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, 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; hypobarcic 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.1,5-6

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).1,5-7 It may also be necessary to administer fluid therapy with saline 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.

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References


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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 sporation, in addition to typical wide, asceptate 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

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Casposfugin, 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-epithelialization 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

Table 1 Published Cases of Saksenaea vasiformis Infection in Spain.

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

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.1 In our search of PubMed, we found 6 cases of S vasiformis infection reported for Spain.3-6 One of these was in the same area as our hospital, 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.
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 and sting by a scorpion. 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. vasiformis* 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.

References


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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 chelitis 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 chelitis.

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/cm²). 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 chelitis that was sometimes cracked and painful and dated from

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