Scientific Research Publishing

Case Reports in Clinical Medicine, 2023, 12, 345-351
https://www.scirp.org/journal/crcm
ISSN Online: 2325-7083
ISSN Print: 2325-7075

Transient Acantholytic Dermatosis: A Case Report and Literature Review

Shuxian Shang[1], Tenglong Li[2], Ying Yan[2], Ping Xia[2*], Xiaoyong Zhou[2*]

[1]School of Medicine, Jianghan University, Wuhan, China
[2]Dermatological Department, Wuhan No.1 Hospital, Wuhan, China
Email: ssx19961108@126.com, ltl2006912@126.com, 19745459@qq.com, *445814434@qq.com, *zhouxuefeng1970@126.com

How to cite this paper: Shang, S.X., Li, T.L., Yan, Y., Xia, P. and Zhou, X.Y. (2023) Transient Acantholytic Dermatosis: A Case Report and Literature Review. *Case Reports in Clinical Medicine*, **12**, 345-351.
https://doi.org/10.4236/crcm.2023.129048

Received: July 16, 2023
Accepted: September 15, 2023
Published: September 18, 2023

Abstract

Transient acantholytic dermatosis (TAD, Grover disease) is a benign skin disease with a nonspecific rash that may present as a reddish-brown, dermatomal edematous papule or herpetiform rash with a partially central horny plug. This disease is usually self-limiting and can subside within weeks or months, but it may also have a chronic progression with a trend towards long-term recurrence. The patient is an 80-year-old male who visited the clinic due to recurring erythema and blisters all over his body that had been present for over 2 years. Based on the patient's course, laboratory tests and histopathological findings were consistent with the diagnosis. The final diagnosis was transient acantholytic dermatosis. The patient has a long course of the disease, a wide range of skin lesions and he has the different subtypes of pathological manifestations. This case and corresponding literature review help us have a clear understanding of Grover's disease, which has received reference value for the diagnosis and treatment of this disease in clinical work.

Keywords

Acantholysis Cell, Transient Acantholytic Dermatosis

1. Introduction

Transient acantholytic dermatosis (TAD, Grover disease) was first described in 1970 as a self-limited eruption of pruritic, discrete, and edematous papules and vesicles which appeared on the trunk and extremities following non-specific irritation. TAD is a benign skin disease with a nonspecific rash that may present as a reddish-brown, dermatomal edematous papule or herpetiform rash with a partially central horny plug. Scattered or aggregated in clusters, most patients present with pruritus of varying intensity, and episodes of severe pruritus may

*Corresponding authors.

precede rash attacks [1]. This disease is usually self-limiting and can subside within weeks or months, but it may also have a chronic progression with a trend towards long-term recurrence and persistent itching, which may even last for several years or longer [2]. The etiology of this disease is not yet clear. Hot environments, sweating, dry air, kidney failure, malignant tumors, and other triggering factors may trigger the disease. Other triggering factors include sunlight, ionizing radiation, and ultraviolet radiation [3] [4]. Other triggering factors include sunlight, ionizing radiation, and ultraviolet radiation. It has been reported abroad that most cases occur in middle-aged men [5] and that the male-to-female sex ratio ranges from 3:1 to 7:1 [6]. The trunk and proximal end are the most common affected areas of TAD, usually limited to the front of the chest, upper back, and lower part of the chest, widely dispersed, without a tendency to merge, while the scalp, palms, and soles of the feet are usually not affected [7]. It has been reported that up to 50% of patients experience recurrence [8]. The patient has a long course of disease, a wide range of skin lesions, and has pleomorphism, variability, and confusion. The pathological manifestations also show transitions between different subtypes, which are somewhat different from the clinical characteristics of Grover's disease. This case and corresponding literature review help us have a clearer understanding of Grover's disease, which has a certain reference value for the diagnosis and treatment of this disease in clinical work.

2. Case Report

2.1. First Diagnosis and Treatment

The patient is an 80-year-old male who visited the clinic due to recurring erythema and blisters all over his body that had been present for over 2 years. He initially developed erythema, blisters, scaling, and severe itching in the right popliteal fossa (the hollow behind the knee) 2 years ago, which would ooze after he scratched and irritated it. Over time, the rash gradually spread to his abdomen and both upper limbs. He had been prescribed intermittent oral or intravenous methylprednisolone, which provided some improvement but the symptoms returned after he stopped the medication. He was also given oral azathioprine, 40mg, twice daily for over two weeks, but he did not notice significant improvement and thus discontinued the medication.

