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# Crusted Scabies as a Complication of Severe Endogenous Hypercortisolism Due to Occult Ectopic ACTH Syndrome: A Case Report

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**Case Report**

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## Abstract

Crusted scabies is a severe, highly contagious variant of scabies that occurs predominantly in immunocompromised individuals. Severe endogenous hypercortisolism caused by ectopic adrenocorticotropic hormone (ACTH) syndrome results in profound immune dysfunction and increases susceptibility to opportunistic infections. However, crusted scabies associated with occult ectopic ACTH syndrome has not previously been reported.

A 52-year-old man presented with progressive weight gain, peripheral edema, and cushingoid features. Biochemical and dynamic endocrine testing confirmed ACTH-dependent hypercortisolism, while pituitary evaluation and extensive imaging failed to identify the ACTH source, leading to the diagnosis of occult ectopic ACTH syndrome. During hospitalization, hyperkeratotic lesions on the hands and feet were diagnosed as biopsy-confirmed crusted scabies. The patient was treated with oral ivermectin and topical permethrin, while hypercortisolism was managed with staged bilateral adrenalectomy and metyrapone therapy. Cutaneous lesions regressed following antiparasitic treatment, accompanied by progressive metabolic improvement.

This case highlights crusted scabies as a rare infectious complication of severe endogenous hypercortisolism and emphasizes the importance of early recognition of atypical parasitic infections in immunocompromised patients.


## Introduction

Crusted scabies is a rare and highly contagious variant of Sarcoptes scabiei var. hominis infestation characterized by hyperkeratotic skin lesions containing an extraordinarily high mite burden.[1] It occurs predominantly in immunocompromised individuals and has been associated with conditions such as diabetes mellitus, HIV infection, organ transplantation, hematological malignancies, and prolonged corticosteroid or immunosuppressive therapy. Owing to its atypical clinical presentation, crusted scabies may mimic other dermatological disorders, leading to delayed diagnosis and increased risk of secondary bacterial infection and disease transmission.[2,3]

Cushing syndrome is a disorder of chronic glucocorticoid excess resulting from either exogenous corticosteroid exposure or endogenous cortisol overproduction. Endogenous Cushing syndrome is rare, with an estimated annual incidence of 2–8 cases per million population.[4] It is associated with substantial morbidity due to multisystem complications, including metabolic, cardiovascular, musculoskeletal, neuropsychiatric, and characteristic dermatological manifestations.[5]

The risk of infection is closely related to the severity of hypercortisolism, and opportunistic infections are well-recognized complications of Cushing syndrome.[6] Excess glucocorticoids impair both innate and adaptive immune responses while inhibiting keratinocyte and dermal fibroblast proliferation, resulting in impaired collagen synthesis, skin atrophy, delayed wound healing, and disruption of the cutaneous

barrier.[7,8] Characteristic cutaneous findings include facial plethora, easy bruising, wide violaceous striae, skin fragility, ecchymoses, acne, hyperpigmentation, and superficial fungal infections.[7,9] These immunological and cutaneous alterations provide a plausible biological basis for the development of crusted scabies. Although crusted scabies has been reported in association with exogenous corticosteroid therapy and other immunosuppressive conditions, its occurrence in patients with endogenous hypercortisolism remains exceptionally rare. Accordingly, we describe a patient with occult ectopic ACTH-dependent Cushing syndrome who developed biopsy-confirmed crusted scabies, highlighting severe parasitic infestation as a rare infectious complication of endogenous hypercortisolism.

## Case Presentation

A 52-year-old man was admitted with progressive weight gain, facial and peripheral edema, and cushingoid features. His medical history included hyperthyroidism, type 2 diabetes mellitus, hypertension, hyperlipidemia, previous sleeve gastrectomy, vertebral fracture, and a pulmonary nodule under radiological follow-up. On examination, he had a body mass index of 32 kg/m², facial plethora, supraclavicular fullness, dorsocervical fat accumulation, wide violaceous abdominal striae, skin atrophy with multiple ecchymoses, and hyperkeratotic crusted lesions involving both palms and soles (Fig. 1). Blood pressure was elevated, and laboratory investigations demonstrated severe hypokalemia (2.9 mmol/L) and poor glycemic control (HbA1c 9.5%).

