CASE REPORT

# Grover's disease associated with Sarcoptes scabiei: case report

## *Doença de Grover associada a Sarcoptes Scabiei: relato de caso*

José Henrique Pereira Pinto[1],Chan I Lym[2], Clarissa Maria Serpa Vieira[3], Chan Kun Wa[4], Chan I Thien[4]

DOI: 10.5935/2238-3182.20140063


### ABSTRACT

Grover's disease, or transient acantholytic dermatosis, consists of a papulovesicular pruritic dermatosis histologically characterized by acantholysis. Most cases are represented by white men above 40 years of age. There is no known etiology; however, heat and excessive sweating are frequently described as triggering or aggravating factors. The diagnosis is made by clinical suspicion and confirmed by biopsy of cutaneous lesions. In this study the case of a 53-year-old patient is reported with Grover's disease associated with infestation by *Sarcoptes scabiei*.


**Key words:** *Sarcoptes scabiei*; Scabiei; Acantholysis.


### RESUMO

*A doença de Grover, ou dermatose acantolítica transitória, consiste em uma dermatose papulovesiculosa pruriginosa, caracterizada histologicamente por acantólise. A maior parte dos casos está representada por homens brancos acima de 40 anos de idade. Não existe etiologia conhecida, mas o calor e o suor excessivos são descritos com frequência como fatores desencadeantes ou agravantes. O diagnóstico é feito por suspeição clínica e confirmado pela biópsia das lesões cutâneas. Neste estudo é relatado o caso de um paciente de 53 anos de idade, com doença de Grover associada à infestação por* Sarcoptes scabiei.


***Palavras-chave:*** Sarcoptes scabiei*; Escabiose; Acantólise.*

[1] MD. Full Professor of Immunology at the Medical School of Itajubá – FMIt. Itajubá, MG – Brazil.
[2] MD. Graduate School student in Dermatology by the Federal University of Rio de Janeiro – UFRJ. Rio de Janeiro, RJ – Brazil.
[3] MD. Hospital School at FMIt. Itajubá, MG – Brazil.
[4] Students at the Medical School at UFRJ. Rio de Janeiro, RJ – Brazil.

## INTRODUCTION

The Grover's disease (GD) was described by the American dermatologist Ralph Grover in 1970. In his account, he described the cases of six patients who presented papular and papulovesicular lesions located on the trunk and accompanied by intense itching, which appeared in the changing seasons and regressed after a few weeks. The biopsy of these lesions showed small foci motivating the author to name the disease as transient acantholytic dermatosis (TAD).

The first TAD case described in Brazil was published by Azulay-Abulafia in 1986, and only two other cases were described later by Manfrinato et al.[1] in 1999. Although GD is characterized by a self-limited transitional course[2] with an average duration of 22 months, according to an article by Mark D.P. Davis et al.,[3] some patients present chronic manifestations of the disease.[4,5] In his analysis of 72 cases, 13 patients presented recurrences in varying periods.[3]

*Submitted:* 2011/01/12
*Approved:* 2014/24/04

*Institution:*
Hospital School Medical School of Itajubá – FMIt
Itajubá, MG – Brazil

*Corresponding Author:*
Chan I Lym
E-mail: ilymchan@yahoo.com.br

Most GD cases portray white men over 40 years old[4-6] and the extension and duration of illness worsens with the patients' advanced age.[1,7]

## CASE REPORT

Male patient, Eastern ethnicity, age 53 years old. In October 2007, came into contact with a patient with scabies (*Sarcoptes scabiei*). Acquired this parasitosis showing papulo-eritematosus lesions of approximately 3 mm, followed by intense itching around the trunk and proximal portions of upper and lower limbs. The patient was treated with 15 mg ivermectin (single dose) and 50 mg/mL permethrin topical lotion, with partial resolution of symptoms.

After two weeks, the dermatological framework worsened. The lesions merged and formed erythematous plaques with diameters ranging between 2 and 4 cms, across the trunk and proximal regions of upper and lower limbs (Figures 1 and 2). These lesions were accompanied by intense itching and painful sensation. Heat and sweat were reported as worsening factors; and bath with cold water as an improving aid.

