

Case Report

Norwegian scabies in human immunodeficiency virus and tuberculosis-infected child: A case report

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Abstract

Norwegian scabies is a rare scabies with the manifestation of thick crusts of the extremities of the skin that contain eggs and mites. Several conditions in which scabies infection is easily transmitted include immunocompromised, home nursing, and severe neurological disorder. The aim of this case report was to present a thorough analysis of a comprehensive resource for the management of Norwegian scabies patients, with a specific focus on individuals who also have HIV or other immunocompromising diseases. A 1-year-and-7-month-old boy was presented to the hospital with a chief complaint of a thick crust that he had experienced for four months. It began as a red papule in the lower extremity, then crusted and spread to the whole body. The patient kept scratching due to itching, had a recurrent fever and diarrhea for three months, and cough for one month. The patient was diagnosed with human immunodeficiency virus (HIV) and pulmonary tuberculosis at three months, suspected to get the infection from the parents. *Sarcoptes scabiei* was found from microscopy examination of skin scraping. The patient received holistic treatment, including antiretroviral drugs, antituberculosis medication, scabies treatment, and malnutrition treatment. Appropriate scabies treatment aimed at peeling crusted skin, relieving itching, and increasing the patient ability to use the extremities. Comorbidity conditions caused by HIV and pulmonary tuberculosis should also be treated to optimize the outcome. The patient was discharged in good condition with sanitation education and regular follow-up at the outpatient clinic. This case highlights that *Sarcoptes scabiei* infestation may be a clue to an immunocompromised condition. Holistic therapy aiming to cure underlying infection, infestation and underlying nutrition and psychosocial problems must be addressed to fully cure this high-burden case.

Keywords: Norwegian scabies, HIV, tuberculosis, crusted skin, management

Introduction

Scabies is a highly contagious dermatological condition resulting from the infestation and sensitization of *Sarcoptes scabiei* var. hominis [1]. Adult mites are characterized by their diminutive, oval-shaped bodies that are flattened on the ventral side and exhibit a convex curvature on the dorsal side, similar to the shape of a tortoise. Scabies is estimated to have a global impact on roughly 150–200 million individuals, with an annual prevalence of approximately 455 million cases [2]. Scabies infestation has the potential to occur in all geographical regions. However, there is a larger prevalence of these occurrences in countries with lower income levels, tropical climates, and among younger demographic groups [2]. Several factors contribute to the



high rate of scabies in children, including impoverished economic situations, social behavior, high population density, poor nutrition, poor hygiene practices, and limited access to medical care [3-5].

Norwegian scabies, also known as crusted scabies, is a variant of scabies that predominantly manifests in individuals who have been diagnosed with human immunodeficiency virus (HIV) infection, human T-lymphotropic virus (HTLV)-1 or have had immunosuppression following chemotherapy or organ transplantation [6,7]. This rare medical condition was identified in 1848 when Böck and Danielssen on a group of leprosy patients in Norway [6]. These individuals presented hyperkeratotic skin manifestations alongside a substantial infestation of many mites [6-9].

While scabies may not pose a direct threat to one's life, it can manifest as a severe and persistent condition [10]. The potential for secondary infections, sepsis, and fatal consequences arises from the presence of skin lesions in scabies [10-12]. Therefore, timely diagnosis is imperative to facilitate the administration of appropriate treatment [13]. The aim of this case report was to provide a comprehensive resource for the management of Norwegian scabies patients, with a specific focus on individuals who also have HIV and pulmonology tuberculosis (TB).

Case

A 1-year-and-7-months-old boy was presented to Pediatrics Outpatient Clinic in Haji Adam Malik Hospital, Medan, Indonesia, with a chief-complaint of thickened and scaled skin that persisted for over four months. It initially started as red bumps on the lower extremities, and he has been repeatedly scratching the affected area. A progressive thickening and expansion of the scales, starting from lower legs and extending to interdigital spaces, palms, and elbows, began to appear and eventually covered the entire body (**Figure 1**). The patient was referred to the hospital due to a confirmed diagnosis of HIV and TB. The double infections were suspected at the age of three months due to a history of repeated fever that resolved with antipyretic drugs and a history of diarrhea. The parents were subsequently checked, and the results yielded to be HIV-positive. The parents rejected the diagnosis procedure in the first place when the patient was approximately three months old; until six months ago, the body weight did not improve, and the parents allowed for the diagnosis. The patient has been on anti-TB therapy for the past six months.



