Scabies

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Key Points

- Scabies is a common infestation of the skin caused by *Sarcoptes scabiei*. Scabies can affect individuals of any age and socioeconomic status. Crowded conditions increase the risk of scabies infestation.
- Female scabies mites create burrows in the skin where they lay two to three eggs per day.
- The two major clinical variants of scabies are classic scabies and crusted scabies. Classic scabies, the most common form, is associated with a relatively low mite burden. Crusted scabies usually occurs in immunocompromised individuals and is associated with a much higher mite burden (up to millions).
- Transmission of scabies usually occurs through direct and prolonged skin-to-skin contact. Transmission via fomites is uncommon in classic scabies but is more likely to occur in crusted scabies.
- The characteristic clinical findings of classic scabies are intense pruritus and multiple small, erythematous papules that are often excoriated. Burrows may be visible as serpiginous lines. Larger papules (nodular scabies) appear less commonly.
- Crusted scabies typically manifests as erythematous patches that develop prominent scales, crusts, and fissures. Pruritus may be minimal or absent.
- A diagnosis of scabies may be strongly suspected based upon patient history and physical examination. The diagnosis is confirmed through the detection of scabies mites, eggs, or feces through microscopic examination. Dermoscopic examination is a very helpful diagnostic tool.
- Simultaneous treatment of both the patient and close personal contacts is recommended. Topical 5% permethrin lotion and oral ivermectin are the most common first-line treatments.

General Principles

Scabies is an ectoparasitic dermatosis caused by *Sarcoptes scabiei* var. *hominis*, an obligate microscopic parasitic mite that lives its entire 10–14 day life cycle in the human epidermis.

Globally, more than 200 million people are affected with a particularly high prevalence in resource-poor tropical regions. In developed countries, scabies outbreaks are frequent in resi-

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dential and nursing care homes where they cause significant morbidity and distress.

The life cycle of the scabies mite begins with the pregnant female burrowing into the stratum corneum and laying its eggs. They continue to extend their burrow and lay two to three eggs per day before dying after 4–6 weeks. Larvae emerge after 48–72 h and form new burrows. Human scabies mites are capable of surviving in the environment, outside of the human body, for 2 or 3 days in normal room conditions; during this time, they remain capable of infestation.

Prolonged skin-to-skin contact is the primary mode of transmission. Transmission via fomites may be more important in crusted scabies, wherein mites are more numerous and survive in large numbers.

Scabies mites induce a cutaneous hypersensitivity reaction to themselves and their products. This results in generalized pruritus that is worse at night. Pruritus may be mediated by non-histaminergic itch mechanisms, including tryptase, its receptor, protease-activated receptor-2 (PAR), ion channel transient receptor potential cation channel subfamily V member 1 (TRPV1), and transient receptor potential cation channel subfamily A member 1 (TRPA1).

Pruritus can be severe, negatively impacting the quality of life. However, sensitization to mite antigens occurs 4–6 weeks after the initial manifestation and therefore asymptomatic carriage is common during this period. With reinfestation, itching begins within days and presentations may be more severe.

Clinical Presentation

Scabies presents with multiple morphologies and the differential diagnosis varies by clinical subtype. The major clinical variants of scabies are classic scabies and crusted scabies.

Classic Scabies

Classic scabies is typically described as an intense, intractable, generalized pruritus, which is worse at night. Occasionally, patients are asymptomatic.

Clinically, it is characterized by a generally symmetrical erythematous papulo-vesicular rash, with a predilection for the volar surface of the wrists, interdigital web space, axillae, periumbilical area, groins, buttocks, genitals, and the breasts in women. It does not affect the head and face in adults.

In infants and young children, the palms, soles, and head are more commonly involved.

The pathognomonic lesions are the skin burrows and scabietic nodules.

The burrow is visible as a short serpiginous gray line culminating with an intact or eroded vesicle/pustule containing the mite. It usually can be found on the hands and feet, particularly on the finger web spaces.

Nodular scabies is a less common manifestation of classic scabies. Nodular scabies is characterized by persistent, firm, erythematous, extremely pruritic, nodules located on the glans, scrotum, thighs, and axillae. The nodules may represent a hypersensitivity reaction to prior or currently active scabies infestation.

More commonly, nonspecific secondary lesions are seen, including excoriated papules, eczematous plaques, and impetigo. Prolonged scratching can result in lichenification and prurigo nodularis.

Lesions in children are usually more inflammatory than in adults and often are vesicular or bullous. Clinically, brownish to pinkish nodules, vesicles, often accompanied by plaques, pustules, or nodules, are found. Acral pustules are common. The appearance of the rash is often altered secondary to a bacterial superinfection or topical steroid use. Very young babies do not scratch and may look irritable and feed poorly.

