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REVIEW ARTICLE

Scabies: A comprehensive review and current perspectives

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Abstract

Human scabies is a contagious skin infestation caused by the parasitic mite *Sarcoptes scabiei* var. *hominis*. It is a common skin disease worldwide that occurs not only in the underprivileged sections of society but also in developed countries. In 2009, World Health Organization (WHO) recognized scabies as "neglected tropical disease (NTD)" or NTD thus emphasizing the need for community awareness and proper treatment strategies. This review attempts to summarize the varied clinical presentation of the disease and describes the advances in diagnosis and management including the ongoing search for novel agents to overcome the problems associated with conventional treatments. The literature research includes peer-reviewed articles (clinical trials or scientific reviews). Studies were identified by searching electronic databases (MEDLINE and PubMed) till February 2020 and reference lists of respective articles. Only articles published in English language were included.

KEYWORDS

Sarcoptes scabiei var. *hominis*, scabies, treatment

1 | INTRODUCTION

Human scabies is caused by the mite *Sarcoptes scabei* var. *hominis*, which is an obligate ectoparasite that lives in the human epidermis.[1] The disease is common in overcrowded populations and is transmitted by close skin-to-skin or sexual contact. The cutaneous signs and symptoms occur due to hypersensitivity reaction to mite antigens. Patients present with an intensely itchy eruption that disturbs sleep and affects the quality of life. Diagnosis can be challenging due to the varied presentation and lack of diagnostic criteria, which have come up only recently.[2,3]

2 | ETIOLOGY—SCABIES MITE

Sarcoptes scabiei var hominis, the scabies mite causing infection in humans, is an obligate human parasite that is creamy white in color. Though the mite was discovered in the year 1687, the earliest references to scabies date back to the year 482 BC in Egypt.[4] The scabies mite belongs to the family *Sarcoptidae* that has three subfamilies. Sarcoptes belongs to the *Sarcoptinae* subfamily.[5,6] The female mite measures 0.4 × 0.3 mm whereas the female is smaller. The adult mite has four pairs of legs compared to the larval form that has three pairs of legs.

The female mite burrows into the stratum corneum at a speed of 0.5 to 5 mm per day and lays eggs after copulation. In 3 to 4 days, eggs hatch into larvae that leave the burrow and mature on the surface of skin into adults. The life cycle is around 14 to 21 days. The life span of a female mite is 4 to 6 weeks during which it lays around 40 to 50 eggs.[7,8]

The mite avoids areas that have high density of sebaceous glands. There are around 12 mites on an average in an individual with classical scabies. Adult females live in the host for up to 1 month. Outside the human host, mites can survive for around 24 to 36 h at room

conditions. This time is longer in cold environmental conditions. The ability to infest a host decreases as the time outside the host increases.[9,10] The minimal time necessary for skin-to-skin transmission is 5 min.[11]

3 | EPIDEMIOLOGY

According to the 2015 Global Burden of Disease Study, around 204 million people are affected with scabies worldwide.[12] The infection is more common in underprivileged and resource poor settings where there is overcrowding and lack of hygiene. In developed countries, scabies frequently occurs as institutional outbreaks. The prevalence is higher in children.[13,14] The main mode of transmission of classical scabies is skin-to-skin transmission, spread via fomites is important in cases of crusted scabies. Sexual transmission can also occur and scabies is considered a sexually transmitted disease.[15]

4 | CLINICAL PRESENTATION

Symptoms occur 3 to 6 weeks after primary infestation but can occur as early as 1 to 2 days in cases of re-infestation.[16,17] Pathognomonic lesion of scabies is the burrow seen as short, linear tracks that end with an intact vesicle or erosion containing the mite. Burrows are usually seen in the finger web spaces, hands, wrists, axillae, feet, buttocks, and genitalia (Figure 1). Presence of burrows is classical of scabies. However, they are observed only in few cases.[18] Non-specific secondary lesions in the form of excoriated papules, eczematous or lichenified plaques, nodules are more commonly seen (Figure 2). Patients complain of generalized itching that is worse at night.

5 | IMMUNOLOGY IN SCABIES

In classical scabies, there is a mixed Th1/Th2 (protective) immune response whereas in crusted scabies there is a pronounced Th2 (non- protective) allergic response.[19] Th2 response promotes growth and activation of eosinophils and also causes increase in IgE along with down-regulation of cell mediated immunity. Also, there is activation of Th17 cell response leading to increase in IL-17 and IL-23 in crusted scabies. Immunoglobulin levels (Ig G, E, A, and M) are increased in classical scabies whereas the levels (Ig G, E, and A) are highly elevated in the crusted variant. The latter also shows decrease in serum levels of complement C3 and C4.

