Wolf's isotopic response, presenting as lichen planus*

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Abstract: The term "Wolf's isotopic response" describes the occurrence of a new skin disorder at the site of another unrelated and already healed skin disease. In most cases, herpes zoster is the inicial disease. Different disorders may develop on the same site, most commonly granulomatous and lichenoid reactions, infiltration of hematologic diseases, skin tumors and infections. There are few related cases of lichen planus presenting as isotopic response. We report a case of a 74 year-old woman, with multiple itchy, rose-colored and shiny papules that developed at site of previously healed herpes zoster, on the right arm and shoulder. The pathogenesis of this phenomenon is still unknown and further studies are needed.
Keywords: Herpes Zoster; Lichen planus; Lichenoid eruptions; Lichens

INTRODUCTION

Wolf's isotopic response occurs when a dermatosis appears in a region previously affected by another unrelated and already healed skin disease.[1] The term was created by Wolf et al in 1995, but it had already been described by Wyburn-Mason, an English neurologist who in 1955 reported 26 cases of patients with malignant tumors, developed in the same site of a previous herpes zoster or herpes simplex eruption.[2,3] Primary lesion is usually a herpes zoster, followed then by secondary condition - most commonly a neoplasm or granuloma annulare.[1] It's important to differentiate the isotopic phenomenon from the isomorphic response (or Köebner phenomenon), which defines the appearance of lesions of the same disease after traumatic provocation.

Wolf's isotopic response is an uncommon phenomenon in clinical practice and even in the literature, so its recognition is very important. We report a case of this phenomenon.

CASE REPORT

Female patient, 74 years old, with pruritic skin lesions on the right shoulder and arm for a week (Figures 1 and 2). She states that the eruption occurs exactly in the area where she had been affected by herpes zoster for 3 weeks, which was diagnosed and treated with resolution of the lesions. Physical examination revealed slightly elevated papules, rose-colored and shiny, with zosteriform distribution following the thoraco-brachial dermatome. Clinical hypothesis of lichen planus was made and a biopsy was performed (Figures 3 and 4). Histological analysis showed epidermis with hyperkeratosis, mild increase in the granular layer, acanthosis areas interspersed with atrophic areas, foci of degeneration in the basal layer, subepidermal cleft (Max Joseph space), mild lymphohistiocytic infiltrate in the superficial dermis. These findings confirmed the diagnosis of Wolf's isotopic response.

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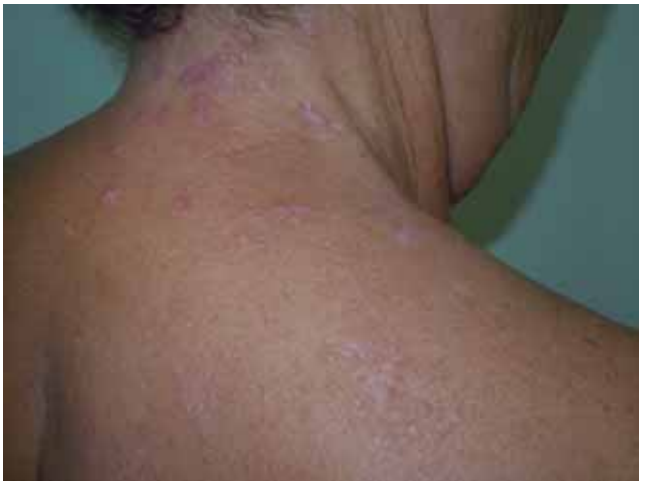

Figure 1: Slightly elevated papules, rose-colored and shiny, with zosteriform distribution

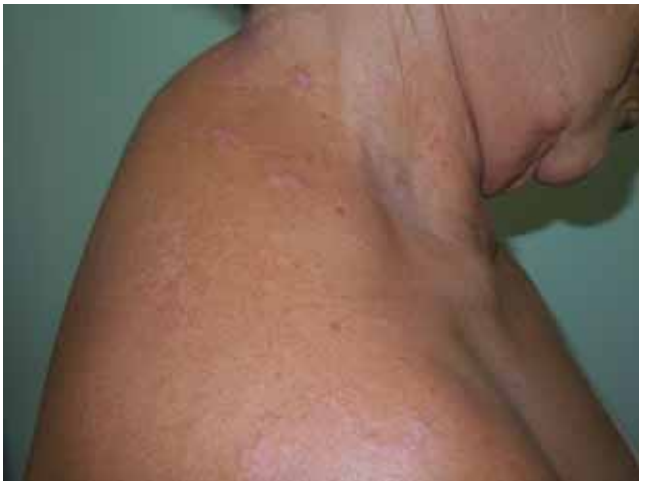

Figure 2: Papules following the thoraco-brachial dermatome, area previously affected by herpes zoster

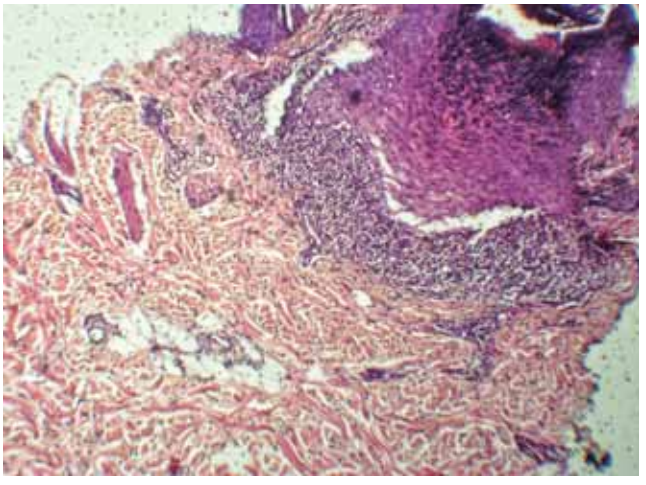

Figure 3: Epidermis with hyperkeratosis, mild increase in the granular layer, foci of degeneration in the basal layer, subepidermal cleft (Max Joseph space) and lymphohistiocytic infiltrate in the superficial dermis

