

Cutaneous Excoriations and Severe Lung Injury Associated with Chronic Methamphetamine Abuse: A Case Report

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ABSTRACT

Background: Methamphetamine (METH) abuse is a growing public health problem with complex, multisystemic complications. While central nervous system and cardiovascular side effects are well known, cutaneous manifestations such as "meth sores" and non-cardiogenic toxic lung injury remain underrecognized, often leading to misdiagnosis or inappropriate microbial therapy. **Case Presentation:** A case is presented of a 43-year-old male with a history of chronic METH abuse and poorly controlled type 2 diabetes, admitted with progressive dyspnea and severe hypoxemia. Thoracic computed tomography demonstrated extensive alveolar consolidation and bilateral patchy alveolo-interstitial opacities. Concurrently, dermatological consultation and skin biopsy confirmed METH-induced skin excoriations ("meth sores"). Despite broad-spectrum antimicrobial coverage and intensive supportive care, the patient experienced progressive respiratory failure secondary to severe METH-induced toxic pneumonitis and ultimately succumbed to the illness. **Conclusions:** METH toxicity can present with severe, overlapping dermatological and pulmonary complications. Recognizing "meth sores" provides a crucial clinical key to suspecting substance abuse, avoiding unnecessary diagnostic procedures, and identifying METH-induced toxic pneumonitis in patients with refractory respiratory failure.

Keywords: *Methamphetamine, Meth sores, Excoriations, Toxic lung injury, Acute respiratory distress syndrome, Case report.*

INTRODUCTION

Methamphetamine (METH) abuse has established itself in recent decades as a critical public health issue with growing global repercussions. It is estimated that approximately 37 million people worldwide are active users of this psychomotor stimulant, ranking second among the most consumed illicit substances internationally. Chronic METH abuse not only leads to severe neuropsychiatric and behavioral alterations, but also produces a multisystemic organic impact characterized by cardiovascular, pulmonary, hepatic, and dermatological toxicity. Despite the constant increase in the availability and purity of this substance, many of its systemic clinical manifestations remain underdiagnosed in emergency departments and intensive care units.¹

At the cutaneous level, methamphetamine use triggers a broad spectrum of dermatological manifestations that frequently constitute the first visible stigmata of addiction. Among the most characteristic skin effects are marked xerosis, intense neurogenic pruritus, and the development of compulsive skin picking and scratching behaviors. These lesions, commonly known in the medical literature as "meth sores," result from a complex combination of central dopaminergic stimulation, underlying psychotic phenomena, tactile hallucinations (formication), and direct mechanical damage. Furthermore, the resulting excoriations usually present heterogeneous evolutionary stages on the face and extremities, often associated with secondary soft tissue infections and atrophic scarring.² In the psychiatric and psychodermatological fields, methamphetamine abuse is closely linked to the

emergence of delusional parasitosis or Ekbom syndrome. Under the effect of the drug-induced dopaminergic burst, patients experience persistent tactile hallucinations, describing the sensation of insects or parasites crawling under their skin a phenomenon popularly known as "meth mites" or "cocaine bugs." This false belief drives the individual to perform deep excoriations and intentional self-harm to "extract" the parasites, clinically deriving into conditions described as Morgellons disease. Due to the lack of specific histopathological or parasitic findings, early identification of this condition is essential to avoid unnecessary antiparasitic or biological treatments.³

Historically, the erroneous perception that cutaneous alterations due to methamphetamine constituted an exclusive disorder of modern medicine has been challenged by medical evidence. Clinical literature indicates that the psychosomatic phenomenon of delusional parasitosis and scratch lesions were conceptually observed since the 17th century by the English physician Sir Thomas Browne in 1674. Since then, various investigations in internal medicine and dermatology have categorized the "matchbox sign" or "specimen sign," where the patient attends consultation carrying small boxes or jars with crusts, fibers, or cellular debris as proof of their infestation. This behavior, added to traumatic excoriations, emphasizes the need for a multidisciplinary and comprehensive approach from hospital admission.⁴