CD4+ T cells, which form the predominant inflammatory infiltrate in classical scabies have a protective role. HIV patients are associated with severe disease due to a decrease in CD4+ T cells. Aging is associated with decrease in Th1 cell function and enhanced Th2 function explaining the increased incidence of scabies in elderly population in nursing homes.[20]

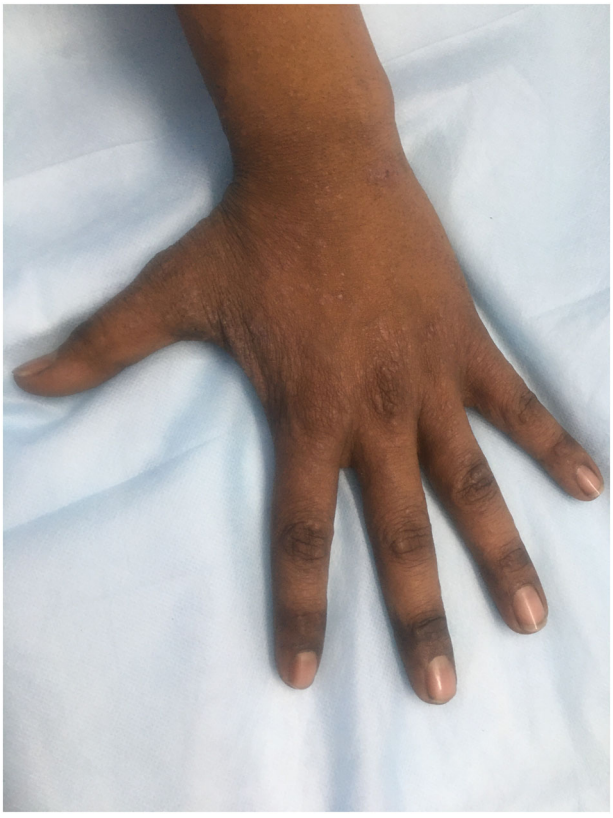

FIGURE 1 Interdigital involvement with lesions on hands in a young male with scabies

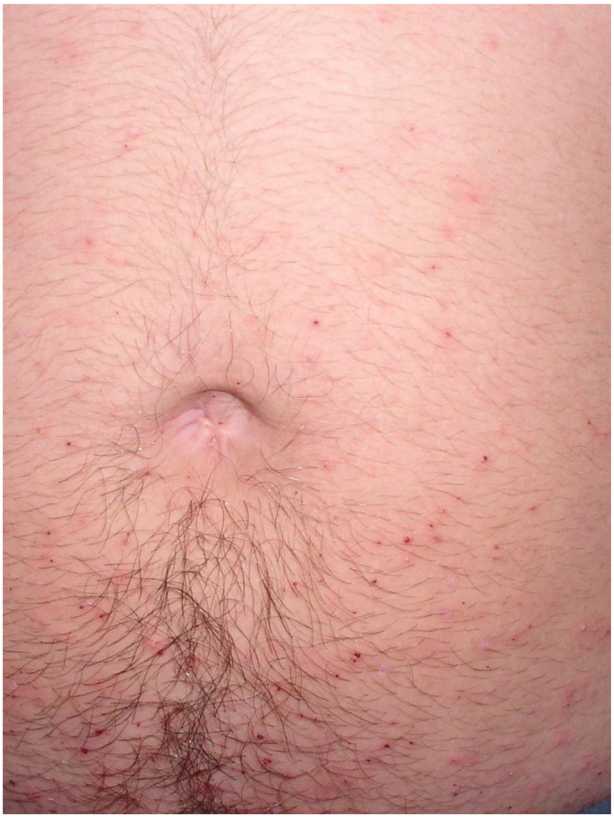

FIGURE 2 Classical features of scabies in an immunocompetent man

6 | PRURITUS IN SCABIES

Scabies was previously described as "the worst itch" of a patient's life emphasizing the considerable itching that occurs in this disease. Pruritus in scabies can occur either due to direct action by scabies mite or due to immune response elicited by the host against it. *Sarcoptes scabiei* can trigger itching by the following mechanisms:

i. Activation of TLR-3,4,7 expressed on primary sensory neurons.[21]
ii. Proteases present in mite feces activate protease activated receptor-2.[22] Direct action on keratinocytes leading to activation of pruritus receptors.

Itch, caused by the immune response against scabies, occurs due to:

a. Complement system activation that causes stimulation of mast cells causing release of histamine, tryptase, and TNF-alpha.[23,24]
b. Macrophage activation leads to production of prostaglandins and leukotrienes that potentiate the itch.
c. Th1 response in classic scabies leads to production of IFN-gamma, TNF-alpha, and IL-2 that activate prurireceptors.[23] There is a Th2 response in crusted scabies that causes B cell activation and production of IL-4, IL-5, IL-13, and IL-31 that activate prurireceptors. IgE is produced that causes mast cell activation.
d. Th2 cells demonstrate an increased generation of thymic stromal lymphopoietin and periostin stimulating hereby Arginase-1(+)/CD163(+) M2 macrophages.[24] M2 macrophages generate IL-31 which causes pruritus.

7 | ATYPICAL PRESENTATION

Scabies can have atypical presentation depending on the site of involvement, morphology of lesions and age group affected (Table 1).

8 | SCALP INVOLVEMENT

Scalp involvement is seen in infants, children, elderly, immunocompromised patients, and crusted scabies. It may resemble seborrheic dermatitis.

