

Grover Disease in an 83-Year-Old Patient: A Clinicopathological Case Report

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Received: 01 Sep 2026; **Accepted:** 03 Oct 2026; **Published:** 14 Oct 2026

Citation: Camargo-Goñi Alba Regina, García-Crespo Ashley, González Maribet. Grover Disease in an 83-Year-Old Patient: A Clinicopathological Case Report. American J Pathol Res. 2026; 5(10): 1-4.

ABSTRACT

Grover disease, or transient acantholytic dermatosis, is a benign acquired dermatosis whose diagnosis can be challenging due to the variability of its clinical and histopathological manifestations and its resemblance to other acantholytic dermatoses. It has gained particular relevance in oncologic patients, in whom it may present idiopathically or in association with radiotherapy and other antineoplastic treatments.

We report the case of an 83-year-old man with a history of prostate cancer treated with radiotherapy 16 years earlier, who presented with a 3-month history of a chronic, asymptomatic dermatosis involving the chest, abdomen, and left arm, characterized by erythematous plaques with thick scales and surrounding pustules. Given the lack of response to initial treatment with oral prednisone and minocycline, as well as the persistence of the lesions despite tapering doses of systemic corticosteroids, two incisional biopsies were performed. Histopathologic examination revealed an acanthotic, hyperkeratotic epidermis with suprabasal acantholytic clefts and dyskeratotic keratinocytes, including corps ronds and grains, findings consistent with Grover disease.

The patient achieved sustained clinical control following gradual tapering of oral corticosteroids and transition to a combined topical regimen consisting of a corticosteroid, calcipotriol, emollients, and keratolytics. Progressive improvement was observed, with no new lesions after one month of follow-up, although complete resolution was not achieved.

This case highlights the importance of clinicopathologic correlation in the diagnosis of Grover disease in older patients with a history of malignancy, as well as the potential to achieve clinical control with topical therapy alone, thereby minimizing exposure to systemic corticosteroids in this age group.

Keywords

Acantholysis, Acantholytic dermatosis, Dyskeratosis, Grover disease, Transient acantholytic dermatosis.

Abbreviations

TAD: Transient Acantholytic Dermatitis.

Introduction

Transient acantholytic dermatosis (TAD), universally known as Grover disease, is a benign acquired skin condition originally

described in 1970 by Dr. Ralph Wier Grover. In his initial report, Grover characterized a group of six patients presenting with an intensely pruritic papulovesicular eruption on the trunk with a self-limited clinical course, establishing a clinicopathological entity distinct from the hereditary acantholytic disorders known at the time [1].

In recent years, Grover disease has gained particular relevance in the oncologic patient, in whom it may present idiopathically, in association with radiotherapy, induced by antineoplastic

treatments, or, less frequently, as a paraneoplastic manifestation. In this setting, it may take on extensive and atypical forms, particularly in immunosuppressed patients [2,3].

Diagnosis can be challenging owing to the variability of its clinical and histopathological manifestations and its resemblance to other acantholytic dermatoses, drug eruptions, and cutaneous infections [4]. Clinicopathological correlation is therefore essential to establish a timely diagnosis and avoid unnecessary therapeutic interventions.

We present a case of Grover disease in an 83-year-old man with a history of prostate cancer treated with radiotherapy. This case highlights the clinical and histopathological features of Grover disease and emphasizes the importance of clinicopathological correlation in establishing an accurate diagnosis.

Case Presentation

An 83-year-old male patient, weighing 80 kg, with a history of prostate cancer diagnosed in 2008 and treated with 23 sessions of radiotherapy in 2009, achieving complete remission and currently off treatment for this condition. He had no other relevant comorbidities or concomitant pharmacologic treatments prior to the onset of the current dermatologic condition.

He presented to our department with a disseminated dermatosis affecting the anterior chest in the inframammary region and the left side of the abdomen, as well as the anterior aspect of the left arm, characterized by multiple erythematous plaques, some with thick surface scale and small surrounding pustules, with a chronic course of 3 months and an asymptomatic presentation (Figure 1A and 1B).

