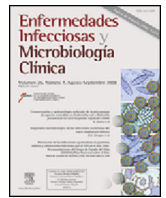




Enfermedades Infecciosas y Microbiología Clínica

www.elsevier.es/eimc



Diagnosis at first sight

Ulcers on psoriatic plaques in a patient with advanced Human Immunodeficiency Virus infection[☆]



Úlceras sobre placas psoriásicas en paciente con infección avanzada por el virus de la inmunodeficiencia humana

Carlos González-Cruz^{a,*}, Juan Espinosa Pereiro^b, Gloria Aparicio Español^a, Vicenç Falcó Ferrer^b

^a Servicio de Dermatología, Hospital Universitari Vall d'Hebron, Barcelona, Spain

^b Servicio de Enfermedades Infecciosas, Hospital Universitari Vall d'Hebron, Barcelona, Spain

Case description

A 49-year-old woman admitted to the infectious disease department with a fever of one week's onset and general state of deterioration. Her history included a 22-year history of HIV infection with poor adherence to treatment, resulting in poor control of her disease. On admission she had a viral load of 135,000 copies of RNA/ml and a CD4 count of 30 cells/ml. She presented complications of repeated oropharyngeal and oesophageal candidiasis. She also had generalised psoriatic plaques of 8 years' onset, with poor adherence to treatment (acitretin and topical corticosteroids), and exacerbated by the advanced stage of HIV infection.

Physical examination revealed generalised large erythematous desquamative plaques affecting the chest and limbs bilaterally and symmetrically (PASI score 25, BSA 50%, PGA 5).

Diagnosis and outcome

The patient was admitted to investigate her fever with no apparent focal point of infection and antiretroviral treatment was restarted with topical corticosteroids for the psoriasis. The initial test results showed neutropenia and chest X-ray was normal.

The patient's general state of health deteriorated 72 h after admission with the onset of confluent, violaceous macules/papules on the psoriasis plaques gradually progressing to necrotic ulcers (Fig. 1). The patient made poor progress, with reduced level of consciousness, hypotension, oliguria and signs of poor peripheral perfusion, in addition to the progression of the skin lesions (Fig. 2). Despite starting intravenous cloxacillin and meropenem, intensive saline therapy and vasoactive drug perfusion, the patient died. Skin



Fig. 1. Necrotic ulcers on psoriatic plaques 72 h after admission.

biopsy revealed superficial and deep necrosis with pronounced acute inflammatory infiltration of the dermis and subcutaneous tissue, with vascular thrombosis (Fig. 3). It was not possible to observe bacteria by Gram staining around the vessels. *Staphylococcus aureus* sensitive to cloxacillin and *Pseudomonas aeruginosa* were isolated in blood cultures and the biopsy culture with no relevant resistance. The PCR of the exudate from the lesions was negative for herpes simplex virus 1 and 2 and the varicella zoster virus.

Commentary

Although the prevalence of psoriasis in HIV-infected patients is similar to that in the general population, in these patients the disease tends to be more severe and worsen as their

DOI of original article: <http://dx.doi.org/10.1016/j.eimc.2016.04.007>

[☆] Please cite this article as: González-Cruz C, Espinosa Pereiro J, Aparicio Español G, Falcó Ferrer V. Úlceras sobre placas psoriásicas en paciente con infección avanzada por el virus de la inmunodeficiencia humana. *Enferm Infecc Microbiol Clin.* 2017;35:605–606.

* Corresponding author.

E-mail address: carlos.gonzalez@vhebron.net (C. González-Cruz).



Fig. 2. Generalised necrotic ulcers on psoriatic plaques 72 h after admission.

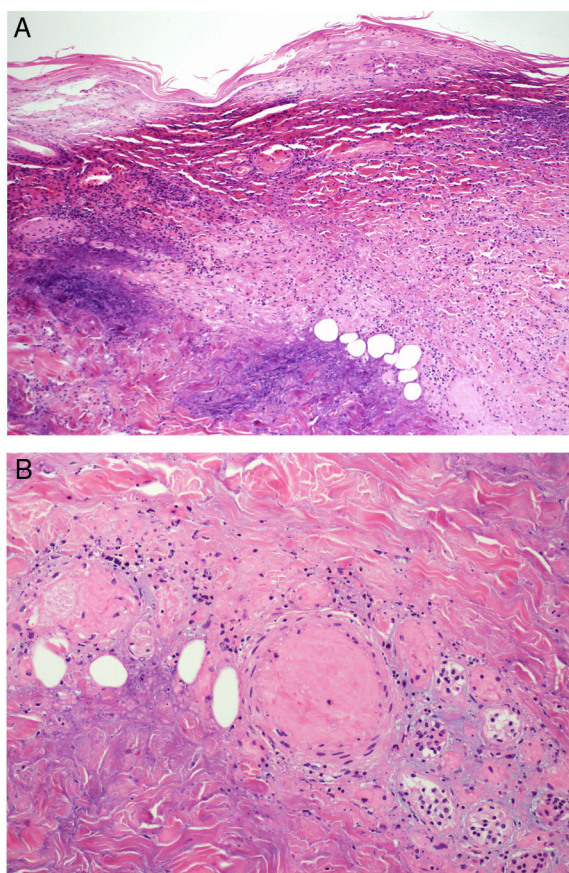


Fig. 3. (A) Skin biopsy of one of the necrotic ulcers showing necrosis of the superficial and deep dermis, with a large inflammatory dermal component. (B) On enlargement of the image, in addition to the necrosis and the polymorphonuclear inflammatory infiltration, we can observe dermal blood vessels with vascular thrombosis, associated with the inflammatory process.