Figure 1. Clinical condition of patient on hospital admission showing thick crusts on whole body.

The cardiovascular, pulmonary, and abdominal functions were observed to be within normal parameters during the physical examination. The patient was severely malnourished, as evidenced by a Z-score of less than negative three on all growth charts. The patient had a global developmental delay (GDD) with deficits across domains, including personal, social, fine motor, and gross motor skills. A comprehensive dermatological assessment revealed generalized, diffuse, hyperkeratotic lesions accompanied by excoriation and erosion marks that covered the entire body (**Figure 1**). The Auspitz sign, Koebner's phenomenon, and waxy spots yielded negative results. A mild itching was reported.

Laboratory examination revealed hemoglobin level of 11 g/dL, thrombocyte count of 74,000/mm³, leukocyte count of 12,390/mm³, hematocrit of 21.5%, MCV of 80 fl, RDW of 19.4 fl, absolute neutrophil count of 11.93, and procalcitonin level of 1.2 ng/nL. The count of CD4 T-lymphocytes was 388 cells/μL, while the proportion of CD4 T-lymphocytes was 23.2%. A skin scraping was conducted with a potassium hydroxide (KOH) 10% solution and revealed the presence of *S. scabiei* mites (**Figure 2**).



Figure 2. Microscopic examination of skin scraping showing *Sarcoptes scabiei*.

The patient was managed in the isolation room to restrict the transmission of the disease. The room exclusively accommodates patients, with a daily routine of changing bed sheets and pillows and soaking in hot water. The treatment regimens involved two topical scabidical medicines, a 5% permethrin cream, and a combination of 2% salicylic acid with 4% sulfur precipitate. Additionally, an oral scabidical medication, ivermectin, was administered. The permethrin 5% cream was uniformly distributed on the entire body during nighttime, followed by two hours of leaving the cream on the skin before rinsing. Since the mites persisted in the patient's skin scrapings following the fourth day of permethrin application, the administration of permethrin was prolonged for seven days. A solution containing 2% salicylic acid and 4% sulfur precipitate was administered topically to the entire body once per 24 hours in the morning, following the rinsing off of permethrin, for three consecutive days. Following the administration of scabidical treatment during the initial days, a noticeable reduction in crusting was observed. However, substantial crusting persisted on the soles of the feet and hands. Subsequently, the family received immediate treatment alongside the patient.

In addition to the prescribed scabies medication, antibiotic therapy was also administered, ampicillin-sulbactam and gentamicin ointment, due to the discovery of a secondary infection. Antihistamines were given to alleviate the pruritus.

The patient's progress was evaluated on the eighth day post-treatment, revealing positive outcomes such as decreased itchiness, thinning of pre-existing lesions, and absence of any new lesions. The previously observed efflorescence, consisting of hyperkeratotic plaque with a thick scale, has dissolved into a thin layer of white scale (**Figure 3**). The subsequent skin-scraping analysis did not indicate the presence of *S. scabiei*. Two administrations of ivermectin were administered with an interval of eight days between each dosage. The administration of the third or subsequent dose was postponed due to the absence of a persistent thick scale during the evaluation. Anti-scabies medication has been stopped, and antihistamines only need to be taken

when the skin itches. The family was provided with educational information pertaining to comprehensive scabies treatment, including the persistence of itching after one month, the potential for reinfection, and preventive measures. The health staff responsible for scabies-infected individuals did not exhibit any symptoms.



Figure 3. Clinical condition improvement after appropriate treatment.

