

# An outbreak of scabies: a forgotten parasitic disease still present in Switzerland

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## Summary

**Introduction:** Scabies, a contagious parasitic dermatosis, has a worldwide distribution but is considered a “disease of the poor” in resource-rich countries. However, it can cause major public health problems following outbreaks in industrialised countries. The following study describes a large outbreak of scabies involving several health care institutions in the canton of Neuchâtel, Switzerland.

**Patients and methods:** After reporting a case of crusted scabies hospitalised for several months (for other comorbidities) in various health care institutions, a “scabies task force” was created in order to detect further cases by contact tracing. Suspected cases were reported to public health authorities, with notification of the health care institutions where cases or exposed patients had been transferred, and information to general practitioners and dermatologists of the entire area (100,000 inhabitants),

**Results:** Three health care institutions (a rehabilitation clinic, a 200-bed acute care hospital, a small hospital with a haemodialysis unit) were involved. Overall, 24 cases of scabies were detected, 12 among inpatients after exposure within the

health care institutions, and 12 among household or other close contacts. 116 health care providers exposed to cases within the health care institutions were investigated with negative results for scabies. After the creation of the task force, no further transmission of scabies was observed. Prolonged misdiagnosis of crusted scabies as well as frequent transfers of cases between various health care institutions facilitated the outbreak. Barrier precautions for health care workers caring for patients with skin lesions even in the absence of a diagnosis of transmissible disease appeared to be efficacious since no transmission to health care workers could be detected.

**Conclusions:** This is the first reported observation of a large scabies outbreak involving health care institutions in Switzerland. Our outbreak demonstrates that it is not an obsolete disease and that a high index of suspicion must be maintained in order to promptly detect difficult cases and to curb potential outbreaks.

**Key words:** scabies outbreak; crusted scabies; nosocomial transmission

## Introduction

Scabies, first described in 1687 [1], is caused by the acarine itch mite *Sarcoptes scabiei*. Faecal mites penetrate the skin and burrow at a rate of 0.5–5 mm per day. These channels are considered pathognomonic of the disease. Clinical symptoms and signs actually result from the host’s immune response, leading to the visible, polymorphous manifestations of papules and vesicles which progress into lesions such as excoriations, nodules and eczema. Indeed, the “incubation period” has been estimated to range from 2 to 6 weeks. Hypersensitivity reactions in case of re-infestation occur more rapidly, within a few days [2–4]. Secondary pyoderma-like infection is frequent, especially in resource-poor countries. The distribution of the lesions in the immunocompetent host is typical, with lesions most frequently seen in the finger webs, on the flexor surfaces of the wrists, in

the axillae, on the buttocks, in the genital area and around nipples of women [4, 5] (figure 1).

Usually, the parasitic burden in the immunocompetent host is low with around 10 mites in these lesions. Person-to-person transmission is limited in these cases, requiring close skin contact (sexual transmission, overcrowding) [6, 7]. By contrast, scabies in immunodeficient individuals (HIV-associated immunodeficiency, elderly people, other T-cell deficiencies) can lead to crusted (“Norwegian”) scabies, first described in Norwegian patients with leprosy, a hyperkeratotic diffuse skin disease similar to psoriasis (figure 2). In these cases, as many as several million mites may be present in these lesions and the exfoliating scales. Crusted scabies is highly contagious and close skin contact is no longer required for transmission which can also occur through fomites on

**Figure 1**

Classic scabies.

**Figure 2**

Crusted scabies.



infected bedding, clothing, chairs or other inanimate objects. Survival of the parasite outside the host is possible for 24 to 36 hours (at 40–80% relative humidity) but lower temperature and higher humidity may prolong survival, thus increasing the likelihood of transmission via fomites [8]. Furthermore, this “atypical” presentation of crusted scabies may lead to delayed diagnosis thus contributing to increased transmission and outbreaks.

Scabies is not an “innocent” disease. It carries a socially negative connotation and is often considered shameful for those affected. This is well illustrated by the French term “gale” which is still used synonymously for “bad” or “vicious” (“méchant comme une gale”). This social prejudice may also contribute to extension of transmission and, eventually, outbreaks by underreporting of the disease.

Here we report a large outbreak of scabies involving three health care institutions in the canton of Neuchâtel which emphasises the need for a high index of clinical suspicion and for an improved public health awareness.