9 | BULLOUS SCABIES

It usually occurs in elderly males and presents as intensely itchy bullae that may be flaccid or tense with or without classical lesions of scabies. There are various mechanisms that have been postulated for the development of bullous lesions in scabies. These are:

i. Blisters can occur secondary to superinfection of the scabietic lesion with *Staphylococcus aureus* similar to bullous impetigo.
ii. Bullae formation can be autoantibody mediated. This can occur due to the lytic destruction of basement membrane zone (BMZ) mediated by enzymes produced by the mite which leads to production of Bullous Pemphigoid (BP)-like antigens that trigger formation of BP antibodies causing complement cascade activation and dermo-epidermal separation.[25,26] Mites have been found within the blister cavity hence supporting this hypothesis.

Autoantibody formation can also be explained by the phenomenon of antigen mimicry that is cross reactivity between mite antigens and the BMZ antigens that leads to production of BP antibodies.[25] Few cases in literature have demonstrated the presence of antibodies against BP180 or BP230 in patients with bullous scabies, thereby supporting this theory.[27,28] An id reaction to scabies mite, called as scabid, can occur leading to bulla formation.[29]

iii. Scabies can trigger the bullous eruptions of BP via a Koebner phenomenon explaining the occurrence of both scabies and BP in a single patient.

Linear deposition of C3 and IgG is often seen on DIF in bullous scabies.[30,31]

Treatment remains the same as classical scabies. Some cases may require a short course of oral steroids.

TABLE 1 Atypical presentation in scabies

Site	Scalp involvement
Morphology	Bullous scabies Nodular scabies Crusted scabies
Special groups	Infantile scabies Scabies in elderly Scabies in immunocompromised patients

10 | NODULAR SCABIES

Chronic infestation due to hypersensitivity leads to severe eczematous changes and nodule formation especially on male genitalia (Figure 3) and breasts. These are intensely itchy and persist for weeks or months even after effective treatment.[32]

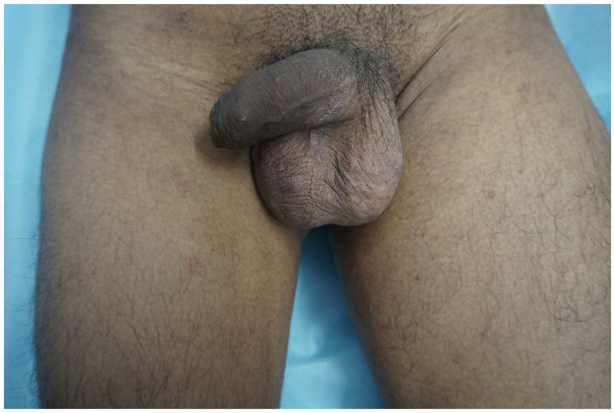

FIGURE 3 Nodules over the scrotum

11 | CRUSTED/NORWEGIAN SCABIES

It was first described by Boeck and Danielssen in leprosy patients in Norway in the year 1848 and named as Norwegian scabies by Von Hebra.[33]

Crusted scabies is characterized by dense hyperkeratosis of skin, which occurs due to proliferation of mites because of altered host response. Crusted scabies is seen in three types of patients:

a. Defective T cell immunity—HIV, leukemia, and lymphoma.[34,35]
b. Reduced cutaneous sensation—leprosy, neurological disorders like tabes dorsalis and syringomyelia.
c. Reduced ability to debride the mites mechanically—critical illness, Down syndrome, and senile dementia.

Crusted scabies has also been seen in Australian aborigines with normal immunity and could be associated with HLA-A11.[36,37]

There is an imbalance of inflammatory response in the dermis leading to increased levels of IL-4 that is responsible for the dense hyperkeratosis of skin.[37] This along with failure to mount an effective response results in uncontrolled proliferation of the mite which in case of crusted scabies can reach a number in millions. Unlike classical scabies, transmission via fomites is commonly seen in the crusted variant. Itching is minimal or absent. The lesions are insidious in onset and progression with formation of yellow-brown or yellow green, gray crusts that are firmly adherent and on removal show a porous appearance. There may be fissuring over the extensors. Crusts are commonly seen over the scalp, ear, extensor aspect of elbows, and on soles. Nails show subungual hyperkeratosis similar to psoriatic nails and are an important source of relapse. Rarely, crusted scabies can present as erythroderma.[38] It is likely to be complicated by secondary bacterial infection by *Staphylococcus aureus*, septicaemia, and generalized lymphadenopathy.

Crusted scabies should be differentiated from psoriasis, eczema, seborrheic dermatitis, and pityriasis rubra pilaris.

