Indonesian Journal of
Tropical and Infectious Disease
Vol. 6. No. 2 Mei–Agustus 2016

Case Report

NORWEGIAN SCABIES IN AIDS PATIENT: A CASE REPORT

Meita Ardini Pratamasari[1], Indropo Agusni[1a], Cita Rosita Sigit Prakoeswa[1], Linda Astari[1], Willy Sandhika[2]
[1] Department of Dermatology & Venereology
[2] Department of Pathology Anatomy
Faculty of Medicine Universitas Airlangga/Dr. Soetomo General Hospital
[a] Corresponding author: indropo49@gmail.com

ABSTRACT

Scabies is a skin infection caused by Sarcoptes scabiei var. hominis. This disease may present severe clinical manifestations in immune-compromised patient, well-known as Norwegian scabies or crusted scabies.A 36-year old man with AIDS had chief complaint thick crust almost all over his body in this case. History of household member infected by scabies before was present. Clinical findings show hyperpigmented macules unsharply marginated, covered with thick scales and accompanied by papules, fissures, and erotion. T cell CD4 level was 12 cell/μL. Scraping examination showed scabies infection and so did the histopathology examination. This patient was treated by topical Permethrin 5% combined with 2-4 ointment application in between permethrin usage. Before topical scabicide was given, thick crust was previously treated by topical urea 10% and wet dressing by normal saline. On day 14 after the patient first came there was lesion improvement.

Key words: *Norwegian scabies, immunocompromised, AIDS, thick crusts*

ABSTRAK

Skabies adalah suatu penyakit infeksi kulit yang disebabkan oleh tungau Sarcoptes scabiei var. hominis. Penyakit ini bisa bermanifestasi klinis yang hebat pada pasien dengan sistem imun yang rendah dan biasa disebut "Norwegian Scabies" atau skabies berkrusta. Dilaporkan seorang laki-laki, usia 36 tahun, penderita AIDS, yang datang dengan keluhan keropeng yang tebal dan gatal pada sekujur badannya. Beberapa anggota keluarga juga menderita gatal pada malam hari, namun tidak separah pasien. Pemeriksaan klinis menunjukkan adanya bercak hiperpigmentasi yang menebal, disertai adanya erosi dan fisura pada beberapa tempat. Pemeriksaan sel Limfosit CD4 menunjukkan kadar yang rendah (12 sel/ul). Pada pemeriksaan kerokan kulit ditemukan adanya infeksi scabies dan ditunjang oleh pemeriksaan histopatologi. Pengobatan diawali dengan kompres NaCl fisiologis dan salep urea 19%, selanjutnya diberikan salep Permethrin 5% secara berkala, diselingi dengan kombinasi salep campuran asam salisilat dan sulfur (" salep 2-4 "). Setelah 14 hari diobati, lesi kulit berkurang dan menunjukkan banyak kemajuan.

Kata kunci: *Norwegian scabies, immunocompromised, AIDS, krusta tebal*

INTRODUCTION

Acquired Immunodeficiency Syndrome (AIDS) is a group of clinical symptoms due to the decreasing of lymphocyte T-CD4+ cell count, caused by Human Immunodeficiency Virus (HIV) infection. This virus belongs to genus *lentivirus*, family *Retroviridae* or commonly known as the retroviral group. It destroys the lymphocyte T-CD4+ cells, causing the cell count to decrease below 200 cells/μL and the patients become prone to infection.[1] One of infection that could affect HIV/AIDS patient is scabies. Scabies is a disease caused by *Sarcoptes scabiei var. hominis* parasite infestation, family *Sarcoptidae*, class *Arachnida* on the skin.[2] This disease is one of major skin health problem in development countries associated with poverty, with estimated 300 million people worldwide are affected.[3] The prevalence of Scabies in Indonesia varies from 2–65% and it relates with geographical, seasons and communities. High prevalence are reported in certain communities (pondok pesantren,

dormitory, jails).[4] Scabies infection is very contagious and could be the source of infection to the surrounding environment through direct skin or clothing contact.[2,3] Clinical symptoms such as itchy, especially at night time accompanied by papular skin eruptions. Pathognomonic lesion in scabies infection is a burrow, which is a thin, thread-like, linear structure 1-10 mm in length. Burrow is actually a tunnel caused by the movement of the mite in the stratum corneum, with predilection at interdigital webs, periumbilical, and genital areas.[2,3]

