Portuguese Journal of Dermatology and Venereology

Permanyer

Check for updates

Port J Dermatol and Venereol.

REVIEW ARTICLE

The differential diagnoses and complications of scabies variants

Os diagnósticos diferenciais e as complicações das variantes da sarna: uma breve revisão

Jacob Al-Dabbagh[1a], Razan Younis[2], and Rasha Sliman[1]*

[1]Faculty of Medicine, Tishreen University, Latakia; [2]Faculty of Medicine, Tartous University, Tartous. Syria
[a]ORCID: 0000-0002-5596-9738

Abstract

Scabies is a common infection that affects people all over the world and has various presentations and impacts depending on the clinical situation. It is caused by the mite *Sarcoptes scabiei* var. hominis, an obligate ectoparasite that resides in the epidermis of the human skin. In this article, we present a review of the differential diagnoses and complications of scabies and scabies variants. We also present a summary of the etiology and the current diagnostic methods of scabies.

Keywords: Scabies. *Sarcoptes scabiei* var. hominis. Infestation. Parasitism. Differential diagnosis. Complications.

Resumo

A sarna é uma infeção comum que afecta pessoas em todo o mundo e tem várias apresentações e impactos, dependendo da situação clínica. É causada pelo ácaro Sarcoptes scabei var. hominis, um ectoparasita obrigatório que reside na epiderme da pele humana. Neste artigo, apresentamos uma revisão dos diagnósticos diferenciais e das complicações da escabiose e das variantes da escabiose. Apresentamos também um resumo da etiologia e dos métodos de diagnóstico actuais da sarna.

Palavras-chave: Sarna. *Sarcoptes scabiei* var. hominis. Infestação. Parasitismo. Diagnóstico diferencial. Complicações.

Introduction

Scabies is a neglected tropical cutaneous disease that affects people of all ages[1]. Children, adolescents, and elderly individuals are the most frequently affected age groups with no gender predilection[2,3]. However, children are more likely than other age groups to have scabies[2]. The pervasiveness of scabies in children is approximated to be 5-10%, with the highest predominance in children under 2 years old[4].

Poor hygiene, poverty, homelessness, overcrowding, lack of access to medical care, immunodeficiency, indiscriminate sexual activity, and demographic forces (including wars and migration) are significant risk factors for scabies infection[2,5,6]. Several family members may be infested at the same time, as scabies mites are transmitted by direct and indirect transmission[1].

The most prevalent regions of scabies infections are the tropical and subtropical areas, such as Latin America, the Pacific islands, South-east Asia, sub-Saharan Africa, and Northern and Central Australia[7-9].

Correspondence:
Jacob Al-Dabbagh
E-mail: jacobaldabbagh0@gmail.com

Received: 20-07-2023
Accepted: 19-09-2023
DOI: 10.24875/PJDV.23000062

Available online: 20-12-2023
Port J Dermatol and Venereol. 2023;81(4):259-266
www.portuguesejournalofdermatology.com

Etiology

Animal mites can be found in six genera: *Chorioptes*, *Cnemidocoptes*, *Notoedres*, *Psoroptes*, *Otodectes*, and *Sarcoptes*, with just the last, addressed in this article[6]. *Sarcoptes scabiei* causes scabies in humans, livestock, and wild animals[6].

The scabies mite (*S. scabiei* var. hominis) is an obligate human parasite that belongs to the *Sarcoptidae* family which is a member of the class *Arachnida* in the subclass *Acari* of the order Astigmata[2]. It is white-brown in color and lives in linear burrows that are dug into the stratum corneum and can be seen by the naked eye as a speck, and skilled dermatologists may be able to notice it using dermoscopy[2,3].

The female mite has a size of approximately 0.4 × 0.3 mm while the male is about two-thirds of the female size[10,11]. The adult mite has four pairs of legs contrasted with the larva which has three pairs of legs[10]. At the beginning of the scabies mite life cycle, which takes about 14-21 days, the fertilized female burrows into the stratum corneum at a rate of 0.5-5 mm/day and lay 2-4 eggs each day that hatch after 48-72 h into larvae and create new burrows[2,5,10]. The larvae take approximately 10-14 days to reach adulthood[10,11]. The life range of a female mite is 4-6 weeks, during which it lays about 40-50 eggs[10]. Adult male mites may enter the burrows in search of food and unfertilized female mites for mating and die shortly after mating[2,3].

