Norwegian Scabies Complicated by Fatal Brain Abscess in a Renal Transplant Patient

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Dear Sir,

Scabies is a contagious, global disease that is characterized by persistent, pruritic papules, which may be linear in the interdigital areas. Individuals whose normal body defense mechanisms are modified or compromised may have the severe generalized infestation, termed, 'Norwegian scabies with crusted or papulosquamous lesions' [1]; because of its associated pruritus, which is probably induced by an IgE-mediated phenomenon [2], it is easy to mistake scabies for simple renal pruritus in renal patients [3]. We report a case of scabies in a patient after renal transplantation, which was complicated by a fatal brain abscess, illustrating the difficulty of diagnosing scabies in the immunocompromised, as it is not unusual for it to mimic the clinical presentation of a specific dermatosis.

A 50-year-old Sudanese male with end-stage renal failure due to hypertensive nephrosclerosis was treated with regular intermittent hemodialysis for 18 months prior to a successful HLA-matched renal transplantation from his son. Postoperatively and for more than 1 year he had no episodes of transplant rejection. He had had an infection with hepatitis B and C and schistosomiasis in the past. Before renal transplantation, a needle liver biopsy revealed chronic persistent hepatitis, but liver function tests were normal. Before the operation, he had sustained a hemorrhagic cerebrovascular accident with left hemiplegia, but he made a full recovery. He remained in good health for 1 year after the operation on prednisolone, cyclosporin A, azathioprine, atenolol and ranitidine. Thereafter, while on vacation in the Sudan, he developed a nonitchy, painless, indurated ulcer on the calf of the left leg. Punch biopsy of the ulcer wall revealed granulomata with intracellular Leishmania organisms. This was treated at our hospital with cryotherapy and topical fusidic acid. He was later admitted with a 3-month history of generalized resistant pruritus, progressive polyuria and polydipsia. On examination he had a generalized papular eruption, with a few areas of lichenification and some pustules over the medial aspect of the left thigh, thickened keratotic axillary skin of the back and nodules over the neck. The differential diagnosis was pyoderma, lichenoid eruption or a drug-induced rash. The only other findings were hepatosplenomegaly and an old operation scar. He did not respond to topical fusidic acid and steroids; however, his newly diagnosed diabetes mellitus responded well

to insulin. His generalized pruritus persisted with marked excoriation, and he developed a fluctuant perianal abscess which was drained. He was treated with parenteral flu-cloxacillin and vancomycin. The perianal abscess and a swab of his excoriated skin both grew *Staphylococcus aureus*. A punch biopsy of the keratotic, dry, thickened skin in the middle of the back showed focal hyperkeratosis, acanthosis, and a cross-section of a *Sarcoptes scabiei* mite (fig. 1). This result became only available just before his final deterioration and he had no treatment for scabies. His electrolytes, urea and creatinine were all normal but his hepatic enzymes were elevated. Abdominal ultrasound revealed only a thickened gallbladder wall, and no other abnormality. Throughout his admission, he was unhappy, despite the control of his diabetes mellitus and his perianal abscess, and the good function of his renal transplant, but he continued to have persistent pruritus. He deteriorated psychologically, refusing his medications, and developed mood swings. Three weeks after admission he developed a sudden flaccid right hemiplegia with no alteration of consciousness. A CT scan of the brain showed only the scar of his previous cerebrovascular accident, but MRI showed two circumscribed hyperdense areas in the left anterolateral cerebral hemisphere suggestive of an infective abscess (fig. 2). His CSF examined on the same day showed protein of 80 mg/dl (reference range 15-45 mg/dl), cell count 5·10³/UL (80% monocytes, 20% polymorphs) with no malignant cells while gram stain and AFB were negative. Blood culture was negative. IgG and IgM antibodies to CMV were positive but he was HIV-negative; mycology: negative, in particular *Cryptococcus neoformans*. His presumed brain abscess was treated with intravenous benzylpenicillin and chloramphenicol. Because his toxoplasma serology result was not immediately available, he was also given cotrimoxazole and pyrimethamine in case he had *Toxoplasma encephalitis* (serology subsequently proved to be negative). Unfortunately his condition deteriorated, he lapsed into a coma and died 24 h later.

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Fig. 1. Cross-section of a *S. scabiei* mite.

Fig. 2. MRI showing two circumscribed hyperdense areas in the left anterolateral cerebral hemisphere.

Scabies is a chronic disease caused by *S. scabiei*, which causes continual scratching with a keratotic, dry skin [4]. Pathologically, *Sarcoptes* infestation is a deep epidermal lesion with the distal end of its burrow sunk in the malpighian layer, creating a variety of skin swellings and excoriations [5]. Scabies may simulate lichenoid eruption, contact dermatitis, infective dermatoses or lichen nitidus. Such mimicry is aggravated in immunocompromised patients in whom the host parasite relationship is deranged [6] and this contributed to the late diagnosis in our patient. Cutaneous damage in scabies, and poorly controlled and newly diagnosed diabetes mellitus probably predisposed to *S. aureus* colonization in our patient. This was similarly described in an 88-year-old lady with a 2-year history of generalized pruritic exfoliative dermatitis, later found to be scabies complicated by staphylococcal infection [7]. The presence of multiple opportunistic infections is not unusual. Presumably the brain abscess originated from his staphylococcal skin infection. T2-weighted MRI scans are often more sensitive than CT scans in detecting cerebral abscesses [8], as it has been shown in our case. This case demonstrates that the clinician should be aware of the atypical clinical features of scabies in an immunocompromised patient,

complaining of intractable pruritis. When there is doubt skin scraping for microscopy is mandatory. If this fails, a skin biopsy of the keratotic, papular skin lesion or examination of the subungual areas or scalp may reveal the diagnosis, as early diagnosis in immunocompromised patient is of crucial importance to avoid potential serious complication as in our unfortunate patient. Most importantly, patients with an organ transplant must be educated to improve their personal hygiene so as to reduce the likelihood of opportunistic infections [9].

References

- Brickling AS, Musgnug RH: Norwegian scabies: A new complication of immunosuppressive therapy. *Cutis* 1978;22:581-22584.
- Falk ES, Bolle R: In vivo demonstration of specific immunological hypersensitivity to scabies mite. *BrJ Dermatol* 1980;103:367-373.
- Lempert KA, Baltz PS, Welton WA, et al: Pseudo-douremic pruritis: A scabies epidemic in a dialysis unit. *Am J Kidney Dis* 1985;5:117-119.
- Orken M: Today's scabies. *JAMA* 1975;233: 882-885.
- Van Neste D: Intraepidermal localisation of scabies mites overlooked. *J Am Acad Dermatol* 1984;10:676.
- Lachepelle JM, Van Neste D: Host-parasite relationship in hyperkeratotic (Norwegian) scabies: Pathological and immunological findings. *BrJ Dermatol* 1981;105:667-678.
- Shelley B, Shelley E, Dordinda E, et al: Staphylococcus aureus colonization of burrows in erythrodermic Norwegian scabies. *J Am Acad Derm* 1988;19:673-678.
- Levy RM, Mills CM, Kaposin JP, et al: The superiority of cranial magnetic resonance imaging to computed tomographic (CT) brain scan for the diagnosis of cerebral lesions in patients with AIDS (abstract 146); in proceedings of the 2nd International Conference on Aids, Paris, 1986.
- Mandell GL, Douglas R, Bennet JE: Principles and Practice of Infectious Diseases, ed 3. New York, Churchill Livingstone, 1991, chap 108, pp 106-107.