Diagnosis can be confirmed by microscopic examination of skin scrapings. Eosinophilia and increase in serum IgE can occur.[39]

Davis et al proposed a severity grading scale based on the distribution and extent of crusting, history of past episodes and condition of skin. The scores were graded into mild (grade 1, score 4-6), moderate (grade 2, score 7-9), and severe (grade 3, score 10-12).[40]

Roberts et al recommended a 5-dose regimen of oral ivermectin for patients with crusted scabies on day 1, 2, 8, 9, and 15 with additional two doses on 22nd and 29th day in severe cases.[38]

12 | INFANTILE SCABIES

Infants develop very few excoriations due to their immature pincer grasp. Due to the inability to itch, the number of burrows are numerous and inflammatory and appear as red, oedematous, crusted serpiginous papulovesicles, and nodules. The mite load is higher reaching a few hundred mites in one patient.[41-43] Also the early eruption in infants is often localized compared to older children and adults who present with widely disseminated lesions.[44,45] A study has shown that infants with scabies have more nodules and greater lower limb involvement especially of soles as compared to older children and adults.[46]

Delay in diagnosis can lead to superficial skin infections, ecthyma, and cellulitis. Infantile scabies should be differentiated from other similar disorders presenting in this age group like papular urticaria, atopic dermatitis, and infantile acropustulosis. Though topical permethrin is safe and effective in children, FDA has not approved it for infants younger than 2 months.

13 | SCABIES IN ELDERLY

In elderly patients, burrows are more often found on the soles of feet.[47] There may be complete sparing of the finger webs. Scalp and facial involvement is common unlike younger population.[20] Old patients are more prone to develop crusted scabies. This is due to the presence of several factors is this particular group like altered immune response, nutritional deficiency, decline in cognitive function and inability to maintain personal hygiene.

Treatment remains the same. However, patients may have difficulty in applying topical treatment. In such cases, oral ivermectin may be preferred.

14 | SCABIES IN IMMUNOCOMPROMISED PATIENTS

Severe involvement can occur in immunocompromised patients. Crusted scabies can occur and itching may be mild or absent.

15 | COMPLICATIONS

The most common complication seen in scabies is secondary infection and impetiginization that is most often due to *Streptococcus pyogenes* and *Staphylococcus aureus*. Impetigo due to *S. pyogenes* can lead to toxin mediated diseases like scarlet fever, streptococcal toxic shock syndrome, rheumatic fever and post-streptococcal glomerulonephritis. The latter leads to more than 160 000 deaths in tropical and subtropical countries per year and is a risk factor for chronic renal disorders in adulthood.[48]

Modification of clinical picture, called as scabies incognito, can occur due to inappropriate use of topical steroids causing delay in diagnosis.[49] Severe eczematization can also occur (Figure 4).

16 | DIFFERENTIAL DIAGNOSIS

Depending on the clinical presentation, scabies can mimic a variety of common and uncommon dermatoses including atopic dermatitis, papular urticaria, folliculitis, dermatitis herpetiformis, prurigo nodularis, pityriasis rosea, and insect bites.[50,51]

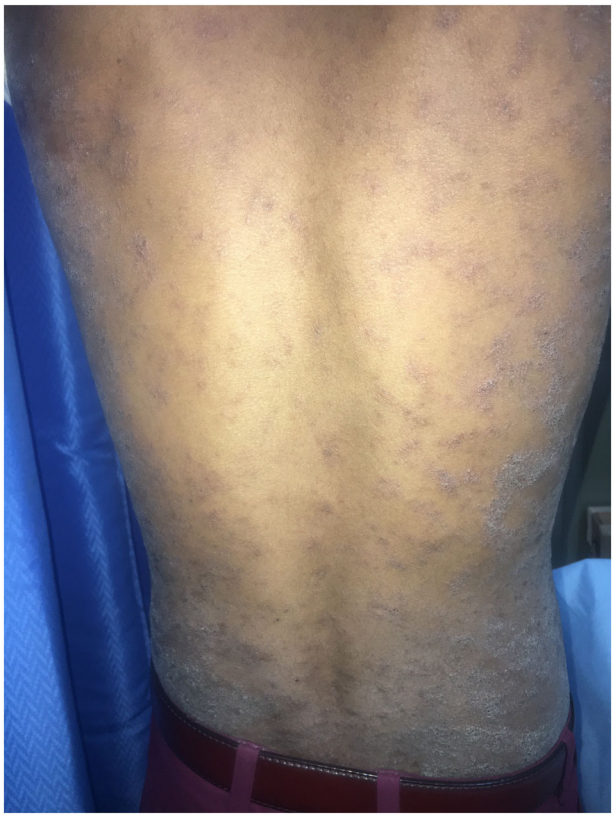

FIGURE 4 Eczematization in scabies

17 | INVESTIGATIONS

There are various invasive and non-invasive methods to diagnose scabies as outlined in Table 2.

18 | SCRAPINGS

Direct visualization of mite, its eggs or feces forms the gold standard for diagnosis. For this, a drop of mineral oil is placed at the end of a burrow and the lesion is gently scraped with a sterile blade.[52] The material is placed on a glass slide and covered with potassium hydroxide (KOH) and examined after few minutes.

The sensitivity of this test is low and repeated tests from different sites may be needed. This is because of the limited number of mites found in a patient with classical scabies. In addition, the test will be difficult to perform in children who may not cooperate. The presence of fecal pellets or scyballa should not be considered diagnostic, as these resemble artifacts or debris.

