



SCABIES: DERMATOLOGICAL DISORDER

Muddasir Ahmad Rather*, Shubam Sehgal, Tanvay Jaithliya, Wasim Fayaz, Tawseef Hassan Shiekh and Atul Tiwari¹

*Faculty of Pharmaceutical sciences, Mewar University, 312901, Chittorgarh, India.

¹Assistant Professor, Faculty of Pharmaceutical Sciences, Mewar University, Chittorgarh, 312901, India.

***Corresponding Author: Muddasir Ahmad Rather**

Faculty of Pharmaceutical sciences, Mewar University, 312901, Chittorgarh, India.

Article Received on 19/03/2018

Article Revised on 09/04/2018

Article Accepted on 29/04/2018

ABSTRACT

Scabies is a skin infestation caused by a mite known as the *Sarcoptes scabiei*. The characteristic symptoms of a scabies infection include intense itching and superficial burrows. As a symptom, itching is less common in the elderly. The characteristic symptoms of a scabies infection include intense itching and superficial burrows. It is caused by the mite *Sarcoptes scabiei* variety hominis and transmitted by person-to-person contact.

KEYWORDS: *Sarcoptes scabiei*.

INTRODUCTION

Scabies is a skin infestation caused by a mite known as the *Sarcoptes scabiei*.^[1] It is caused by the mite *Sarcoptes scabiei* variety hominis and transmitted by person-to-person contact. The name *Sarcoptes scabiei* is derived from the Greek word "sarx" (flesh) and "koptein" (to smite or to cut) and the Latin word "scabere" (to scratch). Scabies was first described more than 2500 years ago.^[2] Scabies was referred to in the Old Testament and by Aristotle,^[3] but it was not until 1687 that the causative organism was identified by Bonomo and Cestoni using light microscopy.^[4-7] Although the infectious agent is ubiquitous, it is endemic in impoverished communities, such as underprivileged suburban villages, where up to 9% of the population and 19% of those attending a primary healthcare centre are infested.^[8] By contrast, in industrialised countries, outbreaks occur in hospitals, kindergarten, and other institutions.^[9] Despite common belief, scabies is only infrequently acquired from contaminated fomites (eg, clothing, towels, and bedding).^[10]

SIGNS AND SYMPTOMS

The characteristic symptoms of a scabies infection include intense itching and superficial burrows.^[11] The burrow tracks are often linear, to the point that a neat "line" of four or more closely placed and equally developed mosquito-like "bites" is almost diagnostic of the disease. Because the host develops the symptoms as a reaction to the mites' presence over time, typically a delay of four to six weeks occurs between the onset of infestation and the onset of itching. Similarly, symptoms often persist for one to several weeks after successful eradication of the mites. As noted, those re-exposed to

scabies after successful treatment may exhibit symptoms of the new infestation in a much shorter period—as little as one to four days.^[12]

Itching

In the classic scenario, the itch is made worse by warmth, and is usually experienced as being worse at night, possibly because distractions are fewer.^[11] As a symptom, it is less common in the elderly.^[11]

Rash

The superficial burrows of scabies usually occur in the area of the finger webs, feet, ventral wrists, elbows, back, buttocks, and external genitals.^[11] Except in infants and the immunosuppressed, infection generally does not occur in the skin of the face or scalp. The burrows are created by excavation of the adult mite in the epidermis.^[11] In most people, the trails of the burrowing mites are linear or S-shaped tracks in the skin often accompanied by rows of small, pimple-like mosquito or insect bites. These signs are often found in crevices of the body, such as on the webs of fingers and toes, around the genital area, in stomach folds of the skin, and under the breasts of women.^[13] Symptoms typically appear two to six weeks after infestation for individuals never before exposed to scabies. For those having been previously exposed, the symptoms can appear within several days after infestation. However, symptoms may appear after several months or years.^[14] Acropustulosis, or blisters and pustules on the palms and soles of the feet, are characteristic symptoms of scabies in infants.^[13]

