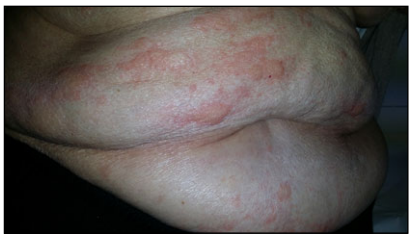

Figure 1 Yellow weals on the abdomen.

alcohol.[1] With a total of ten cases reported in literature by now, we considered relevant to report another case we recently came across.

A 55-year-old woman with 3-year history of cryptogenic cirrhosis was admitted to our Dermatology service because of the appearance of abdominal pruriginous lesions for the past 2 months. Clinical examination revealed yellowish confluent weals affecting her abdomen and flanks (Figs 1 and 2). Blood tests revealed hyperbilirubinemia. She was diagnosed with idiopathic yellow urticaria and was given antihistamines with clearance of the rash and symptoms, without scarring.

The cause of yellow urticaria remains unknown, but it is suggested to be the result of increased blood vessels permeability due to capillary vasodilation, as described in common urticaria, and accumulation of excessive bilirubin into the surrounding dermis, responsible of the yellow colour of the skin.[2] The oedema fluid associated with urticaria in patients with jaundice may appear yellow because of hyperbilirubinemia.[3] Most reported cases were related to chronic hepatic dysfunction (end-stage liver disease, metastatic disease of breast and colon, hepatic cirrhosis associated with alcohol, virus, haemochromatosis).[1,3–8] Only one case was related to cholestasis in a patient with acute hepatic dysfunction due to haemorrhagic shock.[2] Liver dysfunction is characterized by a decrease in the capacity to conjugate bilirubin, with several other problems as dyscrasia. This impairs the normal process of excretion of bilirubin, with consequent bilirubin crystal deposition in tissues.[5] The jaundice may persist for several days, despite a normalized bilirubin, due to its affinity for elastin.[5] The diagnosis is clinical and does not require histopathological examination. Nevertheless, some of the reported cases demonstrated mild dermic oedema and perivascular inflammatory infiltrate composed of lymphocytes, neutrophils and eosinophils.[3,5] Dermic deposition of bilirubin crystals was demonstrated in one case.[3] Treatment is the same of acute urticaria and includes antihistamines and steroids.[1]

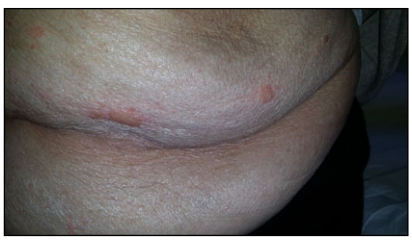

Figure 2 A particular of the lesions affecting the flanks.

Although yellow urticaria is an exceptional event, we can assert that it could be the only scenario in which aetiology of urticaria can be intuited on the basis of the clinical morphology.

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Sporotrichosis masquerading as pyoderma gangrenosum

Editor

Sporotrichosis is a subcutaneous mycosis with a worldwide distribution caused by species of the *Sporothrix schenkii complex*. Since 1998, it has reached epidemic proportions in the city of Rio de Janeiro, Brazil, via transmission from infected cats to humans. The species identified as the primary cause was *Sporothrix brasiliensis*.[1]

The increased incidence led to a rise in the frequency of atypical and severe clinical forms of the disease, such as disseminated and systemic sporotrichosis, that generally occur in immunosuppressed patients.[1,2]

Pyoderma gangrenosum (PG) is an uncommon neutrophilic dermatosis that presents as a painful ulcerative skin disorder. Despite the characteristic clinical appearance of PG, other ulcerating conditions may have a similar aspect causing diagnostic