From the perspective of public health and clinical epidemiology, the systemic implications of methamphetamine consumption extend beyond isolated dermatosis and reach a high rate of pulmonary morbidity and mortality. Large-scale epidemiological studies have shown that individuals with methamphetamine use disorder present a significantly higher risk of developing pulmonary arterial hypertension, as well as chronic parenchymal pathologies such as emphysema, lung abscesses, and empyema. Exposure to methamphetamine generates coronary and pulmonary vasoconstriction, oxidative stress, and endothelial remodeling that predispose to severe respiratory complications, substantially increasing hospitalization rates and the requirement for invasive ventilatory support.⁵

At the hospital level, pulmonary toxicity induced by methamphetamine abuse represents a priority diagnostic challenge due to its ability to mimic community-acquired infectious processes. Retrospective evaluations of hospital discharges demonstrate a solid statistical association between methamphetamine use and the development of acute pneumonia and acute respiratory failure in both sexes. The disruption of adaptive immune barriers and the impairment of pulmonary mucociliary clearance caused by methamphetamine vapor or smoke favor the deterioration of lung function. This non-cardiogenic toxic pneumonitis typically debuts with ground-glass

opacities and an alveolo-interstitial pattern that responds poorly to conventional antimicrobial regimens.⁶

Pathophysiologically, vascular and parenchymal damage secondary to methamphetamine consumption involves complex cellular mechanisms that condition a somber prognosis. The selective accumulation of the substance in lung tissue induces direct cellular toxicity, mitochondrial dysfunction, and overproduction of reactive oxygen species, leading to pulmonary vascular endothelial dysfunction. Prospective clinical studies have shown that methamphetamine-associated pulmonary arterial hypertension (Meth-APAH) presents a more aggressive phenotype, with worse right ventricular hemodynamics, greater dilation of right cavities, and a significantly lower event-free survival compared to idiopathic forms.⁷

Finally, the spectrum of acute lung injury induced by methamphetamine can lead to fulminant conditions such as acute respiratory distress syndrome (ARDS) and diffuse alveolar damage. Crystalline methamphetamine inhalation promotes the breakdown of the alveolocapilar barrier, increased vascular permeability, and rapidly installing acute non-cardiogenic pulmonary edema. In clinical practice, these patients usually present with severe refractory hypoxemia, requiring mechanical ventilation and urgent hemodynamic support. Timely recognition of the association between torpid respiratory failure and cutaneous stigmata ("meth sores") is essential to establish the diagnosis of exclusion, rule out unfounded infectious suspicions, and optimize intensive medical management.⁸

CASE PRESENTATION

A 43-year-old male presented with progressive dyspnea, dry cough, and severe hypoxemia (SpO₂ 84% on room air, requiring oxygen via a non-rebreather mask at 6 L/min). His history was remarkable for poorly controlled type 2 diabetes, occupational organic dust exposure, and daily crystalline methamphetamine inhalation over five years.

Initial laboratory workup revealed leukocytosis ($12.93 \times 10^3/\mu\text{L}$), marked procalcitonin elevation (PCT: 6.48 ng/mL), and respiratory alkalosis with moderate hypoxemia (PaO₂/FiO₂ 140 mmHg). Empirical antimicrobial coverage for severe community-acquired pneumonia was initiated with intravenous levofloxacin. During the first week of hospitalization, the patient required parenteral electrolyte replacement for severe hypokalemia (nadir 2.1 mEq/L) and glycemic optimization with basal-bolus insulin.