CAUSE

Scabies mites

S. scabiei is a member of the family Sarcoptidae within the class of Arachnida. They are easily distinguished from other arachnids by the position of a distinct gnathosoma (head with mouth parts) and the lack of a division between the abdomen and cephalothorax. Adult females are around 0.4 mm long and 0.3 mm wide with males being smaller. Adult nymphs have eight legs and larvae have six. Although the mites cannot fly or jump, they may crawl as fast as 2.5 cm per min on warm skin.^[15,16] After mating, the male mite dies, and the female begins to lay up to three eggs per day in skin burrows within the stratum granulosum. A single mite can produce up to 40 ova. Larvae hatch at 2–4 days and also dig burrows (so-called “moulting pouches”). 3 to 4 days later, the larva moults into a protonymph, which after 2–5 days moults into a tritonymph, from which an adult male or female emerges after another 5–6 days. In total, mature adults develop within 10–14 days. Skin entry occurs in less than 30 min and can take place during every stage by secreting enzymes that dissolve the skin, which is then ingested by the mite as nutrient. The average infested adult human being has an estimated ten to 15 adult female mites on the body surface at a given time.^[16] *S. scabiei* is responsible for epizootic disease in livestock animals and wild populations of dogs, cats, ungulates, boars, wombats, koalas, great apes, and bovids.^[17] Current estimates indicate that between 50% and 95% of pig herds worldwide are infested with *S. scabiei*.^[18,19] Animal scabies can be transmitted to human beings, which can also result in pruritic papules (eg, pig handler’s itch, cavalryman’s itch).⁸ In general, animal scabies is self-limiting in human beings, since the mites cannot complete their life cycle.^[20,21] Research on scabies has historically been limited because of the difficulty in obtaining sufficient numbers of the causative organism. Recent molecular approaches have enabled substantial advances in the study of population genetics and transmission dynamics of *S. scabiei*.^[18] Promising data from *S. scabiei* cDNA libraries have enabled the identification of several unique genes.^[22] Of particular interest was the identification of *S. scabiei* homologues of the known house dust mite allergens glutathione-S transferase, paramyosin, and cathepsin-L.^[22] By comparison with trypsin-like serine proteases (members of group-3 allergens), which are excreted in faecal pellets,^[23] one homologue has been termed Sar s 3 in analogy to Der p 3. Their most remarkable feature is that they usually do not contain an intact catalytic site, thus rendering these proteases non-functional. These inactivated proteases were designated scabies mite inactivated protease paralogues (SMIPPs). SMIPPs might, therefore, act as antagonists of active proteases by competing for peptide substrates. House dust mite code-3 and code-9 allergens that are also serine proteases are known to bind and activate protease-activated receptor (PAR)-2 on the surface of human pulmonary epithelial cells, inducing cytokine release.^[24] Because PAR-2 is also present in the surface of keratinocytes, SMIPPs

might be capable of binding to, but not activating, PAR-2, thus protecting the scabies mite from the inflammatory response.

Transmission

Scabies is contagious and can be contracted through prolonged physical contact with an infected person. This includes sexual intercourse, although a majority of cases are acquired through other forms of skin-to-skin contact. Less commonly, scabies infestation can happen through the sharing of clothes, towels, and bedding, but this is not a major mode of transmission; individual mites can only survive for two to three days, at most, away from human skin at room temperature. As with lice, a latex condom is ineffective against scabies transmission during intercourse, because mites typically migrate from one individual to the next at sites other than the sex organs. Healthcare workers are at risk of contracting scabies from patients, because they may be in extended contact with them.

Diagnosis

Scabies may be diagnosed clinically in geographical areas where it is common when diffuse itching presents along with either lesions in two typical spots or itchiness is present in another household member. The classical sign of scabies is the burrow made by a mite within the skin. To detect the burrow, the suspected area is rubbed with ink from a fountain pen or a topical tetracycline solution, which glows under a special light. The skin is then wiped with an alcohol pad. If the person is infected with scabies, the characteristic zigzag or S pattern of the burrow will appear across the skin; however, interpreting this test may be difficult, as the burrows are scarce and may be obscured by scratch marks. A definitive diagnosis is made by finding either the scabies mites or their eggs and fecal pellets.^[25] Searches for these signs involve either scraping a suspected area, mounting the sample in potassium hydroxide and examining it under a microscope, or using dermoscopy to examine the skin directly.^[11]