## Materials and methods

### Case definition

A case of classic scabies was defined as a pruritic rash that worsens at night with a typical localisation (sides and webs of the fingers, flexor aspects of wrists, extensor aspects of elbows, axillary folds, skin adjacent to the nipples, periumbilical areas, waist, penis, extensor surface of the knees, buttocks and adjacent thighs, lateral and posterior aspects of feet), with or without findings of mites at scraping. A clinical response to specific scabidicidal medication was also required for definition in the case of clinical diagnosis.

A case of crusted (“Norwegian”) scabies was defined as erythematous lesions and scales, involving scalp, hands and feet up to the entire skin surface, with or without itching. The cases were confirmed by detection of mites at scrapings or in skin biopsy.

All presumptive diagnoses of scabies were confirmed by a dermatologist.

### Active case finding

The reporting of a case of crusted scabies (WR 27) to the acute care hospital prompted the creation of a “scabies task force” in order to identify other cases. Members

of the task force were the head of the department of medicine (RM, director of task force, infectious disease physician), the head of the infection control committee (PE), a public health official of the Canton, a dermatologist (FG), the head of the nurses’ committee and an infection control nurse, the director of employee health committee of the hospital, and a medical resident (LAJ) who coordinated the investigation and the data flow. The task force undertook the following steps: Contact tracing of patients staying at the health care institutions with the patient in the same room or in immediately adjacent rooms during the current and previous hospitalisations by telephone interviews including their primary care physicians; personal and telephone interviews with all family and household contacts of the cases; general information about emergence of scabies cases to the general practitioners and dermatologists of the entire area (100,000 inhabitants); implementation of infection control measures for patients with itchy dermatitis; personal interviews with nurses involved in care of patients with a diagnosis of scabies regarding the emergence of pruritic skin lesions.

## Results

Three health care institutions, *hospitals A, B and C*, were involved in the epidemic. The dynamic of the epidemic is shown in the figure 3.

**Hospital A:** this is a rehabilitation clinic with 36 beds. Patients are mostly suffering from alcohol or opiate dependence as well as from psychiatric disorders. The investigation performed by the task force in *hospital B* showed that the epi-

demic had started in *hospital A* nine months before the task force was created.

The index case (PK 55) was a 44 year-old chronic alcohol and opiate addict who stayed in *hospital A* from October 1998 to September 1999. During his hospitalisation he was diagnosed with crusted scabies and treated with 0.3% lindane emulsion (Jacutin®). The index case lived in a sin-

gle room. Strict isolation procedures, however, were not implemented. Four more patients living in *hospital A* at the same time as the index patient developed itchy skin disease weeks later. Two of these patients shared the same room, and all four patients shared the same bathroom. Three of these 4 patients underwent dermatologic consultation and were diagnosed with scabies and were successfully treated with lindane emulsion. The fourth case (WR 27) was hospitalised from November 1998 to April 1999 in *hospital A* for depression. Itchy dermatitis appeared in January 1999 and dermatological consultation was performed in April 1999. Disseminated eczema and spongiform skin disease in a patient with chronic renal failure were diagnosed and topical corticosteroid treatment was administered. The patient was suffering from end-stage renal disease, COPD and ischaemic heart disease and was eventually transferred for hospitalisation in the acute care *hospital B*.

Two additional patients in *hospital A*, neither of whom had shared the same building as the index patient, developed itchy cutaneous lesions during the stay of the index patient. One of these subjects was diagnosed with scabies and was treated successfully with permethrin lotion (Loxalol®), the other was investigated without a clear diagnosis. Finally, 33 care providers having been in contact with these cases in *hospital A* were investigated for itchy skin lesions with a negative result.

**Hospital B:** this is an acute care, 200-bed hospital. Patient WR 27, after transfer from *hospital A*, was hospitalised for 3 weeks in *hospital B*. He presented with widespread, eczematous and mildly itching skin lesions. He again underwent dermatological consultation with a diagnosis of diffuse eczema and local corticosteroid treatment was continued. He was eventually diagnosed with