Despite a steady decline in PCT levels (0.57 ng/mL) and broad-spectrum antimicrobial escalation including voriconazole, oseltamivir, meropenem, and clarithromycin after ruling out co-infections via serial negative acid-fast bacilli (AFB) smears, respiratory

viral multiplex polymerase chain reaction (PCR) tests, and non-reactive serologies for human immunodeficiency virus (HIV), hepatitis B virus (HBV), hepatitis C virus (HCV), and Venereal Disease Research Laboratory (VDRL) testing, oxygen dependence persisted. Chest computed tomography (CT) demonstrated extensive left lower lobe alveolar consolidation with air bronchograms, alongside bilateral patchy ground-glass opacities and peribronchovascular thickening, consistent with toxic pneumonitis.

Concurrently, dermatology evaluation was requested for nummular excoriations (0.3–0.7 cm) on the face and hands with hemorrhagic crusts. The patient admitted to compulsive scratching driven by methamphetamine-induced formication. A punch biopsy confirmed diffuse epidermal sloughing and superficial papillary microhemorrhages without vasculitis, granulomas, or specific infection, corroborating the diagnosis of "meth sores."

Upon recognizing methamphetamine-induced toxic lung injury and psychomotor dermatosis, antimicrobials were safely weaned. The clinical evolution across hospitalization phases and corresponding biomarker trajectories are summarized in Table 1.

Table 1. Clinical Phases and Laboratory Trajectory

Parameter / Variable	Phase 1. Admission	Phase 2. Antibiotics Failure	Phase 3. METH Toxicity
Period	Days 1–3	Days 6–10	Days 12–20
Clinical Findings	Dyspnea, hypoxemia, severe Community-Acquired Pneumonia diagnosis. Empirical antibiotic initiation.	Persistent hypoxemia and O ₂ dependence. Escalation to anti fungal/antivirals.	O ₂ dependence, CT pneumonitis findings, and "meth sores" diagnosis.
Procalcitonin (ng/mL)	6.48	1.17 → 0.57	0.93 → 0.48
White blood cells (×10 ³ /μL)	12.93 → 8.92	14.57 → 3.81	2.31 → 15.25
Hemoglobin (g/dL)	14.6 → 11.8	11.4 → 8.9	7.7 → 8.1
Platelets (×10 ³ /μL)	229 → 171	121 → 150	163 → 543
C-reactive protein (mg/L)	—	1.08	254.5 → 82
Interpretation	Marked procalcitonin elevation from acute bacterial process.	Procalcitonin reduction uncoupled from clinical respiratory failure.	Persistent inflammation secondary to non-infectious toxicity.

METHODS

Study Design and Setting

This is a descriptive, single-center clinical case report conducted at a secondary-level hospital. The manuscript was developed following the CARE (CAse REport) guidelines to ensure systematic and transparent reporting of clinical data.

Data Collection and Patient Evaluation

Clinical, laboratory, imaging, and histopathological data were retrieved from the patient's medical records upon admission and throughout the 20-day hospital stay. Diagnostic evaluation included serial arterial blood gas analyses, acute-phase reactants (procalcitonin and C-reactive protein), complete blood counts, metabolic panels, and comprehensive microbiological screening (gastric juice acid-fast bacilli smears, multiplex PCR for respiratory viruses including SARS-CoV-2 and Influenza A/B, and serological testing for HIV, HBV, HCV, and VDRL).

Imaging and Histopathology

Thoracic computed tomography (CT) was performed in axial and coronal reconstructions (lung window) to evaluate parenchymal involvement. Dermatological evaluation was supported by a 4-mm punch skin biopsy of active facial excoriations. Tissue specimens were fixed in 10% buffered formalin, paraffin-embedded, sectioned at 4 μm, and stained with Hematoxylin and Eosin (H&E) for histopathological analysis (10x and 40x magnifications).

Ethical Considerations and Anonymization

Written informed consent was obtained from the patient for the publication of this case report, including relevant clinical details, laboratory findings, radiological images, and histopathological photographs. All personal identifiers, specific dates, and location details were strictly removed or generalized to protect patient confidentiality in compliance with the Declaration of Helsinki and international data privacy regulations (HIPAA/GDPR).

