International Journal of Research in Dermatology
Mythri J et al. Int J Res Dermatol. 2026 Jul;12(4):346-350
https://www.ijord.com

DOI: https://dx.doi.org/10.18203/issn.2455-4529.IntJResDermatol20261935

Case Report

Transient acantholytic dermatosis mimicking pemphigus foliaceus: a report of 2 cases

J. Mythri*, B. Rekha Rani, K. V. T. Gopal, P. V. Krishnam Raju

Department of Dermatology, Maharajah's Institute of Medical Sciences, Vizianagaram, Andhra Pradesh, India

Received: 12 February 2026
Revised: 04 April 2026
Accepted: 18 May 2026

***Correspondence:**
Dr. J. Mythri,
E-mail: jagannadham.mythri@gmail.com

ABSTRACT

Transient acantholytic dermatosis (TAD) is a relatively uncommon acquired acantholytic disorder of unknown etiology, typically characterized by pruritic papulovesicular eruptions predominantly affecting the trunk, however atypical presentations can occur and pose diagnostic challenges, as illustrated in this report of two female patients aged 29 and 50 years who presented with rapidly evolving vesiculobullous lesions that ruptured to form erosions and extensive crusted plaques involving the scalp, face, neck, and trunk, thereby closely mimicking pemphigus foliaceus, while routine laboratory investigations including complete hemogram, serum biochemistry, thyroid profile, lipid profile, viral markers, and serum electrophoresis were within normal limits, histopathological examination revealed features of acantholysis with subcorneal blister formation in one case and suprabasal clefting in the other along with hyperkeratosis and mild dermal inflammatory infiltrate, and direct immunofluorescence studies were negative effectively ruling out autoimmune blistering disorders, based on detailed clinical evaluation and clinicopathological correlation a diagnosis of TAD was established, following which both patients were managed with parenteral corticosteroids, systemic antibiotics, saline soaks, and supportive care leading to near-complete resolution of lesions within a short duration, thus these cases highlight an unusual presentation of TAD with predominant head and neck involvement that can closely resemble pemphigus foliaceus, emphasizing that a high index of suspicion along with appropriate use of histopathology and immunofluorescence is essential for accurate diagnosis, differentiation from other acantholytic disorders, and timely institution of effective management while avoiding misdiagnosis and unnecessary prolonged therapy.

Keywords: Transient acantholytic dermatosis, Subcorneal blister, Acantholysis

INTRODUCTION

Transient acantholytic dermatosis also known as Grover's disease is characterized by the development of pruritic, papulovesicular eruptions over the trunk (anterior part of chest and upper part of back) and proximal. extremities. Heat, sweating, atopic dermatitis and asteatotic eczema have been thought to be aggravating factors.[1] Histological features include focal acantholysis, dyskeratosis and spongiosis.[2] It mostly affects Caucasians in the fifth decade or later and is more common in men. It is associated with numerous conditions, including chronic renal failure, HIV infection, mechanical irritation, prolonged bedrest, heat exposure, and excess sweating.[3] We report two cases of transient acantholytic disorder (TAD) who presented with extensive crusted plaques over the head and neck region which clinically mimicked pemphigus foliaceus.

CASE REPORT

Case 1

A 29-year-old female patient presented with crusted and raw areas over the body for 1 month. The lesions started as minute fluid filled lesions which ruptured spontaneously

within hours to form raw areas and crusting over scalp, retro-auricular area, neck, upper limbs, trunk and lower limbs. There was no history of itching or diarrhoea, photosensitivity, drug intake, application of any topical irritants prior to onset of lesions. No relevant past history and family history non-contributory. On dermatological examination, crusted plaques with scaling were seen over neck and scalp (Figures 1 and 2) retro auricular area. Erosions, crusts, fissures were seen over peri umbilical region and abdomen.

Figure 1: Crusted plaques with scaling over neck.