The average burden in classical scabies is around 10-20 mites, and between 50 and 250 mites can be found in infants and the elderly[3]. However, thousands to millions of mites are carried by crusted scabies patients[3].

The scabies mites are resistant to soap and alcohol and they either reinfest the host at a different location or infect another human host[2]. In normal room conditions (21°C and 40-80% relative humidity), scabies mites can survive outside the human body for 24-36 h, but this period can be increased with lower temperatures and higher humidity[2]; during this time, they remain able to invade[5]. However, as the amount of time spent away from the host increases, the ability to invade a new host decreases[10].

Scabies mites cannot jump or fly and they are less contagious the longer they are separated from their host[2]. The minimum necessary time for skin-to-skin transmission is 5 min[10]. Scabies symptoms typically begin 3-6 weeks after the primary infestation and 1-3 days after a reinfestation due to an immediate or delayed (type IV) hypersensitivity reaction to the mites' products (feces, eggs, and dead parasites)[12].

Variants of clinical scabies

Classic scabies

Classic scabies is typically characterized as an intensive cutaneous rash with generalized pruritus that worsens at night[6,13]. The classic signs include burrows and papules[6].

The burrow is a short, 1-10 mm long, serpiginous gray line found on the hands and feet, especially the interdigital spaces, and wrists[13]. Burrows are rarely visualized to the naked eye, and lesions are often misdiagnosed as excoriated or impetiginized skin[13]. The papule is usually small and erythematous, often excoriated or covered with a tiny blood clot[6].

The erythematous papular rash is generally symmetrical, with a tendency to affect the anterior axillary folds, periumbilical skin, elbows, volar surface of wrists, interdigital spaces, beltline, thighs, buttocks, ankles, areola area in women, penis, and scrotum in men[13]. The scalp, face, and neck areas are usually spared in adults, but infants and immunocompromised patients may be affected[13].

Crusted scabies

Crusted scabies, also known as Norwegian scabies, manifests as hyperkeratotic plaques that contain high numbers of mites[14]. It often occurs in patients who are immunosuppressed, such as those infected with HIV or after solid organ transplantation[14].

Clinically, lesions present as thick, gray, scaly, hyperkeratotic, and crusted plaques that are diffusely distributed and typically cover the hands, elbows, feet, knees, nail beds, trunk, scalp, and in some cases the entire body[13,14]. In addition, crusted scabies has little to no pruritus[6]. It is extremely contagious, and if no precautions are taken, outbreaks can occur among family members and patients in hospital wards[15].

Nodular scabies

Nodular scabies is an uncommon variant of scabies that is characterized by persistent itchy nodules that can persist even after treatment of the primary infestation[16]. These nodules are violaceous, pruritic, and between 2 and 20 mm in size[13,14]. The appearance of nodular scabies is the result of a hypersensitivity response to scabies mites and other products of the infection[16]. Nodular scabies occurs most commonly on the thighs, axillae, glans, and scrotum[13].

Bullous scabies

It is a rare clinical variant that usually presents in the elderly[13]. It manifests as tense vesicles/bullae, which occur most commonly on the arms, legs, and trunk; they may also be generalized[14]. Less frequently, they appear on the genitals, buttocks, inguinal folds, thighs, neck, and feet[14]. Bullous scabies often occurs in patients with pruritic dermatosis who have been previously treated with systemic and/or topical corticosteroids[14].

Nail scabies

Nail scabies is an abnormal clinical presentation[17]. It is often misdiagnosed and can be the initial presentation of scabies[13]. It affects certain populations such as infants, immunocompromised individuals, and the elderly[18].

It may affect multiple fingernails and/or toenails and manifests as nail plate dystrophy, which remains even after successful treatment[13].

Scabies incognito

This variant of infestation appears after application of the topical corticosteroid[13]. The corticosteroid alters the distinctive symptoms and lesions of the scabies infestation[14].

Diagnostic methods of scabies

Under ordinary conditions, the diagnosis of scabies can be easily made on the basis of the clinical distribution, the presence of skin lesions, and the patient's medical history[19]. However, in certain circumstances, such as patients receiving steroid medications for a prolonged time or those with weakened immune systems, scabies may manifest with an atypical clinical pattern[19]. In addition, it can be difficult to diagnose scabies in infants or the elderly because their clinical features may differ from those of normal adults[19]. Therefore, an accurate diagnosis of scabies infection is essential for patient treatment[19].