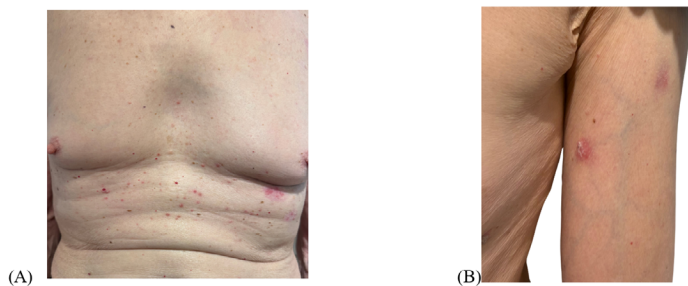


Figure 1: Clinical presentation of Grover disease. (A) Frontal view showing multiple erythematous lesions predominantly involving the inframammary and upper abdominal regions. (B) Close-up of erythematous scaly plaques on the left arm.

Prior to evaluation at our department, the patient had been seen by another physician, who prescribed oral prednisone 20 mg/day and minocycline 100 mg/day for 30 days, with no change in the clinical picture. Due to persistence of the lesions, he presented to our department, where the prednisone dose was adjusted to 0.5 mg/kg/day (40 mg/day), with a tapering schedule of 5 mg every 10 days, combined with topical treatment using a medium-potency corticosteroid (betamethasone) and emollients. This regimen

resulted in improvement of the lesions; however, upon reaching a prednisone dose of 30 mg/day, the lesions recurred.

Given this relapse, the oral prednisone dose was increased again, and, with the patient's informed consent, two incisional biopsies were performed, obtained from the left chest at the midaxillary line and from the abdomen. Subsequently, gradual de-escalation of the oral steroid was carried out, together with continuation of the topical regimen.

At the time of writing this report, the patient is under topical treatment only, with betamethasone, calcipotriol, emollients, and 30% urea as a keratolytic agent, with one month of follow-up under this regimen. No new lesions have appeared, and the pre-existing lesions show improvement, although without complete resolution.

Histopathological examination of both biopsies showed an acanthotic epidermis with hyperkeratosis and foci of parakeratosis, associated with suprabasal acantholytic clefts and dyskeratotic keratinocytes of the corps ronds and grains type. In the superficial dermis, dilated vessels surrounded by a moderate lymphocytic infiltrate were observed. Based on the histopathological findings of acantholytic dyskeratosis and clinicopathological correlation, a diagnosis of Grover disease was established.

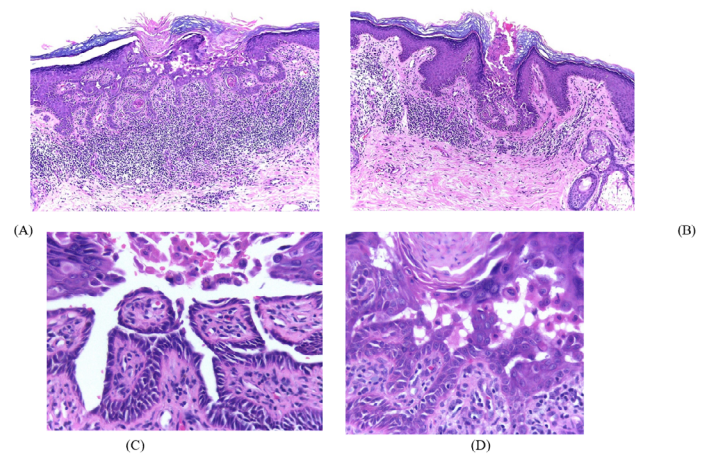


Figure 2: Histopathologic findings of Grover disease. (A) Low-power view showing epidermal acanthosis and hyperkeratosis. (B) Focal epidermal alterations with acantholytic changes. (C) Higher-power view showing prominent acantholysis with loss of cohesion between keratinocytes. (D) Higher-power view highlighting dyskeratotic keratinocytes, including corps ronds (hematoxylin and eosin stain).