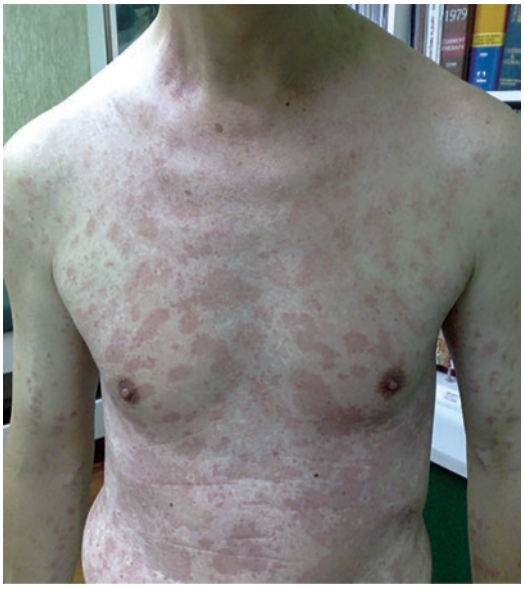

**Figure 1** - Lesions on the trunk.

On physical examination, the patient only presented skin lesions. The laboratory tests were normal. He used 20 mg/day prednisone and 75 mg/day hydroxyzine without improvement. The biopsy of skin lesions and histopathological findings are compatible with Grover's disease.

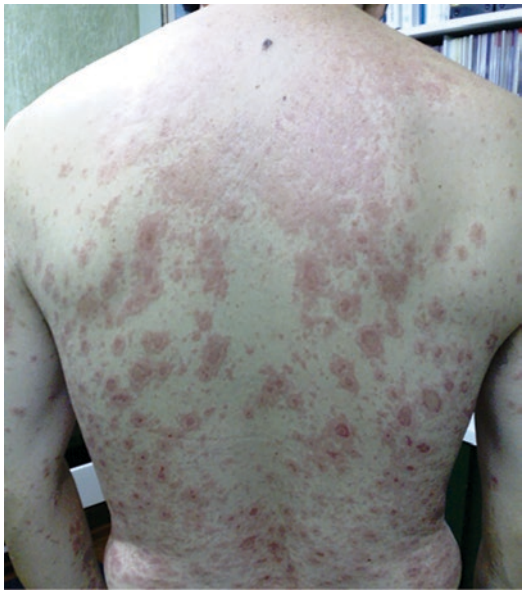

**Figure 2** - Lesions on the trunk.

Treatment with methotrexate was started at the dose of 15 mg per week for 10 weeks; a rapid remission was observed. After six months, however, the recurrence of lesions occurred. We decided to restart the treatment with methotrexate, in the same dose, during 12 weeks, associated with retinol acetate (50,000 IU/day) and the suspension of aerobic physical activity; the lesions regressed promptly.

Currently, the patient continues with the daily use of retinol acetate (50,000 IU/day), showing skin lesions compatible with Grover disease on the trunk and in small numbers (Figure 3).

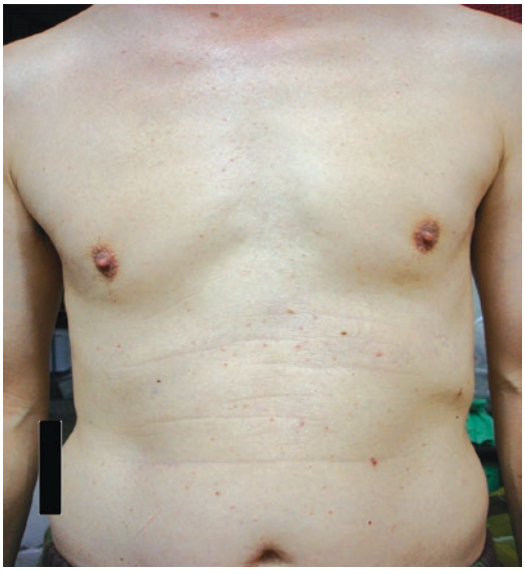

**Figure 3** - Remaining lesions.

## DISCUSSION

The Grover's disease is characterized by papular, papulovesicular, or papular keratosic formations that can vary from 1 to 10 mm.[3-6] Vesiculopustular, bullous, follicular, herpetiformis, and erythematous forms have already been described.[3,5] The most common locations of eruptions are the chest and root of members, while scalp, face, and neck are less affected. There have been rare descriptions in the literature of incidence in mucous membranes and palms and soles.[3-6,8,9] In an exacerbation, the erythematous papules may converge, forming erythematous plaques (Figures 1 and 2). The itching is a frequent complaint, and its intensity is variable. When intense, it is accompanied by a burning feeling that is difficult to control.[4] The age group most affected by GD includes individuals over the fifth decade in age, with predominance of the Caucasian ethnicity, and with a men to women ratio of 3:1, which according to some authors it can be up to 8:1.[3,4]