Past medical history: The patient has a history of hypertension, coronary artery disease, stomach bleeding, and renal insufficiency. The patient denies any familial or hereditary diseases, as well as food and drug allergies. He has a history of smoking for over 40 years, averaging about ten cigarettes per day, and has no history of alcohol abuse.

The patient complained that he was diagnosed with "psoriasis" locally in 1997, and had intermittent recurrent attacks for 3 months. After treatment with topical and oral Chinese medicine, the rash basically subsided, and there was occasional local re-emergence of the rash for more than 20 years, which basically improved after topical medicine.

First hospital system examination: The patient's body temperature, respira-

tion, blood pressure, and heart rate are all within the normal range. But the patient has arrhythmia with premature beats.

Dermatologic conditions: The patient had widely distributed edematous dark red erythema, papules and maculopapular rash on the chest and back, partially fused into large plaques, covered with flaky thin adherent scales; dark red plaques covered with loose flaky thin scales and scabs on both forearms, dorsum of both feet and groin; numerous fissures on the plaques of both lower limbs, no obvious oozing or blistering, no involvement of mucous membranes.

Laboratory examination: erythrocytes: 3.14×10^{12} ($4.5 - 5.5 \times 10^{12}$), HGB: 101 g/L (120 - 160 g/L), urinary microalbumin: >0.15 g/L (<20 mg/L), albumin: 33.5 g/L (30 - 50 g/L), creatinine: 112 umol/L (44 - 132 umol/L), sedimentation: 31 mm/H (0 - 15 mm/H), anti-O: 226.00 KIU/L (0 - 200 KIU/L), humoral immunity: IgG19.3 g/l (7 - 16 g/l), total IgE > 2500 IU/ml (20 - 200 IU/ml), no significant abnormalities were found in fecal routine, electrolytes, lipids, blood glucose, tumor markers and syphilis.

2.2. Three Times Pathological Examinations

The patient was admitted to the hospital several times over a period of 3 years due to recurrent skin rashes, and three pathological examinations were performed: first dermatopathological examination (Figure 1): (right forearm) hyperkeratosis, focal epidermal spicule relaxation, and visible acantholytic cell. Direct and indirect immunofluorescence: negative. Diagnosis: eczema, transient acantholytic dermatosis. Treatment: oral levocetirizine tablets and topical fusidic acid cream and halometasone ointment. The patient was discharged after improvement.

Second dermatopathological examination (Figure 2): (left thigh) histopathological image: hyperkeratosis, focal epidermal spicule relaxation, visible acantholytic cell and dyskeratotic cells. Direct and indirect immunofluorescence: negative.

Third dermatopathological examination (Figure 3): (abdominal) mild hyperplasia and hypertrophy of the epidermis with spiny layer loosening cells visible on the basal layer with a collapsed brick wall-like appearance, edema of the dermal papillae, and infiltration of lymphocytes predominantly in the superficial perivascular layer. Direct and indirect immunofluorescence: negative.

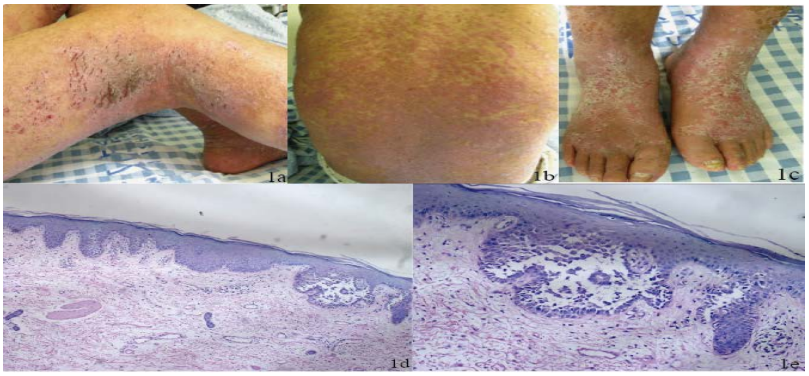

Figure 1. (a) Lesions on the popliteal fossa; (b) lesions on the back; (c) lesions on the feet; (d) HP × 40; (e) HP × 100.

