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Case Report

Pemphigoid gestationis in a primigravida: autoimmune dermatosis of pregnancy — a rare case report

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ABSTRACT

Pemphigoid gestationis (previously termed pemphigus gestationis) is a rare autoimmune vesiculobullous dermatosis unique to pregnancy, with an estimated incidence of approximately 1 in 50,000 pregnancies. It is mediated by maternal IgG autoantibodies directed against BP180 (collagen XVII), resulting in complement activation and subepidermal blister formation. The disease typically presents during the second or third trimester with severe pruritus followed by erythematous urticarial plaques and tense bullous lesions. We report the case of a 22-year-old primigravida at 34 weeks and 3 days of gestation who presented with generalized pruritus and multiple vesiculobullous skin lesions involving the abdomen, trunk, upper limbs, thighs, and face. Histopathology and direct immunofluorescence confirmed the diagnosis of pemphigoid gestationis. The condition was complicated by fetal growth restriction and abnormal Doppler findings, necessitating preterm emergency caesarean delivery. The patient was managed with systemic corticosteroids and multidisciplinary care, resulting in favorable maternal and neonatal outcomes. Although uncommon in primigravida women, pemphigoid gestationis should be considered in the differential diagnosis of pregnancy-associated pruritic dermatoses. Early diagnosis using histopathology and direct immunofluorescence, along with prompt treatment and close fetal surveillance, is essential to improve fetomaternal outcomes.

Keywords: Pemphigoid gestationis, Autoimmune blistering disorder, Pregnancy dermatoses, Vesiculobullous lesions

INTRODUCTION

Pemphigoid gestationis, historically referred to as pemphigus gestationis, is a rare autoimmune blistering disorder specifically associated with pregnancy and the puerperium. It belongs to the spectrum of autoimmune subepidermal bullous diseases and is characterized by circulating IgG autoantibodies directed against basement membrane zone antigens, predominantly BP180 (collagen XVII).[1,2] Binding of these antibodies activates complement pathways, especially complement component C3, leading to inflammation, eosinophilic infiltration, and eventual separation at the dermoepidermal junction with formation of tense subepidermal blisters.

The condition most commonly presents during the second or third trimester, though onset may occur at any stage of pregnancy or even in the postpartum period. Clinically, the disease typically begins with intense pruritus and erythematous urticarial papules or plaques around the periumbilical region, subsequently progressing to widespread vesiculobullous eruptions involving the trunk and extremities.[2,3] Facial and mucosal involvement is uncommon.

The diagnosis of pemphigus gestationis is often challenging because its initial manifestations may resemble more common pregnancy dermatoses such as polymorphic eruption of pregnancy (PUPPP), atopic eruption of pregnancy, or allergic drug reactions.

Histopathological examination usually demonstrates subepidermal blistering with eosinophilic infiltrates, while direct immunofluorescence remains the gold standard for diagnosis, showing linear deposition of C3, with or without IgG, along the basement membrane zone.[1,2]

Maternal morbidity is related to severe pruritus and discomfort, fetal complications include prematurity, low birth weight, fetal growth restriction, and neonatal skin lesions in some cases. Early recognition and multidisciplinary management by obstetricians and dermatologists are essential.[5,6] Treatment primarily includes systemic corticosteroids, antihistamines, and supportive skin care, with therapy individualized according to disease severity.

The present case is notable because of the occurrence of pemphigoid gestationis in a young primigravida with severe cutaneous involvement complicated by fetal growth restriction and abnormal Doppler findings necessitating preterm emergency caesarean delivery.

CASE REPORT

A 22-year-old, booked, primigravida at 34 weeks + 3 days of gestation presented to the outpatient department of tertiary care hospital with complaints of generalized itching and multiple fluid-filled skin lesions. The patient initially developed severe pruritus during the seventh month of pregnancy, predominantly over the abdominal region. Over the following weeks, erythematous urticarial lesions appeared and progressively evolved into vesicular and bullous eruptions of ~ 1×1 cm involving the abdomen, trunk, upper limbs, thighs, and face. The lesions were associated with intense itching, burning sensation, and progressive discomfort, significantly affecting her quality of life.

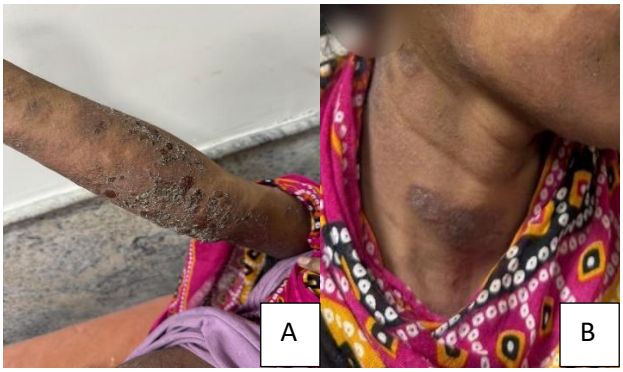

Figure 1 (A and B): Multiple erythematous hyperpigmented crusted plaques of varying size.

