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A Narrative Review on Crusted Scabies: Pathogenesis, Clinical Manifestations, Diagnosis, and Treatment Options

Review Article

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Abstract: Crusted scabies is a rare but severe skin condition that predominantly affects immunocompromised and elderly individuals, with potentially fatal outcomes if untreated. It can also trigger institutional outbreaks, complicating public health responses. Diagnosis can be challenging due to overlapping features with other dermatological conditions. In 2016, crusted scabies was classified as a notifiable disease in Australia's Northern Territory. This review aims to summarize the pathogenesis, clinical features, diagnostic methods, and treatment options for crusted scabies over a period of 20 years ending in December 2022. A critical literature review was conducted using PubMed, Google Scholar, and Research4life for studies published between January 2003 and December 2022. Relevant articles in English discussing pathogenesis, clinical manifestations, diagnostic approaches, and treatment strategies were reviewed. Crusted scabies is characterized by a Th1-to-Th2 immune shift, leading to hyperkeratosis, extensive scaling, and minimal pruritus. Diagnostic methods include microscopy, dermatoscopy, biopsy, PCR, and serodiagnosis. Treatments involve topical scabicides, keratolytic agents, and oral ivermectin. Standardized protocols for diagnosis and treatment are needed to address variability in current practices. Early diagnosis and timely management are critical to improving patient outcomes and preventing complications, including sepsis and nosocomial outbreaks.

1. Introduction

Crusted scabies, also known as Norwegian scabies, is a condition that was initially documented in 1848 by Boeck and Daniel in a cohort of leprosy patients from Norway (Alexander, 1984). It is a severe and unusual presentation of the ectoparasitic hyper infestation caused by the Sarcoptes scabiei mite which is characterized by a non-protective host immune response, the development of hyperkeratotic skin crusts, and skin fissuring (Hengge et al., 2006; Walton et al., 2010). Unlike typical scabies, which have a few dozen mites' infestation, crusted scabies involve an overwhelming infestation characterized by the presence of thousands to millions of mites within the epidermis (Ishii et al., 2008). The presence of hyperkeratotic skin lesions leads to the formation of thick crusts, hence the term "crusted scabies". This condition predominantly affects immunocompromised individuals and those with neurological disorders (Karthikeyan, 2009). Common neurological disorders associated with crusted scabies include dementia, Parkinson's disease, and stroke-related complications, which may impair personal hygiene or cause immobility. These conditions contribute to increased susceptibility due to reduced ability to respond to pruritus or maintain skin integrity.

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The clinical diagnosis of crusted scabies presents challenges, even for experienced clinicians, as there is a lack of reliable rapid diagnostic tests. Moreover, several dermatological conditions, such as psoriasis, eczema, Darier's disease, and cutaneous adverse drug reactions, can mimic the clinical presentation of crusted scabies (Adler et al., 2017). The rarity and complexity of crusted scabies necessitate a deeper understanding of their pathogenesis, clinical characteristics, diagnosis, and therapeutic approaches.

2. Background

Crusted scabies, also known as Norwegian scabies, is a severe and highly contagious form of scabies infestation characterized by a massive proliferation of scabies mites in the skin (Sánchez-Borges et al., 2018). Though it represents a small proportion to overall scabies cases, it poses significant public health challenges, because of its infection not only in individuals but also their families and communities. Individuals with crusted scabies can act as super-spreaders continually reinfecting others (Walton et al., 2010). The disease has been associated with institution-wide outbreaks, especially in key vulnerable populations such as immunocompromised individuals like elderly residents in nursing homes, where undiagnosed cases among elderly residents have been identified as a source of transmission (Roberts et al., 2005).

In recent years, there has been an increase in the use of immunosuppressive or immune-modulator medications, particularly due to the rise in organ transplantation (Organ Procurement & Transplantation Network (OPTN), 2022). This is significant in the fact that use of steroid and immunosuppressive may exacerbate the risk of nosocomial and endemic outbreaks, especially for diseases like crusted scabies with high mortality rates (Glennie et al., 2021; Roberts et al., 2005).

Historically, crusted scabies had a 5-year mortality rate of up to 50%, but the dedicated use of ivermectin and topical scabicides has contributed to a decrease in mortality (Glennie et al., 2021). Nonetheless, the disease remains a concern, necessitating effective management strategies. Misdiagnosis of scabies has been reported in a substantial proportion of cases, highlighting the need for improved diagnostic accuracy (Kosmala et al., 2019).

Crusted scabies is a notifiable disease only in certain regions, such as the Northern Territory of Australia, where its reporting and monitoring have been prioritized (Glennie et al., 2021). From a public health point of view, the lack of notification can imply that if the disease continues to go unnoticed, the population with crusted scabies will become core transmitters. In scabies endemic communities globally, the control efforts, such as mass drug administration (MDA) programs, can be undermined by even a single case of crusted scabies, reducing the efficacy and acceptability of such programs(Roberts et al., 2005).

In terms of a Cambodian perspective, in 2015, the Global Burden of Disease Study reported that the regions that suffer from the burden of scabies the most are East Asia, Southeast Asia, Oceania, and tropical Latin America. Out of 195 countries analyzed, Cambodia ranked sixth in terms of the highest standardized scabies DALY (Disability-adjusted life year) burden per 100,000 people. In 2016, there was a scabies outbreak in the Kampong Cham provincial prison.

Secondary bacterial infections, particularly with Staphylococcus aureus and Streptococcus pyogenes (Group A Streptococcus), further complicate the clinical feature of scabies, crusted scabies, and concurrent tinea infections, leading to increased morbidity, mortality, and socioeconomic costs through invasive infections (May et al., 2019).

Given the multifaceted challenges posed by crusted scabies, there is a need for comprehensive guidelines, standardized protocols, and improved diagnostic accuracy to enhance outcomes for affected

individuals. Though crusted scabies is rare but often occurs due to undiagnosed or poorly treated ordinary scabies. Since ordinary scabies is highly prevalent in Southeast Asia, particularly among vulnerable populations, addressing it may help reduce the progression to severe forms such as crusted scabies. This highlights the importance of having comprehensive scabies control as a public health priority. Furthermore, the development of effective anti-parasitic therapies, along with the better understanding of pathogenesis and transmission, can contribute to the formulation of preventive strategies, including vaccines and immunotherapy. The significance of health education, timely diagnosis, and adequate drug supply cannot be overstated in reducing the prevalence of scabies, particularly its crusted form (Roberts et al., 2005).

3. Aim and Objectives of the Study

3.1. Problem Statement

Despite the growing attention of research on crusted scabies, there remains a paucity of high-quality research studies including double blinded randomized control trials. As a rare condition, crusted scabies pose significant clinical, diagnostic, and management (Aukerman et al., 2019). Current studies have focused on various aspects of crusted scabies, including epidemiology, pathogenesis, clinical manifestations, diagnostic methods, and treatment approaches (Ishii et al., 2017). However, there is a need for a comprehensive narrative review that brings together the available evidence to provide a clear overview of the current knowledge on crusted scabies (Roberts et al., 2005). Additionally, the lack of a comprehensive review on crusted scabies hinders the ability of healthcare professionals to make informed decisions regarding the diagnosis, management and limits the development of evidence-based guidelines and protocols for prevention and infection control, particularly in high-risk institutional settings such as prisons, hospitals, and care facilities, where outbreaks are more likely to occur (Binić et al., 2010; Shimose & Munoz-Price, 2013). This review aims to synthesize existing literature and identify knowledge gaps (Walton, 2010; Walton et al., 2010). This may stimulate further research, be useful for clinical decision-making and for the development of comprehensive strategies for prevention and management of crusted scabies.