RESULTS

Diagnostic Workup and Biochemical Trajectory

Upon admission, initial laboratory testing demonstrated systemic inflammatory response markers, including a PCT level of 6.48 ng/mL, leukocytosis (12.93×10³/μL) with absolute neutrophilia (11.95×10³/μL), and moderate hypoxemia (PaO₂/FiO₂ ratio of 140 mmHg). Serial microbiological investigations—including repeated gastric juice AFB smears, respiratory viral multiplex PCR (SARS-CoV-2, Influenza A/B), and serological panels (HIV, HBV, HCV, VDRL) were negative. Despite escalating empirical antimicrobial coverage to meropenem, clarithromycin, voriconazole, and oseltamivir, the patient exhibited persistent oxygen dependence, contrasting with a steady decline in PCT

to 0.57 ng/mL and a secondary surge in CRP up to 254.5 mg/L.

Thoracic Imaging Findings

Chest computed tomography (CT) confirmed severe, non-cardiogenic pulmonary parenchymal involvement secondary to inhaled methamphetamine toxicity.

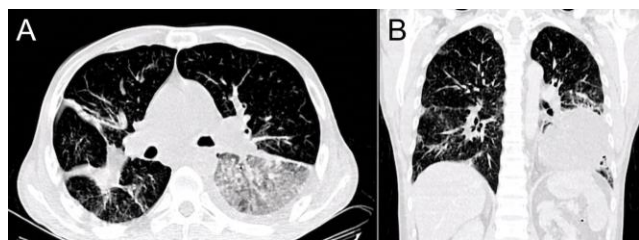


Figure 1. Thoracic Computed Tomography Findings in Methamphetamine-Induced Toxic Pneumonitis (A) Axial and (B) coronal chest computed tomography (CT) reconstructions in lung window demonstrating extensive alveolar consolidation in the left lower lobe accompanied by prominent air bronchograms. Bilateral patchy alveolo-interstitial opacities with ground-glass attenuation and basal peribronchovascular thickening are present in the right lung, showing asymmetric distribution without evidence of cavitation, lymphadenopathy, or pneumothorax.

Dermatological Evaluation and Histopathology

Dermatological consultation identified a widespread eruption predominantly involving the face, chin, forehead, and dorsal hands, characterized by nummular excoriations (0.3 to 0.7 cm in diameter) with irregular borders, hemorrhagic crusts, and post-inflammatory hyperpigmentation in varying evolutionary stages, devoid of primary bullous or vesicular lesions.

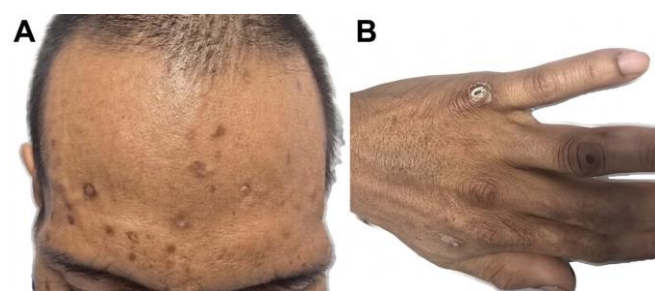


Figure 2. Clinical Manifestation of Methamphetamine-Induced Skin Excoriations ("Meth Sores") (A) Disseminated facial dermatosis displaying multiple erythematous, nummular excoriated lesions with hemorrhagic crusts across the forehead, cheeks, and chin. (B) Dorsal hand view demonstrating discrete, crusted excoriations with surrounding erythema resulting from compulsive skin picking secondary to methamphetamine-induced formication.

Histopathological analysis of a punch skin biopsy confirmed the diagnosis of traumatic excoriations secondary to psychomotor agitation, excluding

infectious dermatoses, vasculitis, or primary autoimmune skin disorders.