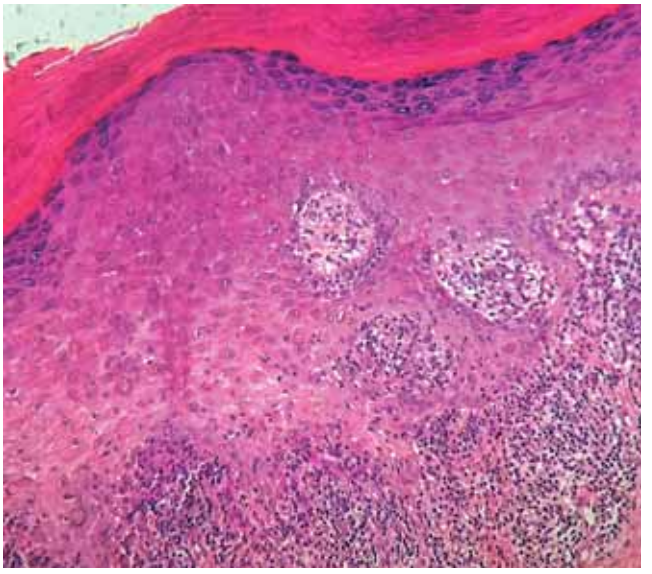

Figure 4: Presence of hyperkeratosis, hypergranulosis, irregular acantosis and lymphocytic infiltrate

DISCUSSION

There are about 200 reported cases of Wolf's isotopic response. The most commonly found initial dermatoses is herpes zoster, but the condition has also been described after herpes simplex, varicella and thrombophlebitis.[4] The most reported secondary diseases are granuloma annulare, malignancies (breast cancer, squamous cell and basal cell carcinoma), leukemic/ lymphomatous infiltration, inflammatory reactions (lichen planus), contact dermatitis, psoriasis and infections (mycosis, molluscum contagiosum, verruca vulgaris, furunculosis).[5,6] There are also reports of cases of perforating collagenosis, comedones and acneiform eruptions, morphea, graft versus host disease (GVHD), eosinophilic dermatoses, Kaposi's sarcoma, angiosarcoma, erythema annulare centrifugum, subcorneal pustular dermatoses and chronic hives.[3,7,8]

A literature review analyzed 176 cases of isotopic response and found herpes zoster (89%) and herpes simplex (11%) as the most common initial diseases.[3] Regarding the secondary diseases, the largest group was granulomatous reactions (31%), mainly granuloma annulare (18%). The study also found 36 cases of malignant tumors (20%), 15 cases of infectious diseases (9%), 10 cases of lymphomas (6%), 9 cases of leukemic infiltration (5%), 9 cases of lichen planus (5 %), 6 cases of morphea (3%) and 4 cases of perforating dermatosis (2%) (Table 1). Jaka-Moreno et al[4] reported in a series of 9 cases the occurrence of 4 granulomatous dermatitis, 2 lichenoid dermatitis, 2 infiltration by B cell chronic lymphocytic leukemia and 1 infiltration by non-Hodgkin's lymphoma.

Interval between initial infection and the development of secondary disease varies widely, from days

Table 1: Key secondary skin diseases and reported frequencies

Granulomatous reactions	32%
Malignant tumors	20%
Infections (viral, bacterial and fungal)	9%
Lymphomas	6%
Leukemic infiltration	5%
Lichen planus	5%
Morphea	3%
Perforating collagenosis	2%

to years.[1,8] Jaka-Moreno et al found intervals from 15 days to 7 months.

The pathogenesis leading to the development of secondary disease is not yet fully understood. It has been suggested that viral particles remaining in the tissues would be directly responsible for the occurrence of secondary disease. Findings of isolated viral DNA in secondary lesions corroborate this hypothesis.[9] However, the presence of viral DNA is rare and has only been documented in cases where there was a short interval between the two diseases (less than 4 weeks).[10] Moreover, it was showed no detectable reduction in the occurrence of the response with the use of systemic antivirals for the treatment of herpetic infections.[4]

Some authors postulate that vascular and immunological changes occur after viral infection and would make the skin more susceptible to a second disease in the same area.[1] The herpesviruses are known for their ability to destroy A-delta and C-nerve fibers in the mid and deep dermis. Damage to peripheral sensory nerves would alter the expression profile of neuropeptides and neurotransmitters (substance P, calcitonin gene-related peptide, neuropeptide Y) of these nerves. These neuropeptides mediate immune functions such as degranulation of mast cells and release of pro-inflammatory cytokines. The neuroimmunologic reaction would create an invisible scar of immune dysregulation confined to the area of initial infection, which has been called "locus minorus resistentiae", where apparently healthy skin is more susceptible to subsequent diseases.[6] In these sites of immune alterations, the hyperreactivity would favor the inflammatory processes as lichenoid and granulomatous dermatitis, and local immunosuppression would lead to tumor infiltration and infections.[1] It has also been suggested that the nerve damage may lead to abnormal angiogenesis, causing development of vascular tumors.[4]

It's still unknown how the herpesvirus infection leads to such a wide variety of secondary diseases and why most people do not present the isotopic response. It is postulated a contribution of genetic, environmental, nutritional and other unknown factors.[6] ❑

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