Treatment

Several topical scabicides are available. Oral antihistamines can be used to help alleviate pruritus. For crusted scabies, crust and scale removal is necessary for scabicides to penetrate and oral ivermectin is also recommended. Topical antipruritics such as menthol (Sarna lotion) or pramocaine hydrochloride (Prax) can additionally be used. Nodular scabies can also be treated with intralesional steroid injections or possibly with topical pimecrolimus.^[26]

Topical therapy

Pyrethrins and pyrethroids

The flowers in the genus chrysanthemum have been used for centuries for their insecticidal properties. The active ingredients in the flowers are the pyrethrins. They were first commercially produced in Dalmatia (Croatia) in the 1840s, reaching the USA in the 1860s as Dalmatian

“insect powder”. The pyrethrins were imported as dry flowers until 1956 when an extract, some 60 times more potent, made shipping cheaper. Permethrin, a pyrethroid, was approved in the USA for the treatment of scabies in 1989. Synthetic pyrethroids including permethrin represent one of the most important insecticides, accounting for over 25% of the world insecticide market.^[27] Absorption of topically applied permethrin is very low, underlining its clinical safety.^[28] Permethrin has a good cosmetical acceptance and lacks allergic potential, shows low percutaneous penetration, and is excreted via the urine. According to a recent updated Cochrane review, permethrin appears to be the most effective topical scabicide, significantly better than crotamiton and lindane.^[29] Permethrin is indicated in babies. Topical 5% permethrin cream can cause erythema, burning, and, rarely, dystonic adverse reactions, including muscle spasms.^[30] This adverse effect is probably related to permethrin’s ability to delay sodium channel closure within nerve cells. Consequently, central nervous system symptoms could occasionally occur, especially in infants.

Lindane (γ -hexachlorocyclohexane)

Lindane is a widely prescribed topical scabicide because of its efficacy and cost-effectiveness. Dermatological adverse events include irritation; allergic contact dermatitis against parabens; and neurological symptoms (upon overdosing and accumulation in fat or milk) include insomnia, irritability, vertigo, convulsions, vomiting, diarrhea, restlessness, and collapse. This drug, therefore, has been banned in the European Union as a pesticide since 2001; it should only be used with caution when other options are not feasible.

CONCLUSION

Patients with scabies should be informed that scabies is benign but transmissible and that several treatments are available. Treatment should be recommended on the basis of a confirmed diagnosis. Topical permethrin is reasonable first-line therapy in the United States. Where permethrin is not available (e.g., in France), topical benzyl benzoate or oral ivermectin are good choices. Oral ivermectin is preferred for patients who cannot tolerate topical therapy and those who are unlikely to adhere to a regimen of such therapy. If ivermectin is used to treat the man described in the vignette, limited data and my professional experience would lead me to recommend administration of a dose of 200 μ g per kilogram, repeated two weeks later. Close contacts of the man should also be treated, and I would provide a prescription to treat his girlfriend at the same time, even if she were asymptomatic. I would also recommend machine washing and drying recently worn clothes and bed linens. The patient should be followed to confirm resolution of itching, which may take up to four weeks.

REFERENCES

1. <https://www.healthline.com/health/scabies>
2. Van Hee R. Jeremy Thriverius (1504–1554):