Crusted Scabies

Crusted scabies was previously known as Norwegian scabies because it was first identified in Norway in 1848 among patients with leprosy.

It is also known as scabies crustosa, Boeck scabies, or keratotic scabies and often occurs in the presence of conditions that compromise cellular immunity, such as acquired immunodeficiency syndrome (AIDS), human T cell lymphotropic virus type 1 (HTLV-1) infection, lymphoma, and in the elderly.

In crusted scabies, the host may be colonized with many millions of mites, in contrast to classical scabies in which the host will harbor on average 10–15 mites.

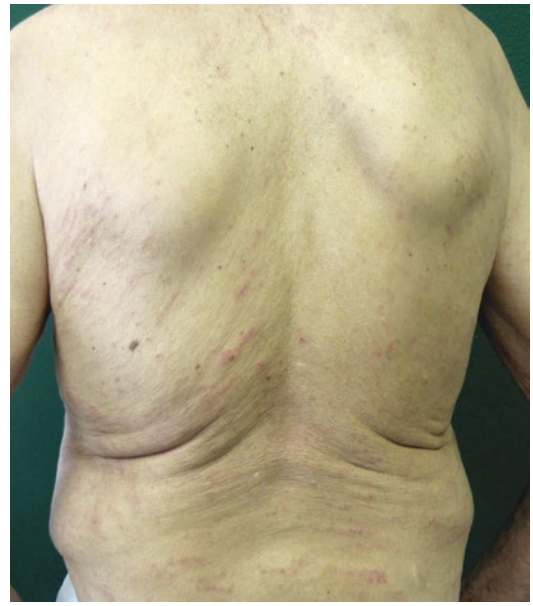
Clinically, crusted scabies presents as a hyperkeratotic dermatosis, typically involving the palms and soles, often with deep skin fissures that provide a portal of entry for bacteria. It begins with poorly defined, erythematous patches that quickly develop prominent scaling.

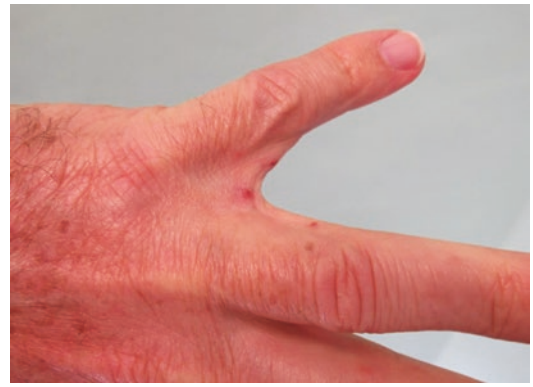
Firm red-brown and violaceous nodules can occur on the axillae, groin, male genitalia, and trunk and often persist for months after treatment. Any skin area may be affected, but the scalp, hands, and feet are particularly susceptible. Because of the altered immune response in immunocompromised subjects, pruritus may be minimal or absent. The crusts contain a large number of mites and so when they flake off they contaminate clothing, linen, curtains, walls, floor, furniture, and the immediate environment, where the mite remains infective for 2–3 days.

If untreated, the disease usually spreads inexorably and may eventually involve the entire integument. Generalized lymphadenopathy, peripheral blood eosinophilia, and raised serum IgE levels are frequently observed, and secondary bacterial infection is common and associated with a significant mortality.

Erythematous rash with excoriated lesions due to prolonged scratching.

Interdigital web space with pathognomonic papulo-vesicular lesions.





Local and Systemic Complications

Scabies infestation is often complicated by *Streptococcus pyogenes* or *Staphylococcus aureus* impetigo because of scratching-induced skin trauma. These bacteria have been isolated from skin burrows and mite products suggesting that mites could contribute directly to the spread of bacteria. In addition, mite and host immune system interactions, including mite production of complement-inhibiting proteins, promote *S. pyogenes* survival and *S. aureus* growth.

Impetigo due to *S. pyogenes* acts as a precursor to a variety of clinical complications, such as acute poststreptococcal glomerulonephritis (APSGN). Although the immediate consequences of APSGN are limited, long-term effects, particularly chronic kidney disease, have substantial morbidity. As with APSGN, streptococcal skin infection is likely an important driver of acute rheumatic fever and subsequent rheumatic heart disease in some settings.

Secondary bacterial infections also predispose those with scabies to bacteremia and sepsis. Untreated crusted scabies carries a high risk of mortality from secondary sepsis.

Abscesses, cellulitis, and, rarely, necrotizing soft tissue infections can also occur as local complications.

Diagnosis

The diagnosis of scabies is made largely on clinical grounds. The combination of physical examination, noting typical skin lesions with characteristic burrows, together with the description of an intensely itchy rash, which is often worse at night, enables a confident diagnosis of scabies to be made. A history of contact with known cases is often present.