Clinical findings of scabies infection in HIV patients is different with immunocompetent patient. Lesions manifest as thick crusts so it's called crusted scabies or commonly known as Norwegian scabies.[5] This type of infection has very enormous mite population so that it's highly contagious even through casual contact. It also affects face, scalp, nail, with minimal pruritic symptom. This uncommon and hyperkeratotic type of scabies infection tends to affect immuno-compromised person due to lack of immune system ability to maintain the mite.[6,7]

CASE REPORT

A thirty-six year old male patient admitted to the Dr. Soetomo General Hospital Surabaya with chief complaint thick crusts almost all over his body since 1 week before. Firstly it was some small papules over his thigh area, felt a bit itchy but no itchy sensation at nighttime. The papules then spread to all over his body, became thick crusts with some cracks in between and causing difficulty when moving. His wife and child ever had similar complaint 1 month before visitation, which were papules over their bodies accompanied with itchy sensation. They had been treated with permethrin cream and their lesions were getting better. Meanwhile, the patient's mother suffered from psoriasis but history of lesion on Koebner area in this patient was denied.

Before his admission, he did the Voluntary Counseling and Testing (VCT) and HIV Rapid Test 2 months before and the result was positive, confirmed by three methods test hence he was diagnosed as AIDS. He had controlled routinely to the HIV outpatient clinic and consumed antiretroviral (ARV) treatment for 1 month. At the outpatient clinic about 3 weeks before, he was diagnosed chronic dermatitis and got topical corticosteroid with emollient, and his complaint was getting better until the last complaint occurred 1 week before his admission.

Physical examination showed weak general condition was but good consciousness with normal vital sign. He had anemic conjunctiva and also slight enlargement of the liver. For the dermatological state, on auricular, axilla, colli, abdominal, inguinal, extremity (interdigitalis), and also gluteus regions there were large hyperpigmented macules, unsharply marginated and covered by thick crusts. There were also some fissures over the thick crusts, erosions, and we could see multiple papules over the eroded area over the scrotum (Figure 1).

The laboratory examination results were: White Blood Cells 3,600 cells/mm^3, Red Blood Cells 4.54 x 10^6 cells/mm^3, Platelet Count 128,000 cells/mm^3, Hemoglobin level 7.8 g/dl, SGOT 126 U/l, SGPT 67 U/l, BUN 7 mg/dl, creatinine serum 0.6 mg/dl, albumin level 2.1 g/dl, random blood glucose 86 mg/dl, natrium 130 mmol/l, kalium 3.8 mmol/l, chloride 100 mmol/l. The CD4 absolute count had been performed before with the result was only 12 cells/µL.Lesion scraping was done to find if there was any mite of *Sarcoptes scabiei*, there were the adult mite with some eggs.

Histopathologic examination was done to exclude psoriasis. Nevertheless, the diagnosis of scabies had been established by the scraping examination, so the therapy was soon started. First the wet dressing by normal saline solution was done to the thick crusts area in orderboth to decrease the crust andaddress the erosion, accompanied by Urea 10% cream application. The thick crusts should