Discussion

This case illustrates several important clinical and diagnostic challenges associated with Grover disease in an elderly patient with a history of prostate cancer. The variable and occasionally atypical clinical presentation of this condition, together with its histopathological resemblance to other acantholytic dermatoses, can generate diagnostic uncertainty, underscoring the importance of clinicopathological correlation in establishing an accurate diagnosis.

Epidemiology and Predisposing Factors

Grover disease markedly predominates in fair-skinned elderly men, with a mean age at diagnosis in the sixth to seventh decades of life [1-3]. Large demographic studies report an estimated male-to-female prevalence ratio of between 1.6:1 and 3:1 [2,4]. This predilection for elderly patients correlates with physiological skin senescence: with aging, there is a marked decrease in the function and density of sweat glands, as well as in stratum corneum lipid levels, predisposing to pronounced cutaneous xerosis and dysfunction of the epidermal barrier [2].

Although the pathogenesis of Grover disease is not fully elucidated, it is considered a polypathogenic process in which individual susceptibility interacts with various physical and environmental factors [2,4]. Among the most frequently described triggers are heat and sweating, possibly related to alterations of the intraepidermal eccrine sweat duct [1-3], as well as xerosis and cold climate, which would explain the paradoxical increase in cases during the winter months [2]. Prolonged immobilization, occlusion, and friction have also been implicated as precipitating factors, particularly in hospitalized patients or those with chronic illnesses [2,3].

Other associated factors include ultraviolet radiation exposure, ionizing radiation (including radiotherapy), chronic kidney disease in patients undergoing hemodialysis, and thermal burns [2]. An association has also been described with agents such as *Malassezia furfur*, *Demodex folliculorum*, and *Sarcoptes scabiei*, possibly through inflammatory mechanisms that promote acantholysis in predisposed individuals [2,4].

Grover Disease in the Oncologic Setting

An association between Grover disease and hematologic and solid malignancies has been described, particularly in extensive or atypical presentations [2,5]. These include genitourinary neoplasms such as prostate cancer, as well as melanoma and lung and breast carcinomas [5]. Cases associated with antineoplastic treatments have also been reported, including chemotherapy, targeted therapies, and immunotherapy [4,5].

Radiotherapy is another associated factor, with cases described predominantly in previously irradiated areas and, more frequently, within the first weeks after treatment, although delayed presentations have also been reported [1-3]. In our patient, radiotherapy for prostate cancer was performed approximately 16 years before the development of the dermatosis; therefore, although this history is relevant, the prolonged time interval limits the possibility of attributing a direct causal relationship, and its role as a predisposing factor remains uncertain.

Diagnostic Challenges: Grover Disease as a Clinical Mimicker

Clinical Presentation

Grover disease predominantly affects the trunk, although it may extend to other anatomical areas [2,6]. It is characterized by erythematous, keratotic, or crusted papules or papulovesicular lesions, usually pruritic, although some patients may remain

asymptomatic [1,2]. In patients with oncologic history, more extensive or atypical clinical presentations have been described, with variations in both the distribution and morphology of the lesions [2,6].

Histopathological Features

Histopathologically, foci of epidermal acantholysis accompanied by variable degrees of dyskeratosis are observed [1,6-10]. Five main patterns have been described: Darier-like, Hailey-Hailey-like, pemphigus vulgaris-like, pemphigus foliaceus-like, and spongiotic-acantholytic, which may coexist within the same specimen or between different lesions [4]. The Darier-like pattern is characterized by suprabasal acantholysis and prominent dyskeratosis, with the presence of corps ronds and grains [4].

Differential Diagnosis

Clinically, the differential diagnosis includes dermatitis herpetiformis, miliaria rubra, scabies, and other acantholytic dermatoses such as Darier disease and Hailey-Hailey disease, which are distinguished by their earlier onset, chronic course, characteristic distribution, and hereditary history [2-7].