The diagnostic procedure of scabies can be divided into two phases: the presumptive diagnosis (history and physical examination) and the definitive diagnosis (investigations)[20].

History and examination

Medical history, physical examination, and history of concomitant infections in family members and close contacts play an important role in the diagnosis of scabies[21].

Based on the history of nocturnal itching and the typical distribution of skin lesions, a preliminary diagnosis can be made[21].

Dermoscopy

Dermoscopy is an accurate method of diagnosing scabies when performed by a trained physician[20]. It is a painless procedure that can lead to better patient compliance[19]. Finding small, dark, and triangular structures at one of the ends of the burrows indicates the existence of the mites[20].

However, darker skin phototypes, hairy areas, and complications caused by scratching (such as excoriations, crusts, bleeding, or microscopic dirt particles) make it difficult to identify the scabies mite with dermoscopy[1]. In crusted scabies, dermoscopy shows multiple burrows and a hyperkeratotic appearance[1]. Anyhow, the main limitation of dermoscopy is low specificity because of lower magnification[10]. Other limitations include low sensitivity in mild disease and operator dependence[10].

Videodermoscopy

Videodermoscopy is a quick, non-invasive diagnostic method with 100% specificity and greater sensitivity than skin scraping[1,10]. Since it does not make the patient uncomfortable, it can be used in patients who are non-cooperative[10]. In addition, it takes less time and reduces the chance of cross-infections[10].

Reflectance confocal microscopy (RCM)

Through RCM, the burrows can be seen as linear segments in the middle of the surrounding epidermis that appear as a "honeycomb" pattern[1,10]. In addition, eggs, feces, larvae, and mites can be seen[10]. RCM is non-invasive and can be used to study the biological behavior of the mite, as it also shows the movement and peristalsis of the mite[10].

Lack of availability and high equipment costs are the limitations of RCM[10]. Furthermore, the high time requirement is another limitation[10].

Optical coherence tomography (OCT)

OCT is comparable to ultrasonography but has a higher resolution and allows visualization of the most

important parts of the skin[1,10]. Mites, burrows, and eggs can be observed and examined[10]. With the ability to detect the mite both vertically and horizontally, OCT can quickly and precisely diagnose scabies *in vivo*[1].

Burrow ink test (BIT)

BIT is helpful for detecting the scabies mite's burrows[1]. A positive BIT results when the ink follows the mite burrow and forms a distinctive, dark, zigzagged line that is easily visible to the naked eye[22]. If one does not have access to a microscope, dermoscopy, or skin biopsy equipment, this test can be helpful[22]. However, this method provides only a partial diagnosis and is unable to distinguish between old and new lesions[1].

Adhesive tape test (ATT)

In outbreaks in nursing homes and other large accommodations, the ATT is a quick and effective procedure[23]. This technique is not recommended for people with fragile skin[23].

Microscopic examination

Microscopic examination of the mites, eggs, or feces from scales obtained by skin scraping or from the skin biopsy confirms the diagnosis of scabies[1].

Skin scrapings at the end of burrows can be used to detect mites, eggs, or feces microscopically[23]. Although the microscopic examination is inexpensive, a negative result does not rule out infestation, as conventional infection contains only 10-15 mites[24].

Skin biopsy is considered one of the most accurate methods for diagnosing scabies[1]. However, skin biopsy is used only to confirm atypical presentations and is not considered part of the standard examination for the diagnosis of scabies[1].

In addition to the previous diagnostic methods, other methods such as modern molecular techniques and serology can help in diagnosing scabies, although standardized laboratory tests for the detection of scabies are not currently available[1].

The following diagram summarizes the currently available diagnostic procedures for scabies (Fig. 1)[1].

Differential diagnoses of scabies variants

The differential diagnosis of scabies is broad and includes various skin disorders[2]. Physicians may use objective evidence to confirm the diagnosis of scabies or subjective observations to meet the criteria for a clinical diagnosis or a suspected diagnosis of scabies when a patient presents with classic symptoms such as itching that worsens at night and lesions suggestive of mite infestation, such as burrows located on the flexor areas[25].

When patients present with atypical symptoms of scabies, physicians may not suspect that it is a mite-associated dermatosis[25]. Therefore, it can be difficult to make a definitive diagnosis of scabies surrepticius (non-classical scabies), especially when the clinical history and morphologic features of the lesions suggest another disease or the lesions are infected with a bacterial or viral infection[25].

