenetic alopecia; premature greying	Microvascular damage; matrix metalloproteinase upregulation; TGF- β 1 depletion; carcinogen exposure
Methamphetamine and other stimulants	Excoriations ("meth mites"); delusional infestation; prurigo nodularis; formication; hyperhidrosis; cellulitis and abscess; fixed drug eruption (MDMA)	Formication and behaviourally mediated excoriation; vasoconstriction; injection-related infection
Cocaine	Acute multifocal skin necrosis; AGEP; "crack hands"; chronic ulceration; nasal septal and palatal perforation; retiform purpura (levamisole); pseudovasculitis; Raynaud phenomenon; midline destructive lesions	Vasoconstriction and ischaemia; adulterant-induced vasculopathy
Opioids and injection drug use	Pruritus and flushing; needle tracks and scarring; abscess and cellulitis; necrotising fasciitis; puffy hand syndrome; wound botulism; delayed skin-popping ulceration; nail and finger staining	Opioid receptor-mediated itch; inoculation of organisms; lymphatic and microvascular injury
Cannabis	Flushing; contact urticaria; angioedema; generalised pruritus; IgE-mediated allergy; cannabis arteritis; oral leukoplakia and erythroplakia; rare severe cutaneous adverse reactions	Vasodilatation; type I hypersensitivity; arteritis; combustion-product carcinogen exposure
Inhalants	Cryogenic (frostbite) burns, typically periorofacial or unilateral hand distribution; perioral dermatitis	Direct cryogenic injury from rapidly expanding propellant

AGEP, acute generalised exanthematous pustulosis; MDMA, 3,4-methylenedioxymethamphetamine; TGF- β 1, transforming growth factor beta 1.

5. Hair and nail changes

Nail changes are frequent in alcohol-related liver disease and can be classified by whether the abnormality lies in the nail plate or the nail bed. Nail bed discolouration is attributed to altered vascular flow and typically blanches with pressure on the fingertip. The best known of these changes is Terry's nails, reported in a high proportion of patients with cirrhosis, in which the proximal two-thirds of the nail appears white and the distal 1–2 mm remains pink. A central spider angioma is occasionally seen alongside depigmentation, indicating a vasoconstrictive state. Acquired haemochromatosis, which produces a greyish-metallic hue accentuated on photo-exposed skin, is distinct from the hyperpigmentation of alcoholic cirrhosis and may coexist with it (9).

Smoking is a recognised risk factor for androgenetic alopecia in men. The mechanism is multifactorial: cigarette smoke may impair the microvasculature of the hair papilla, smoke genotoxins may damage follicular DNA, and smoke-induced protease–antiprotease imbalance with increased pro-inflammatory cytokine production in anagen follicles may drive follicular microinflammation and fibrosis. Premature greying is also more common among smokers of both sexes (10).

6. Skin and soft tissue infection and vascular complications

Cellulitis, an infection of the dermis and subcutaneous tissue, is the commonest skin and soft tissue infection encountered in people who use drugs. Erysipelas, its more superficial counterpart, is usually streptococcal and presents as a raised, sharply demarcated inflamed plaque, characteristically on the face, ears or lower legs, with lymphatic involvement (31). Necrotising fasciitis is the most feared complication in this group; diagnosis remains primarily clinical and the early presentation overlaps with that of uncomplicated skin and soft tissue infection, though poorly defined margins and disproportionate swelling or pain should raise suspicion. Management follows established guidelines for skin and soft tissue infection, with early surgical assessment where necrotising infection is suspected (36).

Chronic venous disease is common in long-term injecting populations and results from injury to the venous system. It presents predominantly in the lower limbs with oedema, ulceration, discomfort, pruritus, skin thickening, pigmentary change and venous thrombosis, although individual presentations vary considerably (33). Cutaneous purpura in a person with a substance use disorder should prompt consideration of a drug-induced inflammatory response; as noted above, pseudovasculitis associated with cocaine or methamphetamine can be difficult to separate from autoimmune vasculitis on clinical and laboratory grounds alone (32,34).

7. The psychodermatology interface

The intersection of substance use, psychiatric illness and skin disease is bidirectional and clinically consequential. Formication and delusional infestation in stimulant users are psychiatric phenomena with dermatological manifestations; treating the excoriations without addressing the underlying perceptual disturbance and substance use rarely achieves durable improvement (11,14). Conversely, chronic inflammatory skin disease carries its own psychiatric burden: a systematic review and meta-analysis found significantly elevated rates of psychiatric comorbidity, completed suicide and substance use among patients with hidradenitis suppurativa (40). Despite this, psychocutaneous disease remains sparsely represented in dermatology training texts, which plausibly contributes to under-recognition in practice (30).

Two practical implications follow. First, any patient presenting with unexplained excoriations, delusional content, labile mood or other indicators of psychiatric disturbance warrants consideration of substance-induced cutaneous disease. The converse also holds. A patient with an established substance use disorder and a new skin eruption should not have every finding attributed to the substance, since a positive toxicology screen does not exclude a concurrent unrelated dermatosis (16,24). Second, dermatologists are well placed to identify psychiatric need that has not otherwise come to attention, and referral pathways should exist to act on that.

8. Screening, diagnosis and diagnostic pitfalls

Substance use screening in primary care relies on brief written, verbal or electronic instruments. The Substance Abuse and Mental Health Services Administration publishes the Treatment Improvement Protocol (TIP) series, of which TIP 24, a guide to substance abuse services for primary care clinicians, is directly relevant here. It sets out how such instruments should be selected and applied. Choice of instrument should account for patient acceptability, cost, ease of administration, and sensitivity and specificity. A significant advantage of screening within routine primary care is the opportunity to follow up a positive result at subsequent visits, and the continuity of the primary care relationship makes brief assessment feasible in a way that episodic specialist contact does not (35). Systematic use of validated instruments allows clinicians to identify at-risk patients and to initiate appropriate action, including referral to addiction services or dermatology.