Discussion

Norwegian scabies is a highly contagious condition characterized by extensive mite infestation. Classic scabies requires fewer than fifteen *S. scabiei* mites and requires 15 to 20 minutes of close contact for transmission, whereas Norwegian scabies typically infects immunocompromised and weakened individuals with tens of thousands to millions of mites [1]. This particular variant of scabies can be identified by the appearance of psoriasiform dermatitis, which is often observed as a diffuse hyperkeratotic plaque accompanied by the thickness and dystrophy of the nails. Patients frequently report experiencing only mild itching, yet the frequent occurrence of secondary bacterial infections has been linked to a high mortality rate among those with scabies [1]. Due to the substantial infestation of mites, Norwegian scabies exhibits a high degree of contagiousness, consequently leading to a markedly elevated risk of transmission to individuals nearby [7,14,15]. In individuals affected by scabies, there is an activation of the cell-mediated immune response, resulting in the presence of various inflammatory cells (such as eosinophils, lymphocytes, and histiocytes) surrounding the burrows created by the mites, as observed through histological analysis. The pruritus seen in scabies is a result of a type IV hypersensitivity reaction to the compounds produced by the mites. Scabies is known to elicit a humoral immune response, which is characterized by the presence of peripheral eosinophilia and elevated levels of immunoglobulin E (IgE) and immunoglobulin G (IgG). This immunological response often leads to the accumulation of IgE in the skin. Scabies is transmitted via direct contact. The degree of contagion of scabies is directly proportional to the extent of crust formation observed by those affected by the condition [1,14].

Individuals diagnosed with Norwegian scabies showed specific IgE allergic reactions against cysteine proteases and apolipoproteins generated by *S. scabiei*. Differences in the antibody and cellular responses to these proteins indicate clinical severity. While Norwegian scabies is frequently linked to prevalent immunosuppressive conditions such as HIV, HTLV-1 infection, or

those who have undergone organ transplantation, there are instances where individuals without known immunodeficiency are susceptible to Norwegian scabies [17,18].

The individual involved in the present case was a child diagnosed with HIV and TB, a known predisposing factor for Norwegian scabies or crusted scabies. The presence of pruritus throughout the body, particularly during nighttime, increases suspicion of scabies. However, it is worth noting that pruritus is not commonly observed in cases with crusted scabies. The observed skin abnormalities in this instance consisted of hyperkeratotic plaques characterized by thick white scales, which were identified as psoriasis vulgaris. According to the parents, the patient denied the presence of thick, red, scaly patches in the psoriasis predilection areas. Upon thorough examination, the absence of the Auspitz sign, Koebner's phenomenon, and waxy spots indicate that the diagnosis of psoriasis vulgaris can be ruled out, and the presence of lesions in the scabies' predilection area with a hyperkeratotic and crusted appearance supports the diagnosis of crusted scabies.

Permethrin 5% is considered a primary topical treatment for scabies, exhibiting a high efficacy rate of approximately 90% and demonstrating a favorable safety profile. The percutaneous absorption of this substance is minimal, and its presence in the bloodstream and brain is found in low amounts, indicating its safety for administration to infants, children, pregnant women, and breastfeeding individuals. Permethrin exerts its effects on arthropod nerve cells by disrupting voltage-gated sodium channels, leading to extended depolarization of the nerve cell membrane [19]. This interference hinders neurotransmission, ultimately causing paralysis and destruction of the mite. Potential adverse reactions might include mild burning sensations, stinging, itching, redness, and tingling, as well as chronic excoriation. Additionally, dystonia and seizures, albeit infrequent, have been reported as unusual side effects [9,19].

Due to the possibility that the crust would turn into a nidus where mites congregate, keratolytic medicines should be included in the therapy of crusted scabies. The application of topical keratolytic medicines, such as salicylic acid or urea 40%, and the practice of soaking the affected area in hot water have been found to reduce skin hyperkeratosis effectively. Additionally, these treatments have shown a capacity to decrease the population of mites and enhance the absorption of topical scabicial agents [7,14]. The use of salicylic acid at concentrations ranging from 3% to 6% has been observed to induce the exfoliation of the outermost layer of the skin, known as the stratum corneum, by a process of softening and dissolution [20]. The intracellular matrix is responsible for loosening the links between corneocytes, making it a potential component for incorporation in scabies therapy [20]. In this case, our treatment regimens involved two topical scabicial medicines, a 5% permethrin cream, and a combination of 2% salicylic acid with 4% sulfur precipitate. An oral scabicial medication, ivermectin, was also administered. Two doses of ivermectin were administered with an interval of eight days between. The administration of the third or subsequent dose was not given due to the absence of a persistent thick scale during the evaluation, which means the anti-scabies treatment was successful.

