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Cutaneous Epstein–Barr virus-associated lymphoproliferative polymorphic disease – AIDS presenting manifestation

Editor

The Epstein–Barr virus (EBV)-associated polymorphic lymphoid proliferation (PLP) in post-transplant recipients is well documented. A similar entity can present in immunodepressed non-transplant patients, associated with congenital or acquired immunodepression.[1] PLP in HIV patients can manifest as extranodal disease – lung, parotid gland, perineum and skin.[2]

We report the case of a 34-year-old Caucasian female referred to our consultation due to a pruriginous plaque on her cervical region, with a 6-month evolution and worsening on the last one and a half months. Recently, the lesion had become painful. She had been medicated before with flucloxaciline and drainage was performed, without improvement.

Her past medical history was irrelevant except for a reactive depression and obstructive sleep apnoea syndrome, medicated with fluoxetine. She had no history of travelling or living abroad.

On examination, the patient had general good condition, with a 6 cm roundish infiltrated plaque on the posterior cervical area, hard, with crust and little oozing (Fig. 1a,b). Another lesion was present on the left anterior axillary line, at the thoracic level, with 3 cm of diameter, with no infiltration and a small crust.

Analytic screening was relevant for a positive HIV antibody by chemiluminescence confirmed by immunoblotting as HIV-1 positive, HIV-2 negative, with a viral load of 962756 copies/mL.

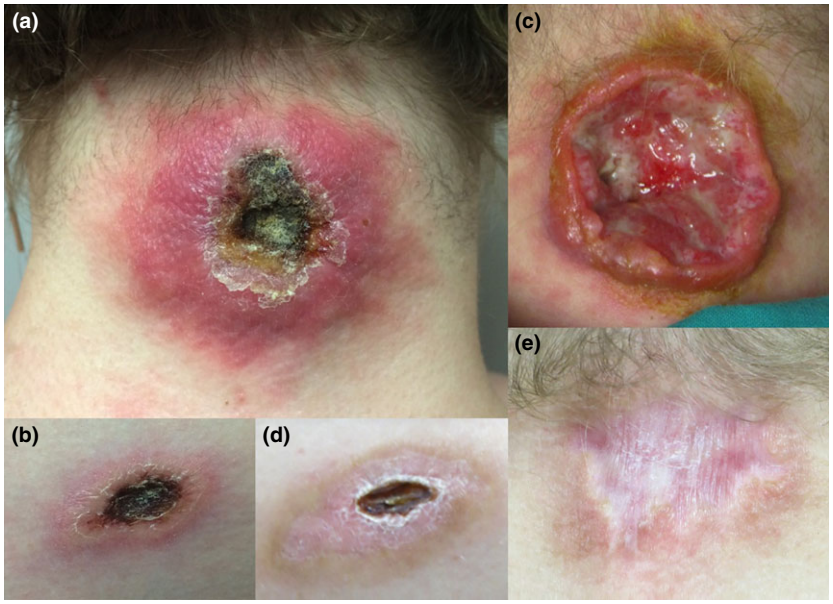

Figure 1 At diagnosis (a) Cervical lesion (b) Left thoracic lesion (c) Enlargement of the cervical lesion – 1 month after and with 1 week of treatment. After 2 months of HAART – (d) Left thoracic lesion (e) Cervical scar.

