Bullous scabies presenting as bullous pemphigoid - a case report

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Abstract
Scabies is a common parasitic infestation in our population which can affect individuals of all ages. More than 300 million cases of scabies are reported annually. It is caused by a mite called Sarcoptes Scabiei. Common clinical manifestations include classic burrows, papules, nodules, crusts, excoriations and rarely vesicles and bullae. We report a bullous scabies case of a 60 years old man with more than 3 months history of itchy vesiculobullous lesions, mimicking bullous pemphigoid.

Key words
Bullous, scabies, bullous pemphigoid.

Introduction
Scabies caused by parasite Sarcoptes Scabiei, usually spreads with physical contact commonly presenting as papules, burrows, nodules, excoriations and crusts. Vesicles and bullae are less common. The lesions are usually present on flexor aspect of wrists, lateral aspects of fingers, finger and toe webs, elbows, external genitalia, umbilicus, buttocks, axillae and areola of breasts.1 The common risk factors for scabies include overcrowding, immigration, poor hygiene, poor nutritional status, homelessness, dementia, close contact and immunocompromised state. The typical symptom is intractable, generalized pruritus that is worse at night. Elderly patients occasionally develop crusted scabies, also known as Norwegian scabies.2 It may present as folliculitis, impetigo, eczema and urticaria.3,6

A rare subtype is bullous scabies which resembles bullous pemphigoid and can even induce true lesions of bullous pemphigoid in affected patients.5

Case report
A 60 years old man presented with more than three months history of intensely pruritic papules starting from the genitals and later involving finger, toe webs, popliteal fossa and axillae (Figure 1). The itching was worse at night and family members were also affected. The patient started to develop multiple clear fluid filled vesicles which slowly enlarged to become tense bullae, after 2 to 3 days the bullae became flaccid and ruptured leaving behind crusted, hemorrhagic erosions and erythematous plaques (Figure 3, 4). There was eczematization between the finger and toe webs, generalized scaling of the back. Eczema involved the flexor surfaces of the arms, legs, inner side of thighs and genitals (Figure 2). Face, scalp and oral mucosae were spared. The Nikolsky’s sign was negative. Rest of the general physical examination showed no significant finding. The patient had history of taking topical and systemic steroids mainly prednisolone, azathioprine and dapsone but with no relief.
Direct microscopy of the scrapings taken from the digital eczematized webs showed multiple mites.

Lab investigations showed Hb 12.6g, TLc 8000, HIV negative, HBsAg, Anti HCV were negative. Renal and Liver functions were normal. Patient was treated with oral ivermectin 6mg repeated after one week along with 10% Sulphur ointment for 3 days alternated with 5% permethrin lotion. Anti histamines were also given. His itching started to decrease after starting the treatment and bullae formation stopped after 5 days.

**Discussion**

Bullous scabies mostly occurs in immune compromised or elderly patients. In all the reviewed 45 cases (including this one) 31 were males and 14 females with age range 1-89, but most of the patients were above 60 years of age.

Bullae are highly itchy, they may be tense to flaccid, or some may have associated hemorrhage. They are mostly found on scabies prone sites with or without usual scabies lesions.

How the bullae are formed in scabies isn’t clear yet but multiple theories do exist in this regard.

According to one theory Staphylococcus aureus is found to be the culprit, causing superinfection of the existing scabies lesions leading to bullae formation.

The second theory proposes auto antibody mediated immune damage which can lead to formation of bullae. The mite enzymes can cause lysis of the basement membrane releasing antigens which are BP like antigens. These antigens interact with BP antibodies thus activating the compliment cascade. Eosinophils and lymphocytes are recruited and secretion of protease enzymes leads to dermo-epidermal cleavage.
The third concept is of antigen mimicry. The mite antigen cross reacts with BMZ antibodies. These antibodies behave like those present in original autoimmune diseases, causing autoimmune damage. A Western blot study revealed circulating antibodies against BP180 and BP 230 in the sera of two scabies patients with bullous eruption.7

The bullae may also form as an iderupture and in that case they are called scabid.3,5

Scabies and bullous pemphigoid both can occur independently in a patient. Scabies can illicit BP like lesions in prone areas via koebnerization.4

On direct immunofluorescence, complement component (C3) and IgG deposition is often seen in bullous scabies. In 25 cases where direct immunofluorescence was performed, 9 had IgG and 13 had C3 deposition. IgG deposition pattern was linear in 8 while granular in 3.4

The treatment of bullous scabies remains the same as typical scabies. The recommended medications for scabies are 5% permethrin cream/lotion, 10% crotamiton lotion/cream and ivermectin. Other antiscabietic agents that are benzyl benzocate, malathion, and 10% sulfur ointment.

In some cases of bullous scabies, a short course of oral steroids may also be required to stop new bullae formation. In serious cases or in cases of steroid intolerance, other immunosuppressive agents, such as methotrexate, azathioprine, mycophenolate mofetil and cyclosporine can be used.6

Conclusion

Bullous scabies should always be considered in differential diagnosis of any case of bullous eruption with papules and nocturnal itch. As there is no definite diagnostic criteria for BS except for the response to anti scabies treatment, so suspecting scabies in a patient who is not responding to immunosuppressive drugs is of paramount importance for proper treatment and prevention of recurrence.

References