immunosuppression progresses.^{1–3} Furthermore, in the generalised stages, it can form a portal of entry for microorganisms that can lead to sepsis.⁴

Sepsis is still one of the main causes of mortality in the world. The frequency of onset of skin lesions associated with sepsis is not well known, although it is essential to be able to identify them

because they tend to express in early stages, which will enable prompt diagnosis and treatment. One of the pathophysiological mechanisms that would explain the onset of these skin lesions is direct invasion of the vascular wall by the microorganism responsible for the sepsis, as occurs in ecthyma gangrenosum.⁵ This disorder is essentially caused by *P. aeruginosa*, although cases caused by different microorganisms have been described including *S. aureus*. Necrotising vasculitis occurs with vascular thrombosis which triggers a cutaneous infarction that progresses to ulcerated lesions, which can be single or multiple and can appear on any part of the body. It is more common in immunosuppressed patients with intense and prolonged neutropenia.^{5–8}

We present the case in this article of a patient infected by HIV in an advanced state of immunosuppression, neutropenia and with poorly controlled psoriasis. Her generalised psoriasis could have formed the portal of entry for *S. aureus*, the most common skin bacteria in HIV infection, and *P. aeruginosa*.^{9,10} The symptoms, histology, blood cultures and the biopsy culture led us to the conclusion that our patient's ulcerated lesions were probably an ecthyma gangrenosum due to a septic vascular disease caused by *P. aeruginosa*. However, we cannot discount the aetiological role played by *S. aureus*, less commonly involved in the pathogenesis of this disorder. This clinical case illustrates the need for strict therapeutic control of both the HIV infection and the psoriasis of patients in whom both diseases coexist, considering the serious systemic complications that can be triggered.

Funding

The authors declare that they have received no source of funding.

Conflict of interest

The authors have no conflicts of interest to declare.

Acknowledgements

Our thanks go to Berta Ferrer and Rosa Cristòfol for their collaboration.

References

- Parker SR. The skin and HIV: no superficial matter. *Top Antivir Med.* 2014;22:680–4.
- Morar N, Willis-Owen SA, Maurer T, Bunker CB. HIV-associated psoriasis: pathogenesis, clinical features and management. *Lancet Infect Dis.* 2010;10:470–8.
- Patel RV, Weinberg JM. Psoriasis in the patient with human immunodeficiency virus. Part 1. Review of pathogenesis. *Cutis.* 2008;82:117–22.
- Ferrándiz C. Dermatitis eritematoescamosas (I). Psoriasis. Eritrodermias. In: Bielsa I, Ferrándiz C, Ferrándiz-Pulido C, Ferrándiz-Pulido L, Fonseca E, Moreno-Ramírez D, et al., editors. *Dermatología Clínica.* 4.ª edición España: Elsevier; 2014.
- Delgado-Jiménez Y, Fraga J, Fernández-Herrera J, García-Díez A. Vasculopatía séptica. *Actas Dermosifiliogr.* 2007;98 Suppl. 1:S22–8.
- Buffière-Morgado A, Demongeot C, Battistella M, Taieb F, Viguier M, Rybojad M, et al. Ecthyma gangrenosum associated with infection involving a methicillin-sensitive, Pantone-Valentine-negative strain of *Staphylococcus aureus*. *Ann Dermatol Venereol.* 2015;142:262–5.
- Meynard JL, Barbut F, Guiguet M, Batisse D, Lalande V, Lesage D, et al. *Pseudomonas aeruginosa* infection in human immunodeficiency virus infected patients. *J Infect.* 1999;38:176–81.
- Vaiman M, Lazarovitch T, Heller L, Lotan G. Ecthyma gangrenosum and ecthyma-like lesions: review article. *Eur J Clin Microbiol Infect Dis.* 2015;34:633–9.
- Human retroviral infections and mucocutaneous manifestations of HIV/AIDS disease. Wolff K, Johnson RA, editors. *Fitzpatrick's color atlas and synopsis of clinical dermatology.* 6.ª edición New York: McGraw-Hill; 2009.
- Vidal F, Mensa J, Martínez JA, Almela M, Marco F, Gatell JM, et al. *Pseudomonas aeruginosa* bacteremia in patients infected with human immunodeficiency virus type 1. *Eur J Clin Microbiol Infect Dis.* 1999;18:473–7.