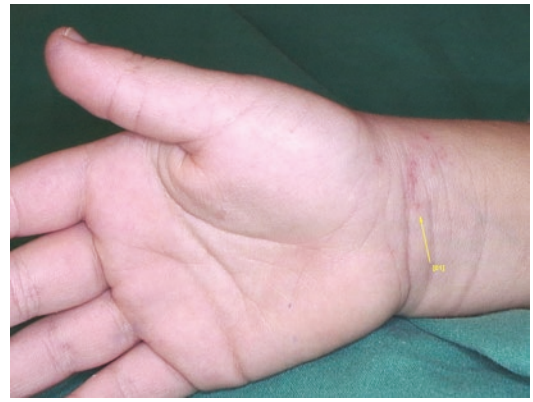
Classically, diagnosis is confirmed by microscopy examination with the visualization of mites, eggs, or feces from skin scraping. To obtain a skin-scraping sample, a drop of mineral oil is placed at the terminal end of a burrow, and the lesion and underlying epidermis are gently scraped away with a surgical blade or sterile needle. The specimen should be applied to a glass slide. The application of potassium hydroxide (KOH) to the slide may be helpful for the examination because it dissolves excess keratotic debris. However, sensitivity can be low, and testing is contingent on the availability of required equipment.

Recently, several noninvasive in vivo diagnostic techniques have emerged. Dermoscopy may be a very useful tool in scabies. Examination with a handheld dermatoscope allows better visualization of the curvilinear scaly burrow, and the mite itself may be seen at the end of the burrow as a dark triangular structure, corresponding to the pigmented head and anterior legs of the scabies mite; this picture is often referred to as "delta wing sign." Additionally, eggs may be seen as small ovoid structures within the burrow.

Several other noninvasive imaging techniques have been used, including videodermatoscopy and confocal microscopy, which provide a more detailed inspection of the mite.

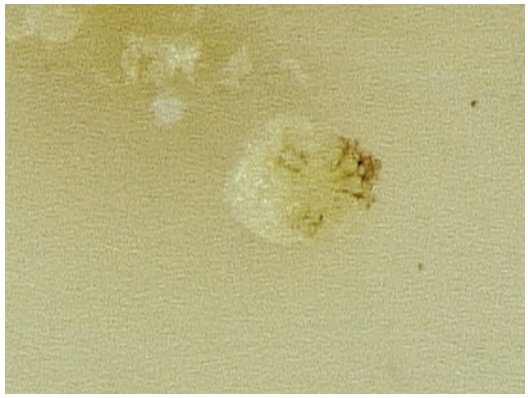
When dermoscopy cannot distinguish burrows from excoriations, the higher magnification of videodermoscopy is useful and contributes to its high specificity.

Reflectance confocal microscopy reliably detects and quantifies classic and crusted scabies, but its cost and availability limit its utility in daily practice.



Characteristic burrow at the volar surface of the wrist of a young patient.

Classic dermoscopic image of triangle or "delta wing sign" (yellow arrow), corresponding to the head of the mite.



Photographic image of scabies mite taken through videodermoscope (Courtesy of Dott. Domenico Di Maria).

Diagnostic Criteria

Scabies is commonly misdiagnosed. Recently, agreed diagnostic criteria were developed using the Delphi method to enable standardization and to compare findings, with validation studies ongoing. This study introduces three categories of diagnosis—"confirmed scabies," "clinical scabies," or "suspected scabies."

Level	Criteria
A: Confirmed scabies	At least one of: A1: Mites, eggs, or feces on light microscopy of skin samples A2: Mites, eggs, or feces visualized on individual using high-powered imaging device A3: Mite visualized on individual using dermoscopy
B: Clinical scabies	At least one of: B1: Scabies burrows B2: Typical lesions affecting male genitalia B3: Typical lesions in a typical distribution and two history features
C: Suspected scabies	One of: C1: Typical lesions in a typical distribution and one history feature C2: Atypical lesions or atypical distribution and two history features
History features	H1: Itch H2: Close contact with an individual who has itch or typical lesions in a typical distribution
Notes: *1. These criteria should be used in conjunction with the full explanatory notes and definitions (in preparation)* *2. Diagnosis can be made at one of the three levels (A, B, or C)* *3. A diagnosis of clinical and suspected scabies should only be made if other differential diagnoses are considered less likely than scabies*	

Summary of 2018 IACS criteria for the diagnosis of scabies.

Treatment

The approach to the eradication of scabies mites is dependent upon the clinical presentation and patient population. Simultaneous treatment of both the patient and close personal contacts is highly recommended in order to prevent reinfestation.