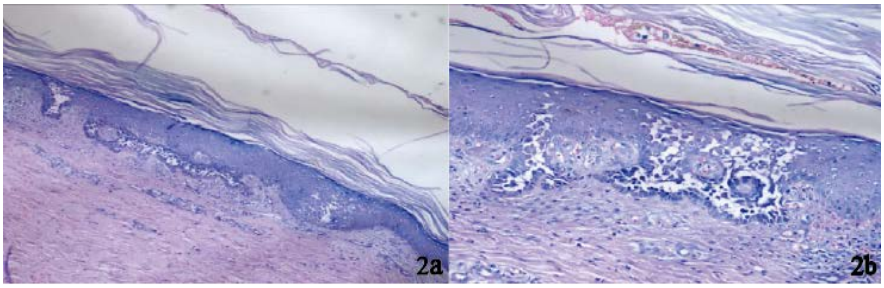

Figure 2. (a) HP × 40; (b) HP × 100.

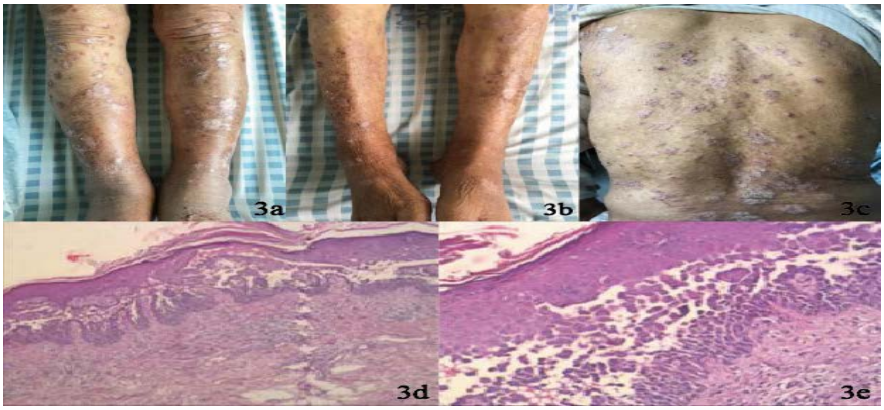

Figure 3. (a) Skin lesions of both lower limbs; (b) back skin lesions; (c) foot skin lesions: lesions; (d) HP × 40; (e) HP × 100.

Based on the patient's course, laboratory tests and histopathological findings were consistent with the diagnosis. The final diagnosis of transient acantholytic dermatosis was made.

3. Discussion

Transient acantholytic dermatoses require histopathologic confirmation of the diagnosis and the exclusion of other differential diagnoses. The typical histopathologic presentation is focal spondylolysis and varying degrees of dyskeratosis. There are six histologic subtypes of the disease: Darier's disease-like, common aspergillosis-like, Hailey-hailey-like, spongiform edema, deciduous aspergillosis-like, and maculopapular. These subtypes can occur separately or simultaneously [8] [9]. It is worth noting that PV and Darier's disease-like patterns are the most common types of reporting [8] [10].

It is necessary to differentiate this disease from psoriasis, eczema, and follicular keratosis. Chronic eczema often persists from acute and subacute eczema, with skin lesions characterized by hypertrophy of the affected area, infiltration or mossy-like changes. The skin lesions are mostly dark red or grayish brown, with dry, rough, scaly areas, and may be accompanied by pigmentation or hypopigmentation [11], However, in this case, the patient's skin lesions showed dark red erythema, papules, and maculopapules, partially fused into large plaques, overlaid with thin adhesive scales. The appearance of the two skin lesions did not match. The pathological manifestations of chronic eczema include excessive