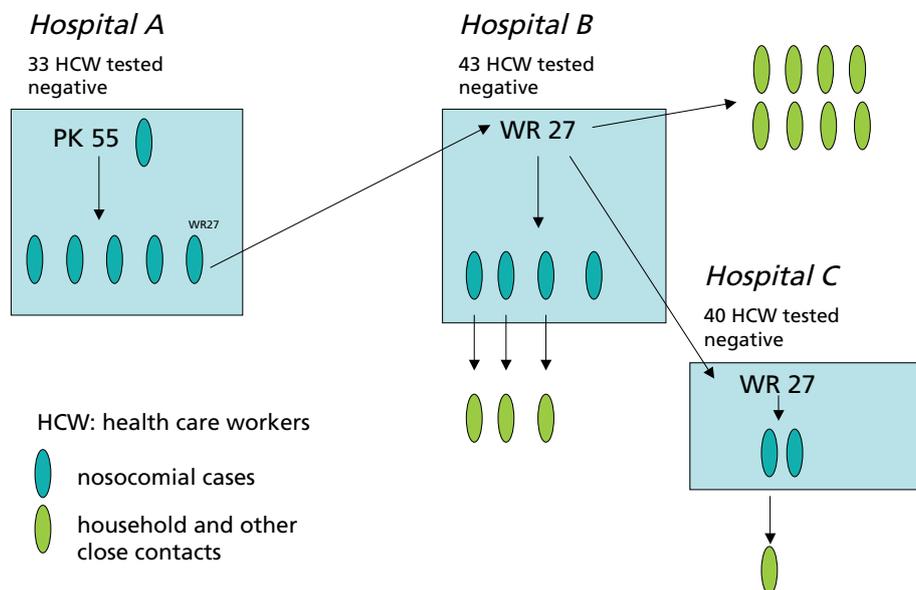
crusted scabies (by the same dermatologist) weeks after transfer to *hospital C*. The clue to diagnosis was that the patient's spouse consulted the same dermatologist with classic scabies. The investigation showed that 5 household members of WR 27 (spouse, children and daughter-in-law, "au pair"), two friends and one spouse were diagnosed with classic scabies. While in *hospital B*, WR 27 transiently shared his room with 4 other patients before being transferred to *hospital C*. These 4 patients stayed in the same room for a minimum of 5 to a maximum of 15 days and they were all subsequently diagnosed with classic scabies. Two of them had their spouse and one a close friend diagnosed with classic scabies. A total of 6 household members or close friends received prophylactic treatment with a single dose of oral ivermectin. Finally, 43 care providers who had been in contact with these patients were investigated for itchy skin lesions with a negative result.

**Hospital C:** this is an acute care, 80-bed hospital with a haemodialysis unit. After the transfer of WR 27 from *hospital B* to *hospital C* for haemodialysis, 2 patients in the haemodialysis unit were subsequently diagnosed with classic scabies. The spouse of one of these patients was also affected. One prophylactic treatment with oral ivermectin was administered to one household member. Forty care providers of *hospital C* were investigated for itchy skin lesions with a negative result.

Overall, there were 24 cases of scabies, 12 of whom had been directly exposed to either PK 55 or WR 27 in the health care institutions, and 12 close contacts from outside these centres who presented with scabies over a 9 month period. One hundred and sixteen health care providers exposed to patients while in *hospital A*, *B* or *C* were investigated and showed no signs or symptoms of scabies.

**Figure 3**

Distribution of scabies cases among the three hospitals. Patients PK 55 and WR 27 suffered from crusted scabies, the other cases had "classic" scabies. Dark circles represent cases with nosocomial transmission of scabies, light circles are cases with household or other close contact. Patient WR 27 was transferred from hospital A to B and C before crusted scabies was eventually diagnosed.



## Discussion

This is the first reported observation of a scabies outbreak involving health care institutions in Switzerland. Scabies is not a notifiable disease in Switzerland. From personal communication with colleagues, we can assume that other scabies outbreaks have been previously observed in Switzerland but were not reported, possibly because of the negative “social connotation” associated with the disease. Scabies is endemic in industrialised countries. In the canton of Neuchâtel (160'000 inhabitants), the number of new scabies cases per year is 15 to 20, the vast majority being young travellers and immigrants. Under certain circumstances, however, scabies can cause epidemics. Indeed, nosocomial outbreaks of scabies have been repeatedly reported from other industrialised countries, more often involving long-term care facilities than acute-care hospitals [9–15]. These reports have emphasised various risk factors for epidemics in industrialised countries which also apply to the outbreak reported here.

First, the role of misdiagnosis has been repeatedly stressed. Diagnosis may be difficult in the most contagious cases of crusted scabies. Indeed, these patients may present with chronic, widespread eczematous lesions that are not immediately recognized as being transmissible disease. Furthermore, these lesions may not be severely itching as in “classic” scabies, due to the poor host immune response in these debilitated hosts [16]. In addition, these patients are frequently treated with topical or systemic steroids which may mask signs and symptoms further increasing the likelihood for transmission. A high index of suspicion for crusted scabies is therefore essential in all debilitated patients with chronic “eczematous” lesions. Strict barrier precautions and isolation is crucial in the hospital setting [12]. Once the diagnosis established, patients should be isolated and treated promptly (see below). In our outbreak, misdiagnosis, failure to isolate for longer periods and delayed treatment were at the origin of the epidemic in all health care institutions.

