

1. Take the tablet with a glass of water (200 mL) first thing in the morning.
2. Not to chew the tablet or allow it to dissolve in his or her mouth.
3. Not to eat, drink, or lie down for at least 30 minutes after taking the tablet.
4. Suspend the medication at the least symptom of dysphagia, pyrosis, or retrosternal pain.

Findings from endoscopic studies in patients with esophagitis caused by alendronate are associated with chemical esophagitis, similar to that caused by acetylsalicylic acid.⁷ The pathophysiological mechanism involved in these cases was direct irritation of the mucosa due to prolonged exposure to the drug. The same mechanism could apply to our patients who allowed tablets to dissolve in their mouths, causing erosion due to prolonged contact of the drug with the oral mucosa.

In view of the increasing use of alendronate sodium we presume this must be quite a common complication despite the paucity of cases cited in the literature. As we are aware that alendronate sodium can cause erosions or ulcerations of the oral mucosa if not ingested properly, we must consider the drug as a possible causal agent in such cases and must question the patient carefully on how they ingested the tablets.^{8,9}

Conflicts of Interest

The authors declare no conflicts of interest..

Correspondence:
Mercedes García Font,
Servicio de Dermatología,
Hospital Mútua de Terrassa,
C/ Castell 25,
08221 Terrassa, Barcelona, Spain
mercedes.garcia.font@telefonica.net

References

1. High WA, Cohen JB, Wetherington W, Cockerell CJ. Superficial gyrate erythema as a cutaneous reaction to alendronate for osteoporosis. *J Am Acad Dermatol.* 2003;48:945-6.
2. Krasagakis K, Kruger-Krasagakis S, Ioannidou D, Tosca A. Chronic erosive and ulcerative oral lesion caused by incorrect administration of alendronate. *J Am Acad Dermatol.* 2004; 50:651-2.
3. Dicko A, Toulemonde A, Hoareau F, Avenel-Audran M, Verret JL. Ulcérations buccales. *Ann Dermatol Venereol.* 2007;134:389-90.
4. Demerjian N, Bolla G, Spreux A. Severe oral ulcerations induced by alendronate. *Clin Rheumatol.* 1999;18:349-50.
5. De Groen PC, Lubbe DF, Hirsch LJ, Daifotis A, Stephenson W, Freedholm D, et al. Esophagitis associated with the use of alendronate. *N Engl J Med.* 1996;335:1016-21.
6. Nightingale SL. Important information regarding alendronate adverse reactions. *JAMA.* 1996;20:1534.
7. Graham DY, Malaty HM, Goodgame R. Primary amino-biphosphonates: a new class of gastrotoxic drugs-comparison of alendronate and aspirin. *Am J Gastroenterol.* 1997;92: 1322-5.
8. Schmutz JL, Barbaud A, Trechot P. Alendronate et ulcérations buccales: d'abord bien interroger le patient. *Ann Dermatol Venereol.* 2005;132:930.
9. García-Bustínduy M, Gantes MA. Corticoides y osteoporosis. *Actas Dermosifiliogr.* 2007;98:526-30.

Primary Cutaneous Cryptococcosis Presenting With a Sporotrichoid Pattern in a Cancer Patient

C. Posada, C. de la Torre, B. González-Sixto, and M.J. Cruces

Servicio de Dermatología, Hospital Provincial de Pontevedra, Complejo Hospitalario de Pontevedra, Pontevedra, Spain

To the Editor:

Cutaneous cryptococcosis is the outcome of infection by *Cryptococcus neoformans*, an opportunistic encapsulated yeast principally found in soil contaminated by bird droppings (mainly pigeons), wood debris, fruit and vegetable waste, and dust.¹⁻³ It usually presents in immunocompromised patients as a secondary disseminated infection, as occurs in 10-15 % of cases.¹⁻⁵ Cutaneous cryptococcosis remains a controversial entity despite

articles on presumed instances of the condition being published since the 1950s. The infection can also occur in immunocompetent patients, in which case prognosis is better.¹ The causal agent in both types of infection is almost always serotype D organisms, as a result of their greater epidermotropism.^{1-4,6}

We present the case of a 66-year-old man referred to us by the oncology department with dermatosis that had been present for 1 month. The patient had received treatment



Figure 1. Subcutaneous erythematous nodular lesions in a sporotrichoid distribution along the forearm.



