CASE REPORT

Grover's disease (transient acantholytic dermatosis): A clinical case report

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SUMMARY

Grover's disease is a transient acantholytic dermatosis with an unknown etiology; however, various triggering and exacerbating factors have been described. This condition primarily affects men over the age of 40. Clinically, it presents with various manifestations ranging from erythematous papules to plaques with thick scaling, accompanied by fissures and erosions. The histopathological finding of acantholysis is key to the diagnosis. Although the disease course is usually benign and self-limited, in some cases, it may be refractory, requiring treatment to be tailored to the extent of involvement.

INTRODUCTION

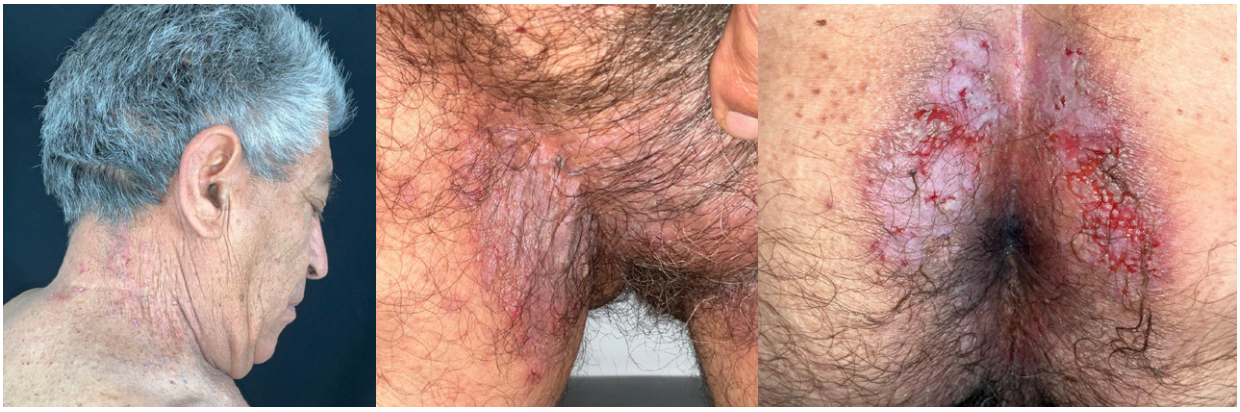
Grover's disease was described by Ralph Grover in 1970 and is classified as an acquired dermatological disorder. Although its etiology is not fully understood, it has been suggested that the activation of helper T lymphocytes, stimulated by inflammatory cytokines, may trigger alterations in keratinocytes. This condition predominantly affects white males in their sixth and seventh decades of life.[1]

Clinically, it can present in various forms, ranging from a monomorphic eruption of erythematous papules or papulovesicles with eventual hyperkeratosis to characteristic plaques with thick scaling, accompanied by fissures and erosions. Histologically, five distinct patterns have been described, with acantholysis and dyskeratosis being the key signs that support the diagnosis.

Although it is also known as transient acantholytic dermatosis, it has been observed that it is not always a transient condition, as recurrent forms have been documented. Due to its wide range of clinical presentations and variable progression, it is crucial to consider multiple differential diagnoses.

CLINICAL CASE

We present a 78-year-old male patient with no significant medical history who presents with a 5-month history of a cutaneous condition. The condition is characterized by intensely pruritic lesions that initially localized to the neck and later spread to intertriginous areas. The patient was treated for body ringworm (tinea corporis) and received oral fluconazole, but no improvement was noted.



On physical examination, disseminated dermatosis was observed on the neck (Figure 1A), trunk, inguinal region, and perianal area, characterized by erythematous papules that confluence to form erythematous plaques (Figure 1B). These plaques were mildly hyperkeratotic and presented maceration and areas of erosion in the skin folds (Figure 1C).

A skin biopsy was performed on the neck and inguinal region (Figure 2).

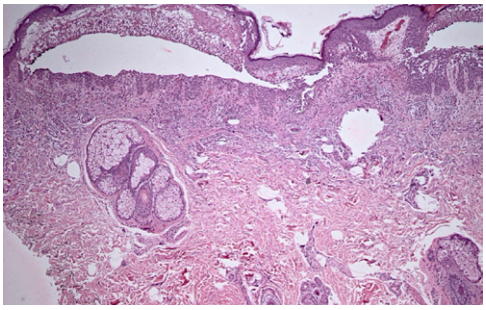


Figure 2A: Corresponding to the scalp, intraepidermal vesicle formation is observed containing cellular debris, exudate, and inflammatory cells, with neutrophils and eosinophils predominating. The epidermis shows ballooned keratinocytes, many with cytopathic changes, multinucleated cells, and nuclear molding. Extensive acantholysis affecting the spinous layer is also observed. In the upper dermis, edema and a dense, diffuse inflammatory infiltrate composed of lymphocytes, histiocytes, eosinophils, and neutrophils are noted.

