# Crusted(norwegian)scabies- two case reports with HIV/AIDS and lepromatous leprosy association

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**Abstract:** Parasitological axiom is that "10% of the population harbours 70% parasites". An estimated 300 million cases of scabies occur annually. The etiological agent of scabies, Sarcoptesscabieivarhominis belongs to class Arachnida and subclass Acarina. Crusted scabies, a severe variant of scabies usually seen in immunocompromised is characterized by hyperkeratotic crusts that contain large numbers of scabies mites and eggs which are highly contagious. Prompt diagnosis, quick and aggressive treatment is needed to prevent outbreaks of scabies. We are reporting two cases of crusted scabies seen in immunocompromised patients. **Key words:** Crusted scabies, Immunocompromised, Mite, HIV/AIDS, Lepromatous leprosy

### I. Introduction

Sarcoptesscabiei, often neglected parasite affects humans and other mammalians world wide<sup>1,2</sup>. Scabies was reported 2500 years back. Epidemics of human scabies occur across the world in 15- 25 year cycles and can lead to outbreaks in institutions and asylums associated with overcrowding. The Latin word 'scabere' means 'to scratch'. Crusted scabies was first reported by Boeck and Danielssen in Norway in 1948 among lepers in an asylum<sup>3</sup>. In 1862, Von Hebra named it "scabies NorwegicBoeckii". In some slums of Bangladesh, the incidence of scabies in children under 5 years was observed to be 952/1000 per year. Data base search revealed nearly 200 case reports and only a few case series of crusted scabies .

Scabies mite is an obligate ectoparasite that lives in the burrows of stratumcorneum eating the dead tissues. Sarcoptesscabiei undergoes four stages in its life cycle i,e egg, larva, nymph and adult over three to four weeks. After mating the male dies and the female mite measuring 0.3 to 0.4mm long and 0.25 to 0.35 mm wide lays 2 to 3 eggs per day. The eggs hatch in 3 to 4 days. The young larva becomes nymph and matures to adult stage in 14 to 17 days. Scratching and immune response of the host limit the mite load in normal scabies and the opposite occurs in crusted scabies. An association has been described between scabies and HLA-A11. In cases of first contact with scabies mite, the incubation period is 3 to 6 weeks, but shorter(1-3 days) in case of reinfestation.

Patients with defective T-cell immune response, those with neurological problems and people who have impaired capacity to debride mite develop crusted scabies. Crusted scabies may be misdiagnosed as psoriasis<sup>4</sup> or eczema. It is highly contagious, because of the mite load which is up to 2 millions. Transmission is usually through skin-skin contact but in crusted scabies, transmission through fomites which are heavily infested can often trigger an epidemic. Hyperkeratosis, a notable feature in crusted scabies may be due to increased interleukin-4 levels<sup>5</sup>. Recent studies have showed that Th 2 response and decreased B cell activity lead to decreased effective immune response<sup>6,7</sup> and hence uncontrolled parasite proliferation in skin. Itching which is quite remarkable in scabies is minimal or absent in crusted scabies. Firm and adherent often porous localized parakeratotic crust, with an erythematous area underneath are hallmarks of crusted scabies. Palms, soles and nails are usually affected. It can involve the entire body leading to erythroderma<sup>8</sup>. These cases are usually diagnosed clinically and by demonstration of mite from crusts<sup>9</sup>. Epiluminescence microscopy and video-dermatoscopy are useful noninvasive techniques to visualize the mite. Mite or parts of the mite may be seen in histopathological sections. Eosinophilia may be seen in 58% of cases and serum IgE levels increased in most cases<sup>10</sup>. An outbreak of scabies in family members or in close contacts can be an indirect evidence of crusted scabies.

## II. Case Reports

**Case 1:** A 32 year old male unmarried PLHA presented with history of thickening and painful fissuring of both palms and soles of 6 months duration(Fig.1&2). Patient had difficulty in walking. Later he observed scaly thickened lesions spreading all over the body associated with mild itching(Fig.3). He was diagnosed and treated as psoriasis at various places without relief before he came to Gandhi hospital. Routine investigations were within normal limits except for anemia (hemoglobin -8 gms/dl) and eosinophilia (eosinophil count -1100 per

cubic millimeter). He was reactive for HIV-1 as per NACO guidelines. HBsAg was negative, RPR was nonreactive and CD4 count was 17 cells/ Cu mm. Superficial inguinal lymphadenopathy was present. On high suspicion, the crusts from skin were dissolved in KOH for half an hour which showed plenty of scabies mites and eggs(Fig.4). He was treated with oral Ivermectin 9 mgs(200µg/kg body weight) single dose, repeated after one week along with local permethrin 5% cream 30 gms applied twice with a week gap, keratolytic agents and oral antihistamines daily. Anti retroviral therapy was started after 2 weeks, ruling out other opportunistic infections. Patient had symptomatic relief and complete clearance of hyperkeratotic lesions within 2 weeks (Fig. 5).