Skin injuries due to frequent scratching often lead to complications in scabies infestation, such as impetigo caused by *Streptococcus pyogenes* or *Staphylococcus aureus*. The reciprocal interaction between the mite and host immune systems, which involves the synthesis of complement inhibitory proteins by the mite, facilitates *Streptococcus pyogenes*' survival and *Staphylococcus aureus*' growth [21]. Local complications might manifest as abscesses, cellulitis, and, in rare cases, necrotizing soft tissue infections [22]. The primary etiology of systemic problems associated with scabies primarily stems from secondary bacterial infections. Infection with *Streptococcus pyogenes* has the potential to induce acute post-streptococcal glomerulonephritis (APSGN), and outbreaks of APSGN have been linked to the occurrence of *S. pyogenes* superinfection in scabies lesions [23,24]. Additionally, secondary bacterial infections have the potential to induce bacteremia and sepsis. The absence of medical intervention for crusted scabies poses a significant mortality risk due to the potential development of secondary sepsis [25,26]. In this case, antibiotic therapy was administered, specifically ampicillin-sulbactam and gentamicin ointment, due to the discovery of a secondary infection. This antibiotic is known to be an empirical therapy for *Staphylococcus aureus*, which might help to prevent systemic infection.

Child growth and development are influenced by a combination of genetic-hereditary and environmental influences, particularly during both the prenatal and postnatal periods. These environmental elements cover a range of essential needs required for the growth and development of children, including care, affection, and stimulation. The optimal fulfilment of basic requirements in this patient was hindered because the patient is a child of divorced parents, both of whom hailed from low socio-economic backgrounds and infected with HIV. The patient received prior care from the mother and afterwards was placed under the guardianship of the patient's paternal uncle. As shown by examination findings, the patient presented with growth and developmental issues, called global developmental delay (GDD), with deficits in personal, social, fine motor, and gross motor skills domains. The etiology of GDD encompasses various factors that can be divided into three main stages: prenatal (genetic predisposition, exposure to infections, and teratogenic influences), perinatal (hypoxia and premature birth), and postnatal (infections, traumatic incidents, as well as instances of neglect or psychosocial deprivation) [27]. In this case, postnatal variables, specifically infection (TB and HIV) and psychological issues (poor parental knowledge), were observed. To address the growth and developmental challenges experienced by the patient, it is imperative to provide appropriate therapeutic interventions for the infections, as well as stimulation and proper nourishment. These interventions are essential for meeting the patient's fundamental needs, such as affection, skill development, and nurturing, which are crucial for children's overall well-being.

Conclusion

A case study has been documented in a child with HIV and TB infection presented with Norwegian scabies, exhibiting widespread crusting across the body. The patient demonstrated improvement following a treatment regimen consisting of oral ivermectin therapy, 5% permethrin, and a combination of 2% salicylic acid with 4% sulfur precipitate ointment, in addition to providing hygiene education to the patient's family. This case highlights that in order to cure this high-burden case, holistic therapy for several factors, such as underlying infection, infestation of *S. scabiei*, and underlying nutritional and psychosocial problems should be done comprehensively and completely.

Ethics approval

The parent signed the written informed consent.

Conflict of interests

The authors declare that there is no conflict of interest.

Acknowledgments

None.

Funding

This study received no external funding.

Underlying data

All data underlying the results are available as part of the article and no additional source data are required.

How to cite

Wijaya H, Kollins F, Lubis IND, *et al.* Norwegian scabies in human immunodeficiency virus and tuberculosis-infected child: A case report. Narra J 2024; 4 (1): e661 - <http://doi.org/10.52225/narra.v4i1.661>.