or incomplete keratinization, obvious thickening of the spinous layer, thickening of the superficial capillary walls of the dermis, and thickening of collagen fibers. However, the pathological examination of the patient, in this case, revealed a symbol of acantholysis, and the pathological phenomena of them are different; follicular keratosis is an autosomal dominant disorder that often develops in childhood, usually caused by sun exposure and other triggers. The lesions are mainly tan-colored warty papules or plaques that occur in seborrheic areas, such as the scalp, forehead, forehead, and axilla. The pathological manifestations of Darier's disease include hyperkeratosis with focal parakeratosis and mild acanthosis. Suprabasal cleft, single dyskeratotic in all layers of the epidermis (corps ronds, grains). Immunofluorescence investigations are negative [12]; about 2/3 of patients with chronic familial benign pemphigus have a clear family history and occur mainly in the folds of the neck, axilla, and groin. The lesions are mainly recurrent blisters or large blisters with thin walls, easily broken crusts, and gradually expanding to form a ring around the skin [13]. The diagnosis of follicular keratosis and chronic familial benign pemphigus was not consistent with the age of onset, rash distribution, lesion morphology, and family history of this patient, so they are excluded; the patient's rash morphology, histopathological examination, and multiple direct and indirect immunofluorescence were negative, so autoimmune bullous disease was not considered.

The patient's condition is different with multiple domestic and foreign reports [14] [15] [16] [17], who has extensive skin lesions, a long course of disease and recurrent seizures. The appearance of the lesion evolves from acute eczema-like, blister exudation chronic to hypertrophic plaques and scales. Multiple pathological examinations all found acantholysis. The evolution from Darier's disease-like to Hailey-hailey like type suggests that the morphology of the lesion and pathological morphology of this disease are not fixed and unchanged, but have variable and transformative properties.

In this case, the patient has a past history of psoriasis, which may be one of the triggers for the development of the disease. Many skin conditions have been reported in the literature that are also associated with transient acantholytic dermatoses, including dry eczema, allergic contact dermatitis, and atopic dermatitis [18]. This disease also sometimes occurs after skin infections resolution, including lichen planus and scabies [19]. There are also some studies indicating that GD may be a paraneoplastic disease, so the occurrence of GD may be related to hematological malignancies [20].

Although the disease has a tendency to heal spontaneously, this patient had a severe condition and first-line treatment included topical glucocorticoids and vitamin D analogs with antihistamines [21]. Sun exposure and strenuous exercise should be avoided. The patient's hypertrophic rash was given antihistamines in combination with acitretin and topical fusidic acid cream and halometasone ointment to obtain a more lasting improvement.

The long-term prognosis remains unclear as transient acantholytic dermatoses are less common clinically.

Acknowledgements

Authors' contributions: P.X. and X.Y.Z. designed the case report and helped to modify the manuscript. S.X.S. drafted the manuscript. T.L.L. and Y.Y. prepared the figures. All authors read and approved the final manuscript.

Informed consent: The patient's written consent has been signed/obtained.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

[1] Heenan, P.J. and Quirk, C.J. (1980) Transient Acantholytic Dermatosis. *British Journal of Dermatology*, **102**, 515-520. https://doi.org/10.1111/j.1365-2133.1980.tb07648.x

[2] Brihan, I., *et al.* (2022) Clinicopathological Correlation of Transient Acantholytic Dermatosis: A Case Report. *Experimental and Therapeutic Medicine*, **23**, Article No. 173. https://doi.org/10.3892/etm.2021.11096

[3] Held, J.L., Bank, D. and Grossman, M.E. (1988) Grover's Disease Provoked by Ionizing Radiation. *Journal of the American Academy of Dermatology*, **19**, 137-138. https://doi.org/10.1016/S0190-9622(88)80229-9

[4] Scheinfeld, N. and Mones, J. (2006) Seasonal Variation of Transient Acantholytic Dyskeratosis (Grover's Disease). *Journal of the American Academy of Dermatology*, **55**, 263-268. https://doi.org/10.1016/j.jaad.2006.01.029

[5] Motaparthi, K. (2017) Pseudoherpetic Transient Acantholytic Dermatosis (Grover Disease): Case Series and Review of the Literature. *Journal of Cutaneous Pathology*, **44**, 486-489. https://doi.org/10.1111/cup.12909

[6] Parsons, J.M. (1996) Transient Acantholytic Dermatosis (Grover's Disease): A Global Perspective. *Journal of the American Academy of Dermatology*, **35**, 653-666. https://doi.org/10.1016/S0190-9622(96)90715-X