In patients with chronic or progressive skin problems, the possibility of scabies should be considered and further diagnostic testing should be performed, especially if the skin is itchy and unresponsive to therapeutic measures[25].

Clinicians should consider the differential diagnoses that may mimic classic scabies, including insect bites, infections (such as tinea corporis, body lice, impetigo, folliculitis, and viral exanthems), drug eruption, and inflammatory or immune-mediated dermatologic conditions (such as papular urticaria and pityriasis rosea)[2,5,10,26].

Insect bites such as fleas, mosquitoes, midges, and bedbugs are commonly found on exposed skin as red, itchy, and clustered papules[26,27]. Papular urticaria manifests as pruritic red and edematous grouped papules/papulovesicles representing hypersensitivity to insect bites[27,28]. The lesions generally prefer the extensor sides of the extremities, although the trunk is also often affected[28].

Folliculitis is identified by erythematous papules and pustules that are asymptomatic, pruritic, or mildly painful, and located around the hair shaft[29]. Although any area that has hair can be affected, folliculitis often affects only the face, scalp, thighs, armpits, and groin[29].

Dermatitis such as eczema, contact dermatitis, and atopic dermatitis is considered also as differential diagnoses of classic scabies[26,27]. Atopic dermatitis manifests as dry, scaly, erythematous, and itchy skin plaques, which commonly affect the face, neck, elbows, and knee extensors of infants, later including the flexures[27].

Gianotti-Crosti syndrome (GCS) is sometimes mistaken for scabies because of the high percentage of acral papules[28]. The extensor sides of the limbs, buttocks, and cheeks all have a symmetrical distribution of papules[28]. In contrast to scabies, GCS is almost never as itchy as scabies; mite burrows and

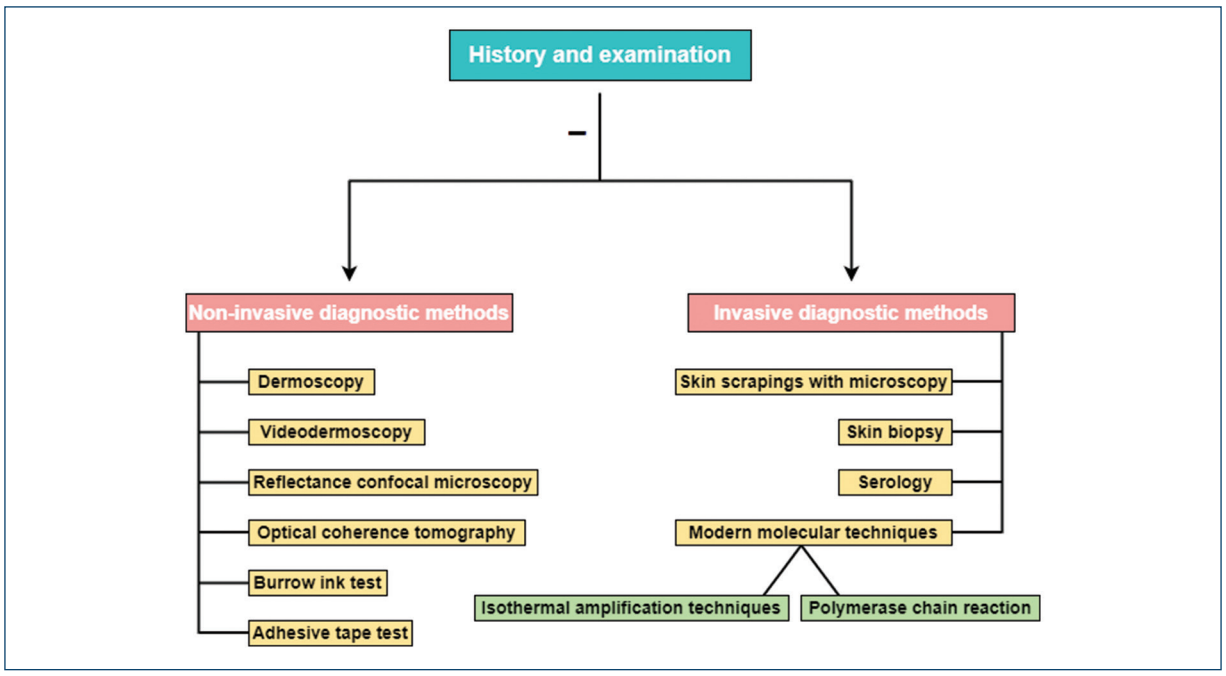

Figure 1. The currently available diagnostic methods for scabies.

excoriations are a regular occurrence in scabies but are the absolute exception in GCS[28].

