Sporotrichoid Cutaneous Infection with *Purpureocillium lilacinum* - Formerly Known as *Paecilomyces lilacinus* - in an 83-Year Old Woman

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**Abstract**

*Purpureocillium lilacinum* is a ubiquity appearing mold fungus that can cause cutaneous and subcutaneous infections and is capable of causing sporotrichosis. We present a case of an 83-year old patient with spreading erythematous lesions on the right hand since one year. Under systemic treatment with oral itraconazole and ciclopirox external the lesions were declining rapidly. Despite initial negative diagnostic findings, a rare pathogen such as *P. lilacinum* must be considered in therapy-refractory cutaneous lesions.

**Introduction**

Chronic rashes and skin diseases resistant to topical therapy are frequent in Dermatology. In most cases skin eruptions belong to the eczema group. Nevertheless bacterial and fungal infections should always be considered as differential diagnosis. Sporotrichosis is a so-called injury mycosis that occurs worldwide but has become rare in Europe. It usually presents as a subacute or chronic cutaneous and subcutaneous infection. The classic sporotrichosis is caused by *Sporothrix schenckii* complex. The pathogen usually enters the body via skin lesions. However, other pathogens can also cause sporotrichoid infections: *Purpureocillium lilacinum* (*P. lilacinum* - formerly known as *Paecilomyces lilacinus*) is a ubiquity appearing mold fungus that can cause severe infections, especially in immunocompromised individuals [1]. It can be found in soil, dead parts of plants and foods [2]. We present a therapy-resistant case with a progressive course over more than one year. After repeated mycological examinations an infection with *P. lilacinum* could be identified.

**Case**

An 83-year old female patient presented with erythematous, infiltrated lesions on the right hand since one year. In the beginning a small nodule occurred and spread from the dorsum of her hand till her forearm over last month leaving erythema with crusts. Only arterial hypertension was reported as a secondary disease. Microbiological testing revealed gram-positive cocci. A first skin biopsy resulted in a superinfection on chronic eczema without any signs of a fungal infection. Nevertheless, tinea could not be completely excluded as a possible cause. After one year as several infiltrated erythematous lesions arose in a sporotrichoid distribution a mycological testing was performed by scrapings of scale suspecting a dermatophyte infection. But the results for dermatophytes, candida or molds were negative. Several external therapies like topical steroids (such as mometasone, betamethasone valerate) or anti-infective agents (such as fusidic acid, mupirocin) did not result in any improvement. On the first visit in our department the patient presented several limited
infiltrated erythematous plaques up to 6 cm in diameter with scaling and crusts reaching from the dorsum of the right hand till half of the forearm (Figure 1). The patient’s anamnesis revealed regular gardening and a stitch injury during cutting roses.

We carried out mycological examinations including mycological smear and scrapings and repeated skin biopsy. Our histological findings showed a pseudoepithelomatous hyperplastic squamous epithelium with regenerative changes and local hyperortho- and parakeratosis. In the underlying dermis a chronic-granulating and locally also suppurative-absceding inflammatory reaction was seen. Tissue sections stained with periodic-acid Schiff (PAS) possibly displayed isolated small PAS-positive structures that could correlate with yeast. Molecular pathologic testing for mycobacterium tuberculosis and MOTT (Mycobacteria other than M. tuberculosis) were negative. Finally the mycological culture and the molecular biological diagnostic revealed Paecilomyces lilacinus (now called Purpureocillium lilacinum) (Figures 2 and 3).

We initiated a topical therapy with ciclopirox (day 0) and added itraconazole 100 mg/d (day 33) after receiving the mycological culture and PCR results. The lesions healed quickly under therapy. After eight months of therapy only a small round plaque on distal forearm with scarred healing remained and still \textit{P. lilacinum} was found in the mycological culture.

After 10 months of therapy a clearly improved cutaneous condition with pale atrophic areas and scarred retractions can be observed.

After 10 months the oral therapy with itraconazole was stopped because of gastric problems (day 271). The topical treatment with ciclopirox was continued. At that time a nearly recovered condition with only discrete erythema and a partially scarred healing was seen (Figure 4). The last culture after nearly 12 months was negative and the topical therapy was terminated (day 338).