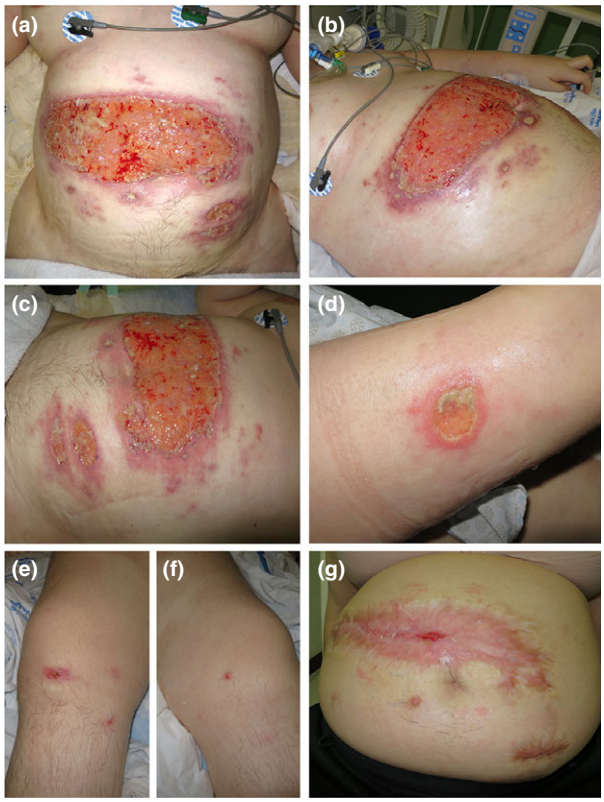

Figure 1 (a) Extensive ulcer on the abdomen with erythematous irregular borders with satellite pustules and ulcers (b) view of the right side (c) close view of the left side (d) round ulcer on the right arm (e–f) erythema and pustules on the knees (g) result at the end of treatment with 6 weeks of liposomal amphotericin B and 12 months of itraconazole.

errors. Some of these alternative diseases may be aggravated by PG-directed treatment.[3,4] Diagnosis of PG rests upon the recognition of consistent clinical and histologic findings and the exclusion of other cutaneous disorders.[3]

We report a case of sporotrichosis masquerading as PG with dissemination after therapy with immunosuppressive drugs and infliximab.

A 39-year-old woman was admitted to the hospital with a painful skin ulcer on her abdomen. She reported that the lesion started after a scratch of a sporotrichosis infected cat two years ago but did not respond completely to the treatment with itraconazole 200 mg/day for nine months. Investigation of systemic diseases including inflammatory bowel disease was negative, except for a positive ASCA. The diagnosis was reviewed to PG. Treatment was started with corticosteroids, immunosuppressive drugs and, lastly, infliximab, causing progressive worsening with severe pain poorly responsive to non-opioid analgesics and morphine, enlargement of the abdominal ulcer, appearance of satellite pustules and ulcers, appearance of an ulcer on the right arm

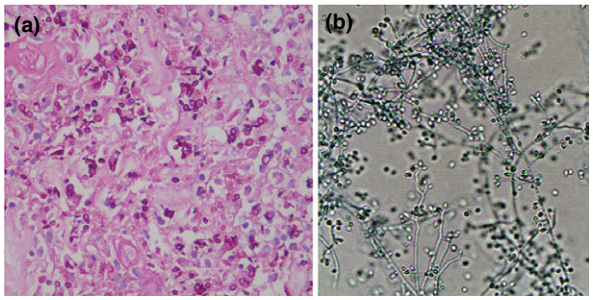

Figure 2 (a) Biopsy from the arm's ulcer showing numerous round, oval and elongated (cigar-shaped yeasts) fungal structures stained in red. Periodic acid–Schiff stain, original magnification ×400. (b) microscopy of fungal colony showing conidia resembling daisy flowers, typical of Sporothrix spp. Lactophenol Cotton Blue, original magnification ×400.

and erythema and pustules on both knees (Fig. 1a–f). The patient evolved with sepsis, pulmonary involvement and respiratory failure that led to ICU admission. Chest CT scan showed extensive diffuse bilateral consolidations. Skin biopsy of the ulcer on the right arm and fungi culture identified *Sporothrix spp.* (Fig. 2). Lung biopsy showed a chronic granulomatous inflammatory infiltrate with focal necrosis. Workup results for microorganisms in lung specimens were negative. These findings led to the diagnosis of disseminated sporotrichosis. Liposomal amphotericin B 400 mg/day was introduced with a good result. It was maintained for 6 weeks, followed by itraconazole 400 mg/day for 12 months, as recommended by the Infectious Diseases Society of America. There was almost complete healing at the end of therapy (Fig. 1g). DNA analysis identified the species as *S. brasiliensis.*