Other diseases characterized by extensive scaling, such as psoriasis, Darier's disease, drug eruption, lichen planus, palmoplantar keratoderma, and seborrheic dermatitis are potential differential diagnoses for crusted scabies[14,15,26].

Scabies masquerading as an adverse drug reaction has rarely been described[25]. In any patient with a suspected drug eruption who develops pruritic dermatosis after discontinuation of the drug and presents with clinical lesions resembling crusted scabies, the physicians should be aware of the possibility of drug eruption-like scabies (scabies surrepticius)[25].

In a patient with persistent nodular rash, nodular scabies must be investigated as part of the differential diagnosis[16]. Nodular scabies might imitate a solitary cutaneous mastocytoma as it can give a positive Darier sign[2]. Nodular scabies may be mistakenly diagnosed as Langerhans cell histiocytosis, non-Langerhans cell histiocytosis, insect bites, lymphoma, or urticaria pigmentosa[14,30].

In case of blistering is present (bullous scabies), other skin disorders should take into account as differential diagnoses include bullous pemphigoid, bullous impetigo, acquired epidermolysis bullosa, arthropod bite reaction, pemphigus, and dermatitis herpetiformis[2,5].

The lesions in pemphigoid scabies resemble bullous pemphigoid clinically, histologically, and on immunofluorescent findings[13,14]. Subepidermal blisters and eosinophilic dermal inflammation can be seen under a microscope with a potential presence of mites in bullous scabies[14].

The diagnosis of nail scabies is frequently mistaken with onychomycosis, nail psoriasis, traumatic nails, and nail dystrophy[17]. However, nail scabies has been documented in the literature in association with periungual scaling and crusting, distal onycholysis, longitudinal nail splitting, subungual hyperkeratotic deposits, and nail plate deformity/hypertrophy[17].

Infants and young children under 2 years of age are particularly susceptible to scabies[30]. Infants may have diagnostic concerns due to low suspicion of scabies, eczematous changes, and inappropriate therapy, particularly topical steroids[30]. It is important to distinguish infantile scabies from other entities that present similarly in this age group such as infantile acropustulosis, papular urticaria, and atopic dermatitis[10]. Many studies have highlighted the possibility that scabies in infants may resemble other skin diseases, such as bullous pemphigoid, Langerhans cell histiocytosis, adverse drug reactions, lymphomatoid papulosis, lupus erythematosus, psoriasis, or an allergic reaction to an insect bite[30]. In addition, infantile seborrheic dermatitis, which manifests as scaly and greasy plaques on the face and scalp, has a similar appearance to scabies[27].

The differential diagnoses of scabies based on each variant are summarized in the following table (Table 1)[2,5,10,14,15,17,26,31].

Table 1. The differential diagnoses of scabies variant

Scabies variants	Differential diagnoses
Classic scabies	– Atopic dermatitis – Contact dermatitis – Seborrheic dermatitis – Folliculitis – Papular urticaria – Tinea corporis – Impetigo – Body lice – Insect bites – Drug eruption – Varicella – Pityriasis rosea – Infantile acropustulosis – Gianotti-crosti syndrome – Psoriasis – Cutaneous mastocytosis – Langerhans cell histiocytosis
Crusted scabies	– Seborrheic dermatitis – Psoriasis – Palmoplantar keratoderma – Darier's disease – Drug eruption – Lichen planus
Bullous scabies	– Bullous pemphigoid – Bullous impetigo – Acquired epidermolysis bullosa – Arthropod bite reaction – Pemphigus – Dermatitis herpetiformis
Nodular scabies	– Solitary cutaneous mastocytoma – Langerhans cell histiocytosis – Non-langerhans cell histiocytosis – Insect bites – Lymphoma – Urticaria pigmentosa
Nail scabies	– Psoriasis – Onychomycosis – Traumatic nails

Complications of scabies

The secondary medical, psychosocial, and economic factors that are related to the burden of disease must be taken into account when estimating the true global burden of scabies[32]. In the context of the impact of the disease, morbidity associated with scabies is commonly underestimated[22].

Severe rubbing or scratching prompts changes such as bleeding, crusting, or excoriations and can lead to secondary bacterial skin infections[10,14,15].