19 | SKIN BIOPSY

On histopathological examination, burrow can be seen within the horny layer. The end of the burrow reaches the Malphigian layer of the skin where the female mite can be seen as a rounded body. Eggs containing larvae, eggshells, fecal deposits (scyballa) with the stratum corneum are indicative of scabies (Figure 5). Spongiosis may be seen in papulovesicular lesions of scabies. Eosinophils can also be seen. Biopsy from nodules shows dense chronic inflammatory infiltrate, which can be pseudolymphomatous.[53]

Thickened horny layer with numerous mites are seen in crusted scabies.

TABLE 2 Various invasive and non-invasive diagnostic modalities for scabies

Invasive	Non-invasive
KOH scraping of burrow	Dermoscopy
Skin biopsy	Videodermoscopy
Burrow ink test	Reflectance Confocal Microscopy (RCM)
Adhesive tape test	Optical Coherence Tomography (OCT)
PCR of scrapings	
Serology	

20 | BURROW INK TEST

Papules are marked with ink and wiped off with an alcohol swab to remove ink from the surface of lesion. Positive burrow ink test is said to occur when a dark, zigzagged line can be seen with the naked eye after wiping with alcohol.[54] This occurs due to tracking down of ink into the burrow.

21 | ADHESIVE TAPE TEST

The adhesive side of tape is firmly applied to a suspicious skin lesion. Tape is pulled off and transferred directly onto a slide for microscopic examination. Though the method is simple its sensitivity is low.[55,56]

22 | VIDEODERMATOSCOPY/ VIDEODERMOSCOPY (VD)

It is a non-invasive technique that allows visualization of burrows and scabies mite using magnification of ×40-×100. VD shows a dark brown triangular structure at the end of a linear segment called "jet with a contrail".[57] The triangular structure corresponds to the pigmented anterior part of the scabies mite whereas the linear segment is the burrow containing eggs or feces.[58-60]

VD is a rapid non-invasive diagnostic tool with a specificity of 100% and sensitivity higher than skin scraping. Moreover, it does not cause any discomfort to the patient hence can be used in non-cooperative patients. It is also less time consuming and also minimizes risk of cross infections.[61]

Another use of VD is for follow-up of patients post therapy. Presence of viable mites in such cases suggests persistence of infection. It

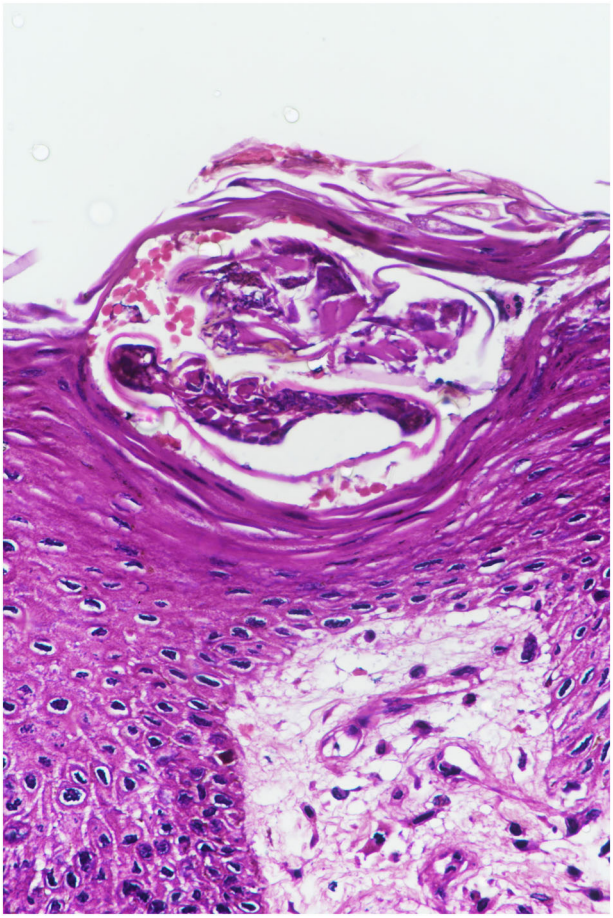

FIGURE 5 Burrow in stratum corneum with *Sarcoptes scabiei* mite (H&E, ×100)

can also be used as a screening tool for asymptomatic family members.

23 | DERMATOSCOPY/DERMOSCOPY

Diagnosis is supported by dermoscopy. It is easier to use and less expensive than VD. Studies have found a sensitivity of 91% and specificity of 86%.[62] The "delta wing jet" sign or kite sign seen on dermoscopy is a whitish structureless line with a dark brown triangular structure in the end (Figures 6 and 7). Another sign described is the "minitriangle sign" refers to scabies eggs that show head of the maturing mite inside. Air bubbles in the track are known as wake sign.[63] Dermoscopy in crusted scabies shows hyperkeratotic appearance with several burrows (Figure 8).

The main limitation is the low specificity due to less magnification. In addition, scratching induced changes like excoriations, bleeding, or crusting can be mistaken for the "jetliner"sign.[64] This sign may not be visible in dark skin and in hairy body areas. Eggs and feces cannot be visualized due to low magnification leading to underdiagnosis.

