Case Report

Grover’s disease: a case report
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Abstract
Grover’s disease, a non-immune acantholytic dermatosis affects mostly adult males over 50 years of age. We present this case as it has not been reported in Pakistani medical literature before and the disease started under 50 years of age in a male patient.

Key words
Grover’s disease, transient acantholytic dermatosis, persistent acantholytic dermatosis.

Introduction
Grover's disease, a non-immune acantholytic dermatosis, is also called transient and persistent acantholytic dermatosis. It is clinically characterized by pruritic papules and vesicles, mainly distributed on the trunk, that affects mostly adult males over 50 years of age. The exact etiology is not known but is precipitated by sweat, fever, heat and sunlight in the genetically predisposed people. Histologically, the disease is characterized by focal acantholytic dyskeratosis.

Case report
A 26-year-old man presented in dermatology OPD with history of recurrent self-healing itchy rash, aggravated in summers, on his trunk, back and arms for the last 3 years. On examination, he was a young healthy male of normal built. He had papules and vesicles over his trunk, back and arms (Figures 1-3). No fluid level was present in the vesicles. His baseline laboratory investigations were normal. In the skin biopsy (Figure 3), epidermis had hyperkeratosis and few intraepidermal clefts. Acantholytic cells were present inside the cleft. This was Darier disease-like picture. There was a dermal inflammatory infiltrate present. Direct immunofluorescence was negative. On the basis of history, examination and biopsy, a diagnosis of Grover’s disease was made. He was advised topical betamethasone dipropionate, which improved his symptoms.

Discussion
Transient and persistent acantholytic dermatosis was first described by Grover in 1970 as a distinctive entity. Clinically, it is characterized by pruritic papules and vesicles, usually occurring on chest, back, and thighs of elderly men. The lesions are usually transient, lasting for days to weeks, but can, in some cases persist for years.

The etiology of Grover's disease is unknown. Triggering factors include sweat, fever, heat, sunlight, xerosis, drugs (sulfadoxine-pyrimethamine, recombinant
human interleukin 4), irradiation, infectious agents (Malassezia furfur, Demodex folliculorum) and immunosuppression (AIDS, immunosuppressive therapy). It also occurs in the febrile bedridden patients and during the post-operative period, with regression of the disease after resumption of normal activity. In our patient, the triggering factor was heat and sunlight in summer season. All these precipitating factors are postulated to act on the genetically predisposed epidermis to cause the disease. Some reports have hypothesized causation by poral occlusion of damaged eccrine in intraepidermal ducts, with spillage of sweat contents and focal acantholysis. Bed confinement can induce an occlusive environment that would elicit the disease. Different other studies have shown that the primary damage is in the proteins of the desmosomal attachment plaque, such as desmoplakin I, II and plakoglobin. Electron microscopy have shown intradesmosomal separation, decreased number of desmosomes and perinuclear aggregation of tonofilaments. These plaque proteins were reported to be dissolved and diffused into the acantholytic cells.

Histologically, the hallmark of the disease is focal acantholysis in the epidermis, at a suprabasal or subcorneal level. Focal acantholysis may be present in the same specimen in four different forms i.e. pemphigus-like, Hailey-Hailey disease-like, Darier's disease-like, and spongiotic. The Darier-like pattern is most commonly seen, especially in persistent cases. Cutaneous adnexa are not usually affected. Spongiosis, dyskeratosis and hypergranulosis also may be seen. Dermis contains an inflammatory infiltrate composed of lymphocytes, histiocytes and eosinophils. Direct immunofluorescence is classically negative which helps to differentiate it from other disorders.
Grover's disease has been reported in association with other diseases like internal malignancy (both solid and hematological) in 25% of patients. Other sporadic associations include human immunodeficiency virus infection, scabies, pyoderma gangrenosum, asteatotic eczema and allergic contact dermatitis, chronic renal failure and haemodialysis.

Clinical differential diagnosis include dermatitis herpetiformis, folliculitis, arthropod reaction, papular urticaria (prurigo simplex), miliaria rubra (history of exacerbation with heat; often more nodular lesions), papular drug eruption, disseminated herpes simplex or herpes zoster, scabies, papular pityriasis rosea, secondary syphilis (scaly, with palmar lesions, pruritus mild or absent).

In mild cases, symptomatic treatment of pruritus and topical steroids usually help. Calcipotriol, retinoids and PUVA have all been proved useful. So, Grover's disease is a common disorder and must be included in the differential diagnosis of papulovesicular disorders. A high index of suspicion for this disease is necessary if the diagnosis is to be made correctly.

References