Secondary infections and impetiginization, most commonly caused by Group A *Streptococcus* (GAS) and *Staphylococcus aureus (S. aureus)*, are the most common complications associated with scabies[10,33]. Staphylococcal and streptococcal growth is promoted by the ability of the scabies mite to disrupt the human complement system by blocking all complement initiation pathways, resulting in decreased neutrophil activity[34]. These bacteria can also be isolated from skin tunnels, and feces indicate that mites may be contributing to the bacteria's distribution[35].

S. aureus can also lead to superficial acute impetiginization, abscesses, ecthyma, cellulitis, paronychia, staphyloderma, erysipelas, and furunculosis[8,34]. However, it can also progress to endocarditis, osteomyelitis, and bacterial sepsis, which can be life-threatening[8]. Whereas, local skin and soft-tissue infections, such as superficial pyoderma, skin abscesses, and cellulitis, as well as more severe necrotizing fasciitis, can be caused by GAS[8].

Impetigo is a common complication of scabies itching, especially in children and patients who live in overcrowded conditions[36]. Impetigo that is caused by *Streptococcus pyogens* can lead to toxin-mediated diseases such as scarlet fever, rheumatic fever, streptococcal toxic shock syndrome, and post-streptococcal glomerulonephritis[10]. *S. pyogens* infection of the skin may also cause reactive arthritis-synovitis, necrotizing fasciitis, and pediatric autoimmune neuropsychiatric disorder[2].

Infectious complications seem to be more severe in crusted scabies[36]. This may be caused by the patient's comorbidities, such as immunosuppression, as well as deeper excoriations that result in invasive infections and severe sepsis, which lead to a high risk of mortality[36,37]. Furthermore, crusted scabies cases may develop generalized lymphadenopathy and eosinophilia[3,20].

Crusted scabies can also cause malodor as a secondary bacterial infections[2]. Colonization of the burrow by *S. aureus* can lead to erythroderma, septicemia, and superinfection with *S. pyogenes*, which causes glomerulonephritis and rheumatic fever[38].

In addition, scabies herpeticum may occur when crusted scabies is superinfected in association with herpes simplex[2].

Systemic complications such as bacteremia, acute post-streptococcal glomerulonephritis, streptococcal and staphylococcal sepsis, acute rheumatic fever, and rheumatic heart disease can be associated with a high risk of mortality[4,33]. Patients with a previous history of scabies are more likely, according to some studies, to develop bullous pemphigoid and chronic kidney disease[39].

Table 2. Scabies complications

Secondary to skin infection		Secondary to itching/ scratching	Psychosocial	Other
Cutaneous	Systemic			
– Superficial acute impetiginization – Superficial pyoderma – Abscesses – Ecthyma – Cellulitis – Paronychia – Furunculosis – Erysipelas – Staphyloderma – Scabies herpeticum – Bullous impetigo – Malodor	– Endocarditis – Osteomyelitis – Bacterial sepsis – Pernicious anemia – Acute post-streptococcal glomerulonephritis – Acute rheumatic fever – Scarlet fever – Rheumatic heart disease – Streptococcal toxic shock syndrome – Chronic kidney disease – Reactive arthritis-synovitis – Necrotizing fasciitis – Pediatric autoimmune neuropsychiatric disorder – Lymphadenopathy – Eosinophilia	– Bleeding – Crusting – Excoriations – Sleep disturbances – Tiredness – Reduced productivity – Reduced ability to concentrate – Increased risk and severity of skin conditions (such as psoriasis and atopic dermatitis)	– Low work attendance – Social stigmatization – Feelings of shame – Loss of performance at school – School absenteeism – Fatigue – Lack of concentration or memory in infants – Intellectual disability – Bipolar disorder	– Significant financial burden – Generalized urticaria

Renal damage without symptoms can occur in scabies patients[40]. The morbidity and mortality associated with chronic renal disease are important late complications of scabies[32].

Scabies patients suffer from a deteriorated quality of life that is linked directly to their itchiness[34]. Up to 90% of scabies patients have sleep disturbances caused by itching[36]. Sleep disturbances may be associated with tiredness, reduced productivity, and reduced ability to concentrate[2]. After eradicating the scabies mite, post-scabetic pruritus can be stubborn and debilitating, and it may last for a long period[2].

Scabies can cause psychosocial complications such as low work attendance, social stigmatization, and feelings of shame[36]. In addition, it can cause loss of performance at school, school absenteeism, fatigue, and lack of concentration or memory in infants[36].