Other limitations are low sensitivity in mild disease and operator dependence. Hence, it is recommended that dermoscopy should be used when VD is not available or for screening of suspicious lesions prior to scraping.[65]

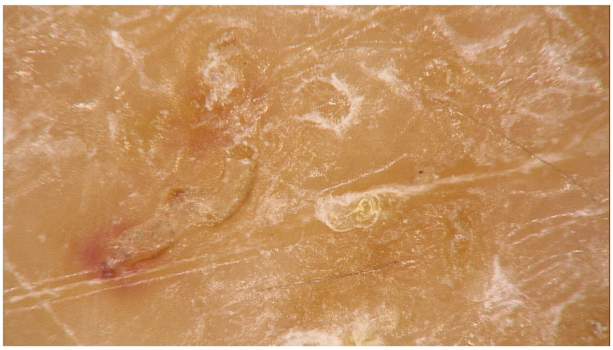

FIGURE 6 Dermoscopy of scabies shows a small dark brown triangular structure located at the end of whitish curved or wavy line. This feature has been called "delta-wing jet with contrail" as shown in the image (70-fold magnification)

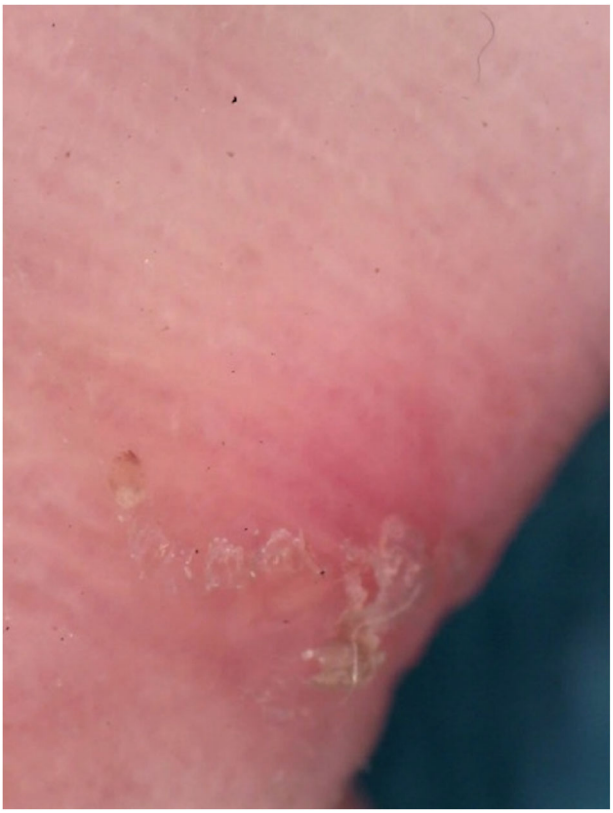

FIGURE 7 Dermoscopy of another patient with scabies. Web space showing a curvilinear burrow and triangular mite at the end of the burrow (jet with contrail sign, Dinolite AM413ZT, polarized ×0)

24 | REFLECTANCE CONFOCAL MICROSCOPY

It is a new non-invasive technique used for scanning the skin at different layers using a reflected laser beam. It allows visualization of

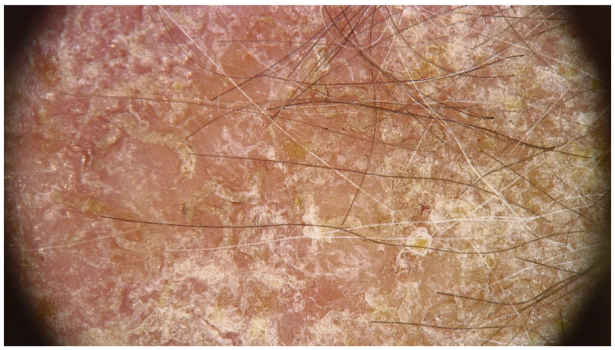

FIGURE 8 Dermoscopy of an immunosuppressed patient with Norwegian scabies (×20-fold magnification)

burrows, which are seen as a linear segment amidst the surrounding epidermis that shows a "honeycomb" pattern. Mite, larvae, eggs, and fecal material can also be seen.[66,67] The mite's biological behavior can be studied with RCM as it also shows the movement and peristalsis of mite. Therefore, it can also be used as a tool to detect mite viability after treatment with scabicide.[68]

The limitation of RCM is its non-availability and expensive equipment. In addition, it is a very time consuming technique.

25 | OPTICAL COHERENCE TOMOGRAPHY

It is similar to ultrasonography but with higher resolution and enables visualization of the main skin components. In scabies, burrows, mites, and eggs can be seen and studied. It is mainly utilized as a research tool for studying mite biology and also for monitoring treatment.[69]

26 | OTHER TESTS

Various other tests that are in progress and currently unavailable are the enzyme-linked immunosorbent assay (ELISA) to detect antibodies, or intradermal skin test.[70]

The most sensitive test is nested polymerase chain reaction (nested-PCR) of the gene for cytochrome C-oxidase subunit (cox1) of *Sarcoptes scabiei var. hominis* on skin scrapings.[71]

27 | DIAGNOSTIC CRITERIA

Until recently, there were no criteria established for the diagnosis of scabies. Not only is the condition misdiagnosed in clinics but also the interpretation in research trials and epidemiological studies are limited. Recently, the International Alliance for the Control of Scabies (IACS) established the consensus diagnostic criteria for scabies using the Delphi method (Table 3).[72] The criteria require validation through various epidemiological studies.