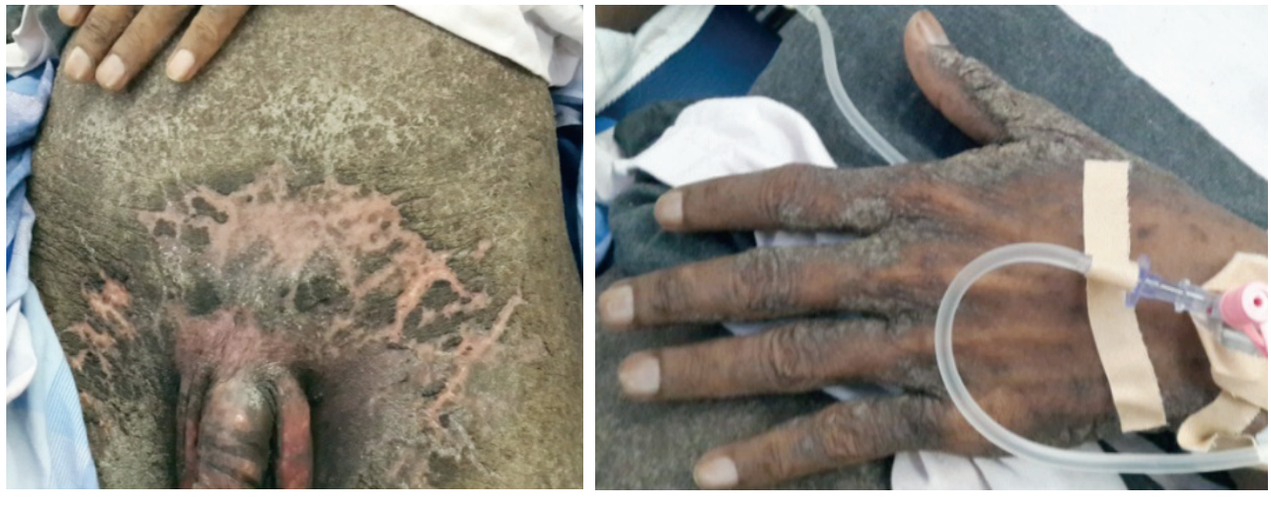

Figure 1. Thick crusted lesion over abdomino-inguinal and interdigital region.

be removed because it could inhibit topical antiscabies penetration, and the erosion should be treated soon because the applicationof topical antiscabies over the erosion could cause irritation. After 4 days of wet dressing treatment the thick crusts and the erosion were decreased, application of Permethrin 5% cream once a week at night was started, with exception wet dressing and Urea application were still done for area with thick crusts. Besides dermatology therapy, the patient also continue the antiretroviral (ARV) treatment, include terafovir, lamivudine and neviral; concomitant with supportive treatment.

Histopathology examination showed hyperkeratotic, parakeratosis, acanthosis and burrow in stratum corneum layer of epidermis. While in dermis there were capillary vessels with a little inflammatory cell, so the conclusion from the histopathology examination was scabies infection.

After hospitalized for 1 week and there were progress of his lesion, this patient was discharged from hospital. Before went home, he and his family were given education to repeat the use of Permethrin 5% cream 1 week after the first use if there was any lesion persist either crusts or papules and to visit hospital afterwards, to wash all clothes, towels, and bedding by hot water. If there were any other household members that have the same complaint, they should be treated soon.

DISCUSSION

Norwegian scabies or crusted scabies is a rare manifestation of scabies characterized by uncontrolled proliferation of mites in the skin. This disease was first described by Boeck and Danielssen among leprosy patients in Norway in 1848.[8,9] High risk groups for this infection such as they who are taking systemic glucocorticoid therapy or using potent topical glucocorticoid therapy, organ transplant recipient, having mental or physical disability, infected by HIV or human T-lymphotrophic virus-1, and also people with malignancy.[10] This severe variant

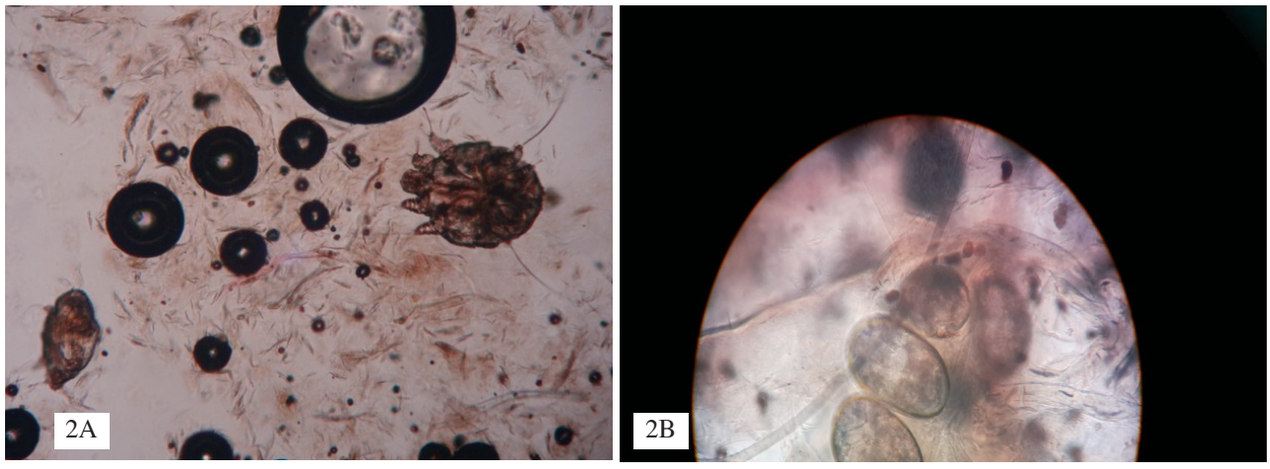

Figure 2. *Sarcoptes scabiei* mite from the lesion scraping: adult mite (2A), eggs (2B)