Scabies can cause a significant financial burden, particularly on patients with severe systemic complications[2]. Rarely, scabies patients may develop generalized urticaria[2].

In rare cases, scabies can have unusual complications such as cutaneous vasculitis, vascular purpura, and glomerulonephritis, which may occasionally foreshadow the original disease[41].

Scabies patients also have a higher risk of pernicious anemia, intellectual disability, and bipolar disorder, according to some studies[2].

The complications of scabies are demonstrated in the following table (Table 2)[2-4,8,10,33,34,36,37,39,42].

Conclusion

The physicians must take into account the differential diagnoses of scabies variants to make the proper diagnosis. The physician must also be aware of the complications of scabies and perform an appropriate treatment to achieve a complete recovery from scabies and its complications.

Authors' contributions:

Jacob Al-Dabbagh: wrote the original draft, performed the literature review, and was the supervisor. Razan Younis and Rasha Sliman: performed the literature review and edited the manuscript.

Funding

None.

Conflicts of interest

None.

Ethical disclosures

Protection of human and animal subjects. The authors declare that no experiments were performed on humans or animals for this study.

Confidentiality of data. The authors declare that no patient data appear in this article.

Right to privacy and informed consent. The authors declare that no patient data appear in this article.

Use of artificial intelligence for generating text. The authors declare that they have not used any type of generative artificial intelligence for the writing of this manuscript, nor for the creation of images, graphics, tables, or their corresponding captions.