- humanist doctor, born 500 years ago. *Rev Med Brux*, 2005; 26: 475–78.
3. Alexander JO. Scabies. In: Arthropods and human skin. Berlin: Springer-Verlag, 1984; 227–92.
4. Montesu MA, Cottoni F, GC Bonomo and D Cestoni. Discoverers of the parasitic origin of scabies. *Am J Dermatopathol*, 1991; 13: 425–27.
5. Holness DL, DeKoven JG, Nethercott JR. Scabies in chronic health care institutions. *Arch Dermatol*, 1992; 128: 1257–60.
6. Chosidow O. Scabies and pediculosis. *Lancet*, 2000; 355: 819–26.
7. Orkin M. Scabies: what’s new? *Curr Probl Dermatol*, 1995; 22: 105–11.
8. Heukelbach J, Oliveira FA, Feldmeier H. Ecoparasitoses and public health in Brazil: challenges for control. *Cad Saude Publica*, 2003; 19: 1535–40.
9. Burgess I. *Sarcoptes scabiei* and scabies. *Adv Parasitol*, 1994; 33: 235–92.
10. Mellanby K. The development of symptoms, parasitic infection and immunity in human scabies: *Parasitology*, 1944; 35: 197–206.
11. Hay RJ. "Scabies and pyoderma-diagnosis and treatment". *Dermatol Ther*, 2009; 22(6): 466-74. doi:10.1111/j.1529-8019.2009.01270.x. PMID 19889132.
12. Markell, Edward K.; John, David C.; Petri, William H. (2006). Markell and Voge's medical parasitology (9th ed.). St. Louis, Mo: Elsevier Saunders. ISBN 0-7216-4793-6.
13. "Scabies" (PDF). DermNet NZ. New Zealand Dermatological Society Incorporated. Archived (PDF) from the original on, 2009-03-27.
14. Bouvresse, S.; Chosidow, O. "Scabies in healthcare settings". *Curr Opin Infect Dis*, Apr 2010; 23(2): 11118. doi:10.1097/QCO.0b013e328336821b. PMID 20075729.
15. Sterling GB, Janniger CK, Kihiczak G, Schwartz RA, Fox MD. Scabies. *Am Fam Physician*, 1992; 46: 1237–41.
16. Haag ML, Brozena SJ, Fenske NA. Attack of the scabies: what to do when an outbreak occurs. *Geriatrics*, 1993; 48: 45–46, 51–53.
17. Pence D, Ueckermann E. 2002. Sarcoptic mange in wildlife. *Rev Sci Tech*, 2002; 21: 385–98.
18. Walton SF, Dougall A, Pizzutto S, et al. Genetic epidemiology of *Sarcoptes scabiei* (Acari: Sarcoptidae) in northern Australia. *Int J Parasitol* 2004; 34: 839–49.
19. Cargill CF, Pointon AM, Davies PR, Garcia R. Using slaughter inspections to evaluate sarcoptic mange infestation of finishing swine. *Vet Parasitol* 1997; 70: 191–200.
20. Menzano A, Rambozzi L, Rossi L. Outbreak of scabies in human beings, acquired from chamois (*Rupicapra rupicapra*). *Vet Rec*, 2004; 155: 568.
21. Walton SF, Choy JL, Bonson A, et al. Genetically distinct dog-derived and human-derived *Sarcoptes scabiei* in scabies-endemic communities in northern

- Australia. *Am J Trop Med Hyg*, 1999; 61: 542–47.
22. Fischer K, Holt DC, Harumal P, Currie BJ, Walton SF, Kemp DJ. Generation and characterization of cDNA clones from *Sarcoptes scabiei* var *hominis* for an expressed sequence tag library: identification of homologues of house dust mite allergens. *Am J Trop Med Hyg*, 2003; 68: 61–64.
 23. Stewart GA, Ward LD, Simpson RJ, Thompson PJ. The group III allergen from the house dust mite *Dermatophagoides pteronyssinus* is a trypsin-like enzyme. *Immunology*, 1992; 75: 29–35.
 24. Sun G, Stacey MA, Schmidt M, Mori L, Mattoli S. Interaction of mite allergens Der p3 and Der p9 with protease-activated receptor-2 expressed by lung epithelial cells. *J Immunol*, 2001; 167: 1014–21.
 25. Andrews RM, McCarthy J, Carapetis JR, Currie BJ (December 2009). "Skin disorders, including pyoderma, scabies, and tinea infections". *Pediatr. Clin. North Am.*, 56(6): 1421–40. doi:10.1016/j.pcl.2009.09.002. PMID 19962029.
 26. Almeida HL Jr. Treatment of steroid-resistant nodular scabies with topical pimecrolimus. *J Am Acad Dermatol*, 2005; 53: 357–58.
 27. Georgiou, GP. Overview of insecticide resistance. In: Green MB, Le Baron HM, Moberg WK, eds. *Managing resistance to agrochemicals*. Washington DC: American Chemical Society, 1990; 421: 18–41.
 28. Tomalik-Scharte D, Lazar A, Meins J, et al. Dermal absorption of permethrin following topical administration. *Eur J Clin Pharmacol*, 2005; 61: 399–404.
 29. Walker GJA, Johnstone PW. Interventions for treating scabies. *Cochrane Database Syst Rev.*, 2006; 3: CD000320.
 30. Coleman CI, Gillespie EL, White CM. Probable topical permethrin-induced neck dystonia. *Pharmacotherapy*, 2005; 25: 448–50.