Figure 2. Residual erythematous lesion showing epithelial healing following treatment with amoxicillin and clavulanic acid.

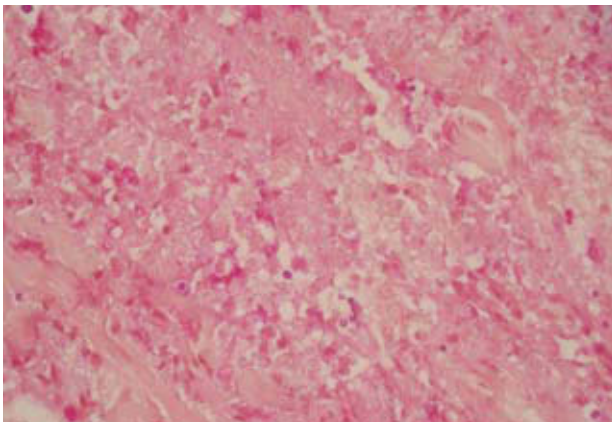


Figure 3. Abundant mycotic eosinophilic round or oval structures of 4-7 µm can be seen in the dermis, surrounded by a characteristic clear halo (hematoxylin-eosin, ×200).

including surgery and radiotherapy for adenocarcinoma of the rectum with hepatic metastases diagnosed 2 years previously. In the course of developing the condition—before the consultation in our dermatology department—a single brain metastasis appeared, and the patient was given a tapering course of phenytoin and dexamethasone, accompanied by whole-brain radiotherapy and stereotactic radiosurgery. The condition started with an erythematous lesion in the palm of the right hand with no preceding trauma that subsequently developed into an ulcer. This was followed by the appearance of subcutaneous, erythematous, and slightly pruritic nodular lesions on the forearm on the same side, in a sporotrichoid distribution (Figure 1). Cultures from the bed of the ulcer revealed the presence of *Acinetobacter lwoffii* and the lesion improved with antibiotics (amoxicillin and clavulanic acid) (Figure 2), although the nodular lesions persisted. A biopsy of the palmar lesion revealed mixed granulomatous and necrotic tissue with abundant encapsulated round mycotic structures of 4-7 µm (Figure 3). A culture of matter aspirated from a nodule confirmed the existence of *C neoformans*, although the serotype was not defined. Blood cultures, and cryptococcal antigen and serological tests, including *Sporothrix schenckii*, were negative. All other results were normal. Imaging studies, including a chest x-ray, abdominal ultrasound, and brain magnetic resonance imaging found nothing unrelated to the tumors and previous surgery. The condition responded in less than a month to oral fluconazole 150 mg/d. The patient died 6 months later from cancer progression with no recurrence of the cryptococcal skin lesions.

Clinical lesions of cutaneous cryptococcosis vary widely with no specific pattern. The sporotrichoid pattern is a very rare presentation. We have only found one case, published by Shuttleworth et al,⁷ that describes symptoms similar to those of our patient: immunodepression, with an initial ulcer on the hand followed by sporotrichoid lesions on the forearm on the same side and a good response to fluconazole. The four main causes of the sporotrichoid pattern are *S schenckii*, *Nocardia brasiliensis*, *Mycobacterium marinum*, and *Leishmania brasiliensis* in endemic areas.⁸ Cryptococcosis is a rare cause, and only a culture can provide a definitive diagnosis. Cutaneous cryptococcosis has been defined in the literature as the identification of *C neoformans* in the biopsy or skin culture in the absence of disseminated disease.^{1,2,9} Other authors have provided clinical and histological criteria (existence of chancriform syndrome and superficial inflammatory infiltrates respectively) for purposes of differential diagnosis.⁵ Neville et al¹ proposed a series of differential factors for the primary form: greater age, lower percentage of immunosuppression, a history of frequent previous trauma and solitary lesions, or lesions confined to the hands or uncovered areas. Our patient was elderly and

immunocompromised as a result of corticotherapy—the most common risk factor in developing cryptococcosis^{3,4}—although he had no history of trauma. The skin lesions, although uncommon due to their sporotrichoid appearance, were confined to uncovered areas. Serotype D organisms were not identified in our case. Our patient showed no systemic symptoms other than those related to his cancer, and the physical examination and complementary studies did not reveal any extracutaneous diseases. This fact, combined with the rapid resolution of the condition following administration of fluconazole, makes us consider cutaneous cryptococcosis as a primary diagnosis.

