to decompression sickness include intense physical activity before, during, or after the dive; diving in cold water (vasoconstriction); associated malformations, in particular patent foramen ovale,[2] which may be asymptomatically present in up to 40% of the population (as it was in our patient); abnormal arteriovenous communications or other cardiocirculatory alterations; successive dives of between 10 minutes and 12 hours after the first dive; obesity due to increased solubility of nitrogen in adipose tissue; hypobaric exposure after diving; female sex; and repetitive dives in a short period of time.

There are 2 clinical variants of decompression sickness. The first variant, type 1, is the least serious type and is characterized by cutaneous involvement in the form of a purpuric macular-papular rash (which needs to be distinguished from an allergic reaction), joint pain, or edema. Type 2 is a more severe variant characterized by neurological, respiratory, and/or cardiocirculatory involvement. Rapid diagnosis and treatment is essential as it can considerably reduce the risk of complications and death.[1,3–6]

In more severe cases, basic care consists of treatment in a hyperbaric chamber with delivery of 100% oxygen. Institution of hyperbaric oxygen therapy should not delay the performance of complementary tests (complete blood count, full biochemistry, gasometry, electrocardiogram, chest radiograph).[1,6,7] It may also be necessary to administer fluid therapy with saline solution to treat hypovolemia and antiplatelet therapy to counteract platelet aggregation. Associated complications should also be treated.[1] In mild cases, such as ours, treatment is symptomatic provided that relevant tests have ruled out the involvement of other organs.

In conclusion, cutaneous manifestations of decompression sickness may be the first sign of a series of events associated with high morbidity and mortality, particularly in cases of delayed diagnosis and treatment. The lack of reports in the literature of cutaneous manifestations of decompression sickness should not lead us to underestimate the potential gravity of this situation.

Acknowledgments

We thank Rosa Taberner for her advice and guidance.

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Primary Cutaneous Mucormycosis Due to *Saksenaea vasiformis* in an Immunocompetent Patient☆

Mucormicosis cutánea primaria por *Saksenaea vasiformis* en paciente inmunocompetente

To the Editor:

A 76-year-old man with a history of hypertension, dyslipidemia, and cerebral vascular accident presented with a necrotic lesion and intense inflammation of the surrounding soft tissues on the left forearm. He attributed the lesion to a sting or bite of unknown origin during a hunting outing in the month of July. The ulcer worsened despite treatment with oral doxycycline and intravenous amoxicillin-clavulanic acid, and the patient was administered intravenous broad-spectrum empiric antibiotic therapy with imipenem and amphotericin B (Fig. 1). Hematoxylin-eosin staining of a biopsy specimen showed branching hyphae in the subcutaneous tissue together with necrosis and an intense inflammatory infiltrate. Cultures were negative for aerobic and anaerobic bacteria and mycobacteria. Fungal culture in Sabouraud-dextrose agar permitted the identification of the microorganism responsible for the infection after 48 hours incubation at 30 °C. Microscopic examination with lactophenol cotton blue revealed the growth of a white downy colony, without sporulation, in addition to typical wide, aseptate hyphae with right-angle branching characteristic of Mucorales fungi. The strain was sent to the Mycology Laboratory at Instituto de Salud Carlos III, where it was identified as *Saksenaea vasiformis* with a minimum inhibitory concentration of 2 μg/mL for amphotericin B, > 8 μg/mL for itraconazole and voriconazole, 2 μg/mL for posaconazole; > 16 μg/mL for

☆ Please cite this article as: Coronel-Pérez I, Rodríguez-Rey E, Castilla-Guerra L, Domínguez M. Mucormicosis cutánea primaria por *Saksenaea vasiformis* en paciente inmunocompetente. Actas Dermosifiliogr. 2015;106:516–518.

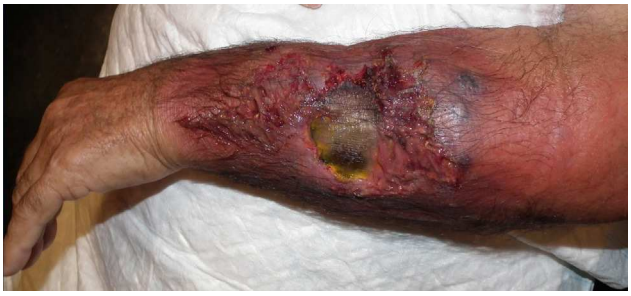

Figure 1 Necrotic 5-cm ulcer surrounded by friable tissue, areas with a lumpy cotton-like appearance, and swelling of the forearm and dorsum of the left hand.

caspofungin, and 0.03 μg/mL for terbinafine. No accumulation of liquid or gas was observed on ultrasound. The ulcer started to heal after treatment with amphotericin B 100 mg daily for 10 days combined with surgical debridement of the wound. Re-epithelization occurred 3 months after topical application of silver sulfadiazine (Fig. 2).