Different treatments for scabies are available. Topical permethrin, benzyl benzoate lotion, and oral ivermectin are the most common first-line treatments.

Because of its high efficacy and tolerability, 5% permethrin has been approved by the US Food and Drug Administration for scabies treatment in individuals >2 months of age, and it is considered safe in pregnancy due to its low absorption.

Second-line treatments for pregnant women include topical sulfur and benzyl benzoate. Although the risk associated with oral ivermectin may be low, data on use in this population are limited.

Permethrin is a topical synthetic pyrethroid agent that impairs function of voltage-gated sodium channels in insects, leading to disruption of neurotransmission.

Permethrin 5% cream should be applied from neck to the soles of the feet including areas under the fingernails and toenails and washed after 8–12 h. In young children, scalp involvement is common. Therefore, permethrin should also be applied to the scalp and face (avoiding the eyes

and mouth) in this population. The treatment must be repeated after 7–14 days.

When permethrin is unavailable, 10%–25% benzyl benzoate can be used. Treatment regimens vary. It may be applied once daily at night on 2 consecutive days with re-application at 7 days. It is considered safe in infancy and pregnancy, and it is widely used globally.

Oral ivermectin is a safe and efficacious systemic option with the benefit of simple administration and low cost. It is not recommended for use in pregnant women or young children (< 5 years of age or < 15 kg) because of insufficient safety data. Ivermectin therapy consists of a 200 mcg/kg single dose followed by a repeat dose after 1–2 weeks. This mode of treatment may be particularly useful for large scabies outbreaks in nursing homes and other facilities where topical therapy can be impractical.

Potential side effects of topical treatments generally consist of burning and eczematous eruption. Pruritus commonly persists for 1–4 weeks, even after effective treatment, and it is due to ongoing inflammation. It can be managed with emollients, oral antihistamines, and low-potency topical corticosteroids.

Alternative topical treatment options for scabies include topical sulfur, lindane, crotamiton, malathion, and ivermectin lotion. These agents have not been shown to be more effective than the others. Topical ivermectin is a recent, high-cost, agent that appears to have efficacy for scabies. Lindane is no longer recommended because of its potential to cause neurotoxicity.

In crusted scabies, a combination treatment with permethrin and oral ivermectin is considered the preferred first-line treatment. Oral ivermectin 200 mcg/kg must be taken on days 1, 2 and 8. For severe cases, additional ivermectin treatment might be necessary. The topical scabicide should be repeated daily for 7 days then twice weekly until the patient is cured.

In endemic scabies, mass drug administration has been shown to be an effective control strategy. This involves repeat administration of single doses of therapeutic agents to the entire community. Oral ivermectin is the preferred intervention given the drug's efficacy and ease of administration.

Environmental Measures

Environmental Measures Include the Following Recommendations

Close personal contacts of infested patients (parents, caregivers, people involved in bathing or lifting, sexual partners, and family members) who have had prolonged skin-to-skin contact in the preceding 6 weeks are at high risk of acquiring the infection and all should be treated simultaneously regardless of symptomatology to avoid an endless cycle of transmission and reinfestation.

Clothes and bed linen should be machine washed at least at 60 °C. Clothes that cannot be washed should be kept in a bag for at least 72 h because some studies have documented that mites cannot survive more than 3 days after separation from their human hosts.

There are data suggesting that fomites are important in the spread of scabies and populations of mites have been recovered from linen, chairs, floor, dust, etc. However, evidence supporting this precautionary intervention is not yet available, so the advice remains somewhat controversial. In fact, several studies in low-resource settings achieved scabies control without concomitant environmental decontamination. However, the risk of transmission or reinfestation via fomites is probably not negligible in the severest forms of crusted scabies, given the associated high mite burden (up to millions of mites). Therefore, in severest forms of scabies and in settings where linen washing above 50 °C is feasible, it is reasonable to recommend doing so.

Assessment for Cure and Treatment Failure

Therapy is considered successful if active lesions resolve and nocturnal pruritus ceases by 1 week after treatment. It is important to note that some pruritus often persists for 2–4 weeks after successful treatment as it can take this long for symptoms and signs of hypersensitivity to resolve.

A follow-up visit 2 weeks after completion of treatment is recommended.

Most cases of treatment failure are likely to result from inadequate treatment, poor adherence to the treatment regimen, or reinfestation. If resistance appears to be the most likely cause of treatment failure, treatment with an alternative anti-scabietic agent should be attempted.

However, alternative diagnoses also should be considered. The differential diagnosis should include psoriasis, atopic eczema, and lichen planus. In infants and young children, the differential diagnosis might include Langerhans cell histiocytosis, papular urticarial, and infantile acropustulosis.

Further Reading

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