Treatment of the primary form is not well established at present.² Initial management tends to be medical, or a combination of medical treatment and surgical excision. Fluconazole (200–400 mg/d) is the most common agent prescribed, on average, for 32 days. For maintenance treatment in immunocompromised patients it is recommended that this drug be replaced with a less toxic alternative.¹ The effectiveness of the therapy was remarkable in our patient, with full healing obtained in less than a month despite immunodepression and the absence of surgical intervention.

In conclusion, we present a new case of primary cutaneous cryptococcosis with an uncommon sporotrichoid presentation in a patient with metastatic cancer on high doses of corticosteroid treatment. We also stress the excellent response to fluconazole.

Correspondence:
Celia Posada García,
Servicio de Dermatología,
Hospital Provincial de Pontevedra,
Complejo Hospitalario de Pontevedra,
C/ Loureiro Crespo 2,
36000 Pontevedra, Spain
posada@aedv.es

Acknowledgments

We thank the oncology and microbiology services (Silvia Varela and Victoria Pulían).

Conflicts of Interest

The authors declare no conflicts of interest.

References

1. Neuville S, Dromer F, Morin O, Dupont B, Ronin O, Lortholary O. Primary cutaneous cryptococcosis: a distinct clinical entity. *Clin Infect Dis*. 2003;36:337–47.
2. Revenga F, Paricio JF, Merino FJ, Nebreda T, Ramírez T, Martínez AM. Primary cutaneous cryptococcosis in an immunocompetent host: case report and review of the literature. *Dermatology*. 2002;204:145–9.
3. Hafner C, Linde HJ, Vogt T, Breindl G, Tintelnot K, Koellner K, et al. Primary cutaneous cryptococcosis and secondary antigenemia in a patient with long-term corticosteroid therapy. *Infection*. 2005;33:86–9.
4. Christianson JC, Engber W, Andes D. Primary cutaneous cryptococcosis in immunocompetent and immunocompromised hosts. *Med Mycol*. 2003;41:177–88.
5. Ng WF, Loo KT. Cutaneous cryptococcosis—primary versus secondary disease. Report of two cases with review of literature. *Am J Dermatopathol*. 1993; 15: 372–7.
6. Naka W, Masuda M, Konohana A, Shinoda T, Nishikawa T. Primary cutaneous cryptococcosis and *Cryptococcus neoformans* serotype D. *Clin Exp Dermatol*. 1995;20:221–5.
7. Shuttleworth D, Philpot CM, Knight AG. Cutaneous cryptococcosis: treatment with oral fluconazole. *Br J Dermatol*. 1989;120:683–7.
8. Tobin EH, Jih WW. Sporotrichoid lymphocutaneous infections: etiology, diagnosis and therapy. *Am Fam Physician*. 2001;63:326–32.
9. Noble RC, Fajardo LF. Primary cutaneous cryptococcosis: review and morphologic study. *Am J Clin Pathol*. 1972;57:13–22.

Acrochordons Caused by Friction From Crutch Use

M. Cabanillas,^a B. Monteagudo,^a J. Labandeira,^b C. de las Heras^a and J. Toribio^b

^aServicio de Dermatología, Hospital Básico de la Defensa, Complejo Hospitalario Arquitecto Marcide-Nova Santos, Ferrol, A Coruña, Spain

^bServicio de Dermatología, Complejo Hospitalario Universitario, Santiago de Compostela, A Coruña, Spain.

To the Editor:

Acrochordons, also known as soft fibromas or fibroepithelial polyps, are the most common type of fibrous skin tumor and affect nearly 50% of individuals.¹ From the clinical viewpoint, they can present as solitary or multiple, pedicled, soft, skin-colored, or pigmented lesions. The most frequent locations are the neck, armpits and groin. Despite their high prevalence, there are few references in the specialized literature to their pathogenesis. The possible

role of mechanical friction in their development has been barely mentioned in the literature. We report a case in which this factor played a clear role in their development. The case concerns a 79-year-old man with a personal history of hypertension, peripheral venous failure, fracture of the left hip during childhood with osteolysis of the proximal end of the femur (which, since this time, has required a crutch for walking), and right hip arthroplasty,