References

1. Sunderkötter C, Wohlrab J, Hamm H. Scabies: Epidemiology, diagnosis, and treatment. *Dtsch Arztebl Int* 2021;118(41):695-704.
2. Engelman D, Yoshizumi J, Hay RJ, *et al.* The 2020 international alliance for the control of scabies consensus criteria for the diagnosis of scabies. *Br J Dermatol* 2020;183(5):808-820.
3. Retha R, Sawitri S. Scabies in children: A retrospective study. *Berkala Ilmu Kes Kulit Kelamin* 2020;32(1):55-61.
4. Amro A, Hamarsheh O. Epidemiology of scabies in the West Bank, Palestinian Territories (Occupied). *Int J Infect Dis* 2012;16(2):e117-e120.
5. Haile T, Sisay T, Jemere T. Scabies and its associated factors among under 15 years children in Wadila district, Northern Ethiopia, 2019. *Pan Afr Med J* 2020;37:224.
6. Schlesinger I, Oelrich DM, Tyring SK. Crusted (Norwegian) scabies in patients with AIDS: The range of clinical presentations. *South Med J* 1994;87(3):352-356.
7. Aukerman W, Curfman K, Urias D, Shayesteh K. Norwegian scabies management after prolonged disease course: A case report. *Int J Surg Case Rep* 2019;61:180-183.
8. Karthikeyan K. Crusted scabies. *Indian J Dermatol Venereol Leprol* 2009;75(4):340-347.
9. Vignesh R, Shankar EM, Devaleenal B, *et al.* Atypically distributed cutaneous lesions of Norwegian scabies in an HIV-positive man in South India: A case report. *J Med Case Rep* 2008;2(1):1-3.
10. Subramaniam G, Kaliaperumal K, Duraipandian J, Rengasamy G. Norwegian scabies in a malnourished young adult: A case report. *J Infect Dev Ctries* 2010;4(5):349-351.
11. Esposito L, Veraldi S. Skin bacterial colonizations and superinfections in immunocompetent patients with scabies. *Int J Dermatol* 2018;57(10):1218-1220.
12. van der Linden N, van Gool K, Gardner K, *et al.* A systematic review of scabies transmission models and data to evaluate the cost-effectiveness of scabies interventions. *PLoS Negl Trop Dis* 2019;13(3):e0007182.
13. Leung V, Miller M. Detection of scabies: A systematic review of diagnostic methods. *Can J Infect Dis Med Microbiol* 2011;22(4):143-146.
14. Centers for Disease Control and Prevention. Medications. Available from: https://www.cdc.gov/parasites/scabies/health_professionals/meds.html. Accessed: 12 September 2023.
15. Talaga-Ćwiertnia K. *Sarcoptes* infestation. What is already known, and what is new about scabies at the beginning of the third decade of the 21st century? *Pathogens* 2021;10(7):868.
16. Walton SF, Beroukas D, Roberts-Thomson P, Currie BJ. New insights into disease pathogenesis in crusted (Norwegian) scabies: The skin immune response in crusted scabies. *Br J Dermatol* 2008;158(6):1247-1255.sss
17. Roberts LJ, Huffam SE, Walton SF, Currie BJ. Crusted scabies: Clinical and immunological findings in seventy-eight patients and a review of the literature. *J Infect* 2005;50(5):375-381.
18. Liu X, Walton SF, Murray HC, *et al.* Crusted scabies is associated with increased IL-17 secretion by skin T cells. *Parasite Immunol* 2014;36(11):594-604.
19. Shimose L, Munoz-Price LS. Diagnosis, prevention, and treatment of scabies. *Curr Infect Dis Rep* 2013;15(5):426-431.
20. Federico M, Mellick L. Norwegian scabies: A challenging dermatologic condition. *The Open Emergency Medicine Journal* 2010;3(1):25-26.
21. Swe PM, Reynolds SL, Fischer K. Parasitic scabies mites and associated bacteria joining forces against host complement defence. *Parasite Immunol* 2014;36(11):585-593.
22. Krüger R, Hanitsch LG, Leistner R, *et al.* Scabies, periorbital cellulitis and recurrent skin abscesses due to panton-valentine leukocidin-positive staphylococcus aureus mimic hyper IgE syndrome in an infant. *Pediatr Infect Dis J* 2017;36(12):E347-E348.
23. Streeton CI, Hanna Jn, Messer Rd, Merianos A. An epidemic of acute post-streptococcal glomerulonephritis among aboriginal children. *J Paediatr Child Health* 1995;31(3):245-248.
24. Hoy WE, White AV, Dowling A, *et al.* Post-streptococcal glomerulonephritis is a strong risk factor for chronic kidney disease in later life. *Kidney Int* 2012;81(10):1026-1032.
25. Glover R, Young L, Goltz RW. Norwegian scabies in acquired immunodeficiency syndrome: Report of a case resulting in death from associated sepsis. *J Am Acad Dermatol* 1987;16(2 Pt 1):396-399.
26. Lin S, Farber J, Lado L. A case report of crusted scabies with methicillin-resistant *Staphylococcus aureus* bacteremia. *J Am Geriatr Soc* 2009;57(9):1713-1714.

27. Bélanger SA, Caron J. Evaluation of the child with global developmental delay and intellectual disability. *Paediatr Child Health* 2018;23(6):403.