To understand the pathogenesis and management of crusted scabies, below are the questions that were used throughout the review (review form is in the appendix):

1. What is the pathogenesis of Crusted Scabies?
2. What are the clinical manifestations of Crusted Scabies?
3. What aids in the diagnosis of Crusted Scabies?
4. What are the various treatment options of Crusted Scabies?

3.3. Objectives

3.3.1. General Objective

The aim of the study is to review the literature over a period of 20 years ending in December 2022, regarding pathogenesis, clinical manifestations, diagnosis and treatment options for crusted scabies.

3.3.2. Specific Objectives

- To provide an overview of the pathogenesis of crusted scabies

- To review the dermatological manifestations of crusted scabies
- To review diagnostic approaches and related guidelines
- To review treatment options of crusted scabies

4. Methods

This manuscript adopts a narrative review design based on a focused literature search conducted through PubMed, Google Scholar, and Research4Life databases. Articles published between January 1, 2003, and December 31, 2022, were considered eligible, and over the course of two months, a total of 50 articles were reviewed, including 29 from PubMed, 16 from Google Scholar, and 5 from Research4Life.

Searches were performed using the keywords "Crusted scabies" or "Norwegian scabies" combined with "Pathogenesis," "Clinical manifestations," "Diagnosis," and "Treatment."

All articles reviewed were in English. Data was collected using an article summary form, which categorized the information into four groups: pathogenesis, clinical manifestations, diagnosis, and treatment options, with the form provided in the annex. All eligible articles were screened, synthesized, and categorized according to the objectives of the review, and the extracted data were summarized in tables and analyzed to identify key themes, trends, and knowledge gaps. The findings were then presented in a narrative format, highlighting central concepts, ongoing debates, and areas in need of further research.

5. Results and Discussion

We reviewed a total of 50 articles eligible. The date of publication ranging from 2004 to 2021, spanning over 17 years. Over time, there appears to be an increase in interest in crusted scabies (Figure 1).

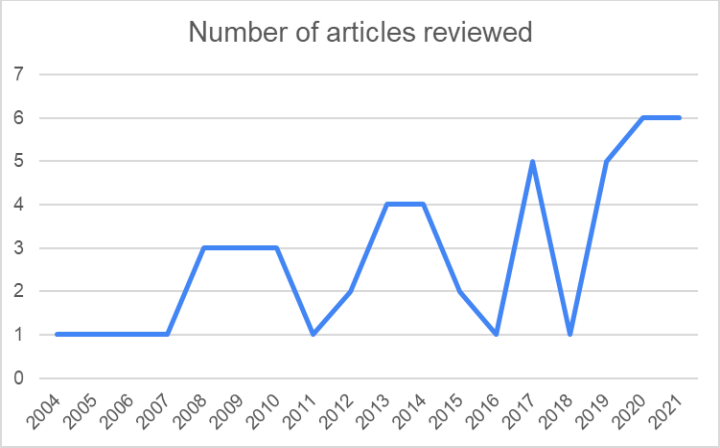

Figure 1. Number of articles reviewed by year of published

Among the 50 reviewed articles, North America had the highest number of articles, followed by Oceania. Asia also had a significant number of articles published (Figure 2). Overall, the articles covered a diverse range of regions, reflecting a global distribution of publication locations.

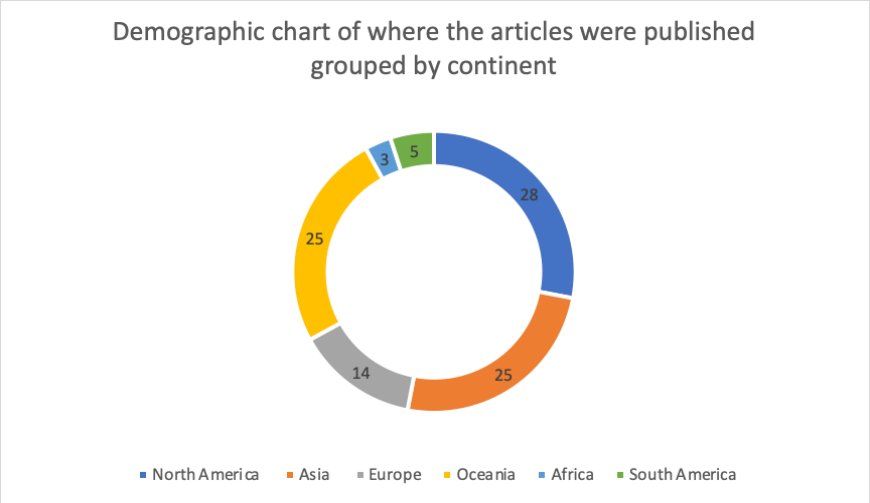

Figure 2. Demographic chart of where the articles were published grouped by continent

5.1. Pathogenesis

Crusted scabies is a severe variant of Sarcoptes Scabiei var. Hominis infestation characterized by overwhelming mite proliferation, extensive hyperkeratosis, and a profoundly dysregulated immune response. Although a complete understanding of its pathogenesis is still evolving, existing human and animal studies have provided meaningful insight into the mechanisms underlying this condition (Chavez-Alvarez et al., 2018; Roberts et al., 2005; Walton et al., 2008). Clinical symptoms typically manifest about four weeks after infestation, with the characteristic crusted plaques appearing approximately eight to twelve weeks later (Chavez-Alvarez et al., 2018). Compared to ordinary scabies, which may have a longer incubation period of one to two months or more in elderly individuals (Ishii et al., 2008), crusted scabies progresses more rapidly. Importantly, this accelerated course does not result from an increase in mite virulence; rather, it reflects host factors that permit uncontrolled multiplication of mites (Ishii et al., 2017).

5.1.1. Overview of Immune Transition and Disease Development

The transition from ordinary to crusted scabies reflects a shift from a predominantly Th1-driven response to a Th2-dominated one. Ordinary scabies is characterized by a protective Th1 immune profile, evidenced by the high IFN-γ to IL-4 ratio observed in stimulated peripheral blood mononuclear cells (Hay et al., 2012). This Th1 response limits mite numbers and prevents extensive proliferation. In contrast, crusted scabies is associated with significantly elevated IL-4, IL-5, and IL-13 levels (Hay et al., 2012; Walton, 2010), promoting eosinophil recruitment, mast cell activation, and IgE production, processes that ultimately enhance inflammation without restricting the mites.

There is also evidence of Th17 involvement in crusted scabies. Increased concentrations of IL-17 and IL-23 have been reported (Hay et al., 2012), while porcine models of crusted scabies show markedly elevated numbers of IL-17-secreting T cells within the skin (Roberts et al., 2005). Although systemic IL-17 may not always be elevated, localized production in the skin appears to contribute substantially to tissue inflammation and epidermal hyperplasia (Liu et al., 2014). The overall immune response in crusted scabies is therefore not deficient but rather hyperactive, though in a direction that fails to control infestation.