There was no history of previous dermatological disease, autoimmune disorder, drug intake, fever, photosensitivity, oral ulceration, or similar complaints in family members. Antenatal course prior to the onset of symptoms had been uneventful, and she had received regular antenatal care. There was no history of hypertension, diabetes mellitus, or thyroid disease. There was no significant family history.

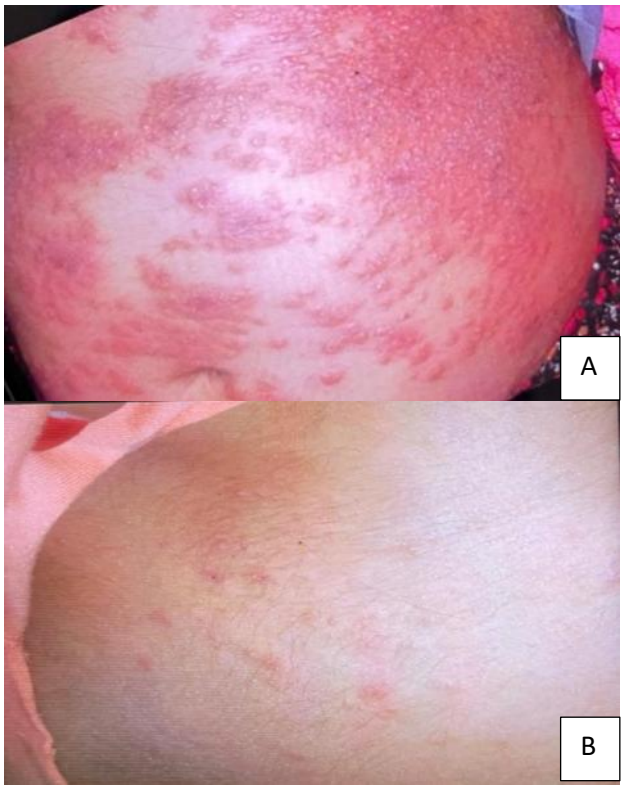

Figure 2 (A and B): Hive like lesions on abdomen.

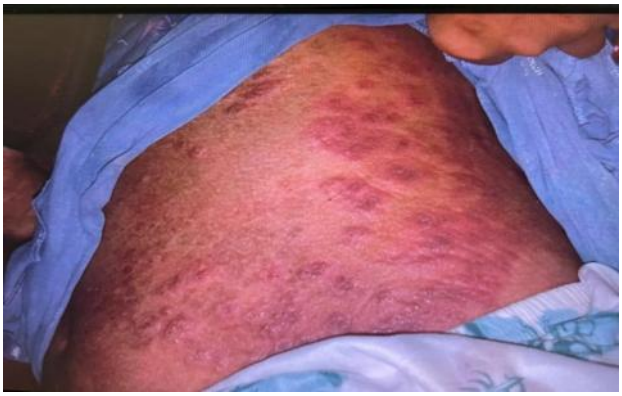

Figure 3: Bullous eruptions on abdomen.

On general physical examination, the patient was conscious, oriented, afebrile, and vitally stable. On examination there were multiple erythematous urticarial plaques, vesicles, and tense bullous lesions distributed over the abdomen, trunk, bilateral upper limbs, thighs, and scattered lesions over the face. Some lesions had ruptured, leaving behind ulcerated erosive areas with crusting. The lesions were intensely pruritic and associated with excoriation marks. Mucosal involvement was absent.

On local examination uterus corresponding to 32 weeks gestation with a singleton fetus in cephalic presentation.

Fetal heart rate was regular at approximately 136 beats per minute and uterus was relaxed

Routine laboratory investigations were within normal limits except for mild anemia. LFT (SGOT/SGPT – 22/9), RFT (UREA/CREATININE- 15/0.65), SERUM ELECTROLYTES (138/3.9/101), Obstetric ultrasonography with Doppler evaluation demonstrated fetal growth restriction with estimated fetal weight below the 10th percentile. Umbilical artery Doppler showed raised pulsatility index with reversal end-diastolic flow, suggestive of severe uteroplacental insufficiency and fetal compromise. Patient was managed with multi-disciplinary approach in HDU.

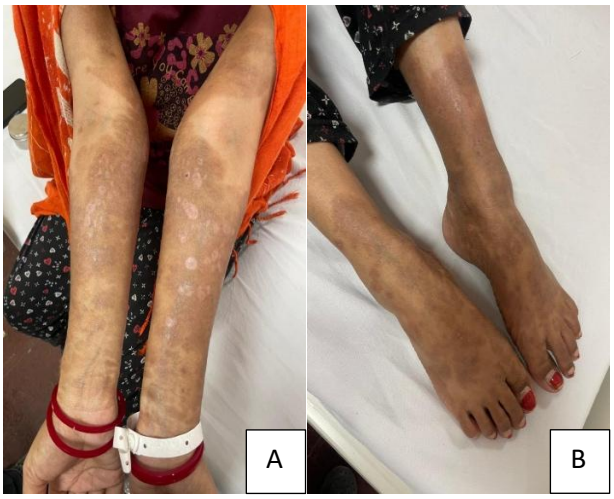

Figure 4 (A and B): Multiple hyperpigmented post-inflammatory plaques and resolving lesions over the bilateral upper and lower extremities.