TABLE 3 Summary of 2018 IACS criteria for the diagnosis of scabies

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

Note: (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.

28 | TREATMENT

28.1 | General principles of treatment

The choice of treatment depends on the type of disease, patient's age, reported efficacy of various treatments and side effect profile.

- Patients should be advised to apply topical agents to the entire body especially the groin, interdigital areas of hands, under the nails, and behind the ears. Repeat application of hands should be done if they are washed before 8 h. In case it is not applied by the patient himself, person applying the topical treatment should wear protective gloves.
- Face and scalp involvement is common in children and elderly, hence these sites should be treated in this group.[73] In cases of failure with initial therapy, these sites should be treated.
- Patients should be advised to cut fingernails and clean the subungual debris as it is a source of relapse. Artificial nails may prevent effective local treatment.
- If there is secondary infection of lesions, topical or systemic antibiotics should be prescribed.
- Pruritus that persists after successful treatment (post scabietic pruritus) is due to hypersensitivity reaction to the mite and can be treated with emollients, antihistamines, and medium potency topical steroids.
- Washing is not necessary for decontamination of fomites. Clothing, bedding, and towels can be dried in a washing machine at 60° for

TABLE 4 Various topical scabicidal agents[78–84]

Drug	Concentration	FDA approved	Mechanism of action	Instructions	Level of evidence	Efficacy	Side effects	Comment
Permethrin	5% cream	Yes	Inhibits sodium channels → neurotoxic to mite	Applied overnight (8-14 h) washed off in morning	IA	90%	Excellent safety profile	Can be used in pregnancy, lactation not approved for use in infants <2 months
GBHC	1% lotion	Yes	CNS stimulant → paralysis and death of mite	Applied at bedtime for 8 h, washed in morning	IB	49-96%	Neurotoxic → irritability, seizures, syncope, vertigo, diarrhea	Carries black box warning, banned in 50 countries, carcinogenic, contraindicated in infants, pregnancy, lactation, crusted scabies and impaired barrier function, treatment failure is due to resistance
Benzyl benzoate	10%-25% lotion 25% adults 12.5% children 6.25% infants	No	Inhibits respiration in mites	Applied for 24 h, then washed off	IB	Cure rates lower than oral ivermectin, in vitro studies have shown better activity than permethrin	Irritant contact dermatitis	Can be used in infants, children and in pregnancy and lactation, avoid alcohol ingestion for atleast 48 h after treatment due to disulfiram like reaction
Crotamiton	10% cream/lotion	Yes	Unknown	Repeat applications for two consecutive days (for 24 h)	IV	Less effective than permethrin	Irritation and pruritus	Low toxicity profile Safe in infancy and pregnancy, repeat applications required, resistance can occur
Sulfur	2%-10% ointment/cream	No	Direct scabicidal activity, keratolytic	Applied consecutively for 3 days (each 24 h)	IB	Limited data	Burning, malodor	Can be used in infancy and pregnancy
Malathion	0.5% lotion	No	Inhibits acetylcholinesterase enzyme → paralysis and death of mite	Two applications 7 days apart	IIA	83%-100%	Safety profile is good, skin irritation, conjunctivitis	Good for treatment of hairy areas due to lotion formulation

10 min. Floors of bedrooms, bathrooms, chairs, and couches should be vacuumed. As the scabies mite can survive for 36 h outside the host at room temperature, contaminated fomites or those that are nonlaunderable should be isolated or not be used. This period can be longer (3 weeks) in cases of crusted scabies and treatment failure as mite can survive up to 19 days in cool, humid environment. Fomites that cannot be washed should be treated with an insecticidal.

- Simultaneous treatment of close contacts, even if asymptomatic, should be advised. Close contacts are defined as those who have extended physical contact, for example, sharing beds, etc.

Topical permethrin and oral ivermectin are considered effective treatments for scabies. Recent Cochrane review has shown that 5% permethrin and ivermectin are equally effective.[74,75]

Five percent permethrin is the gold standard of treatment and is FDA approved for patients older than 2 months. It has shown an efficacy of 90% in most of the studies.[76,77] It has an excellent safety profile and can be used in pregnancy and lactation. US-FDA has approved it for patients more than 2 months of age.

Other topical agents that can be used are listed in Table 4.

Ivermectin is an avermectin macrocyclic lactone that interrupts the gamma-aminobutyric acid induced neurotransmission of mites. It is given at a dose of 200 micrograms/kg as a single dose in patients. As it is not ovicidal the dose needs to be repeated after 7 to 14 days. Ivermectin should be administered with food as the bioavailability increases and greater amount of drug reaches the skin. Ivermectin is not recommended for children <5 years of age or weight <15 kg.[85] It is contraindicated in pregnancy and children.