**Case 2:** A 53 year old lepromatous leprosy patient with recurrent ENL on treatment developed mild itchy scaly lesions on hands and feet(Fig.6) of one month duration which spread to trunk, axillae, buttocks and groins with moderate crusting. Other members of the family had symptoms and signs suggestive of Scabies. Routine investigations were normal, HIV screening was negative. KOH mount from scrapings of crusts showed abundant mites and eggs(Fig. 7). Patient and her family members were treated with tab. Ivermectin 200µgm/kg body weight and topical 5% permethrin cream 30 gms and repeated after one week. Patient had complete clearance of lesions in 2 weeks.

#### III. Discussion

Scabies is commonly seen in HIV infected patients. Crusted(Norwegian) scabies is usually seen in advanced HIV disease<sup>11,12</sup> in typical or atypical forms. Hyperkeratotic, nonpruritic lesions are usually encountered and lesions similar to Darrier's disease may also be seen. Dakar showed in his case series of crusted scabies, an association of 45% with HIV infection. In our present observation one out of two cases is HIV associated. Crusted scabies can sometimes present as psoriasiform dermatitis confused with psoriasis<sup>13</sup> as happened in our first case or super impose on a case of psoriatic erythroderma particularly in patients with AIDS. Bacterial infections like Staphylococcus.aureus complicate crusted scabies and cause life threatening septicemia<sup>14</sup> and death in patients with AIDS if untreated. Erythroderma is another common complication.



Figure 1: Male with HIV infection showing difuse hyperkeratotic plaques – crusted scabies mimicking psoriasis.



Fig. 2: Hyperkeratotic crusts over palms and wrist



Fig. 3: diffuse scaling along posterior axillary fold



Fig. 4 Sarcoptes scabiei from the crusts of Case 1



Fig. 5 Clearing of crusts in case 1 after two weeks of treatment

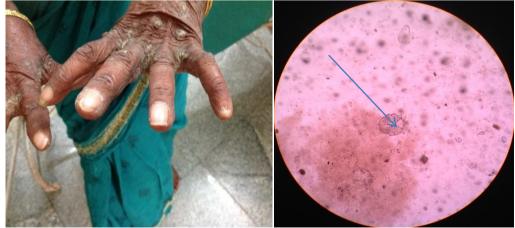


Fig. 6 Crusts in web spaces and dorsum of hand in case 2 Fig. 7Sar

Fig. 7Sarcoptis. scabiei in KOH mount

#### IV. Conclusion

Scabies, a simple condition to diagnose and treat in immunocompetent patients, becomes a great mimicker in immunosuppressed. A high index of suspicion is necessary to avoid misdiagnosis, especially of crusted scabies which is an easily treatable condition thereby preventing patient related and epidemiological complications.

#### References

- Alssad S Ross L HeukelbachJ et al the neglected navigating web of the incomprehensibly emerging and reemerging Sarcoptes mite Infection genetics and evolution2013; 17:253-259.
- Hengge UR, Currie BJ, Jager G, Lupi O, Schwartz RA. Scabies: a ubiquitous neglected skindisease. Lancet Infect Dis. 2006;6:769– 779.
- [3]. Danielsen DG BoeckW treatment of leprosy or Greek Elephentiasis. Paris, JB Ballierre 1848
- [4]. Gach JE, Heagerty A. Crusted scabies looking like psoriasis. Lancet. 2000;356:650.
- [5]. Prens E, Hegmans J, Lien RC, Debets R, Troost R, van Joost T, Benner R. Increased expression of interleukin-4 receptors on psoriatic epidermal cells. Am J Pathol. 1996;148:1493–1502
- [6]. 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;50:375–381.
- [7]. 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;158:1247-55.
- [8]. Mehta V Balachandran C Monga P et al Norwegian scabies presenting as erythroderma Indian J DermatolVenereolLeprol 2009;50:375-381.
- [9]. Guldbakke KK, Khachemoune A. Crusted scabies: a clinical review. J Drugs Dermatol.2006;5:221-227
- [10]. Hay RJ Steer AC Engelman D Swalton Scabies in the developing world- Its prevalence, complications and Management Review Clinical microbiology and infection Vol 18 No 4, April 2012:313-323
- [11]. Brites C, Weyll M, Pedroso C, Badaró R. Severe and Norwegian scabies are strongly associated with retroviral (HIV-1/HTLV-1) infection in Bahia, Brazil. AIDS. 2002;16:1292–1293.
- [12]. Porras-Luque JI, Valks R, Daudén E, Fernández-Herrera J. Localized crusted scabies in a patient with acquired immunodeficiency syndrome. ActaDermVenereol. 1997;77:326–327.
- [13]. KaliaperumalKarthikeyan Crusted scabies Review article Indian Iournal of dermatology, venereology and leprosy 2009 Volume : 75 Issue : 4 : 340-347
- [14]. Walton SF Bart J Curriee Problems diagnosing scabies, a global disease in humans and animal populationClinical Microbiology reviews 2007 Apr,20(2); 268-279.