Several factors make diagnosis difficult. Lesions associated with substance use mimic other dermatoses and may also arise from drug-drug interactions. Patients are frequently reluctant to disclose a substance history, and stigma, including previous negative experiences of healthcare, is a documented barrier to presentation and to candid history-taking (17). Polysubstance use and adulteration mean that attribution of a given lesion to a specific agent is often impossible; histopathology may assist, since injection sites in people who use heroin commonly show multinucleated giant cells, carbon particles, implantation epidermoid cysts, foreign body granulomas and histiocytic infiltrates, and polarised light may reveal birefringent talc or starch introduced with the injected material (21). For some substance classes there is no accessible confirmatory test: inhalant misuse cannot reliably be detected in blood or urine, so a high index of suspicion and, where appropriate, a structured screening inventory are required (29). Methamphetamine and amphetamine can be assayed in urine but are rapidly metabolised and are more prone to cross-reactivity than most other drugs of abuse, so a negative screen does not exclude recent use (11,14).

Once use is identified, cessation is the only definitive treatment for the dermatological component, and clinicians should counsel patients that cutaneous findings may persist for months or longer after abstinence is achieved (11). Screening is also warranted before planned procedures, where unrecognised substance use affects anaesthetic risk, wound healing and post-operative course (9).

9. Integrated and multidisciplinary management

Comprehensive care requires a treatment plan that addresses the substance use disorder and the dermatological disease together rather than sequentially. Interdisciplinary teams comprising primary care physicians, dermatologists, psychiatrists and addiction counsellors permit fuller assessment and more individualised planning, and collaborative care models applied to opioid use disorder in primary care have demonstrated feasibility and benefit (43,44). Coordinated communication across specialties improves both

treatment outcomes and patient satisfaction.

Several concrete measures follow. Harm reduction is central: education on safer injecting technique, wound care and hygiene, together with provision of sterile needles and dressings, reduces the incidence of injection-related skin and soft tissue infection, and syringe service programmes integrated with wound care have been proposed as an efficient way to deliver this (13). Guidance on the initial care of people who inject drugs recommends testing for HIV and hepatitis B and C, discussion of safer injection practice, and offer of opioid agonist therapy with buprenorphine or methadone where indicated (37,38,39,41). Incorporating basic dermatological education into addiction treatment settings raises awareness and may prompt earlier presentation for skin problems. Psychosocial support and counselling address the emotional, social and environmental drivers of use and equip patients with strategies for managing craving and avoiding relapse (45,46). Structured psychological intervention has an evidence base in smoking cessation among people with mental health problems, a group in which quit rates are otherwise poor (47), and integrated treatment of co-occurring substance use and psychiatric disorders is now the accepted standard (48). Dermatologists have a specific role in smoking cessation, since the cutaneous consequences of smoking are visible, personally salient and can be used constructively in motivational conversations (10).

Attention is also owed to settings where dermatological expertise is scarce. Community-oriented training and control programmes have been described as a practical means of managing skin disease in resource-limited environments, and the same approach is applicable to substance-related skin disease in populations with poor access to specialist care (42). Relapse prevention and long-term follow-up, comprising patient education, peer support, medication-assisted treatment and scheduled review, remain essential to sustaining gains in both domains.

10. Limitations

This review has several limitations. It is narrative rather than systematic: the search was not registered, study selection was not duplicated or blinded, and no formal quality appraisal or risk-of-bias assessment was undertaken, so selection and reporting bias cannot be excluded. The literature itself is uneven. Much of the evidence for substance-specific cutaneous findings derives from case reports, small case series and expert reviews rather than from controlled studies, and prevalence estimates therefore carry wide and often unstated uncertainty. Polysubstance use is the norm rather than the exception, which limits attribution of individual findings to individual agents. Publication is heavily weighted towards high-income settings, and the epidemiology cited here is predominantly North American and European; patterns of substance use, adulteration and access to care differ substantially elsewhere. Finally, the composition of illicit drug supplies changes rapidly, and adulterants such as levamisole in cocaine and xylazine in the opioid supply have introduced new cutaneous syndromes within the span of a few years, so any synthesis of this literature dates quickly.

11. Conclusion

The skin offers an accessible and frequently early window onto substance use, and its findings carry information about the agent involved, the route by which it was taken and the circumstances in which it was used. Alcohol, tobacco, stimulants, opioids, cannabis and inhalants each leave a recognisable if overlapping cutaneous signature, arising through vascular, immunological, infectious and behavioural mechanisms that are increasingly well characterised. Recognition is hampered by clinical mimicry, patient

non-disclosure, polysubstance use and the limits of available testing. These obstacles are better addressed by structured screening, non-judgmental history-taking and harm reduction than by dermatological treatment alone.

What follows for practice is a case for closer working between specialties that currently see these patients separately. Dermatologists encounter substance use disorders they are not equipped to treat; psychiatrists and addiction clinicians encounter skin disease they may not recognise; primary care sees both and is well placed to coordinate. Establishing referral pathways between them, embedding brief substance use screening in dermatology practice, and incorporating dermatological education into addiction services are modest, achievable steps. Further research is needed to place this field on firmer evidential ground, particularly prospective studies quantifying the prevalence and natural history of these findings, and evaluations of integrated care models.

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