5.1.2. High Mite Burden in Crusted Scabies

Patients with crusted scabies often harbor extraordinarily high numbers of mites, frequently exceeding one to two million and in some cases reaching five million adult mites(Ishii et al., 2017). This extreme infestation results from failure of the host to regulate mite replication. Impaired cellular immunity has been proposed as a major contributing factor (Leung et al., 2019; Roberts et al., 2005). The absence of normal scratching behavior is another important component; scratching typically disrupts mite burrows, reducing the parasite burden. Individuals with developmental disabilities, neuropathies, or limited mobility exhibit reduced scratching, allowing the mites to proliferate unchecked (Karthikeyan, 2009). Additional external factors, including inadequate hygiene and inappropriate corticosteroid use, can also contribute to the progression toward crusted scabies (Aukerman et al., 2019; Engelman & Steer, 2018; Ishii et al., 2017).

Cases have been reported in individuals without identifiable immunodeficiency (Merad, 2021), indicating that immune dysfunction rather than outright immunosuppression may be key. A possible genetic predisposition has also been noted, especially among Aboriginal Australians, with studies suggesting a relationship to the HLA-A11 allele, which appears in higher proportions within this population (Hay et al., 2012; Roberts et al., 2005). This may partially explain the increased susceptibility to severe disease observed in certain groups.

5.1.3. Adaptive Immune Response

5.1.3.1. Cell-Mediated Immune Response

Cell-mediated immunity plays a critical role in determining the clinical outcome of scabies infestation. In ordinary scabies, the Th1 response dominated by IFN-γ promotes effective parasite control (Hay et al., 2012). In crusted scabies, however, the immune system shifts toward Th2 polarization, characterized by elevated IL-4, IL-5, and IL-13 levels (Hay et al., 2012; Walton, 2010). IL-4 not only enhances IgE production but also stimulates keratinocyte proliferation, contributing to the formation of hyperkeratotic lesions (Walton, 2010).

The role of Th17 cytokines further distinguishes crusted scabies from ordinary scabies. Increased IL-17 and IL-23 levels have been documented (Hay et al., 2012), and porcine models show strong upregulation of IL-17-secreting T cells in the skin during crusted scabies (Roberts et al., 2005). IL-17 promotes neutrophil recruitment and contributes to tissue remodeling and hyperplasia. Mast cells, found in increased numbers, may also release IL-17, amplifying the inflammatory cascade (Liu et al., 2014).

Histologically, crusted scabies lesions demonstrate a predominance of CD8+ T lymphocytes within the dermis, with fewer CD4+ cells present (Walton, 2010; Walton et al., 2008). CD8+ T-cell–mediated keratinocyte apoptosis contributes to epidermal remodeling resembling psoriasis (Hay et al., 2012). Lesions exhibit both IL-1β and TGF-β staining, reflecting simultaneous presence of proinflammatory and regulatory signals, which exemplifies the disorganized and ineffective nature of the immune response (Walton, 2010; Walton et al., 2010).

5.1.3.2. Humoral Immune Response

The humoral response in crusted scabies is excessive rather than protective. Mellanby's reinfestation experiments showed that ordinary scabies can induce partial immunity, as only 40% of previously infected individuals could be reinfested (Hay et al., 2012). However, crusted scabies elicits extraordinarily high levels of circulating antibodies that do not correlate with effective parasite clearance. IgE levels exceeding

1000 mcg/L are seen in 73% of patients, and 10% exhibit levels above 10,000 mcg/L (Roberts et al., 2005). IgA is elevated in 64% of cases (Walton, 2010). These extreme antibody levels reflect chronic immune activation rather than a targeted protective mechanism.

Serologic studies show strong binding to as many as 21 mite proteins, including recombinant glutathione S-transferase (Sánchez-Borges et al., 2018; Walton et al., 2010). IgE responses are particularly robust against recombinant proteins C08, F04, and Ssag1.2 (Walton et al., 2010), while IgG4 responses, especially those directed against apolipoproteins, are frequently elevated, often in association with eosinophilia (Sánchez-Borges et al., 2018). Notably, histopathology reveals an absence of B-cell infiltrates within skin lesions, which instead show dense infiltrates of CD45+ and CD3+ T cells (Walton et al., 2008). This finding indicates that antibody production is systemic and that antibodies do not contribute to local mite control within the skin.

5.1.4. Innate Immune Response

Innate immunity is also significantly altered in crusted scabies. Eosinophilia is a common finding, both systemically and within lesional skin (Walton, 2010). Eosinophils, mast cells, and basophils that support Th2 polarization, are frequently elevated and sustain the chronic inflammatory environment. The complement system also appears to be activated. Patients with crusted scabies often exhibit decreased serum C3 and C4 levels, possibly due to consumption or degradation mediated by SMIPPs, which are produced in abundance given the high mite load (Walton, 2010). Dermal deposition of C3 and fibrinogen in both ordinary and crusted scabies suggests complement activation at the tissue level, and this may be exacerbated by secondary bacterial infections.

ANA positivity is overrepresented in crusted scabies patients (Roberts et al., 2005). While this could indicate a predisposition to connective tissue disease, it may instead reflect nonspecific autoantibody production in response to chronic antigenic stimulation. IL-1β, strongly expressed within lesions, is another prominent cytokine contributing to inflammation, T-cell activation, and macrophage stimulation (Walton et al., 2008).

5.1.5. Mechanisms of Hyperkeratosis

Hyperkeratosis in crusted scabies arises from multiple overlapping mechanisms. CD8+ T-cell–mediated keratinocyte apoptosis disrupts normal epidermal turnover, while IL-4–induced keratinocyte proliferation promotes epidermal thickening (Walton, 2010). Increased expression of amphiregulin and epiregulin, both members of the epidermal growth factor family, further supports hyperproliferation (Walton, 2010). Elevated IL-17 within the skin, derived from both T cells and mast cells, amplifies epidermal hyperplasia and contributes to the development of thick, fissured crusts (Liu et al., 2014). These crusts harbor enormous quantities of mites, facilitating persistent infestation.

5.1.6. Transmission of crusted scabies

The extensive hyperkeratosis and extremely high mite burden contribute directly to the high transmissibility of crusted scabies. The hyperkeratotic crusts contain large populations of viable mites, and transmission can occur through direct skin-to-skin contact as well as through shedding of heavily infested skin scales (Ishii et al., 2008). Even brief contact is sufficient to transmit infestation, making crusted scabies a significant cause of institutional outbreaks. Transmission through bedding or clothing is possible and occurs more readily than in ordinary scabies, due to the large number of mites shed (Engelman & Steer, 2018). Outbreaks in hospitals and long-term care facilities have been well

documented and relate directly to the level of skin shedding (Andrews et al., 2009). Mites remain infective for up to 48 hours at room temperature and moderate humidity, although their mobility decreases at temperatures below 16°C (Sunderkötter et al., 2016). The likelihood of transmission therefore depends on mite density, environmental stability, duration of contact, and the extent of skin shedding (Sunderkötter et al., 2021).

5.1.7. Integration with recent guidelines

Taken together, the pathogenesis of crusted scabies reflects a complex interplay of dysregulated immune mechanisms rather than simple immune deficiency. The absence of an effective Th1 response permits uncontrolled mite proliferation, while exaggerated Th2 and Th17 responses drive inflammation, antibody overproduction, and epidermal hyperplasia. CD8+ T-cell predominance, keratinocyte apoptosis, elevated IL-4 and IL-17, complement dysregulation, eosinophilia, and IL-1β–mediated inflammation all contributes to the severe cutaneous manifestations of the disease. Recent guidelines (UK Health Security Agency, 2025; Uzun et al., 2024) reaffirm these mechanisms and align closely with the evidence from earlier human and animal studies. Crusted scabies therefore represents an immune system that is overactive yet ineffective, producing persistent infestation, marked hyperkeratosis, and heightened transmissibility.

