

Gestational Pemphigoid: A Comprehensive Review of Clinical Features, Differential Diagnosis, and Management Strategies with Case Report

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Abstract

Gestational pemphigoid (GP) is a rare autoimmune blistering disorder that typically occurs in the second or third trimester of pregnancy. Characterized by pruritic, tense blisters primarily affecting the abdomen and extremities, GP can lead to significant discomfort and potential complications for both the mother and fetus. The condition is caused by the production of autoantibodies against basement membrane proteins, resulting in subepithelial blister formation. Differential diagnoses include other pregnancy-related dermatoses, such as pruritic urticarial papules and plaques of pregnancy (PUPPP) and drug-induced bullous eruptions. Histopathological findings typically reveal subepidermal vesicles and lympho-eosinophilic infiltrates, although direct immunofluorescence may be negative in some cases. Management primarily involves symptomatic treatment with corticosteroids and antihistamines, leading to favorable outcomes for most patients. This review highlights the clinical features, differential diagnoses, and management strategies for gestational pemphigoid, emphasizing the importance of early recognition and intervention to ensure maternal and fetal health.

Keywords: Gestational Pemphigoid, Autoimmune Blistering Disorder, Pregnancy Dermatoses, Subepithelial Blisters, Differential Diagnosis, Corticosteroids

Introduction

Gestational pemphigoid (GP) is a rare autoimmune blistering disorder that typically manifests during the later stages of pregnancy, affecting an estimated 1 in 50,000 pregnancies. Characterized by the development of pruritic, tense blisters primarily on the abdomen and extremities, GP can significantly impact maternal quality of life and fetal health. The condition arises from the production of autoantibodies targeting specific components of the skin's basement membrane, leading to subepithelial blister formation. While generally self-limiting and resolving postpartum, gestational pemphigoid poses challenges in diagnosis and management due to its clinical similarity to other pruritic dermatoses of pregnancy, such as pruritic urticarial papules and plaques of pregnancy (PUPPP) and drug-induced bullous eruptions.

Case Report

A 27-year-old primigravida, with no significant medical history, was admitted to the Obstetrics and Gynecology department of Liaquat University of Medical and Health Sciences, Jamshoro, Pakistan. The patient, who is currently 20 weeks pregnant, reported a history of generalized pruritus and the sudden onset of vesicular lesions over the past two weeks.

The patient initially noticed small, fluid-filled blisters on her abdomen, which rapidly spread to her thighs and back within a few days. She described the lesions as intensely itchy and painful, which affected her daily activities. There was no previous history of similar lesions or any autoimmune skin conditions, and she had not experienced any complications in her pregnancy thus far.

On physical examination, the patient exhibited numerous tense vesicles and bullae predominantly located on the abdomen, thighs, and upper arms (**figure 1**). The lesions appeared erythematous, with some showing signs of erosion due to scratching. The oral mucosa was spared, and the scalp, palms, and soles were also unaffected. The patient was hemodynamically stable, with no signs of systemic involvement. During her hospitalization, new lesions continued to develop, particularly on her upper back and shoulders. A few blisters ruptured spontaneously, leading to superficial erosions. Laboratory tests, including a complete blood count and liver function tests, were within normal limits.

The differential diagnoses included gestational pemphigoid and bullous drug eruptions. A skin biopsy was performed to aid in confirming the diagnosis.

Histopathological examination revealed sub-epidermal vesicles characterized by spongiosis and a lympho-eosinophilic infiltrate.

The patient was admitted for management and initiated on a regimen of topical and oral prednisolone at a dose of 30 mg/day, along with loratadine for symptomatic relief. She showed a favorable response to the treatment, with no new blister formation noted during her hospitalization. Consequently, the corticosteroid dosage was gradually tapered.

The patient was discharged after five days in stable condition and was scheduled for follow-up in the outpatient clinic. During her follow-up visits, she reported significant improvement and was monitored closely for any recurrence of symptoms.



Figure 1 :Several scattered erosions on the lower limb.

Discussion:

Gestational pemphigoid (GP) is a rare autoimmune blistering disorder that typically occurs during the second or third trimester of pregnancy. It is characterized by the development of pruritic, vesicular lesions that can progress to bullae, primarily affecting the abdomen and extremities while sparing mucous membranes and the scalp. The pathophysiology of GP involves the production of autoantibodies against components of the basement membrane zone, particularly BP180 and BP230, leading to subepithelial blister formation.

Patients with gestational pemphigoid often present with intense itching and the appearance of tense blisters. The lesions may start in the periumbilical region and can spread to other areas of the body. The condition is self-limiting, with spontaneous resolution typically occurring postpartum. However, it can lead to complications such as prematurity and low birth weight due to placental insufficiency.

The differential diagnosis for GP includes other pruritic dermatoses of pregnancy, such as pruritic urticarial papules and plaques of pregnancy (PUPPP), as well as drug-induced bullous eruptions. Drug-associated bullous pemphigoid (DABP) can present similarly, with the distinction being that DABP is linked to specific medications and may occur in patients with no prior history of blistering diseases.

Histopathological examination of skin biopsies in cases of GP typically reveals subepidermal vesicles with spongiosis and a lympho-eosinophilic infiltrate. Direct immunofluorescence may show linear deposits of IgG and complement C3 along the basement membrane zone, although it can be negative in some cases. This variability can complicate the diagnosis and necessitate careful clinical correlation.

Management of gestational pemphigoid primarily involves symptomatic treatment with corticosteroids. Topical and systemic corticosteroids, such as prednisolone, are commonly used to control symptoms and reduce inflammation. Antihistamines may also be prescribed to alleviate itching. The prognosis for both the mother and the infant is generally favorable, with most patients experiencing resolution of symptoms after delivery.

Conclusion

Gestational pemphigoid is a significant condition that requires prompt recognition and management to ensure maternal and fetal well-being. Understanding the clinical features, differential diagnoses, and appropriate treatment strategies is essential for healthcare providers managing pregnant patients with blistering skin disorders.

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