From a histopathological standpoint, pemphigus vulgaris and pemphigus foliaceus should be considered primarily, and, less commonly, Galli-Galli disease [2-4]. Clinicopathological correlation, together with the focal presence of acantholysis and dyskeratosis, is essential for establishing the diagnosis; direct immunofluorescence, characteristically negative in Grover disease, allows it to be differentiated from the pemphigus group [4].

Treatment and Clinical Course

Therapeutic management is based on an individualized approach according to extent, symptomatology, and treatment response, given the absence of standardized guidelines [3,8]. General measures include avoidance of triggering factors and maintenance of adequate skin hydration, while topical corticosteroids constitute first-line treatment [3].

In patients with an insufficient response to topical corticosteroids, vitamin D analogues such as calcipotriol represent a therapeutic alternative, promoting keratinocyte differentiation and epidermal calcium homeostasis [11]. In persistent, extensive, or refractory forms, systemic retinoids, phototherapy, and other alternatives may be considered, including dupilumab or TNF-alpha inhibitors such as etanercept [12-14].

The clinical course is variable; while some patients experience spontaneous resolution, others develop persistent or recurrent forms requiring maintenance therapy [3]. In our patient, the initial regimen of oral corticosteroid at 0.5 mg/kg/day combined with a topical corticosteroid achieved initial improvement of the lesions; however, relapse during tapering of the oral dose demonstrated dependence on the systemic component. This prompted a second de-escalation of the oral steroid, this time progressively replacing it with a combined topical regimen of betamethasone, calcipotriol, emollients, and keratolytics. With this exclusively topical

management, the patient has remained free of new lesions, with progressive improvement of pre-existing lesions, although without complete resolution to date, after one month of follow-up. This course illustrates the possibility of achieving sustained clinical control with topical management alone, avoiding prolonged exposure to systemic corticosteroids in an elderly patient, in whom the risk of associated adverse effects (e.g., osteoporosis, hyperglycemia, immunosuppression) is particularly relevant.

Comparison with the Published Literature

Published cases of Grover disease in oncologic patients show a heterogeneous presentation, related both to the underlying malignancy and to different therapeutic modalities [4,8]. Among others, a patient with a history of prostate cancer treated with radiotherapy and lung adenocarcinoma under pembrolizumab and chemotherapy has been described, who achieved lesion resolution with topical treatment without the need to modify antineoplastic therapy [4,14].

Demographically, our patient, aged 83 years, lies at the upper limit of the age range (14–83 years; mean 56 ± 15 years) described by Gantz et al. in their systematic review of extensive or atypical cases [2]. Advanced age may contribute to increased cutaneous susceptibility due to aging-related changes and impaired epidermal barrier function, together with other predisposing factors.

In contrast to cases temporally associated with oncologic treatments, our patient had no recent exposure to antineoplastic therapy, which makes it difficult to identify a specific trigger and favors consideration of a multifactorial etiology.

Clinical Implications and Lessons Learned

This case underscores several clinically relevant messages for medical practice:

1. Maintain adequate diagnostic suspicion: Grover disease should be considered in cases of persistent or atypical dermatosis, particularly in elderly patients with an oncologic history.
2. Prioritize clinicopathological correlation: Skin biopsy is essential in atypical presentations or in the absence of therapeutic response, allowing differentiation from other acantholytic dermatoses and drug eruptions.
3. Consider the oncologic context: A history of malignancy and antineoplastic treatments, including radiotherapy, should be assessed within the clinical context, avoiding the establishment of causal relationships without sufficient evidence.
4. Avoid unnecessary interventions: Timely recognition allows appropriate dermatologic management and, in patients under active antineoplastic therapy, helps avoid unjustified modification or discontinuation of oncologic treatment.

Conclusions

This case emphasizes the importance of considering Grover disease within the differential diagnosis of persistent dermatoses in elderly

patients with a history of malignancy. Its timely recognition allows appropriate management, avoids unnecessary interventions, and favors individualized follow-up, particularly in patients with a history of oncologic treatment.

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