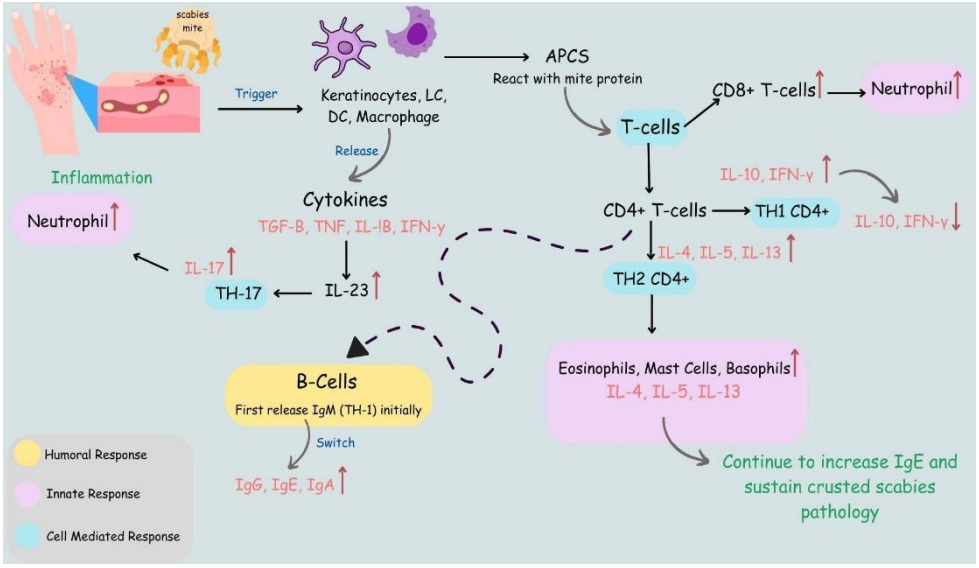

Figure 3. The pathogenesis of crusted scabies (adapted by authors based on reviewed articles)

5.2. Clinical Manifestations

Crusted scabies, often referred to as Norwegian scabies, is an extremely contagious and severe manifestation of scabies infestation. It is distinguished by an extensive proliferation of scabies mites, reaching numbers in the millions within the skin (Sánchez-Borges et al., 2018). Although Crusted Scabies usually affect the elderly with comorbidities, there exists a wide range of age groups reported having this disease from a 4-month-old infant to the old age of 91 years old (Rose et al., 2014; Sil et al., 2020).

Crusted scabies presents with diverse and severe dermatologic manifestations (See Table 2), including generalized dermatitis, erythroderma, and thick hyperkeratotic plaques that may appear as piled-up sand or deeply fissured surfaces (Figure 4) (Aukerman et al., 2019; Binić et al., 2010; Palaniappan et al., 2021). Nail involvement is common, with dystrophic, discolored, and markedly thickened nails mimicking tinea

unguium (Ishii et al., 2008; Vasanwala et al., 2019). Although the entire body may be affected, burrows predominantly appear on the interdigital spaces, nails, wrists, elbows, axillae, umbilicus, belt line, nipples, buttocks, penile shaft, and extremities, especially on extensor surfaces (Andrews et al., 2009; Cuellar-Barboza et al., 2020; Sánchez-Borges et al., 2018). Localized scalp-limited diseases have also been reported, and lesions can occasionally be malodorous (Al -Tarawneh & Shamasneh, 2021; Bimbi et al., 2020; Chiu & Lan, 2020).

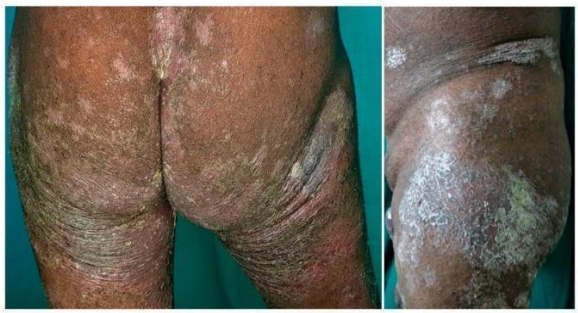

Figure 4. Multiple erythematous hyperkeratotic crusted plaques over the gluteal region and lateral aspect of thighs in a 54-year-old man.
Reprinted with permission from Palaniappan, V., Gopinath, H., & Kaliaperumal, K. (2021). Crusted Scabies. The American journal of tropical medicine and hygiene, 104(3), 787-788. https://doi.org/10.4269/ajtmh.20-1334

Crusted scabies occur most frequently in individuals with severe immunodeficiency, neurologic disorders causing sensory impairment, or immobility that limits scratching (Salavastru et al., 2017; Scott & Chosidow, 2011; Walton et al., 2008). Pruritus may be minimal or absent owing to altered immune responses (Adler et al., 2017; Kosmala et al., 2019; Vasanwala et al., 2019). An atypical form has been described in patients initiating antiretroviral therapy, termed "unmasking crusted scabies–associated immune reconstitution inflammatory syndrome" (Fernández-Sánchez et al., 2012). Additional atypical variants include scabies herpeticum, a rare co-infection with Herpesviridae leading to ulcerative lesions (Sandoval et al., 2019).

Topical or systemic corticosteroids can obscure the characteristic signs of scabies, leading to misdiagnosis, while prolonged local corticosteroid use may exacerbate crusted scabies (Aukerman et al., 2019b; Binić et al., 2010b; Vorou et al., 2007). Crusted scabies is further associated with substantial morbidity and mortality, historically demonstrating 5-year mortality rates of up to 50% due to secondary sepsis (Shimose & Munoz-Price, 2013). Common secondary bacterial infections include impetigo caused by Streptococcus pyogenes or Staphylococcus aureus, as well as cellulitis and lymphangitis (May et al., 2019). Additional systemic complications, such as sleep disturbance, acute post-streptococcal glomerulonephritis, rheumatic fever, and bacterial sepsis, further contribute to the disease burden (Leung et al., 2019; Scheinfeld, 2004b; Sunderkötter et al., 2021; Vasanwala et al., 2019).

It is important to note that the clinical manifestations of crusted scabies can be diverse and may vary depending on the individual case. The severity and extent of the condition can be assessed using a clinical grading scale developed by Davis et al. (2013) (Davis et al., 2013; Hasan et al., 2020). This grading scale categorizes the severity of crusted scabies into different aspects, providing guidance for its management (See Table 1). A study determined that the utilization of the grading scale in this context has been linked to positive results, including reduced hospital stays and decreased use of ivermectin compared to cases managed without the grading scale (Davis et al., 2013) If confirmed in different settings, implementing this scale is expected to enhance the management of crusted scabies, potentially leading to shorter

hospital stays and reduced reliance on ivermectin treatment, all while maintaining favorable outcomes (Davis et al., 2013).