Considering the progressive vesiculobullous lesions, dermatology consultation was obtained. A skin punch biopsy was taken from the left forearm. Histopathological examination revealed multifocal subepidermal blister formation with eosinophilic inflammatory infiltrates. Direct immunofluorescence demonstrated strong linear deposition of C3 and IgG along the dermoepidermal junction, confirming the diagnosis of pemphigoid gestationis.

The patient was started on systemic corticosteroid therapy with oral prednisolone 30mg once daily for 10 days which was then tapered off to 20mg once daily, along with antihistamines, emollients, and topical corticosteroid applications. Symptomatic improvement in pruritus was observed.

In view of severe fetal growth restriction with reversal of end-diastolic flow and evidence of acute fetal compromise, a decision for emergency preterm lower segment caesarean section was taken. A live preterm male baby weighing 1.4 kg was delivered with Apgar score of 7 and 9. No cutaneous lesions were noted in the neonate at birth., Intraoperative findings were otherwise unremarkable.

Skin lesions persisted initially but gradually improved with continuation and tapering of corticosteroid therapy. Wound healing was satisfactory, and no postoperative complications occurred. The patient was discharged in stable condition on post op day 5 with dermatology follow-up, advice regarding gradual steroid tapering, and counselling about the possibility of recurrence in future pregnancies and use of barrier contraception or IUCD insertion.

DISCUSSION

Pemphigoid gestationis is a rare autoimmune blistering disease unique to pregnancy and the puerperium. Despite its historical name “pemphigus gestationis,” it is immunologically and histopathologically distinct from pemphigus vulgaris and is more closely related to bullous pemphigoid.[1,2] The disease is mediated by maternal autoantibodies against BP180, a transmembrane hemidesmosomal protein essential for dermoepidermal adhesion. These antibodies activate the complement cascade, resulting in inflammatory cell recruitment, basement membrane disruption, and subepidermal blister formation.

The disease usually manifests during the second or third trimester; however, atypical presentations such as early onset in the first trimester or postpartum onset have been documented. The present case is unusual because of its occurrence in a primigravida with extensive cutaneous involvement and associated severe fetal growth restriction4. PG is more commonly reported in multigravida women, and recurrence in subsequent pregnancies tends to occur earlier and with increased severity.

Clinically, PG often begins with severe pruritus preceding the appearance of skin lesions. Initial urticarial papules and plaques later evolve into tense vesicles and bullae. Lesions classically originate in the periumbilical region and spread centrifugally to involve the trunk and extremities. Facial and mucosal involvement is relatively uncommon but may occur in severe disease. Differential diagnoses include polymorphic eruption of pregnancy (PUPPP), atopic eruption of pregnancy, erythema multiforme, dermatitis herpetiformis, and drug-induced eruptions.

Histopathology typically demonstrates subepidermal blister formation with eosinophilic infiltrates, while direct immunofluorescence is considered the diagnostic gold standard. Linear deposition of complement component C3 along the basement membrane zone is observed in nearly all cases, with IgG deposition seen variably1. In the present case, histopathology and direct immunofluorescence findings were classical and confirmed the diagnosis.

Maternal prognosis in PG is generally favorable, although symptoms may persist postpartum and relapses are

common during menstruation, subsequent pregnancies, or exposure to oral contraceptive pills. Fetal prognosis depends largely on disease severity. Adverse fetal outcomes including prematurity, low birth weight, fetal growth restriction, and rarely stillbirth have been associated with severe disease activity, likely secondary to placental involvement by autoantibodies4. Transient neonatal lesions due to passive antibody transfer occur in a minority of neonates.

CONCLUSION

Pemphigoid gestationis is a rare autoimmune dermatosis of pregnancy that should be considered in pregnant women presenting with severe pruritus and vesiculobullous eruptions. Although uncommon, especially in primigravida patients, delayed diagnosis can lead to significant maternal morbidity and adverse fetal outcomes. Direct immunofluorescence remains the gold standard for diagnosis, and early initiation of corticosteroid therapy plays a key role in disease control. Careful antenatal monitoring is essential because of the increased risk of fetal growth restriction, placental insufficiency, preterm delivery, and low birth weight. This case emphasizes the need for a multidisciplinary approach involving obstetricians, dermatologists, pediatricians, and pathologists to optimize maternal and neonatal outcomes. Counseling regarding recurrence risk in future pregnancies and the possibility of postpartum exacerbations is also an important component of long-term patient care.

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