A second risk factor derives from the fact that patients are increasingly transferred between different institutions. This is well illustrated by patient WR 27 who first acquired scabies in *hospital A*. He was then transferred to *hospital B* owing to worsening health before being moved to *hospital C* for haemodialysis. Unfortunately, crusted scabies remained undiagnosed for several months during these transfers. In our case, the scabies outbreak in *hospital A* was unknown to *hospital B* and then to *hospital C* when WR 27 was being transferred. Prompt notification to public health authorities and infection control committees would have allowed implementation of proper precautions. Thus, we believe that increasing concentration of “specialised care” in single health

care institutions must be accompanied by a strengthened effort to provide an efficacious information network.

Health care workers are at increased risk of acquiring scabies from patients [12]. In a large tertiary acute-care hospital (Johns Hopkins), an AIDS patient with unrecognised crusted scabies who stayed in the hospital for 2 weeks, apparently infected more than hundred health care workers [12]. The presumptive diagnosis was based upon health care workers reporting new pruritic skin rashes. Interviews with exposed health care providers in our outbreak did not reveal any report of pruritic skin lesions several months after exposure to the cases. Health care professionals do not usually under-report professional disease or accidents when their direct responsibility is not involved. We thus believe that under-reporting was unlikely and that routine barrier precautions in our institutions were well implemented by health care workers when dealing with skin lesions.

Treatment of scabies may be topical or by oral route [4]. Permethrin or lindane are available in Switzerland. Permethrin cream (5%) given as a single overnight application seemed to be more efficacious than lindane emulsion according to a meta-analysis [17, 18]. All household and other close contacts should be treated at the same time. If the materials cannot be washed at these temperatures, putting them into hermetically sealed bags for 3 days is recommended. Topical treatment may be messy and exacerbate cutaneous lesions, especially if excoriation or eczema is present. Thus, many physicians and patients prefer highly active oral treatment with ivermectin (200 µg/kg) in a single dose [19]. Repeating the dose after 14 days increased the success rate of oral ivermectin from 70 to 98% [20]. Two instances of ivermectin resistance of *S. scabiei* have been reported in patients with crusted scabies receiving more than 30 doses of ivermectin over several years [21]. Oral ivermectin is clearly the agent of choice in cases of crusted scabies and up to 5–7 doses are recommended, combined with topical permethrin and a softening agent to treat hyperkeratosis and to increase the efficacy of topical agents [22]. At present, ivermectin is approved in Switzerland only for the treatment of nematode infections.

Our observation has several limitations. First, one could question the value of a purely clinical diagnosis of scabies, but our diagnoses were confirmed by specialists which is accepted clinical practice [5]. In our two difficult cases of crusted scabies diagnosis was confirmed by skin scraping and biopsy. Second, our descriptive study did not allow precise determination of risk factors for transmission within health care institutions, such as the extent of physical contacts between cases

and exposed subjects or the role of location of exposed subjects with regard to cases. For our investigation, we had to rely upon published evidence before investigating subjects with presumed exposure. By limiting our investigation to subjects sharing the same and adjacent rooms with regard to cases we made a conservative assumption that may tend to underestimate the risk. Third, molecular characterisation of the parasites to demonstrate a single source would have been helpful but was not available. This would require a high number of parasites to be recovered from affected persons, which is difficult in classic scabies as discussed above. Moreover, appropriate molecular tools are not readily available. In fact, cDNA libraries of *S scabiei* have been available for a few years only but could, in the future, contribute to better characterisation of outbreaks. In one limited study of patients with crusted scabies and repeated infestations, genotyping at hypervariable microsatellite loci by PCR showed that sequential populations of mites were more similar to each other than to mites from other patients [23]. Im-

munodiagnosis using cDNA clones may possibly help in the future for investigation of outbreaks [5].

In conclusion, misdiagnosis as well as poor notification to public health authorities and infection control committees can facilitate the epidemic spread of scabies. Cases must be isolated and immediately treated, and prophylactic treatment should be offered to all patients with close contacts and those having shared common rooms with cases and, in the case of poor barrier precautions, to all exposed health care workers.

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