Figure 2: Crusted plaques with scaling over scalp.

Based on above clinical picture differentials of Pemphigus foliaceous, Hailey hailey disease, Pemphigus vulgaris were considered. Complete hemogram, serum biochemistry was normal, viral screening for HBsAg, HCV, HIV were found nonreactive VDRL was negative and imaging studies including chest X-ray, ultrasound abdomen was normal.

Histopathological examination showed the following changes in epidermis-thick crust (parakeratosis, serum, neutrophils) with sub corneal blister (Figure 3), granular layer is absent, moderate acanthosis with irregular elongation of rete ridges and basal layer is intact, upper dermis showed dilated vessels, mild perivascular lymphocytic infiltrate along with neutrophils and few eosinophils. The middle and lower dermis are unremarkable. Direct immunofluorescence (DIF) was negative for- IgG, IgA, IgM and C3c.

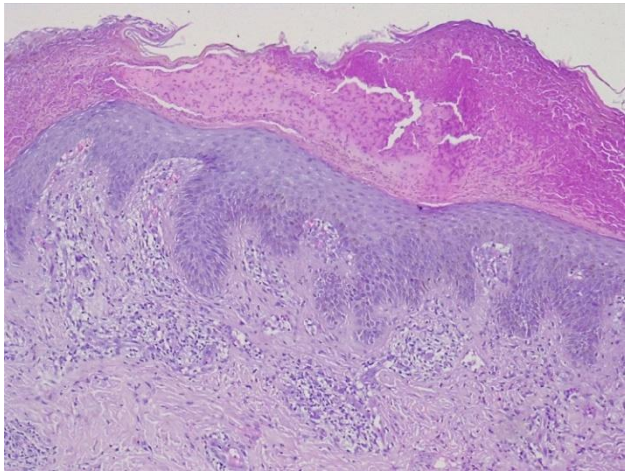

Figure 3: Sub corneal blister.

Case 2

A 50-year-old female presented with redness and flaking of skin over scalp, face, lower back for 15 days initially lesion started as flaccid bullae which rupture in few hours to form raw areas over scalp. Later similar lesions developed over face, upper back, lower back and associated with itching. No involvement of oral mucosa, genital mucosa, No involvement of palms and soles. no history of itching or diarrhoea, photosensitivity, drug intake, application of any topical irritants prior to onset of lesions. No relevant past history and family history non-contributory. On dermatological examination diffuse erythema, scaling and crusting seen all over the face (Figure 4).

Similar lesions are seen over scalp, sides of neck and upper back (Figure 5). Erosions seen over left pre-auricular area, Lichenified plaque with irregular shape and ill-defined margins with erosions, crusts seen over lower back and sacral region. Based on above clinical picture Differentials of pemphigus foliaceous, pemphigus vulgaris, acute cutaneous lupus erythematosus, psoriasis was considered. Complete hemogram, serum biochemistry was normal, viral screening for HBsAg, HCV, HIV were found nonreactive VDRL was negative and imaging studies including chest X-ray, ultrasound abdomen was normal.

Figure 4: Diffuse erythema, scaling and crusting all over the face.

Figure 5: Diffuse erythema, scaling and crusting over the upper back.

Histopathological examination showed the following changes in epidermis stratum corneum mild compact hyperparakeratosis along with serum, cocci and eosinophils (Figure 6), stratum granulosum is preserved, stratum spinosum shows moderate acanthosis with irregular elongation of rete ridges.

In addition, there is evidence of focal supra basal blister along with acantholysis, stratum basalis is intact. The superficial dermis shows dilated blood vessels and mild peri vascular inflammatory infiltrate, composed of eosinophils and lymphocytes. The deep dermis is unremarkable. focal supra basal blister formation along with acanthosis, intact basal layer and unremarkable changes in dermis along with negative DIF. Based on clinicopathological correlation both the cases were diagnosed as TAD.

