Sweet’s syndrome on surgical scar

Síndrome de Sweet em cicatriz cirúrgica

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Abstract: Sweet’s syndrome is a rare dermatosis, characterized by acute tender erythematous plaques or nodules. The case of a 55-year old woman with a skin lesion compatible with Sweet’s syndrome surrounding a surgical scar on the face, after removing an actinic keratosis lesion, and using dipyrone is reported. The reported case points to the possibility of the occurrence of Köebner phenomenon in Sweet’s syndrome, probably triggered by the use of dipyrone.

Keywords: Dipyrone; Surgery; Sweet’s syndrome; Wounds and injuries

INTRODUCTION

Sweet’s syndrome, also called febrile acute neutrophilic dermatosis, was first reported by Robert Sweet in 1964. It is characterized by the acute appearance of erythematous-edematous plaques or nodules, which may be covered by blisters and ulcers. The lesions occur mostly in the trunk, limbs, face and neck. Although occurring in the patients described by Sweet, fever and peripheral leukocytosis are not common findings. Other extra-cutaneous signs include arthralgia, arthritis, conjunctivitis, episcleritis, oral and genital ulcers, pulmonary infiltrate, hepatitis, proteinuria and renal failure. On histopathology, dermal edema and diffuse neutrophilic infiltrate, with no vasculitis, are found. This disorder has been reported in the literature associated to several infectious, inflammatory and neoplastic diseases, and to the use of medications. A case of Sweet’s syndrome associated to the use of dipyrone and showing Köebner phenomenon due to surgical trauma is presented.

CASE REPORT

A 55 year-old woman had a history of a tender lesion on the face for three days. A 6x4 cm, edematous and erythematous plaque surrounding a surgical...
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Scar in the right malar region (Figure 1) was seen. Although she had used cephalexin for seven days, it did not disappear. In the two weeks prior, she had undergone the removal of actinic keratosis in that site, and had used dipyrone for analgesia, despite having previous occurrence of Sweet’s syndrome, 10 years prior, triggered by the use of the same medication. Conjunctivitis was the only constitutional symptom. Complete blood count, erythrocyte sedimentation rate, liver function tests, electrolytes, creatinine, urea and urine analysis were all within normal limits. Histopathological examination of the lesion showed neutrophilic inflammatory infiltrate, vasodilatation and dermal edema resembling the findings of Sweet’s syndrome. The patient discontinued dipyrone and cephalexin, when oral steroids were started. The skin lesions began to subside 48 hours after this treatment was instituted (Figure 2), and the patient has been asymptomatic ever since.

**DISCUSSION**

In 1986, Su and Liu defined the diagnostic criteria for Sweet’s syndrome. They determined that two major and two minor criteria ought to be present for its diagnosis. The two major criteria are the abrupt appearance of tender plaques or nodules and the neutrophilic dermal infiltrate with no signs of vasculitis. The minor criteria are fever or preceding infection; constitutional signs including fever, arthralgia, conjunctivitis or malignancy; leukocytosis and good response to the use of steroids, but not to antibiotics. In 1986, Von den Driesh et al. suggested adding increased erythrocyte sedimentation rate to the list of minor criteria. The patient described here meets the diagnostic criteria. The clinical and histopathological features are compatible with the major criteria, and conjunctivitis and the excellent therapeutic response to steroids are compatible with the minor criteria.

Sweet’s syndrome is a likely hypersensitivity reaction to viral, bacterial, tumor antigens or to external stimuli. Since its original description, it has been associated to infections; vaccines; malignancies, as well as to auto-immune and inflammatory diseases, such as ulcerative colitis and sarcoidosis. Among malignancies, the most commonly associated are the hematological ones, especially acute myeloid leukemia. Some cases triggered by hydralazine, sulphamethoxazole-trimethoprin, celecoxib, oral contraceptives and granulocyte colony stimulating factor have been reported.

In 1975, Guanawardena et al. observed that Sweet’s syndrome may display the Köebner phenomenon, with reports of lesions occurring in photoexposed areas, lesions triggered by chemical irritation and by mechanical trauma (in venopuncture and pressure areas). In 1983, Hams and Saurat suggested that lesions in vaccination and wound sites occurred due to an abnormal neutrophilic response to traumatic stimuli. In the case presented above, a surgical scar was the site of the lesion, corroborating the findings in the literature.

The patient described had no associated malignancy or para-inflammatory condition, but it is likely that the ingested dipyrone caused the lesion to appear, installed over a surgical scar. The patient reported a previous occurrence triggered by medica-
tion containing dipyrone, which improved when dipyrone was stopped and systemic steroids started. Taking into account the many different presentations of this condition, one may conclude that a wide spectrum of endogenous and exogenous influences may trigger this still unknown pathological process. The authors stress that the Köelner phenomenon may occur in Sweet’s syndrome, as well as that medications may not be disregarded as causing this dermatosis.

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REFERENCES