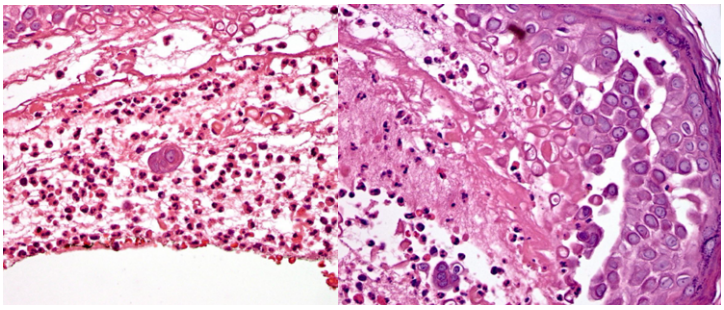


Figure 2B: Corresponding to the inguinal region, moderate epidermal acanthosis is identified, covered by laminar hyperkeratosis and cellular debris, with extensive acantholysis and keratinocytes detached at the spinous level. Focal dyskeratosis is observed in the upper layers of the epidermis. The underlying dermis shows a moderate linfohistiocytic inflammatory infiltrate with eosinophils

DISCUSSION

Transient acantholytic dermatosis is an acquired, generally benign, and self-limiting skin disorder with a variable course. The definitive diagnosis is often delayed because its clinical presentation is frequently confused with infectious processes, as was the case with the patient in our report.

Clinically, the condition presents as a monomorphic, acute eruption of erythematous papules or vesicles, some of which may show hyperkeratosis. Although the lesions commonly appear in photo-exposed areas, they can also occur in various locations, most commonly on the trunk (84-99%), followed by the proximal extremities (35-63%) and the lower extremities (61%). Less common areas include the neck (21%), face/scalp (17%), axilla (4%), and mucous membranes (1%).[2]

In the clinical case presented, the lesions appeared on the chest, neck, and genital region, which are areas frequently affected according to the literature.

Dermatoscopically, the typical findings include a pink background with polymorphic vessels, star-shaped or oval structures that are white-yellow with a white halo, along with scales and superficial erosion in the dermis, which can be a key sign for diagnosis.[3] As the lesions resolve, post-inflammatory hyperpigmentation may occur. This condition is not associated with systemic symptoms, except for pruritus, which is common and can be intense in most patients.

Three clinical variants have been described: transient eruptive, chronic asymptomatic, and persistent pruritic. Several associations have been reported, with the most common being a history of malignancy, tumors, immunocompromised patients, use of medications, other dermatologic conditions, as well as exposure to UV radiation, physical exercise, friction, and sweating, which act as triggering factors.[2]

The diagnosis of transient acantholytic dermatosis is based on the medical history, clinical evaluation, and histopathological findings. Histologically, predominant acantholysis is accompanied by diskeratosis. However, hyperkeratosis, acanthosis, and parakeratosis may also be present in the epidermis. In the dermis, edema and a lymphocytic, eosinophilic, or neutrophilic perivascular inflammatory infiltrate may be observed. Five distinct histological patterns have been described for this condition.[4]

Table 1: Histological Patterns

VULGAR PEMPHIGUS TYPE
DARIER'S DISEASE TYPE
HAILEY-HAILEY DISEASE TYPE
PEMPHIGUS FOLIACEUS TYPE
SPONGIOTIC-ACANTHOLYTIC TYPE

The treatment of transient acantholytic dermatosis depends on the degree of involvement. In mild forms, it is managed with topical corticosteroids, vitamin D analogs, or calcineurin inhibitors. For moderate forms, oral corticosteroids are considered, with prednisone at a dose of 0.5 to 1 mg/kg/day. Isotretinoin (at a dose of 40 mg/day) or acitretin (0.5 mg/kg/day) have also been used with good results.

In severe and refractory cases, the use of phototherapy (PUVA, UVA-1) has been described, and as a last resort, anti-TNF drugs.[6] In the case of our patient, an adequate response was observed to corticosteroid treatment, starting with prednisone at a dose of 60 mg/day, with a progressive reduction in the dose weekly, accompanied by sedating antihistamines. After three weeks, the lesions resolved.

CONCLUSION

In conclusion, transient acantholytic dermatosis is a benign condition, although uncommon, that can sometimes present a diagnostic challenge due to the many diseases that can be considered in its clinical presentation. Therefore, the clinicopathological correlation is essential for accurate diagnosis. Although it does not present systemic alterations, it can affect the quality of life of patients, highlighting the importance of timely diagnosis and treatment.

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