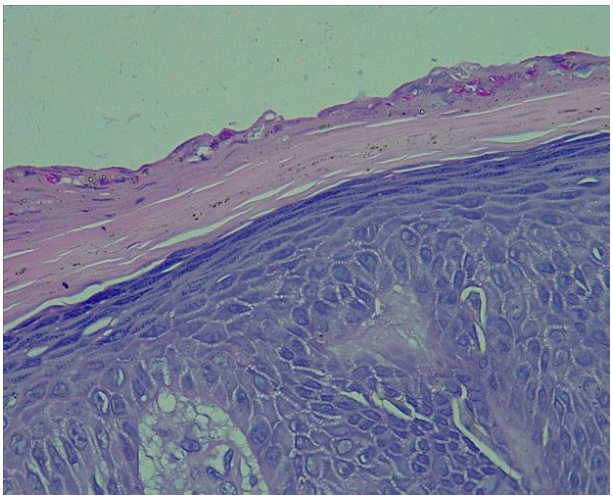

Figure 6: Hyperparakeratosis along with eosinophils.

Follow up of the case

In both cases oral systemic antibiotics, anti-histamines, topical corticosteroids and antibiotic combination cream and supportive treatment were given resulting in complete resolution of lesions in 15 days (Figure 7).

Figure 7 (A-D): Post treatment images of case 1 and case2 with complete resolution.

DISCUSSION

TAD is a rare acquired acantholytic disorder of unknown cause characterized by pruritic papules and papulovesicles distributed mainly on the trunk. Sun exposure, heat, sweating, and ionizing radiation are common predisposing factors. Men are more frequently affected than women, with the reported male-to-female ratio ranging from 3:1 to 7:1.[4] Most patients are older than 40 years of age. The cause of TAD is unknown.

A recent study has demonstrated autoantibodies against a number of proteins involved in keratinocyte development, activation, growth, adhesion and motility using proteomic microarrays to analyse immunoglobulin A (IgA) and IgG autoantibodies.[5] It is still unclear whether this auto-antibodies are causative or are a reaction to the damage to

keratinocytes seen in this disease. Both our cases were females aged 29 and 50 years who were agricultural workers exposed to long hours of UV radiation and hot, humid climate which may be the predisposing factor for development of the blistering eruption.[6]

Five distinct histologic patterns are present in the biopsy specimens: The commonest pattern, showing supra basal acantholysis resembling Pemphigus vulgaris, is most frequently seen in patients who tend to have more persistent eruptions. Other patterns are: PF like with superficial acantholysis, Darier-like in which there is focal acantholysis and dyskeratosis overlying suprabasilar clefts, Hailey-Hailey-like, in which there are numerous acantholytic cells overlying suprabasilar clefts and finally, spongiotic, in which a few acantholytic cells are present within, or contiguous with, spongiotic foci. Some cases show a predominance of one pattern, but more frequently two or more of these patterns can be found in a single biopsy specimen.[7] In the present cases subcorneal cleft formation was observed in case 1, whereas supra basal blister formation along with acantholysis were seen in case 2, hence case 1 and 2 had PF and PV like histological pattern type respectively.

The primary lesions are usually a few, discrete, erythematous, edematous, acneiform, red-brown- or flesh-coloured papules, or papulovesicles, with some appearing to be follicular, rarely bullous lesion. Unusual distributions, including unilateral or zosteriform eruptions, have also been reported as dermatological presentations of TAD.[8] Lesions may resolve with post inflammatory pigmentation. No systemic symptoms are associated with TAD, but oral lesions can develop as slightly painful aphthae. Skin lesions are accompanied by pruritus, with variable degrees of itching from mild to severe. Patients with severe pruritus typically present with multiple, disseminated lesions affecting the neck, shoulders, trunk, arms and leg.[9]

Pruritus of variable intensity occurs in almost all patient. The disorder may be transient which spontaneously resolves within weeks to months, persistent which lasts for even a few years, or recurrent. While the disease is active, there may be considerable fluctuation of the extent and severity of the eruption. Palms and soles are usually not affected.10 In the present cases, skin lesions were predominantly seen over head and neck region and completely resolved within 3 weeks which favoured transient type of TAD.