Table 1. Severity grading scale for crusted scabies adapted Davis et al. (2013)

A: Distribution and extent of crusting

1. Wrist, web spaces, feet only (<10% Total Body Surface Area)
2. Above plus forearms, lower legs, buttocks, trunk, or 10-30% TBSA
3. Above plus scalp OR>30% TBSA

B: Crusting/Shedding

1. Mild Crusting (<5mm depth of crust), minimal skin shedding
2. Moderate (5-10mm) crusting, moderate skin shedding
3. Severe (>10mm), profuse skin shedding

C: Past Episodes

1. Never Had it Before
2. 1-3 prior hospitalizations for crusted scabies OR depigmentation of elbows, knees,
3. >=4 prior hospitalizations for crusted scabies OR depigmentation as above PLUS legs/back or residual skin thickening/ichthyosis

D: Skin Condition

1. No cracking or pyoderma
2. Multiple pustules and/or weeping sore and/or superficial skin crackling
3. Deep skin cracking with bleeding, widespread purulent exudates

Grade 1: Total score 4-6

Grade 2: Total score 7-9

Grade 3: Total score 10-12

Treatment: Ivermectin 200 mcg/kg rounded up to nearest 3mg

Grade 1: 3 doses - Days 0,1,7

Grade 2: 5 doses - Days 0,1,7,8,14

Grade 3: 7 doses - Days 0, 1, 7, 8, 14, 21, 28

Table 2. Essential findings of clinical features from reviewed articles

Items	Essential Findings of Clinical Features
Skin Lesions	Extensive local or diffuse hyperkeratosis on an erythematous background with crusting and fissures that varies in thickness form about 3 to 15mm.
Variety of crusts or plaques display	• Yellowish-green crusts • Gray to yellow-white papules fissured plaques and nodules • Yellow-to-brown crusted plaques • Parakeratotic crust that varies in thickness form about 3 to 15mm

	• Creamy, grey yellow brown or yellow green in color, adherent, and firm • Porous appearance resembling a pumice stone when removed.
Common areas affected	The lesions are typically located on different areas of the body including face, scalp, neck, auricular region, trunk, and extremities which are commonly affected. Moreover, the infestation tends to be exaggerated on the palms, soles, dorsum of the fingers, interdigital spaces, extensor surface of the elbows, buttocks and nails.
Minimal or Absent Pruritus	Due to changes in their immune responses, patients with crusted scabies frequently experience little to no itching. Nevertheless, a minimum of half of the patients do experience some level of itchiness, which typically decreases over time.

5.3. Diagnosis

Diagnosis of crusted scabies necessitates a multifaceted approach combining clinical evaluation, direct visualization methods, histopathology, and supportive laboratory findings to ensure accuracy and early detection. Clinically, patients often present with widespread hyperkeratotic or crusted lesions, severe pruritus, and visible burrows. However, due to the atypical or extensive nature of lesions, particularly in immunocompromised individuals, misdiagnosis is common and may delay appropriate management (Aukerman et al., 2019; Kosmala et al., 2019). Misdiagnosis as psoriasis, eczema, or drug eruption is well-documented as presented in several case reports (See Table 3), and inappropriate steroid therapy may lead to altered clinical presentations referred to as "scabies incognito" (Hasan et al., 2020; Sánchez-Borges et al., 2018; Shrestha & Bischof, 2021). Early and accurate diagnosis is crucial for preventing complications, including bacterial superinfection, sepsis, and nosocomial transmission (Vasanwala et al., 2019). Direct microscopy of skin scrapings remains a foundational and cost-effective diagnostic technique, wherein samples placed in potassium hydroxide dissolve keratinous debris to reveal mites, eggs, eggshells, and fecal pellets (scybala) (Chiu & Lan, 2020; Shimose & Munoz-Price, 2013). To maximize sensitivity, multiple superficial scrapings from burrows and vesicles are recommended (Shimose & Munoz-Price, 2013). In cases of crusted scabies, skin crust specimens often contain numerous viable mites measuring approximately 400 μm by 300 μm, identifiable by their rounded bodies with four pairs of legs (Karthikeyan, 2009; Vignesh et al., 2008). Nonetheless, negative microscopic findings do not exclude scabies, underscoring the method's limitations including considerable time consumption and diagnostic error rates (Hay et al., 2012; Salavastru et al., 2017).

Dermatoscopy has emerged as a valuable, non-invasive diagnostic tool that enables magnified visualization of mites, eggs, and characteristic burrows, demonstrating high sensitivity and specificity (Ishii et al., 2017; Sunderkötter et al., 2021). This approach is particularly useful when infestations are in early disease stages, where skin scrapings may fail to reveal mites, offering an efficient alternative with fewer false negatives and greater time efficiency compared to microscopy, positioning it as an effective first-line diagnostic tool for scabies (Andrews et al., 2009; Chavez-Alvarez et al., 2018; Hay et al., 2012; Sunderkötter et al., 2021).

In complex or atypical presentations, skin punch biopsy provides definitive histopathological confirmation. Typical findings of crusted scabies include epidermal acanthosis, spongiosis, hyperkeratosis, dermal fibrosis, perivascular infiltration by lymphocytes, plasma cells, eosinophils, and presence of mites within skin layers (Hasan et al., 2020; Sánchez-Borges et al., 2018). Such confirmation is crucial to differentiate crusted scabies from clinically similar dermatoses such as plaque psoriasis, hyperkeratotic eczema, or drug reactions differentiation through microscopic confirmation (Shrestha & Bischof, 2021).

Laboratory tests serve as supportive diagnostic tools, with eosinophilia and elevated IgE frequently observed in crusted scabies, reflecting the host's immunologic response to mite antigens (Hengge et al., 2006; Shrestha & Bischof, 2021). Other laboratory abnormalities, including leukocytosis, elevated CRP and ESR, hypoalbuminemia, and renal function changes, though these findings are often confounded by coexisting comorbidities or systemic inflammation (Binić et al., 2010; Cuellar-Barboza et al., 2020; Kosmala et al., 2019; Veraldi et al., 2015; Vignesh et al., 2008).

Overall, recent literature highlights the incorporation of molecular and serological methods, such as PCR and serodiagnostic assays, to enhance sensitivity in identifying S. scabiei DNA or antibodies in atypical or low-mite infestations (Sunderkötter et al., 2021; Vasanwala et al., 2019). These advanced approaches, alongside classical microscopy and dermatoscopy, improve diagnostic accuracy and reduce missed diagnoses. Prompt and precise diagnosis is crucial to prevent complications including secondary infection, sepsis, and outbreaks, particularly in institutional settings. In summary, a multimodal diagnostic strategy, integrating clinical assessment, microscopy, dermatoscopy, and laboratory findings, is recommended for optimal detection of crusted scabies and improves patient outcomes.