Endocrine evaluation confirmed ACTH-dependent hypercortisolism. Basal serum cortisol, ACTH, late-night salivary cortisol, midnight serum cortisol, and 24-hour urinary free cortisol levels were markedly elevated. Cortisol levels failed to suppress adequately following both low-dose and high-dose dexamethasone suppression tests, suggesting ectopic ACTH syndrome.

Pituitary magnetic resonance imaging revealed a 6.5 × 3.5 mm pituitary lesion; however, bilateral inferior petrosal sinus sampling excluded a pituitary source of ACTH secretion. The absence of a central-to-peripheral ACTH gradient on IPSS indicated that this lesion most likely represented a non-functioning incidentaloma. Extensive investigations, including contrast-enhanced thoracoabdominal computed tomography, adrenal magnetic resonance imaging, fluorodeoxyglucose positron emission tomography-computed tomography (FDG PET-CT), gallium-68 PET-CT, upper gastrointestinal endoscopy, colonoscopy, and laboratory evaluation for malignancy failed to identify an ACTH-secreting tumor. The patient was therefore diagnosed with occult ectopic ACTH syndrome.

During hospitalization, persistent pruritus and progressive hyperkeratotic lesions of the hands and feet prompted dermatological consultation. Clinical examination suggested crusted scabies, which was confirmed by skin biopsy demonstrating intracorneal scabies mite structures, epidermal acanthosis, orthokeratosis, and eosinophil-rich inflammatory infiltration. Because of the highly contagious nature of the disease, the patient was immediately placed under contact isolation.

Treatment consisted of topical keratolytic therapy followed by 5% permethrin cream and oral ivermectin (200 μg/kg, 18 mg), with repeated administration after one week. Progressive regression of the cutaneous lesions was observed during follow-up (Fig. 2).

Meanwhile, severe endogenous hypercortisolism was managed with potassium replacement, spironolactone, thromboprophylaxis, and trimethoprim-sulfamethoxazole prophylaxis. Despite prophylactic measures, the patient developed bacterial pneumonia during hospitalization, which was managed with targeted antimicrobial therapy and supportive care. Because hypertension, hypokalemia, and hypercortisolism remained difficult to control despite intensive medical management, bilateral adrenalectomy was planned. Owing to intraoperative hemodynamic instability, only left adrenalectomy was initially performed. Histopathological examination demonstrated adrenal cortical hyperplasia and myelolipoma. Persistent postoperative hypercortisolism necessitated metyrapone therapy as bridging treatment. Following metabolic stabilization, completion contralateral adrenalectomy was successfully performed approximately one month later. At follow-up, blood pressure, serum potassium, and glycemic control had improved substantially, while the crusted scabies lesions had almost completely resolved after antiparasitic treatment (Fig. 3).

## Discussion

Occult ectopic ACTH syndrome represents one of the most challenging forms of endogenous Cushing syndrome because the source of ACTH secretion may remain unidentified despite extensive biochemical and radiological evaluation. Approximately 20% of ectopic ACTH-secreting tumors remain occult despite advances in diagnostic modalities.[10] In our patient, bilateral inferior petrosal sinus sampling excluded a pituitary source of ACTH secretion, while comprehensive radiological investigations failed to identify an ectopic tumor, supporting this diagnosis.

Crusted scabies develops almost exclusively in individuals with impaired immune function. Excess glucocorticoids suppress both innate and adaptive immune responses, impair keratinocyte proliferation and collagen synthesis, and disrupt skin barrier integrity, thereby increasing susceptibility to opportunistic infections.[6,9] These mechanisms likely contributed to the development of crusted scabies in our patient, who exhibited severe biochemical hypercortisolism accompanied by uncontrolled diabetes mellitus, hypokalemia, skin atrophy, easy bruising, and extensive violaceous striae.

Although crusted scabies has been reported in association with exogenous glucocorticoid exposure and other immunosuppressive conditions, reports occurring in the setting of endogenous hypercortisolism remain exceptionally rare.[3] To the best of our knowledge, this is the first reported case of biopsy-confirmed crusted scabies in a patient with occult ectopic ACTH syndrome. Our findings therefore expand the spectrum of infectious complications associated with severe endogenous hypercortisolism.