Patients are advised to avoid exposure to exacerbating factors such as excessive sun exposure, sweating, occlusive fabrics, and heat. Topical therapy is indicated for mild cases and involves use of a potent topical steroid and symptomatic treatment of pruritus. Systemic therapy becomes necessary when the eruption is severely pruritic and extensive and persists despite topical therapy. Oral vitamin A is often effective in a dose of 50,000 three times a day for 2 weeks, reduced to 50,000 IU daily for up to 12 weeks. Isotretinoin (40 mg daily tapered to 10 mg daily administered for 12 weeks) and acitretin (0.5 mg/kg per day) have also been found to be beneficial.

Oral corticosteroids are effective, with triamcinolone (16 mg daily tapered to 2 to 4 mg daily) reported to be more effective than prednisolone. In the present case, autoimmune bullous disorder was ruled out based on negative DIF and diagnosis of TAD was made based on clinic pathological correlation. In both our cases, rapid involvement with complete resolution of lesions were achieved in 3 weeks with use of oral steroids, topical corticosteroids and supportive treatment.

CONCLUSION

Meticulous clinical examination, laboratory investigations including HPE and DIF study helped in ruling out autoimmune blistering diseases and confirmed diagnosis of TAD. Patients with TAD should be counselled regarding adequate precautionary measures to be taken in the season with high temperatures in view of its summer exacerbations. The present cases highlights presentation of TAD with extensive involvement over head and neck region clinically resembling pemphigus foliaceus where pinpoint diagnosis was achieved with immune-fluorescence study.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Das A, Sil A, Khullar G. Transient dermatoses: leaving a lasting memory. Indian J Dermatol Venereol Leprol. 2022;88(2):265-7.
2. Meshoyrer D, Granda E, Sharma K. Grover's disease: an uncommon diagnosis and a simple fix. J Am Med Dir Assoc. 2024;25(5):B6-7.
3. Brihan I, Fekete GL, Turda C, Tica O, Venter AC, Ianosi S, et al. Clinicopathological correlation of transient acantholytic dermatosis: a case report. Exp Ther Med. 2022;23(2):1-6.
4. Chalet M, Grover R, Ackerman AB. Transient acantholytic dermatosis: a reevaluation. Arch Dermatol. 1977;113(4):431-5.
5. Sousou JM, Fritsche JM, Fernandez BR, Tummala MR, Scott R, Fernandez B, et al. Management and treatment of Grover's disease: a case report and review of literature. Cureus. 2022;14(4):e24067.
6. Joshi R, Taneja A. Grover's disease with acrosyringeal acantholysis: a rare histological presentation of an uncommon disease. Indian J Dermatol. 2014;59(6):621-3.
7. Kaprive JF, Washburn S, Emerson CM, Mullins T. Successful treatment of resistant Grover's disease with dupilumab. Int J Womens Dermatol. 2024;10(2):e140.
8. Mansur AT, Demirkesen C. Transient acantholytic dermatosis: new dermatoscopic features. Indian J Dermatol. 2021;66(6):683-5.

9. Grover RW, Rosenbaum R. The association of transient acantholytic dermatosis with other skin diseases. J Am Acad Dermatol. 1984;11(2 Pt 1):253-6.
10. Galamgam J, Lee DJ. Treatment of transient acantholytic dermatosis with liquid nitrogen. JAAD Case Rep. 2020;6(4):341-3.

Cite this article as: Mythri J, Rani BR, Gopal KVT, Raju PVK. Transient acantholytic dermatosis mimicking pemphigus foliaceus: a report of 2 cases. Int J Res Dermatol 2026;12:346-50.