Meta-analysis has shown that oral ivermectin may be less effective in treating scabies compared to permethrin cream at 1 to 2 weeks following treatment initiation.[85,86] However, its efficacy is similar to permethrin at later time points. It has been found to be more effective than crotamiton, malathion, and lindane. Lindane has been banned in many countries due to toxicity. Trials have found no difference in efficacy between topical ivermectin and permethrin.[87,88] But the former is associated with higher incidence of adverse effects compared to synthetic pyrethrins.

Persistence of symptoms in spite of treatment could be due to various factors enumerated in Table 5. It can take at least 6 weeks for signs and symptoms to resolve. Hence, possibility of treatment failure should be considered only after 6 weeks of completion of treatment. Post-scabies eczema should be treated with topical corticosteroids, in case of secondary impetiginization topical fusidic acid combined with a topical corticosteroid is helpful.

TABLE 5 Factors responsible for persistence of symptoms after treatment

Treatment related	Incorrect application, non-adherence
Incorrect diagnosis	Made on initial visit, skin biopsy may be helpful Delusions of parasitosis should also be considered
Reinfestation	Close contacts are not being treated
Resistance	Reported with topical lindane, crotamiton and oral ivermectin in crusted scabies

29 | TREATMENT IN PREGNANCY

Permethrin, benzyl benzoate (only 12 h application), and sulfur are safe in pregnancy. Ivermectin is contraindicated.

30 | TREATMENT OF CRUSTED SCABIES

Treatment of crusted scabies involves isolation of the patient as it can be a source of an epidemic outbreak. Keratolytic agents (5%-10% salicylic acid, 40% urea) must be used along with topical scabicidal as the crusts prevent the penetration of topical agents. While prescribing permethrin, patient should be advised to apply it under the nails, toe creases, navel, cleft of buttocks. Nails should be clipped. Two or more agents can be used and clearing of lesions is slower. Topical gamma benzene hexachloride (GBHC) should be avoided as the barrier function is compromised and toxicity can occur.

Recommended treatment according to the disease severity as follows.[40,89]

Grade 1: three doses of ivermectin over a period of 1 week.
Grade 2: five doses of ivermectin over 2 weeks.
Grade 3: seven doses over 4 weeks.

31 | TREATMENT OF NODULAR SCABIES

Scrotum and shaft of penis are common sites of involvement for nodular scabies. Various treatment modalities that have been tried with limited results include high potency topical steroids, intralesional triamcinolone injections, tacrolimus, and pimecrolimus. Topical crotamiton alone or in combination with hydrocortisone can be used with good response.[90] Liquid nitrogen cryotherapy has been tried with good results.[91]

32 | RESISTANCE IN SCABIES

Resistance to conventional therapy is increasing throughout the world. Studies have shown four different mechanisms of resistance to scabicide. These are voltage-gated sodium channels, ligand-gated chloride channels, glutathione S-transferase (GST), and ATP-binding cassette transporters.[92] The emergence of resistance has led to search for adjunct therapies that can be combined with conventional scabicides to counteract the resistance.

33 | NEWER AGENTS

Several newer agents are being developed for the treatment of scabies. Moxidectin, a milbemycin macrocyclic lactone, has a longer half-life of 20 days compared to 14 h for ivermectin. It is more lipophilic leading to greater tissue retention. Hence, it does not require a second dose. Also the chances of re-infestation are expected to be lower after treatment with moxidectin. [93,94] It is also less toxic than ivermectin.

Various insect growth regulators are being tried in various studies. Chitin is a major component of the exoskeleton of scabies mite. Fluazuron blocks synthesis of chitin hence prevents growth of new larvae within the eggs. However, it has no activity against adult mites.[95] Studies done in pigs with scabies have shown good results. Fluralaner is an isoxazoline that inhibits the nervous system of the scabies mite. A study has shown that a single dose of fluralaner is as effective as oral ivermectin.[96]

Afoxalaner is also an isooxazoline that has shown positive results in porcine model.[97] Tea tree oil, due to its antimicrobial property, has also shown efficacy. Other plant products tried are clove, turmeric, *Lippia*, camphor and neem oils.[95,97,98]

Scabies vaccine is being developed as it has been seen that second infections of scabies are milder than first.[99] For protective immunity to occur a vaccine should trigger the Th1 immune response. It may take years before an optimal scabies vaccine comes in the market.

34 | INSTITUTIONAL OUTBREAKS

Outbreak is defined as two or more cases of classical scabies or a single case of crusted scabies occurring in the same environment.[100] Management requires education of all staff members, recognition of index case, implementation of infection control measures, treatment of cases and contacts, and environmental disinfection.[101]

35 | CONCLUSION

Scabies is a prevalent parasitic skin disease that leads to substantial morbidity globally. Thorough scabies control strategies are needed, such as a community-based mass drug administration approach, in addition to more systematic approach to the monitoring of disease burden.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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