The etiology and GD pathogenesis remain unknown. Many factors have been associated with this disease and are presented as triggering and/or aggravating symptoms. Physical factors such as: heat, exposure to sunlight, ionizing radiation, excessive sweating, and fever are described with frequency.[4,10] Others such as skin irritation and inflammation, skin infections caused by Malassezia furfur, Demodex folliculorum, and Sarcoptes scabiei, trauma, pregnancy, bed rest in prolonged hospitalizations, and polyester apparel have also been reported. To a lesser number of descriptions, are non-infectious skin diseases that would trigger GD found in literature such as: asteatotic eczema, contact dermatitis, atopic dermatitis, bullous and foliaceus pemphigus, lichen planus, psoriasis, pyoderma gangrenosum, seborrhea dermatitis, and versicolor pityriasis.[10,11]

Diseases associated to the development of GD: some medications, related systemic diseases: benign monoclonal gammopathy, thymoma, glomerulonephritis, chronic gastritis, rheumatoid arthritis, HIV infections, poliovirus, carcinoid syndrome, and progressive systemic sclerosis. Malignant solid tumors described: tumors of the genitourinary tract, stomach, lung, prostate, colon, breast, and larynx adenocarcinomas and ovarian carcinoma. Hematologic neoplasias reported: acute and chronic myelogenous leukemia, acute and chronic lymphocytic leukemia, multiple myeloma, myelodysplastic syndrome, and peripheral T-cell lymphoma.[11-16]

In its original publication, Grover suggested that the GD would be a form of irritant dermatitis in people genetically predisposed.[2,17] According to some authors, a dermatological reaction to a paraneoplastic syndrome would also be a possible explanation.[3]

In regards to the present case, it is possible that an allergic reaction to the mite *Sarcoptes scabiei* or its products have exacerbated an immediate hypersensitivity reaction and late in the patient causing skin lesions and producing the symptoms.

The GD diagnosis is based on histopathological finding of focal acantholysis, with or without spongiosis, in the epidermis and supra-basal level or subcorneal in skin specimen submitted to biopsy.[4,10,18] in some cases, it is histologically indistinguishable from other acantholytic diseases (for example, the Darier disease), and the clinical and histological correlation in all cases needs to be performed.[4]

The GD is an under-diagnosed disease, probably due to the large number of oligosymptomatic cases and/or transient or due to the fact that the disease clinically co-exists or is confused with other illnesses making diagnosis difficult.[3]

It was in 1977 that Chalet et al. described the four histological forms currently accepted and which are characterized by acantholysis with formation of vesicles: similar pattern to vulgaris or foliaceus pemphigus, Darier disease-like pattern, Hailey-Hailey disease-like pattern, and a pattern with focal spongiosis. The pemphigus uulgaris-like and Darier-simile patterns are the most common.[3]

In the case of our patient, the biopsy and histopathological examination of two skin segments showed findings compatible with pemphigus foliaceus-like forms, Darier-like, and Hailey-Hailey-simile.

As for the GD treatment, there is no standardization of conduct. Measures to prevent excessive sweating should be introduced early such as avoiding exposure to the sun, heat, and intense physical activity. It is important, also, to minimize skin irritation by using mild soaps, light-colored cotton fabrics, and adequate hydration.

In mild manifestations, the use of topical substances has shown a reduction of lesions and improvement of pruritus. Normally, vitamin A derivatives, calcipotriol, lactic acid lotions, urea ointments, zinc oxide ointment, and corticosteroids are used for these purposes.[4,6-9,19]

Systemic therapy is recommended in the most extensive and persistent lesions. The literature describes the use of retinoids (isotretinoin and etreti-

nate) and cytostatics (methotrexate) besides steroids (prednisone and triamcinolone) for the remission of lesions. It is observed that the recurrence of lesions is frequent at the end of treatment with corticosteroids. Some studies show that the use of phototherapy with psoralen and ultraviolet A (PUVA) may be a useful resource; however, its mechanism of action is not fully understood.[3,5-7] In cases where the itching is very intense, antihistamines (hydroxyzine, cetirizine) can be used as adjunctive therapy. Antibiotics (tetracycline and erythromycin) and antifungals (itraconazole) are indicated when there is secondary infection.[8,20-22]