References

1. Al-Dabbagh J, Younis R, Ismail N. The currently available diagnostic tools and treatments of scabies and scabies variants: an updated narrative review. Medicine (Baltimore). 2023;102:e33805.
2. Leung AK, Lam JM, Leong KF. Scabies: a neglected global disease. Curr Pediatr Rev. 2020;16:33-42.
3. Richards RN. Scabies: diagnostic and therapeutic update. J Cutan Med Surg. 2021;25:95-101.
4. Lobo Y, Wheller L. A narrative review of the roles of topical permethrin and oral ivermectin in the management of infantile scabies. Australas J Dermatol. 2021;62:267-77.
5. Chandler DJ, Fuller LC. A review of scabies: an infestation more than skin deep. Dermatology. 2019;235:79-90.
6. Currier RW, Walton SF, Currie BJ. Scabies in animals and humans: history, evolutionary perspectives, and modern clinical management. Ann N Y Acad Sci. 2011;1230:E50-60.
7. Siddig EE, Hay R. Laboratory-based diagnosis of scabies: a review of the current status. Trans R Soc Trop Med Hyg. 2022;116:4-9.
8. Thompson R, Westbury S, Slape D. Paediatrics: how to manage scabies. Drugs Context. 2021;10:2020\-12-3.
9. Micali G, Lacarrubba F, Verzì AE, Chosidow O, Schwartz RA. Scabies: advances in noninvasive diagnosis. PLoS Negl Trop Dis. 2016;10:e0004691.
10. Arora P, Rudnicka L, Sar-Pomian M, Wollina U, Jafferany M, Lotti T, et al. Scabies: a comprehensive review and current perspectives. Dermatol Ther. 2020;33:e13746.
11. Fischer K, Holt D, Currie B, Kemp D. Scabies: important clinical consequences explained by new molecular studies. Adv Parasitol. 2012;79:339-73.
12. Salavastru CM, Chosidow O, Boffa MJ, Janier M, Tiplica GS. European guideline for the management of scabies. J Eur Acad Dermatol Venereol. 2017;31:1248-53.
13. Hicks MI, Elston DM. Scabies. Dermatol Ther. 2009;22:279-92.
14. Cohen PR. Scabies masquerading as bullous pemphigoid: scabies surrepticius. Clin Cosmet Investig Dermatol. 2017;10:317-24.
15. Goyal NN, Wong GA. Psoriasis or crusted scabies. Clin Exp Dermatol. 2008;33:211-2.
16. Yanes DA, Faith EF. Nodular scabies: a persistent nodular eruption. Dermatol Online J. 2018;24.
17. Tempark T, Lekwuttikarn R, Chatproedprai S, Wananukul S. Nail scabies: an unusual presentation often overlooked and mistreated. J Trop Pediatr. 2017;63:155-9.
18. Abdullah L, Abbas O. Common nail changes and disorders in older people: diagnosis and management. Can Fam Physician. 2011;57:173-81.
19. Park JH, Kim CW, Kim SS. The diagnostic accuracy of dermoscopy for scabies. Ann Dermatol. 2012;24:194-9.
20. Alsyali Z, Alaithan A, Almubarak K, Alibrahim H, Almansour A, Albalawi T, et al. A comprehensive review study on scabies and its associated impact on psycho-social health of an individual. Int J Contemp Res Rev. 2019;10:20700-8.
21. Shimose L, Munoz-Price LS. Diagnosis, prevention, and treatment of scabies. Curr Infect Dis Rep. 2013;15:426-31.
22. Golant AK, Levitt JO. Scabies: a review of diagnosis and management based on mite biology. Pediatr Rev. 2012;33:e1-12.
23. Sunderkötter C, Feldmeier H, Fölster-Holst R, Geisel B, Klinke-Rehbein S, Nast A, et al. S1 guidelines on the diagnosis and treatment of scabies - short version. J Dtsch Dermatol Ges. 2016;14:1155-67.
24. Agyei M, Ofori A, Tannor EK, Annan JJ, Norman BR. A forgotten parasitic infestation in an immunocompromised patient-a case report of crusted scabies. Pan Afr Med J. 2020;36:238.
25. Cohen PR. Drug eruption-like scabies surrepticius: an uncommonly described variant of scabies appearing in a non-classic clinical presentation. Dermatol Online J. 2022;28.
26. Hardy M, Engelman D, Steer A. Scabies: a clinical update. Aust Fam Physician. 2017;46:264-8.
27. Chan OB, O'Brien T, Yazdabadi A, Su JC. The diagnostic challenge of infantile scabies. Hong Kong J Dermatol Venereol. 2020;28:63-6.
28. Brandt O, Abeck D, Gianotti R, Burgdorf W. Gianotti-crosti syndrome. J Am Acad Dermatol. 2006;54:136-45.
29. Durdu M, Errichetti E, Eskiocak AH, Ilkit M. High accuracy of recognition of common forms of folliculitis by dermoscopy: an observational study. J Am Acad Dermatol. 2019;81:463-71.
30. Cölgeçen-Özel E, Ertaş R, Utaş S, Kontaş O. Scabies mimicking mastocytosis in two infants. Turk J Pediatr. 2013;55:533-5.
31. Hill TA, Cohen B. Scabies in babies. Pediatr Dermatol. 2017;34:690-4.
32. Cox V, Fuller LC, Engelman D, Steer A, Hay RJ. Estimating the global burden of scabies: what else do we need? Br J Dermatol. 2021;184:237-42.
33. Kim HS, Hashimoto T, Fischer K, Bernigaud C, Chosidow O, Yosipovitch G. Scabies itch: an update on neuroimmune interactions and novel targets. J Eur Acad Dermatol Venereol. 2021;35:1765-76.
34. Ständer S, Ständer S. Itch in scabies-what do we know? Front Med (Lausanne). 2021;8:628392.
35. Putra IB, Jusuf NK. Scabies with secondary infection resembling kerion-type tinea capitis. Int J Gen Med. 2021;14:163-7.
36. Jannic A, Bernigaud C, Brenaut E, Chosidow O. Scabies itch. Dermatol Clin. 2018;36:301-8.
37. Thomas C, Coates SJ, Engelman D, Chosidow O, Chang AY. Ectoparasites: scabies. J Am Acad Dermatol. 2020;82:533-48.
38. Niode NJ, Adji A, Gazpers S, Kandou RT, Pandaleke H, Trisnowati DM, et al. Crusted scabies, a neglected tropical disease: case series and literature review. Infect Dis Rep. 2022;14:479-91.
39. Khalil S, Abbas O, Kibbi AG, Kurban M. Scabies in the age of increasing drug resistance. PLoS Negl Trop Dis. 2017;11:e0005920.
40. Hay RJ, Steer AC, Engelman D, Walton S. Scabies in the developing world--its prevalence, complications, and management. Clin Microbiol Infect. 2012;18:313-23.
41. Gunarić A, Jurišić K, Šimić D, Penavić JZ, Jozić S, Goluža I. Scabies mimicing child abuse - a case report. Psychiatr Danub. 2017;29 Suppl 2:145-7.
42. Inam W, Walton S, Khan S, Mahmood W. Molecular drug targets for scabies: a medicinal chemistry perspective. Future Med Chem. 2020;12:2225-38.