\section*{Discussion}

\textit{Purpureocillium lilacinum} (\textit{P. lilacinum}) is a saprophyte found in soil, air, and as a common laboratory contaminant [3]. It is used as biological plant protection because it is capable of infesting nematode eggs [4]. In contrast to many other mycological infections \textit{P. lilacinum} is able to sporulate in infected tissue, called “adventitious sporulation” [1,5]. The factor responsible for the bio-control of pests is also responsible for skin infection [6].
mold was already described over a century ago and was named *Paecilomyces lilacinus*. However, it was shown some time ago that it does not belong to the Paecilomyces family but to *Penicillium*. The new genus name *Purpureocillium* was introduced [6].

Because of its flocculent vinaceous to violet colonies on malt agar it was called lilacinus. On Sabouraud dextrose agar brownish colonies can be found [1]. On microscopic evaluation *P. lilacinum* is an anamorphic fungus with septate, non-pigmented hyphae. The conidiophore has delicate bottle-shaped phialides and oval conidia attached in chains [5,7]. This pathogen is often found in biofilms on moist surfaces of water distribution systems. *P. lilacinum* has even been detected in the water pipes of a bone marrow transplantation station. In addition, colonization can occur on plastic materials such as catheters and implants. Infections via contaminated lotions and creams have also been described [6,8]. *P. lilacinum* has keratinophilic characteristics and can rarely cause severe infections, especially in immunocompromised patients [2,8].

In most cases *P. lilacinum* causes cutaneous and subcutaneous infections and onychomycosis but also ocular infections can occur. Usually a breakdown of the skin barrier exists at the entry site. This leads to endophthalmitis or keratitis, especially after ophthalmic surgery like after lens implantation [1]. Arnolder et al described a case with *Paecilomyces lilacinus* fungal keratitis in a contact lens wearer [9]. But also pneumonia, sinusitis or osteomyelitis have been seen [2]. However, atypical localizations due to hematogenic spreading without skin infection such as endocarditis after cardiac valve transplantation or *P. lilacinum*-associated peritonitis due to a peritoneal catheter have also been described [3,7,10]. Particularly at risk are patients after transplantation, with ongoing pharmaceutical immunosuppression or other immune deficiencies. Cancer and diabetes also seem to be predisposing factors [1,8]. Trinh et al. reported a *P. lilacinum* infection on the forearm of a kidney transplant recipient after tattooing [5]. Clinically, solitary or disseminated skin lesions with erythematous maculae or nodi with necrotic center are found in cutaneous infections. The soft tissue can also be affected similar to cellulitis. Unfortunately negative mycological results including PAS staining are found frequently [1,3].

The therapy of a *P. lilacinum* infection is complicated by the high resistances to the most common antifungals such as fluconazole, terbinafine or amphotericin B. Moreover, *P. lilacinum* is resistant to sterilization [1,11]. Depending on the localization, surgical debridement in addition to antifungal therapy is recommended. Data on the efficacy of older azoles (ketocnazole, miconazole, clotrimazole and itraconazole) are diverging. Overall, favorable results were obtained in cutaneous and ocular infections with voriconazole. Skin infections can be successfully treated in almost three quarters of the cases by triazole antifungals, itraconazole and voriconazole. In most cases more than one antifungal had to be used sequentially [1]. Both medications are triazole antifungals. These inhibit the ergosterol synthesis in the cell membrane of fungi. Side effects of itraconazole include nausea, vomiting, hepatic dysfunction, itching and heart failure. Treatment with voriconazole is indicated for severe funga infections and high-risk patients. Under this therapy, in addition to gastrointestinal complaints and liver function disorders, visual disorders, central nervous system dysfunctions and QT time extensions are described. Our patient recovered after one year of combined systemic and topical treatment with daily oral itraconazole and cyclopirox from the sporotrichoid infection with *P. lilacinum*. Due to its pathogenicity and resistance to many antifungal agents, *P. lilacinum* is a great therapeutic challenge. Besides well-known opportunistic pathogens such as Candida, Aspergillus and Cryptococcus, *P. lilacinum* should be kept in mind.

**Conflict of Interest**

Authors have no conflict of interest.

**References**