

An unusual presentation of genital herpes in a patient affected by lichen sclerosus et atrophicus: A case report and a combined treatment proposal

Dear Editor.

Herpes simplex virus (HSV) infection is a common cause of genital ulcers (Looker, Garnett, & Schmid, 2008). Clinical presentation includes vesicles, erosions and ulcers, usually bilaterally with local adenitis.

An overlapping common disease in elderly women is lichen sclerosus et atrophicus (LSA), characterized by white atrophic areas with erosions, painful ulcers and permanent scar tissue on genital areas (Resende, Conforti, Giuffrida, de Barros, & Zalaudek, 2018). In this context, herpes genital lesions may be difficult to identify and can be confused with other diseases (Fergus et al., 2020).

An 82-year-old woman came to our clinic complained of a 3-years history of burning sensation and painful ulcers over the vulvar area. On clinical examination white atrophic patches on the labia majora were observed, with two 0.5 cm nonhealing ulcers on the left labia majora and 1 cm nonhealing ulcer on the right labia majora (Figure 1). Furthermore, in the vaginal introitus, a diffuse mucosal inflammation with multiple erosions was present. Nonmalodorous cervical discharge and inguinal lymphadenopathy were noted. No urinary or systemic symptoms nor history of oral ulcers were reported. The genital symptoms started 3 years earlier, after a hysteronannessiectomy for metrorrhagia and pyometra. A long history of ineffective therapies (topical steroids, metronidazole/clotrimazole cream, tacrolimus cream, and oral antibiotics) for LSA and bacterial vaginosis was reported. Therefore, a biopsy was performed. Histopathology showed areas of LSA and ulcerated vulvar wall with fibrohematic areas, neutrophils, eosinophils and plasma cells, parakeratosis and signs of herpetic infection. Vaginal swab detected HSV-2 DNA.

Genital diseases can produce painful erosions and/or ulcers, but diagnosis based only on physical examination sometimes is inaccurate, especially if clinical features are atypical or the patient's history does not give the right clues. If there are suspicious lesions resistant to first-line therapy, a biopsy should be performed.

Clinical manifestations of our patient included symptoms and signs of LSA and herpes genitalis (HG).

The goal of therapy was to control both diseases, considering that topical corticosteroids alone can lead to reactivation of HG and delay the healing of HSV lesions. Therefore, it is important to associate a systemic antiviral therapy (Grinde, 2003). We treated the patient with acyclovir 400 mg thrice daily for 7 days, as proposed by Patel et al. (2017),

and in addition clobetasol propionate 0.05% cream for 10 days. Then, the patient was treated with acyclovir 400 mg twice daily and clobetasol propionate 0.05% cream for 1 month. This combined treatment allowed to achieve healing after 30 days (Figure 2).

In conclusion, for patients affected by concomitant LSA and GH, we recommend a combination of therapies of the coexisting diseases: first, a treatment of LSA with clobetasol propionate 0.05% cream one-to-two times daily and, second, the control of viral reactivation with acyclovir 400 mg twice/thrice daily for 1 month. In our case, a good response with clinical remission of all symptoms was observed after 1 month of treatment. Topical corticosteroids can be applied for two



FIGURE 1 White atrophic patches on the labia majora with two 0.5 cm nonhealing ulcers on the left labia majora and 1 cm nonhealing ulcer on the right labia majora (black arrows)



FIGURE 2 Restitutio ad integrum after 1-month therapy with clobetasol propionate 0.05% cream one-to-two times daily and acyclovir 400 mg twice/thrice daily

additional months (Resende et al., 2018), while the suppressive antiviral therapy can be prolonged for a maximum of 6 months; renal and hepatic function should be monitored monthly (Sauerbrei, 2016).

CONFLICT OF INTEREST

The authors declare no potential conflict of interest.

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