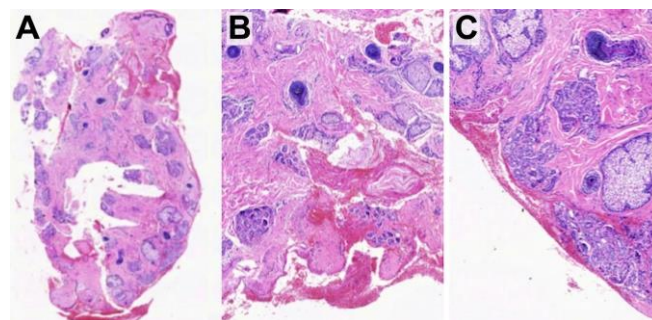


Figure 3. Histopathology of Methamphetamine-Induced Excoriations ("Meth Sores") (A–C) Photomicrographs of skin biopsy sections stained with Hematoxylin and Eosin (H&E, 10x magnification) showing diffuse epidermal sloughing with complete loss of the stratum corneum and spinous layer. Superficial microhemorrhages and focal erythrocyte extravasation extending into the papillary dermis are observed. Dermal appendages retain normal histological architecture, and the reticular dermis exhibits discrete thickening of collagen bundles without evidence of leukocytoclastic vasculitis, granulomatous inflammation, or specific microbial pathogens.

DISCUSSION

Methamphetamine (METH) is a potent psychomotor stimulant whose global consumption continues to increase, posing a severe public health concern. Although central nervous system and cardiovascular side effects are widely recognized, cutaneous manifestations remain poorly documented in the medical literature. "Meth sores" represent a characteristic dermatological finding resulting from a multifactorial mechanism involving neurogenic pruritus, central dopaminergic stimulation, tactile hallucinations (formication), and compulsive skin-picking behaviors. These excoriations typically present on the face and extremities in varying evolutionary stages with hemorrhagic crusts, often posing diagnostic confusion with infectious dermatoses, ecthyma, or dermatitis artefacta.^{1,2}

The psychodermatological spectrum of METH abuse frequently overlaps with delusional parasitosis and Ekbom syndrome, where patients experience intense tactile hallucinations of insects crawling under the skin, colloquially termed "meth mites" or "cocaine bugs". In severe cases, this somatic delusion drives patients to self-inflict deep excoriations to remove imagined fibers or organisms, culminating in Morgellons disease. Historically, the perception of skin infestation and specimen-collecting behavior known as the "matchbox sign" was described as early as the 17th century by Sir Thomas Browne. Early recognition of these cutaneous stigmata provides a critical clinical clue to identify active substance abuse,

avoiding unnecessary diagnostic workups or inappropriate antiparasitic and biological therapies.^{3,4}

Beyond dermatological manifestations, chronic METH exposure induces severe systemic toxicity, with a substantial burden on the pulmonary system. Population-based studies demonstrate that individuals with METH use disorder face significantly higher risks of developing pulmonary arterial hypertension (PAH) as well as parenchymal lung diseases, including emphysema, lung abscesses, empyema, and pneumonia. The pharmacokinetics of METH show selective accumulation and high uptake within pulmonary tissue, predisposing the lung vasculature and parenchyma to direct toxic injury, severe oxidative stress, and increased susceptibility to pulmonary infections.⁵

Hospital discharge evaluations confirm a strong statistical association between METH use and acute respiratory failure, acute pneumonia, and COPD exacerbations. Inhaled crystalline METH promotes mucosal breakdown, impairs adaptive T-cell immunity, and alters mucociliary clearance, facilitating opportunistic microbial invasion and non-cardiogenic pulmonary edema. When evaluating patients with severe, non-responsive respiratory distress, obtaining a thorough METH exposure history is essential, as METH-induced toxic pneumonitis can closely mimic community-acquired pneumonia and result in refractory oxygen dependence despite broad-spectrum antimicrobial coverage.⁶