Table 3. Diseases misdiagnosed prior to crusted scabies diagnosis from reviewed case reports

Title	**Patient's age**	**Gender**	**Comorbidity**	**Disease misdiagnosed prior to Crusted Scabies diagnosis**
Crusted Scabies	71 years old	Female	Otherwise, Healthy	Eczema
Norwegian Scabies management after prolonged disease course: A case report	56 years old	Male	No significant comorbidities	Psoriasis
Treatment for Crusted Scabies: Limitations and Side Effects of Treatment with Ivermectin	In his 90s	Male	Diabetes	Xerotic Skin
Treatment of crusted scabies with acitretin	83 years old	Female	Arterial Hypertension, Parkinson's Disease	Allergic Contact Dermatitis
Treatment of crusted scabies with acitretin	78 years old	Male	Essential Arterial Hypertension, Type 2 Diabetes Mellitus, Benign Prostatic Hypertrophy	Allergic Contact Dermatitis, Psoriasis
Crusted (Norwegian) Scabies Following Systemic and Topical corticosteroid therapy	62 years old	Female	Hypothyroidism	Skin-related changes due to endocrine disorder

Crusted (Norwegian) Scabies Mimicking Psoriasis: A Case Report and Literature Review	49 years old	Male	primary myelofibrosis being treated with ruxolitinib, splenic vein thrombosis, primary hyperthyroidism (currently being treated with levothyroxine).	Psoriasis
Case Report: Crusted Scabies in a patient with systemic disorders- evaluation of ivermectin treatment results	19 years old	Male	Down Syndrome	Erythroderma
Crusted Scabies Misdiagnosed as Erythrodermic Psoriasis in a 3-Year-Old Girl with Down Syndrome	3 years old	Female	Down Syndrome	Erythrodermic Psoriasis

5.4. Treatment Options

The management of scabies relies on both topical and systemic treatments, with specific considerations for crusted scabies due to its severity, extensive mite burden, and high transmissibility. Although ordinary and crusted scabies share similar therapeutic agents, the dosage, frequency, and overall regimen differ substantially between the two. The principles of topical application remain generally the same; however, in crusted scabies, the presence of hyperkeratotic crusts necessitates additional measures such as keratolytic therapy or even surgical debridement to ensure adequate penetration of medications (Aukerman et al., 2019). Crusted scabies also presents challenges because patients often have comorbidities, immunosuppression, or are diagnosed at an advanced stage of disease (Sunderkötter et al., 2016). Identification and early treatment of individuals with crusted scabies is important due to their high parasite loads and potential role as core transmitters (Hay et al., 2012).

A longstanding debate concerns isolation protocols for patients with crusted scabies. Historically, strict isolation was recommended due to the high mite burden and risk of outbreaks. More recent guidance, however, has varied. The 2025 guideline from the UK Health Security Agency does not recommend isolation for crusted scabies, whereas the 2024 Turkish clinical guideline continues to advocate isolation as an important measure to prevent institutional outbreaks (UK Health Security Agency, 2025; Uzun et al., 2024). Regardless of isolation practices, it is essential to treat all close contacts to prevent reinfection and to institute environmental decontamination measures to reduce the risk of recurrence.

5.4.1. Removal of the Crusts and Keratolytic Agents

The removal of crusts is an essential component of treating crusted scabies because hyperkeratotic plaques can prevent penetration of topical agents. Keratolytic treatments such as salicylic acid or urea

help soften and destroy hyperkeratotic lesions, allowing scabicides to reach the mites effectively (Aukerman et al., 2019). In cases of extensive or prolonged disease, surgical debridement may be necessary as an adjunct procedure to remove bulky crusts and enhance therapeutic efficacy.

Although no randomized trials have evaluated the specific contribution of crust removal or keratolytic agents in crusted scabies, clinical experience, particularly in northern Australia, supports their use. A prospective cohort study in Australian Aboriginal patients demonstrated moderate-quality evidence that a regimen combining ivermectin administered on days 0, 14, and 28, together with daily topical permethrin alternating with keratolytic therapy (urea 10% and lactic acid 5%), resulted in a complete cure rate of 40% within four weeks (May et al., 2019). Although this evidence is limited, it suggests that keratolytic therapy plays a meaningful role in improving treatment outcomes in crusted scabies.

5.4.2. Topical Scabicides

Topical scabicides remain an important component of therapy for both ordinary and crusted scabies. In Cambodia, guidelines from the National Center for HIV/AIDS, Dermatology, and STDs recommend permethrin 5% cream and benzyl benzoate 25% lotion as the primary topical agents (National Center For HIV/AIDS, 2011). These medications are typically applied from the neck down, although in cases of relapse or in debilitated institutionalized patients, application to the scalp is also advised to ensure complete eradication (Chiu & Lan, 2020). Crusted scabies, with its dense scale and extensive skin involvement, often requires repeated and intensified application of topical scabicides alongside systemic therapy.

5.4.2.1. Permethrin 5%

Permethrin is widely used in developed countries and is considered one of the most effective topical treatments for scabies. It belongs to the pyrethroid class of insecticides and is safe for human use due to its minimal toxicity and lack of accumulation (Fujimoto et al., 2014). A Cochrane review comparing failure rates found permethrin to be the most efficacious treatment for scabies (Hay et al., 2012). Although these data are derived from ordinary scabies, they remain the best available evidence, as randomized controlled trials in crusted scabies have not yet been performed.

Permethrin exerts its effect by paralyzing motor nerves in mites through the disruption of voltage-dependent sodium channels (Sunderkötter et al., 2021). It is effective against adult mites but does not kill eggs. Correct application is essential; recommended doses are at least 25–30g for adults, 15g for children aged 6–12, and 7.5g for children aged 2 months to 5 years (Sunderkötter et al., 2021). The skin should be dry for at least 30 minutes before application. The cream should remain on the skin for 8–12 hours, and additional applications after 7–10 days are recommended. Permethrin is generally safe for infants and for use during pregnancy and lactation (Hay et al., 2012).

5.4.2.2. Benzyl Benzoate (10–25%)

Benzyl benzoate acts by paralyzing the respiratory muscles of mites (Fujimoto et al., 2014) and is highly effective, affordable, and commonly used as first-line therapy in several regions. While often requiring only a single dose, some guidelines recommend a second application after 2–7 days (Hay et al., 2012). Dosing regimens vary widely; Japanese guidelines advise daily application for 2–4 weeks (Fujimoto et al., 2014; Leung et al., 2019), whereas other sources recommend daily application for seven days followed by weekly applications until lesions clear.

However, benzyl benzoate may cause skin irritation, particularly in children, which limits its use. Dilution to 12.5% for children or 6.25% for infants reduces irritation but may also reduce efficacy (Hay et al., 2012). Despite this, benzyl benzoate remains a widely used and effective agent, achieving cure rates over 90% in ordinary scabies (Fujimoto et al., 2014). It is the primary topical preparation used in France, although it is less commonly used in the United States (Ishii et al., 2008).

5.4.2.3. Malathion 0.5%

Malathion inhibits cholinesterase, leading to accumulation of acetylcholine at nerve endings and paralysis of the mite (Walton et al., 2010). It is typically applied for 24 hours and serves as an alternative for patients who cannot tolerate benzyl benzoate (Aukerman et al., 2019).

5.4.2.4. Sulfur Ointment (6%, range 2–10%)

Sulfur ointment is one of the oldest treatments for scabies (Hay et al., 2012). It requires 24-hour application followed by washing and reapplication. In regular scabies, it is used for three consecutive days (Aukerman et al., 2019). Japanese guidelines recommend 2–5 days of use, up to seven days if necessary. Although safe for infants and young children, sulfur ointment is messy, has an unpleasant odor, and frequently causes irritation (Hay et al., 2012).