## REFERENCES


1. Manfrinato LC, Mulinari-Brenner FA, Santamaria JR, Pizzol AS, Lima LR. Dermatose acantolítica transitória (doença de Grover): relato de dois casos. Anais Bras Dermatol. 1999 jul/ago; 74(4):379-81.
2. Azulay-Abulafia L, Azulay DR, Azulay RD. Dermatose acantolítica transitória: apresentação de um caso. Med Cult ILA. 1986; 14:345-8.
3. Grover RW. Transient Acantholytic Dermatosis. Arc Dermatol. 1970; 101:426-34.
4. Davis MD, Dineen AM, Landa N, Gibson LE. Grover´s disease: clinicopathologic review of 72 cases. Mayo Clin Proc. 1999 Mar; 74(3):229-34.
5. Santisteban MM, Stengel FM. Enfermedad de Grover: Dermatosis acantolítica transitoria: 30 años después. Arc Arg Dermatol. 2000; 50(1):233-43.
6. Gon AS, Guerra DMG. Caso para diagnóstico. Anais Bras Dermatol. 1999 jul/ago; 74(4):405-7.
7. Parsons JM. Transient acantholytic dermatosis (Grover´s disease): a global perspective. J Am Acad Dermatol. 1996; 35:653-70.
8. Quirk CJ, Heenan PJ. Grover´s disease: 34 years on. Australasian J Dermatol. 2004 May; 45(2):83-8.
9. Kaddu S, Müllegger RR, Kerl H. Grover´s disease associated with Sarcoptes scabiei. Dermatology. 2001; 202:252-4.
10. Morán MAG, Salas JS, Nieves C, Ribas MT. Dermatosis acantolítica transitoria (Grover). Actas Dermo-Sifilográficas. 1993; 84:244-6.
11. Rosina P, Melzani G, Marcelli M, Zakaria W, Colato C, Barbat A. Grover´s disease (transient acantholytic dermatosis) associated with atopy. JEADV. 2005; 19:380-99.
12. Tscharner GG, Bühler S, Borner M, Hunziker T. Grover´s disease induced by cetuximab. Dermatology. 2006; 213(1):37-9.
13. Antunes I, Azevedo F, Mesquita-Guimarães J, Resende C, Fernandes N, Macedo G. Grover´s disease secondary to ribavirin. Br J Dermatol. 2000 Jun; 142(6):1257-8.
14. Zvulunov A, Grunwald MH, Avinoach I, Halevy S. Transient acantholytic dermatosis (Grover´s disease) in a patient with progressive systemic sclerosis treated with D-penicillamine. Int J Dermatol. 1997; 36:473-8.
15. Bolaños-Meade J, Anders V, Wisell J, Farmer ER, Vogelsang GB. Grover´s disease after bone marrow transplantation. Biolf Blood Marrow Transp. 2007; 13:1116-7.
16. Villalon G, Martin JM, Monteagudo C, Alonso V, Ramon D, Jorda E. Clinicopathological spectrum of chemotherapy induced Grover´s disease. J Eur Acad Dermatol Venereol. 2007 Sep; 21(8):1145-7.
17. Hashimoto K, Fujiwara K, Harada M, Setoyama M, Eto H. Junctional proteins of keratinocytes in Grover´s disease, Hailey-Hailey disease and Darier´s disease. J Dermatol. 1995; 22:159-70.
18. French LE, Piletta PA, Etienne A, Salomon D, Saurat JH. Incidence of transient acantholytic dermatosis (Grover´s disease) in a hospital setting. Dermatology. 1999; 198(4):410-1.
19. Hanson M, Hsu S. Pruritic papules on the chest and back. Am Fam Physician. 2006 Aug 15; 74(4):641-2.
20. Keohane SG, Cork MJ. Treatment of Grover´s disease with calcipotriol (Dovonex). Br J Dermatol. 1995; 132:832-3.
21. Julliard KN, Milburn PB. Antibiotic ointment in the treatment of Grover disease. Cutis. 2007 Jul; 80(1):72-4.
22. Breuckmann F, Appelhans C, Altmeyer P, Kreuter A. Medium-dose ultraviolet A1 phototherapy in transient acantholytic dermatosis (Grover´s disease). J Am Acad Dermatol. 2005 Jan; 52(1):169-70.