[7] Fantini, F., Kovacs, E. and Scarabello, A. (2002) Unilateral Transient Acantholytic Dermatosis (Grover's Disease) along Blaschko Lines. *Journal of the American Academy of Dermatology*, **4**, 319-320. https://doi.org/10.1067/mjd.2002.120596

[8] Davis, M.D., Dinneen, A.M., Landa, N. and Gibson, L.E. (1999) Grover's Disease: Clinicopathologic Review of 72 Cases. *Mayo Clinic Proceedings*, **74**, 229-234. https://doi.org/10.4065/74.3.229

[9] Fernandez-Figueras, M.T., *et al.* (2010) Grover Disease: A Reappraisal of Histopathological Diagnostic Criteria in 120 Cases. *The American Journal of Dermatopathology*, **32**, 541-549. https://doi.org/10.1097/DAD.0b013e3181c80cf9

[10] Weaver, J. and Bergfeld, W.F. (2009) Grover Disease (Transient Acantholytic Dermatosis). *Archives of Pathology & Laboratory Medicine*, **133**, 1490-1494. https://doi.org/10.5858/133.9.1490

[11] Yang, Z.B., Liu, Q., *et al.* (2018) Consensus of TCM Diagnosis and Treatment Experts on Eczema (Eczema) (2016). *Chinese Journal of Dermatovenereology of Integrated Traditional and Western Medicine*, **17**, 181-183.

[12] Xiao, Y., Lan, Y., *et al.* (2019) A Case of Dermatomyositis Presenting with Keratosis Follicularis-Like Lesions and Literature Review. *The Chinese Journal of Dermatovenereology*, **33**, 569-571.

[13] Chen, W.W., Zhang, D.W., *et al.* (2020) Advances in the Pathogenesis of Familial Chronic Benign Pemphigus. *The Chinese Journal of Dermatovenereology*, **34**, 466-469.

[14] Long, J., Yan, H., *et al.* (2018) A Case of Transient Acantholytic Dermatosis. *Journal of Clinical Dermatology*, **47**, 107-108.

[15] Chen, B.R. (2012) A Case of Transient Acantholytic Dermatosis. *The Chinese Journal of Dermatovenereology*, **26**, 724-725.

[16] Wiersma, G.L., Saavedra, A.P., Yang, F.C., Nandi, T.R., Levine, D. and Murphy, G.F. (2014) Pseudoherpetic Grover Disease: Report of 2 Cases and Review of the Literature. *The American Journal of Dermatopathology*, **36**, 746-750. https://doi.org/10.1097/DAD.0b013e31828b10c1

[17] Cohen, P.R., Paravar, T. and Lee, R.A. (2014) Epidermal Multinucleated Giant Cells Are Not Always a Histopathologic Clue to a Herpes Virus Infection: Multinucleated Epithelial Giant Cells in the Epidermis of Lesional Skin Biopsies from Patients with Acantholytic Dermatoses Can Histologically Mimic a Herpes Virus Infection. *Dermatology Practical & Conceptual*, **4**, 21-27. https://doi.org/10.5826/dpc.0404a03

[18] Grover, R.W. and Rosenbaum, R. (1984) The Association of Transient Acantholytic dermatosis with Other Skin Diseases. *Journal of the American Academy of Dermatology*, **11**, 253-256. https://doi.org/10.1016/S0190-9622(84)70160-5

[19] Kaddu, S., Mullegger, R.R. and Kerl, H. (2001) Grover's Disease Associated with *Sarcoptes scabiei*. *Dermatology*, **202**, 252-254. https://doi.org/10.1159/000051647

[20] Gantz, M., Butler, D., Goldberg, M., Ryu, J., McCalmont, T. and Shinkai, K. (2017) Atypical Features and Systemic Associations in Extensive Cases of Grover Disease: A Systematic Review. *Journal of the American Academy of Dermatology*, **77**, 952-957, E1. https://doi.org/10.1016/j.jaad.2017.06.041

[21] Aldana, P.C. and Khachemoune, A. (2020) Grover Disease: Review of Subtypes with a Focus on Management Options. *International Journal of Dermatology*, **59**, 543-550. https://doi.org/10.1111/ijd.14700