5.4.2.5. Lindane (0.5–1%)

Lindane is applied for 6–8 hours before washing off (Hay et al., 2012; Ishii et al., 2008). Although effective against both adult mites and nearly hatched eggs, it has been discontinued in many countries due to safety concerns and is considered second-line therapy in developed nations. It should be avoided in pregnant or lactating women (Hay et al., 2012), children under 10 years old, individuals with epilepsy, and those with low body fat (Ishii et al., 2008). Lindane can suppress GABA and may interact with other drugs. Despite its nearly 100% efficacy, its lack of antipruritic effect and safety concerns limit its use (Fujimoto et al., 2014; Shimose & Munoz-Price, 2013).

5.4.2.6. Crotamiton 10%

The mechanism of action of crotamiton remains unclear. Initially developed as an insecticide, it is now used as a scabicide and antipruritic agent (Ishii et al., 2017). It is safe and well-tolerated, making it a reasonable option for infants, but requires repeated applications over 10–14 days to be effective (Walton et al., 2010). Its scabicidal effects are weak (Fujimoto et al., 2014).

5.4.2.7. Phenothrin

Phenothrin has demonstrated an efficacy rate of 92.6% in a clinical study of ordinary scabies (Ishii et al., 2017). It possesses a high safety margin compared to permethrin and is applied to the entire body, often with occlusive dressings such as Vaseline containing salicylic acid or zinc oxide to soften crusts. Weekly treatment cycles are recommended, with keratolytics applied between treatments. Nail trimming and removal of thickened keratin can improve drug penetration (Ishii et al., 2017).

5.4.3. Ivermectin

Ivermectin is a cornerstone of crusted scabies treatment due to its systemic activity and ability to reach mites in deeper layers of the skin. It binds to glutamate-gated chloride channels, causing paralysis and

death of mites (Fujimoto et al., 2014). A single oral dose of 0.2 mg/kg is effective in ordinary scabies; however, multiple doses are required in crusted scabies because ivermectin does not affect eggs.

Ivermectin tablets contain 3 mg of the drug and may be used in patients over 15 kg. A syrup formulation (0.4 mg/ml) exists for children under 15 kg (Sunderkötter et al., 2021). Dosing intervals vary; a two-week interval is commonly recommended (Fujimoto et al., 2014), although more intensive regimens have been used for HIV patients with crusted scabies, such as treatment on days 1, 2, and 8 (Del Borgo et al., 2015).

More complex regimens have also been proposed. Molecular genotyping suggests that even three doses given 14 days apart may be inadequate in severe cases (Roberts et al., 2005). A five-dose regimen on days 1, 2, 8, 9, and 15, with optional additional doses on days 22 and 29, has been recommended for Grade 3 crusted scabies (Roberts et al., 2005). Another study tailored dosing based on severity, recommending 3, 5, or 7 doses for Grades 1, 2, and 3 respectively (Davis et al., 2013; Hasan et al., 2020).

Ivermectin has a serum half-life of 18 hours and undergoes hepatic metabolism and renal excretion (Ortega-Loayza et al., 2013). Liver function and platelet counts should be monitored. Although generally safe, ivermectin should not be used in children under five years old or during pregnancy and lactation (Hengge et al., 2006). It is ineffective for nail scabies (Fujimoto et al., 2014; Ishii et al., 2008).

5.4.4. Supportive Treatment and Environmental Measures

Supportive treatments aim to reduce symptoms and prevent secondary infections. Antihistamines may provide relief from itching (Hay et al., 2012), and topical antipruritic such as menthol or pramocaine may be helpful. Secondary bacterial infections should be treated promptly; severe infections may require systemic antibiotics, including cloxacillin, clindamycin, cephalosporins, or macrolides (Leung et al., 2019).

Environmental measures are essential to reduce reinfestation. Clothes, towels, and bed linens should be changed regularly until at least one day after the second treatment or until hyperkeratosis resolves (Sunderkötter et al., 2016). Furniture and textiles that come into prolonged contact with the patient should be thoroughly vacuumed and avoided for seven days. Items that cannot be adequately cleaned may be autoclaved.

5.4.5. Integrated Treatment Strategy for Crusted Scabies

Effective management of crusted scabies requires a multimodal approach combining oral ivermectin, topical scabicides, keratolytic therapy, and environmental decontamination. Experience from northern Australia highlights a regimen of multiple doses of ivermectin combined with topical permethrin and keratolytic agents as particularly effective (Hay et al., 2012). Healing is confirmed when microscopy detects no mites in two consecutive tests performed 1–2 weeks apart, and the absence of new burrows is confirmed (Ishii et al., 2008). Assessment should be performed one month after the last observation.

Reasons for treatment failure often include incorrect application, inadequate repetition of treatment, reinfection due to incomplete environmental decontamination, lack of treatment for contacts, or insufficient patient education (Sunderkötter et al., 2021). No randomized controlled trials exist comparing treatment regimens for crusted scabies (Hay et al., 2012), and current recommendations are therefore based on expert consensus.

5.4.6. Interest in Novel Drug Development

Emerging concerns regarding potential drug resistance have stimulated interest in new treatments. In veterinary medicine, resistance to ivermectin has already been documented (Ishii et al., 2017). Although

human cases of permethrin or ivermectin resistance have not been confirmed outside Australia and New Zealand as of 2016, reports of decreased responsiveness are increasing (Sunderkötter et al., 2021). Several natural products, such as tea tree oil containing terpinene and the essential oil of Lippia multiflora, have demonstrated scabicidal properties (Hengge et al., 2006). Acitretin has been used successfully in two cases of persistent crusted scabies refractory to standard therapy (Veraldi et al., 2015), although research is insufficient to recommend its routine use. Moxidectin, a drug currently undergoing clinical testing, has a longer half-life than ivermectin and may be effective as a single-dose therapy, but its safety and efficacy against scabies require further evaluation (Sunderkötter et al., 2021).

6. Limitations

A notable limitation of this study is the paucity of comprehensive clinical trials and studies available on Crusted Scabies. Given its status as a rare and often neglected disease, the quantity and quality of academic literature and research in this field are considerably limited. Consequently, the review primarily relies on a diverse range of sources, including case reports, observational studies, and retrospective analyses. The inherent heterogeneity in these sources, coupled with the scarcity of large-scale randomized controlled trials, can introduce variations in the reliability and robustness of the data examined. Furthermore, only three main databases, namely PubMed, Google Scholar, and Research4Life, had been utilized to look for articles, as we surmised that these three databases would be enough for this small-scale narrative review. Despite these limitations, this review endeavors to synthesize the available evidence to gain insights into the pathogenesis, clinical manifestations, and immunological responses associated with Crusted Scabies.

7. Conclusions

Crusted scabies remain a serious public health and clinical concern, particularly among vulnerable populations. Key risk factors include immunosuppression (e.g., HIV infection, use of corticosteroids, and hematologic malignancies), neurological conditions that impair itch perception, and institutionalized living conditions. Genetic predispositions may also contribute to some populations.

Evidence-based management requires a systemic approach, prioritizing early recognition and diagnosis, strict infection control measures, and combination therapy. Oral ivermectin, used in multiple doses alongside topical scabicides and keratolytic agents, represents the current standard of care. Integrating severity grading scales, such as the Davis et al. (2013) classification, into routine clinical decision-making can enhance treatment precision, optimize resource use, and improve outcomes. Moreover, isolating affected individuals and treating close contacts is crucial to preventing outbreaks. Future directions should focus on the development of standardized protocols, resistance surveillance, and the inclusion of crusted scabies in public health guidelines to better control transmission and reduce morbidity and mortality.