Recognition of crusted scabies in patients with severe hypercortisolism may be challenging because hyperkeratotic lesions can mimic psoriasis, chronic eczema, palmoplantar keratoderma, or other

inflammatory dermatoses. Delayed diagnosis may result in secondary bacterial infection, nosocomial transmission, and increased morbidity. This case highlights the importance of early dermatological consultation, prompt histopathological confirmation, implementation of isolation precautions, and timely initiation of antiparasitic therapy.

Management required close collaboration between endocrinologists, dermatologists, pathologists, surgeons, and infectious disease specialists. While antiparasitic treatment led to rapid regression of the cutaneous lesions, definitive improvement depended on effective control of hypercortisolism through staged bilateral adrenalectomy and bridging metyrapone therapy. Because the primary ACTH-secreting tumor may become detectable during follow-up, continued clinical and radiological surveillance remains essential.

## Conclusions

This case expands the spectrum of infectious complications associated with endogenous hypercortisolism and demonstrates that crusted scabies may represent a rare but clinically significant dermatological complication of severe cortisol excess. Furthermore, given the possibility that an underlying occult tumor source may become apparent following the control of hypercortisolism, long-term clinical and radiological follow-up should be maintained in these patients. Clinicians should maintain a high index of suspicion for crusted scabies in patients with severe endogenous hypercortisolism presenting with atypical dermatological findings. A multidisciplinary and coordinated approach is essential for early diagnosis, timely initiation of effective treatment, implementation of appropriate isolation measures, and prevention of nosocomial transmission.

## Declarations

# Conflict of Interest Statement

The authors declare no conflicts of interest.

**Ethics approval and consent to participate**

This case report was conducted in accordance with the principles of the Declaration of Helsinki. According to the institutional policy of the University of Health Sciences, Prof. Dr. Cemil Taşcıoğlu City Hospital, Istanbul, ethics committee approval was not required for the publication of this single-patient case report.

**Consent for publication**

Written informed consent was obtained from the patient for publication of this case report and any accompanying clinical images.

# Competing interests

The authors declare that they have no competing interests.

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No funding was received for this study.

# Author Contribution

Z.A.K. and K.Ş. contributed to the clinical care and follow-up of the patient during hospitalization. Ü.A.T. contributed to the outpatient follow-up of the patient. E.A. performed the dermatological evaluation of the patient. H.D.Y. drafted the manuscript, and S.D.Ç. contributed to its critical revision. M.A. performed the final critical review of the manuscript. All authors read and approved the final manuscript.

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Not applicable.

# Data Availability

The data supporting the findings of this case report are included within the article. Additional patient-level data are not publicly available to protect patient privacy and confidentiality.

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## Figures




**Figure 1**

Cutaneous manifestations of hypercortisolism at initial presentation. (A) Abdominal view demonstrating wide violaceous striae, diffuse ecchymoses, and central obesity. (B) Dorsocervical fat accumulation (buffalo hump). (C) Bilateral upper extremities showing extensive ecchymoses and markedly thinned skin, reflecting severe skin fragility and impaired collagen synthesis secondary to hypercortisolism.




**Figure 2**

Clinical findings of crusted scabies at diagnosis. (A–B) Hyperkeratotic crusted lesions on the plantar surfaces with periungual involvement, demonstrating the characteristic mite burden distribution of crusted scabies. (C) Bilateral dorsal hand involvement with hyperkeratotic plaques and scattered ecchymoses reflecting underlying skin fragility. (D) Auricular involvement with crusted hyperkeratotic lesions, representing an atypical but well-recognized distribution site of crusted scabies.




**Figure 3**

Resolution of crusted scabies lesions following completion of antiparasitic therapy. (A–B) Marked regression of hyperkeratotic plantar lesions with residual desquamation. (C) Significant improvement of bilateral hand lesions with persistence of underlying skin atrophy secondary to hypercortisolism. (D) Complete resolution of auricular crusted lesions.