Fungi of the order Mucorales are ubiquitous in nature, and can be found in soil, organic substrates (wood, fruit, excrements, etc.) or as pathogens in animals and plants.[1] Approximately 70% to 80% of Mucorales infections in humans are caused by *Rhizopus*, *Mucor*, or *Lichtheimia* genera and tend to affect immunodepressed individuals. Infections progress fast, do not respond to standard antifungals, and have high morbidity and mortality. The remaining 20% to 30% of cases are caused by the rarer genera *Cunninghamella*, *Rhizomucor*, *Saksenaea*, *Apophysomyces*, *Syncephalastrum*, *Cokeromyces*, and *Actinomucor*. Infections in these cases tend to run a benign course, with exclusive skin and subcutaneous tissue involvement. They are associated with low mortality and respond well to amphotericin B and azoles.[2]

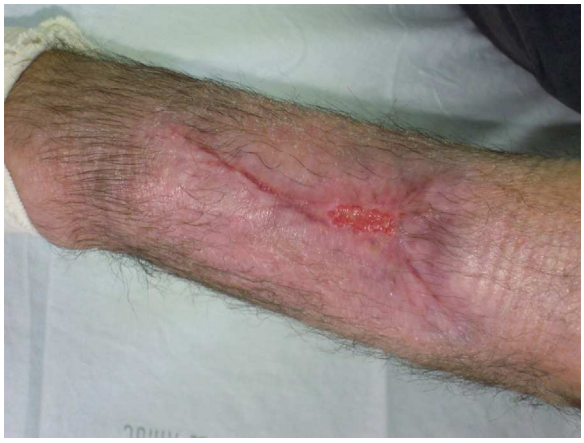

Figure 2 Forearm 3 months later. The ulcer had almost completely re-epithelialized following treatment with amphotericin B, surgical debridement of the wound, and topical application of silver sulfadiazine.

Infections due to *S vasiformis* have been reported worldwide, although most cases have been described in the United States, Central America, Brazil, Europe, India, and Australia.[2] In our search of PubMed, we found 6 cases of *S vasiformis* infection reported for Spain.[3–8] One of these was in the same area as our hospital,[6] and they all occurred in adults aged over 45 years (Table 1). In most of the cases, the fungus had penetrated the skin following an accident involving contact with soil. The cases involving local cutaneous involvement were resolved by administration of specific treatment or amputation of the affected limb.[3,4] The patients with noncutaneous forms of *S vasiformis* infection forms died regardless of their underlying immune status.

Table 1 Published Cases of *Saksenaea vasiformis* Infection in Spain.

Reference	Sex/Age, y	Mechanism of Infection	Presentation	Immune Status	Outcome
Cefai et al.,[3] 1987	Woman/55	Fall with elbow fracture	Gangrenous cellulitis	Not specified	Resolution after amputation
Gómez Merino et al.,[4] 2003	Man/66	Cranioencephalic trauma due to traffic accident	Cellulitis	Immunocompetent	Resolution
García Martínez et al.,[5] 2008	Man/71	Possible inhalation of spores (gardener)	Invasive rhinocerebral mucormycosis	Immunodepressed (diabetes, metastatic gastric adenocarcinoma, corticosteroid therapy)	Death
Domínguez et al.,[6] 2012	Woman/82	Unknown	Disseminated infection	Immunocompetent	Death
Mayayo et al.,[7] 2013	Woman/46	Traffic accident	Necrotizing fasciitis	Immunocompetent	Death
Gómez Camarasa et al.,[8] 2014	Man/58	Farm accident	Cutaneous mucormycosis with subsequent dissemination	Immunodepressed (diabetes)	Death
Present case	Man/76	Bite/sting of unknown origin	Cellulitis	Immunocompetent	Cure

These opportunistic fungi gain entry through injuries or wounds caused by trauma, with most cases involving major trauma, such as traffic accidents, farming accidents (wound contamination) and surgery. There have, however, also been descriptions of infections by Mucorales fungi following minor trauma, including bites and stings. There have been reports of *S vasiformis* infection in a patient pecked by a magpie[9] and stung by a scorpion.[10] The first case was resolved by wound debridement and administration of amphotericin B, although a skin graft was required to repair the wound defect. In the second case, amputation of the affected leg was necessary.

Infections due to *S vasiforms* are probably underdiagnosed as these fungi do not easily produce spores in standard fungal media. A high index of clinical suspicion is therefore necessary to ensure early treatment and avoid amputations and fatal outcomes.

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Photoallergic Contact Dermatitis Due to Chlorpromazine: A Report of 2 Cases☆

Queilitis fotoalérgica de contacto por clorpromazina: descripción de 2 casos

Case 1

The patient was a 64-year-old woman referred to the skin allergy unit of our dermatology department with a 1-year history of chronic pruritic fissured cheilitis on the lower lip (Fig. 1). The physical examination also revealed dermatitis at the outer margin of the right lower eyelid that appeared in outbreaks, as well as cracked and dyshidrotic dermatitis on the tip of the right thumb that had been present for as long as the cheilitis.

Patch testing was performed with the standard series of the Spanish Contact Dermatitis and Skin Allergy Research Group (GEIDAC) and a cosmetics series. The results were positive for cobalt chloride (+++) with no present relevance.

The patient had been taking Largactil drops (chlorpromazine) 5 mg/24 h to treat irritable bowel syndrome for 1 year.

Photopatch testing was performed with chlorpromazine 0.1% in petrolatum (irradiation, 5 J/cm^2). The result for the patch was negative (−), and that of the photopatch was positive (++). Phototesting was not performed. Given the suspicion of contact photoallergy to chlorpromazine, the drug was switched to levopromazine after patch testing with levopromazine at 1% and 0.1% in petrolatum (patch and photopatch negative).

The patient was free of lesions at a follow-up visit a few weeks later. The condition has been controlled for more than 4 years, with no new outbreaks.

Case 2

A 52-year-old woman was referred for possible contact dermatitis on the right lower eyelid that had begun 2 years previously (Fig. 2). She also reported pruritic chronic cheilitis that was sometimes cracked and painful and dated from

☆ Please cite this article as: Esteve-Martínez A, Ninet Zaragoza V, de la Cuadra Oyanguren J, Oliver-Martínez V. Queilitis fotoalérgica de contacto por clorpromazina: descripción de 2 casos. Actas Dermosifiliogr. 2015;106:518–520.