8. Recommendation

This review highlights the need for guidelines and standardized protocols to improve outcomes for patients with crusted scabies. Crusted scabies isn't caused by a different parasite. It's the same Sarcoptes scabies, but it presents much more severely in people whose immune systems are already under stress. As more patients around the world receive treatments like chemotherapy, organ transplants, or long-term steroids, especially in the post-COVID-19 era, conditions like crusted scabies is becoming more common in hospitals and care homes (Glennie et al., 2021). The problem is that corticosteroids and other

immunosuppressive therapies can mask the typical signs of scabies, allowing the mites to multiply unchecked. For this reason, clinicians need to consider crusted scabies in their differential diagnosis when they encounter thickened, crusted, or scaly lesions, especially in older, institutionalized, or immuno-compromised patients, and particularly when eosinophilia is present in blood work.

In the context of Cambodia, there is a lack of hospital records and research regarding patients with crusted scabies; therefore, documentation and research initiatives must be encouraged. Furthermore, even though, most evidence points to permethrin 5% as the topical agent of choice in Crusted Scabies, Benzyl Benzoate is more suitable as the first-line topical treatment in Cambodia due to it being more cost-effective and comparable in efficacy to Permethrin (Bachewar et al., 2009); however, Phenothrin being the newly emerging scabicide with good efficacy should also be included in the recommended regimen, which points to a need in expanding the drug supply in Cambodia. In combination with topical scabicides, we also recommend the regimen of keratolytic agents and Ivermectin for the treatment of crusted scabies.

The development of effective anti-parasite therapies is crucial due to emerging resistance to available treatments and concerns about drug residues on consumer health. Additionally, an improved understanding of crusted scabies pathogenesis can aid in the development of vaccines or new therapeutic treatments for susceptible populations. This review also emphasizes the heterogeneity in methods and outcome measurements among treatment trials, stressing the need to standardize protocols and clinical assessment for scabies infestation. The burden of crusted scabies and their role in sustaining endemic transmission is not fully quantified, but it is considered a significant factor.

Further research is needed to determine the roles of specific immune cells and antibody levels in crusted scabies, develop sero-diagnostic tests, and advance immunotherapy and vaccine strategies. This review underscores the importance of health education, improved diagnosis, and drug supply in reducing scabies prevalence.

Author contributions

NP, MT, and SP contributed to the design and implementation of the research, to the analysis of the results and to the writing of the manuscript.

Data availability

Data is available on request.

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Conflicts of interest

The authors declare no conflicts of interest related to the research, or the publication of this manuscript.

Supplementary materials/Appendix

Appendix A. Sample Article Summary Form

Article Summary Form

We will undertake a keyword search in Google Scholar and PubMed of articles published from 2003 to 2022. Only English language articles will be included.

Title: A Narrative Review on Crusted Scabies: Pathogenesis, Clinical Manifestations, Diagnosis, and Treatment Options

To help with our review writing, we will compile a summary report of the key findings and eventual recommendations for each article, as below:

Keyword search term (through Google Scholar, PubMed)	**Article citation**	**Pathogenesis** (number of mites, humoral immune response, cellular immune response)	**Clinical Manifestations** (skin lesions, distribution, systemic symptoms)	**Diagnosis** (Grading scale; invasive, non-invasive techniques)	**Treatment Options** (Oral, Topical treatment, Injections)	**Eventual recommendations of the Authors**

Appendix B: Complete Article Summary Form (50 articles

Crusted Scabies Article Summary

We will undertake a keyword search in Google Scholar and PubMed of articles published from 2003 to 2022. Only English language articles will be included.

Title: A Narrative Review on Crusted Scabies: Pathogenesis, Clinical Manifestations, Diagnosis, and Treatment Options

To help with our review writing, we will compile a summary report of the key findings and eventual recommendations for each article, as below:

Keyword search term (through Google Scholar, PubMed)	**Citation for article**	**Publication Date**	**Pathogenesis** (number of mites, humoral immune response, cellular immune response)	**Clinical Manifestations** (Lesions, Distribution)	**Diagnosis** (Grading scale; invasive, non-invasive techniques)	**Treatment Options** (Oral, Topical treatment, Injections)	Eventual recommendations of the Authors
GOOGLE SCHOLAR keywords: Scabies, Clinical Manifestation Title: Review: Nosocomial scabies	Vorou R, Remoudaki HD, Maltezou HC. Nosocomial scabies. Journal of Hospital Infection. 2007 Jan;65(1):9–14.	1/1/2007	-Immunocompromised or elderly institutionalized patients admitted with unrecognized crusted scabies are the main source of nosocomial transmission. -About two million of mites found in crusted scabies while in classic scabies around 10-15 mites. -The numerous mites found in crusted scabies facilitate transmission through the environment and explain why it is highly contagious. Patients with crusted scabies serve as a reservoir for the mite. The increased morbidity and mortality.	Crusted scabies is a scaly dermatosis with mild or no pruritus, occasionally accompanied by generalized lymphadenopathy. Nails are commonly involved. Crusted scabies may also manifest as an erythematous eruption. Subsequent long-term local corticosteroid	-A compatible presentation and a family history, residence or working in a nursing home, or a cluster of non-specific pruritic rashes among HCWs should raise the suspicion for scabies. -An oil preparation of skin scraping or material retrieved from underneath the nails enable visualization of mites, eggs, or feces. -Epiluminescence microscopy is an in vivo disposable gloves, gowns and shoe covers. -Patients with crusted scabies should be cared for by the minimum number of HCWs. - Difficulty diagnosing: Most HIV/AIDs patients were misdiagnosed with seborrhoeic dermatitis or eczema, and scabies was suspected when no response occurred the following treatment.	-Ivermectin is indicated for crusted scabies at one to three doses of 200ug/kg each. -Ivermectin is recommended along with local scabicides and keratolytic. -Ivermectin is 100% effective with rare relapses and it is cost comparable with local agents; for immunocompromised patients.	
GOOGLE SCHOLAR keywords: Crusted, Norwegian, Scabies, Clinical Manifestation Title: Atypically distributed cutaneous lesions of Norwegian scabies in an HIV-positive man in South India: a case report	Vignesh R, Shankar EM, Devaleenal B, Balakrishnan P, Thousen SM, Sekar R, et al. Atypically distributed cutaneous lesions of Norwegian scabies in an HIV-positive man in South India: a case report. Journal of Medical Case Reports. 2008 Mar 14;2(1).	3/14/2008		-16-year-old man with HIV infection was admitted to the inpatient department of th YRG Centre for AIDS Research and Education. -He presented with severe crusted cutaneous lesions all over the body (face, ear lobes, shoulder blades and entire trunk). There was extensive, generalized, thick, hyperkeratotic, crusting, yellowish papule lesions with squamous lesions not sparing any region..	-skin crusts were collected and mounted on 10% KOH preparaion and observed under low and high-poer objectives. -Numerous live and motile, adult A.scabiei mites that measured about 400 um long and 300 um wide were seen, which confirmed the diagnosis of Norwegian scabies. - Laboratory investigations revealed elevated erythrocyte sedimentaion rate (ESR) of 20mm (normal 0-14mm)	-The patient was started with Ivermectin (6mg) for 15 days and topical Permecthrin cream with meticulous scrubbing and cleansing of the skin which eventually resulted in complete resolution 4 weeks later.	

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