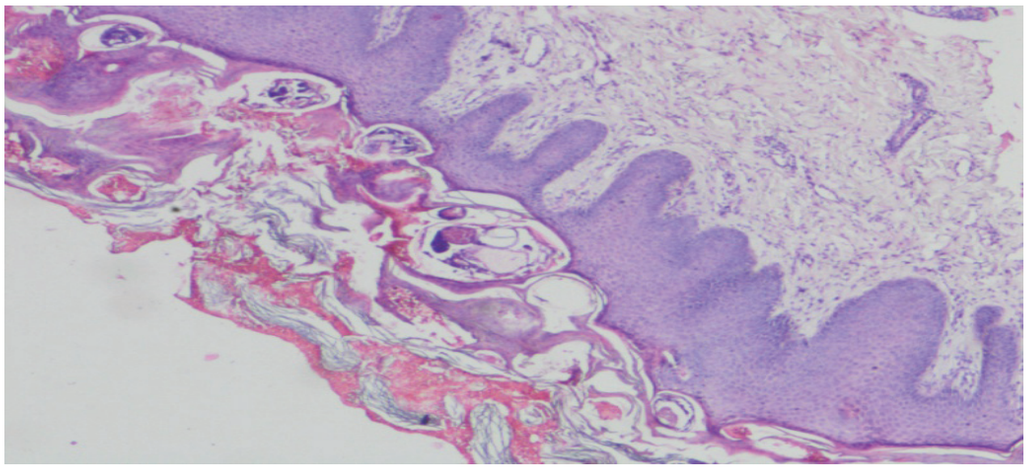

Figure 3. Histopathology slides with 40x magnification

of scabies occurs as widespread hyperkeratotic crusted lesions, hence the name "crusted scabies" is preferred as the synonym of "Norwegian scabies".[11]

The causative agent, mite *Sarcoptes scabiei var. hominis*, is an obligate parasite that lives in burrowed tunnels in the stratum corneum. In the skin, the mite survives on a diet of dissolved human tissue but does not feed on blood. It makes a sloping burrow, in the stratum corneum to the boundary of stratum granulosum every day. The mite lives in the burrow for a 30 day-period, consisting cycle as follows. The female mite lays 2 - 3 eggs daily and the eggs hatch in 10 days, then the young larva leaves the burrow to become mature adult mite in 14 – 17 days.[2,3,9] In normal patient, it is estimated that only 10% of the eggs that develop into adults with total average mite is about 11. However, the number of the mites is very enormous in crusted scabies because of uncontrolled infection.[8,9,10] Recently there is increasing occurrence of this case due to various immunosuppressive agent and increasing case of HIV patient (Figure 2).

Cutaneous manifestations of scabies are due to the burrowing of the female mite followed by humoral and delayed hypersensitivity of the host.[2,3] The mite antigens that trigger the immune response are probably in the mite's saliva. Combined with scratching, the immune system in the healthy host will reduce the mite load but rarely eliminates the mite. The failure of the immune system to suppress the proliferation of the mite is considered to play role in crusted scabies development, though incidence of crusted scabies in Australian aborigines with normal immunity has been reported.[13] In this case, HIV stadium IV that the patient suffered from made the level of CD4+ T cells dropped until 12 cells/μL so that the patient was susceptible to infection. While less itchy sensation occurred as the result of inadequate immune system, a huge amount of mites makes this disease very contagious.[7,8]

Definitive diagnosis of crusted scabies is equal with common scabies, which is the presence of mite, egg, eggshell or fecal material from skin lesion scraping, demonstrated by potassium hydroxide 10% solution under light microscopy examination. In this patient, we found the presence of the mite and egg so that antiscabies treatment could be initiated without waiting for the histopathology result. Later, the histopathology examination revealed that there was burrow in the stratum corneum that is surrounded by inflammation cells, showed that cellular immunity plays role in this disease's pathogenesis. Burrow was pathognomonic sign that we can find in scabies infection (Figure 3).[2,3]

Scabies management involves the use of scabicide agent and mite control. Antiscabies agent might be taken orally and topically as discussed above, meanwhile mite control needs education for the patient and his family. All family members that live together with the patient should be treated at the same time to prevent asymptomatic carrier's reinfestation. If possible, during the time of application of the topical scabicide, all linens, bedding, and clothing in the house that has been used should be soaked with warm/hot water before washed, and then ironed with high temperature to eradicate mite.[2,3]