Prospective clinical and hemodynamic studies demonstrate that methamphetamine-associated pulmonary arterial hypertension (Meth-APAH) exhibits a distinct, highly aggressive phenotype compared to idiopathic PAH. Patients with Meth-APAH present with worse right ventricular dysfunction, higher right atrial pressures, lower stroke volume index, and markedly reduced event-free survival. Molecular investigations suggest a genetic "two-hit" model, where loss-of-function variants in drug-detoxifying enzymes, such as carboxylesterase 1 (CES1), impair endothelial protection against METH-induced reactive oxygen species and autophagy, accelerating severe vascular remodeling.⁷

Acute inhalation of crystalline METH can trigger rapid alveolar-capillary membrane disruption, presenting as acute respiratory distress syndrome (ARDS), diffuse alveolar hemorrhage, or acute eosinophilic pneumonia. Due to its high lipophilicity, inhaled METH achieves rapid systemic absorption and peak concentrations, driving free radical-mediated pulmonary epithelial damage. In clinical scenarios where empirical antibacterial, antiviral, and antifungal therapies fail to resolve respiratory failure, identifying concurrent "meth sores" provides the decisive diagnostic bridge to confirm METH-induced multisystem toxicity, allowing timely de-escalation of unnecessary antimicrobials and initiation of supportive care.⁸

CONCLUSION

Cutaneous excoriations ("meth sores") serve as a critical diagnostic biomarker of active methamphetamine abuse. Recognizing these characteristic skin lesions in patients presenting with severe, unexplained respiratory distress provides an essential clinical key to suspecting substance-induced toxicity rather than attributing the clinical picture exclusively to a primary microbial infection.

Methamphetamine-induced toxic pneumonitis can closely mimic severe community-acquired pneumonia, leading to persistent oxygen dependence, rapid progression to respiratory failure, and high mortality despite broad-spectrum antimicrobial regimens. Early identification of substance-induced non-cardiogenic lung injury is crucial to prevent redundant diagnostic workups, avoid unnecessary antimicrobial escalation, and optimize early supportive care.

Optimal management of methamphetamine multisystem toxicity requires an integrated, multidisciplinary approach involving internal medicine, pulmonology, dermatology, critical care, and addiction psychiatry. Prompt recognition of these overlapping toxic complications is vital to attempt timely intervention in this highly lethal condition.

LEARNING POINTS

- Methamphetamine abuse can produce simultaneous cutaneous and pulmonary manifestations, which may provide important diagnostic clues to systemic drug toxicity.
- Characteristic excoriated lesions ("meth sores") should raise suspicion of methamphetamine-associated formication and compulsive skin-picking behavior in the appropriate clinical setting.
- Methamphetamine-induced toxic pneumonitis may closely mimic severe community-acquired pneumonia and can result in persistent hypoxemia despite broad-spectrum antimicrobial therapy.
- A declining procalcitonin level together with persistent respiratory failure and negative microbiological investigations should prompt consideration of non-infectious toxic lung injury.
- Early recognition of methamphetamine-associated multisystem toxicity and coordinated management involving relevant clinical specialties may help avoid unnecessary antimicrobial escalation and facilitate appropriate supportive care.

DECLARATIONS

Ethics Approval and Consent to Participate

Written informed consent was obtained from the patient for the publication of the clinical case and associated medical information. The case was reported in accordance with the principles of the Declaration of

Helsinki and applicable institutional and international ethical standards.

Consent for Publication

Written informed consent was obtained from the patient for publication of this case report, including relevant clinical details, laboratory findings, radiological images, and histopathological photographs. All potentially identifying information was removed or generalized to protect patient confidentiality.

Availability of Data and Materials

The clinical data supporting the findings of this case report are contained within the manuscript. Additional patient-level information is not publicly available because of patient privacy and confidentiality considerations.

Competing Interests

The authors declare that they have no competing interests related to this case report.

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Authors' Contributions

All authors contributed to the clinical management, interpretation of clinical findings, preparation and critical revision of the manuscript, and approved the final version for publication.

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