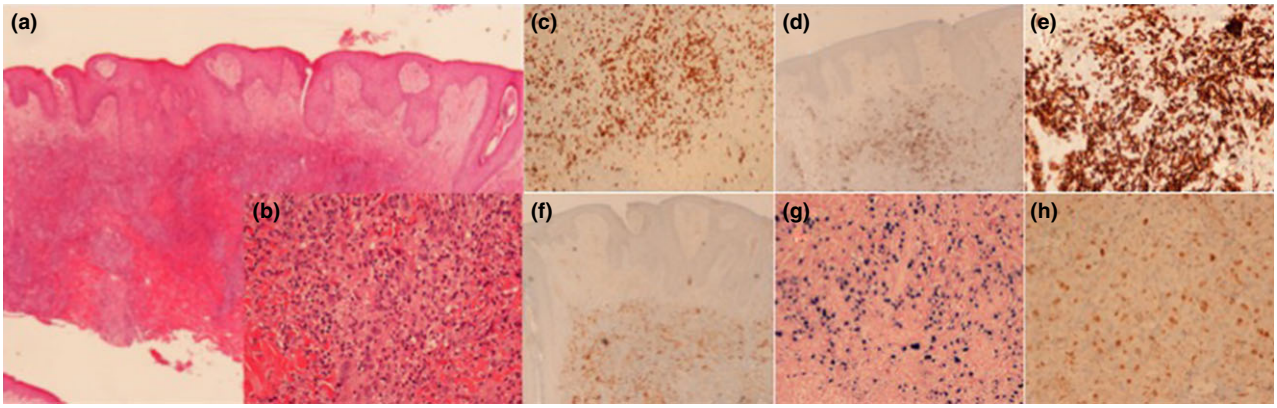

Figure 2 (a) HE, ×40 – Reticular dermis difuse infiltrate with relatively spared epidermis and dermis (b) HE, ×200 – Polymorphic infiltrate with small and intermediate lymphoid cells, large Reed-Sternberg-like cells (inset), histiocytes, eosinophils and neutrophils (c) CD3 (×200) (d) CD15 (×100) (e) CD20 (×200) (f) CD30 (×100) (g) EBER (×200) (h) PAX5 (×200).

The CD4 + lymphocyte count was of 35 cells/μL. Hepatic cytolysis was mild with AST 49 U/L and ALT 47 U/L. IgG, IgA and beta-2-microglobulin were raised (24 000 mg/dL, 614 mg/dL and 3.5 mg/dL respectively). C- reactive protein was 0.55 mg/dL. TPHA and RPR were non-reactive.

Mycobacterial and fungal cultures were negative.

Cutaneous biopsy showed a CD20 + /PAX5 + large B-cell proliferation with EBER e LMP-1 expression on a polymorphic background (Fig. 2).

The diagnosis of EBV-associated PLP in an AIDS patient was made.

On CT pericentimetric laterocervical adenopathies were visualized.

Bone marrow biopsy excluded marrow involvement.

Therapy was initiated with itraconazol for oral candidiasis, and 2 weeks later with emtricitabine, tenofovir, darunavir/r, cotrimoxazole and azithromycin.

The cervical lesion worsened developing a deep central crater (Fig. 1c), just stabilizing after 4 weeks of highly active antiretroviral therapy (HAART).

After 10 weeks of therapy, the lesion involuted greatly (Fig. 1d,e) and due to the good response it was decided to withhold chemotherapy and to keep the patient under surveillance and HAART. The thoracic lesion did not change. In the mean time, immune reconstitution was achieved with CD4 + count of 353 cells/μL, and a reduction of viral load to 453 copies/mL. An acneiform eruption of the face and upper trunk developed, suggestive of eosinophilic folliculitis – biopsy confirmed. It was treated with doxycycline 100 mg, fexofenadine 180 mg and hydrocortisone cream od.

As in our case, PLP is usually associated with profound immunosuppression[3] and it is an AIDS stage defining disease.

To our knowledge, there are just two reported cases of skin manifestation of EBV PLP[1,2] but without clinical iconographic documentation.

As there are less than 20 cases described, its behaviour and treatment remains still largely unknown. It seems to fill in the lower end spectrum of lymphoproliferative entities in HIV patients, without an oncogene or tumour suppressor gene mutations identified. Nonetheless, many cases are monoclonal based and transformation to diffuse large B-cell lymphoma has been described.[2]

As systemic involvement was not identified and due to the involution observed, immunomodulation was chosen over chemotherapy,[4] regarding other cases also controlled with HAART alone.[3,5]

Although HIV lymphoproliferative disorders in HIV patients tend to be less frequent after the HAART era, a high index of suspicion is required to figure it, as it can be the inaugural HIV manifestation with important individual and epidemiological consequences.

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