10

A 55-Year-Old Indigenous Woman from Australia With a Widespread Exfoliating Rash and Sepsis

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Clinical Presentation

History

You are working in a remote indigenous community in tropical northern Australia, and the community health worker asks you to visit a house to assess an elderly woman who has been living in the crowded back room. Her family are worried that she has become increasingly withdrawn and hasn't been getting out of the house much at all.

Clinical Findings

The patient is a 55-year-old indigenous Australian woman with a widespread exfoliative rash involving all limbs and especially the armpits, buttocks and thighs (Fig. 10.1). Many flakes of skin cover the mattress she is lying on. In addition, she has fissures over her wrists and knees. She also looks pale, is clammy and poorly responsive. Her temperature is 39.5°C (103.1°F), heart rate 110 bpm, respiratory rate 28 breath cycles per minute and blood pressure 85 mmHg systolic to radial pulsation. Oxygen saturation by pulse oximetry is 92% on room air.

Laboratory Results

You take blood cultures, full blood count, CRP and biochemistry. Samples are sent into the regional laboratory, with results expected the next day. You also take skin scrapings, which you can look at yourself using the community clinic microscope.

Questions

1. What is your provisional diagnosis?
2. What is your initial management?

Discussion

A 55-year-old indigenous Australian woman has a widespread exfoliating rash and is clearly systemically unwell with signs of sepsis.

Answer to Question 1

What is Your Provisional Diagnosis?

The exfoliating rash is classical for crusted scabies, which is uncommon but well recognized in remote indigenous communities where scabies remains endemic. Undiagnosed cases can be 'core transmitters' of scabies; and in this case many household members, and in particular the children, had multiple scabies lesions, some with secondary pyoderma. In addition, this woman has secondary sepsis which is likely to be from *Staphylococcus aureus* or *Streptococcus pyogenes* bacteraemia, with inoculation through her skin fissures. However, Gram-negative or polymicrobial sepsis is also occasionally seen in patients with crusted scabies.

• **Fig. 10.1** Severe crusted scabies of the right axilla and chest wall.

Answer to Question 2

What is Your Initial Management?

She is transported to the community health centre where you put her on oxygen, insert a wide-bore cannula, take an additional set of blood cultures, give her 1 litre of normal saline over 30 minutes and commence her on intravenous (IV) ceftriaxone 2 g and gentamicin 320 mg (her weight is 62 kg).

The Case Continued…

After the IV fluids, her blood pressure rose to 95 mmHg systolic and her oxygen saturation was 97% with oxygen by nasal prongs. She was evacuated to the regional hospital. In the clinic, microscopy of her skin scrapings under low-power magnification confirmed the diagnosis, showing multiple scabies mites of varying maturity and multiple eggs. In the hospital, the patient was managed in a single room with enhanced contact precautions to prevent transmission of scabies to staff and visitors.

Treatment consisted of a five-dose course of ivermectin (200 µg/kg/dose) on days 0, 1, 7, 8 and 14, together with a topical scabicide (benzyl benzoate 25%) applied every second day for the first week; then twice weekly for the second week, with a keratolytic cream (lactic acid and urea in sorbolene cream) after bathing on the days the topical scabicide was not administered. Her blood cultures taken in the community grew *S. aureus* resistant to flucloxacillin and clindamycin, but sensitive to gentamicin, co-trimoxazole and doxycycline (i.e. community-acquired MRSA). She was therefore given 2 weeks of IV vancomycin. A transthoracic echocardiogram showed no features of endocarditis. Repeated blood cultures 48 hours after admission were negative, and by that stage she was afebrile, off oxygen and eating well. While she was in the hospital, all her family members were treated in the community with topical permethrin 5%, given as two doses 1 week apart, and the children with skin sores were each given a single dose of intramuscular benzathine penicillin, with excellent response.

• **Fig. 10.2** Fissure over the wrist with underlying less severe crusted scabies.

The household clothes, linen and furniture were put out in the sun for a day, and the rooms were treated with a commercial residual insecticide. The woman returned to the community after 3 weeks in the hospital, with her skin showing no residual hyperkeratosis or shedding. She and the family are followed up regularly by the health staff to allow early diagnosis and treatment should she again be infected with scabies.

SUMMARY BOX

Crusted Scabies

Although *Sarcoptes scabiei* infection and mite reproduction is usually self-limiting, hyperinfestation can develop in susceptible individuals. Crusted or Norwegian scabies was first described in patients with leprosy in Norway. Crusted scabies can occur after immunosuppressive therapy in transplantation, rheumatological conditions, chemotherapy for malignancy and in infection with HIV and HTLV-1. It can also occur in malnutrition, Down's syndrome and in the elderly and institutionalized, especially those with physical or cognitive disability who are unable to scratch. Patients with crusted scabies can have many thousands of mites in skin lesions and can serve as 'core transmitters' for continuing outbreaks of scabies in communities and nursing homes. Mites from crusted scabies cases are not genetically distinct, with ordinary scabies occurring in those infected from these cases.

Crusted scabies results from unfettered mite reproduction and host reaction, resulting in formation of hyperkeratotic skin crusts that may be loose and flaky or thick and adherent. Skin flakes with thousands of mites can be shed onto bed linen and floors. Although hands and feet are most commonly involved, the distribution is often extensive, including neck, face and scalp as well as axillae, trunk, buttocks and limbs, especially knees and elbows. Thick deposits of debris with mites accumulate beneath the nails, which are often thickened and dystrophic. Crusting can be limited to one or two limbs, hands or fingers. Unlike ordinary scabies, where itching is usually intense, the presence of itch is variable in crusted scabies. Fissuring and secondary bacterial infection are common (Fig. 10.2). A peripheral blood eosinophilia is common but not always present, and serum IgE levels are often extremely high. There is high mortality in crusted scabies cases from secondary bacterial sepsis, including with Gram-negative organisms such as *Pseudomonas aeruginosa* in addition to *S. pyogenes* and/or *S. aureus*.

The differential diagnoses of crusted scabies include psoriasis, extensive tinea corporis, skin malignancies such as the T-cell lymphomas mycosis fungoides and the Sézary syndrome, nutritional deficiencies such as pellagra, pemphigus, kava dermopathy, onchocerciasis, lepromatous leprosy and secondary syphilis.

Further Reading

1. Currie BJ, McCarthy JS. Scabies. In: Farrar J, editor. Manson's Tropical Diseases. 23rd ed. London: Elsevier; 2013 [chapter 58].
2. Currie BJ, McCarthy JS. Permethrin and ivermectin for scabies. N Engl J Med 2010;362(8):717–25.
3. Roberts LJ, Huffam SE, Walton SF, et al. Crusted scabies: clinical and immunological findings in seventy-eight patients and a review of the literature. J Infect 2005;50(5):375–81.