This patient was treated initially with wet dressing (2-3days), using normal saline combined with 10% urea cream to remove the thick crusts. Then a 5% Permethrin cream was applied intermittently combined with ointment contains 2% salicylic acid plus 4% sulfur ("2-4 ointment) daily in between the permethrin. After 14 days application of this topical medication there is mark improvements. Theoretically oral ivermectine could be used since this drug acts on nerve synapses utilizing glutamate or γ-aminobutyric acid.[14] But this oral medication could not penetrate into the thickness of the keratinous debris and this drug is not available in Indonesia. Conventional wet dressing method using NaCl 0.9% solution was used, then followed by topical Urea 10% use for softening the crusts. After the crusts already minimal and thinned, Permethrine 5%, a topical antiscabies agent, was applied to this patient. This topical agent will be effective in such situation due to better absorption in the skin.

Scabies management involves the use of scabicide agent and mite control. Antiscabies agent might be taken orally and topically as discussed above, meanwhile mite control needs education for the patient and his family. All family members that live together with the patient should be treated at the same time to prevent asymptomatic carrier's reinfestation. If possible, during the time of application of the topical scabicide, all linens, bedding, and clothing in the house that has been used should be soaked with warm/hot water before washed, and then ironed with high temperature to eradicate mite.[2,3]

REFERENCES

1. Murtiastutik D. HIV & AIDS. In: Barakbah J, Lumintang H, Martodihardjo S, editors. Buku ajar infeksi menular seksual. Surabaya: Airlangga University Press; 2008. p. 211–69.
2. Burkhart CN and Burkhart CG. Scabies, other mites, and pediculosis. In: Goldsmith LA, Katz SI, Gilchrest BA, Paller AS, Leffell DJ, Wolff K, editors. Fitzpatrick's Dermatology in General Medicine. 8th ed, vol 2. New York: McGraw-Hill, 2012. p. 2569–77.
3. Burns T, Breathnach S, Cox N, Griffiths C, editors. Diseases caused by arthropods and other noxious animal. In: Burns T, Breathnach S, Cox N, Griffiths C, editors. Rook's Textbook of Dermatology. 8th ed. West Sussex: Wiley-Blackwell; 2010.
4. Hilmy F. Prevalensipenyakit scabies dan hubungannya dengan karakteristik santri pesantren X Jakarta Timur [skripsi]. Fakultas Ilmu Kesehatan Masyarakat, Universitas Indonesia; 2010.
5. Chan CC, Lin SJ, Chan YC, Liao YH. Infestation by Norwegian scabies. CMAJ. 2009, 181(5).
6. Perna AG, Bell K, Rosen T. Localised genital Norwegian scabies in an AIDS patient. Sex Transm Infect. 2004, 80: 72–3.
7. Karthikeyan K. Crusted scabies. Indian J Dermatol Venereol Leprol. 2009, 75: 340–7.
8. 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): 349–51.
9. Fernandez-Sanchez M, Saeb-Lima M, de la Barrera CA, Reyes-Teran G. Crusted scabies-associated immune reconstitution inflammatory syndrome. BMC Infectious Diseases. 2012, 12: 323.

10. Binic I, Jankovic A, Jovanovic D, Ljubenovic M. Crusted (Norwegian) scabies following systemic and topical corticosteroid therapy. J Korean Med Sci. 2010, 25: 188–91.
11. Davis JS, McGloughlin S, Tong SYC, Walton SF, Currie BJ. A novel clinical grading scale to guide the management of crusted scabies. PLoS Negl.Trop. Dis. 2013, 7(9).
12. Workowski KA and Berman S. Sexually transmitted diseases treatment guidelines, 2010. In: Centers for Disease Control and Prevention [Internet]. Morbidity and mortality weekly report. [cited 2013 Aug 20] Available from: www.cdc.gov/mmwr
13. Walton SF and Currie BJ. Probles in diagnosing scabies, a global disease in human and animal populations. Clin Microbiol Rev. 2007, 20(2): 268.
14. .Ly F, Caumes E, Ndaw CAT, Ndiaye B, Mahe A. Ivermectin versus benzyl benzoate applied once or twice to treat human scabies in Dakar, Senegal: a randomized control trial. Bull World Health Organ. 2009, 87: 424–30.