In this patient, despite the epidemiologic history of a sporotrichosis infected cat scratch, the lack of complete response to itraconazole and the ulcerative and severely painful pattern of the lesion led to the misdiagnosis as PG and the consequent immunosupressive therapy that caused the fungus dissemination. The initial lack of response to itraconazole is related with the higher virulence of the species *S. brasiliensis*.[1,5] The technique used for the species identification was T3B PCR fingerprinting. Studies had shown that it is a simple, reliable and rapid methodology.[6]

The misdiagnosis of sporotrichosis as PG is an extremely serious condition, especially due to fungus dissemination caused by immunosuppressive therapy, mainly infliximab in our case. There are five previous case reports of sporotrichosis mimicking PG. Four patients became seriously ill after receiving immunosuppressive therapy and two of them died.[4,7–10] Our patient was also severely compromised with a high risk of fatal outcome.

The epidemic of cat-transmitted sporotrichosis in Rio de Janeiro, Brazil, has brought some unusual clinical manifestations as ulcerative forms that can mimic PG.[1,2,4] Biopsies, cultures and a careful exclusion of ulcerating skin disorders are imperative to

define the diagnosis before starting an immunosuppressive therapy for PG.

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Congenital dermatofibrosarcoma protuberans clinically mimicking a melanocytic naevus treated with serial excisions

Editor

A 9-year-old female patient was referred to our department with the clinical diagnosis of a congenital melanocytic naevus on the back for serial excisions. The child's parents confirmed that the lesion existed since birth. Only little changes in colour and size were observed over the years. The lesion was painless.

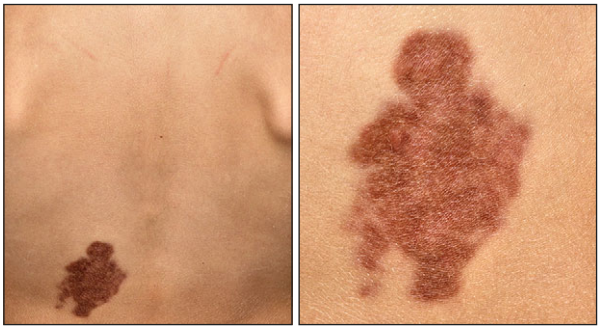

Figure 1 Clinical appearance.

Physical examination revealed a 8 × 4 cm irregular shaped and pigmented macula on the left back (Fig. 1). The histological examination of the first serial excision showed surprisingly a spindle-cell tumour which infiltrated the whole dermis and superficial parts of the subcutaneous fat. Tumour cells were reactive with CD34 but negative for melanocytic markers. The content of small vessels was remarkable. In accordance with the clinically appearance, the epidermis was strongly hyperpigmented (Fig. 2).

Due to histological difficulties, the diagnosis was delayed for several months. Therefore, the second serial excision of the tumour took place four months after the first operation. Finally, the diagnosis of congenital dermatofibrosarcoma protuberans (DFSP) was made. The tumour showed similarities to a recently described vascular variant of DFSP.[4] The COL1A1/PDGFB translocation on chromosomes 17 and 22 was detected in significant number of assessed nuclei, which proved the diagnosis of DFSP (Fig. 2).

Bednar tumour, as a rare pigmented variant of DFSP, could be ruled out due to the absence of pigment-laden dendritic cells within the tumour.

Finally, the last excision with a complete micrographical control of excision margins and a safety margin of 2 cm was performed 7 months after the first serial excision. The wound could be closed primarily with an advancement flap. Up to now, no relapse has occurred.

The paediatric dermatologist is confronted with a variety of predominantly benign skin tumours which rarely require excision. Such common congenital benign skin tumours are, e.g., melanocytic naevi and infantile hemangiomas. However, only 1 to 2 per cent of all skin tumours in children are malignant.[6] The incidence of DFSP in the child's age is not exactly known as there are only case reports or small case series.[2,3,5] In our case, a melanocytic naevus was first clinically diagnosed because the lesion showed homogeneous pigmentation, no elevation and no significant growth, so typical warning signs for a malignant

