

Urticarial Lesions in a Pregnant Woman

Dear Editor,

Gestational pemphigoid (GP) is a rare autoimmune bullous dermatosis in pregnancy. GP usually occurs during the second or third month of pregnancy. It clinically manifests as the development of either early-onset urticarial lesions or late-onset subepidermal blisters that may linger for weeks or even months. Herein we report the case of a 45-year-old woman with the distinctive clinical onset of GP.

A forty-five-year-old woman, gravida I, para 0, at 27 weeks gestation, was referred for evaluation to our Department with an extensive pruritic eruption that had developed over the previous 7 days. The lesions had first appeared on the proximal thighs and extended progressively to the abdomen.

On physical examination, numerous round urticarial plaques of approximately 1 cm in diameter were noted on her abdomen, involving the periumbilical area. Her thighs and back were also affected (Figures 1 and 2). The palms and soles were spared. No mucosal involvement was seen.

The patient medical history was unremarkable, and she denied use of any other medications or herbal remedies at the time the symptoms started or since. No other symptoms but pruritus were referred.

Laboratory studies, including complete blood cell count, coagulation tests, and renal and hepatic func-

tion were all normal. A punch biopsy was taken from an urticarial plaque and stained with hematoxylin and eosin. Histological examination found spongiosis in combination with an intraepidermal eosinophilic infiltrate, without the development of blisters (Figure 3). Direct immunofluorescence of perilesional skin showed linear deposition of complement (C3) along the basement membrane zone (Figure 4).

Serum antibody titers for BP180NC16a were detected by enzyme-linked immunosorbent assay (ELISA). We established a diagnosis of gestational pemphigoid.

Our patient was treated with systemic glucocorticoids, no blisters developed, and lesions cleared 8 weeks after delivery. The newborn girl did not develop neonatal gestational pemphigoid.

Gestational pemphigoid, originally misnamed herpes gestationis, is a rare autoimmune bullous dermatosis in pregnancy. Single cases have been also described in patients with molar pregnancies and trophoblastic tumors (1). Its etiology is based in the development of autoantibodies against the fetoplacental unit, triggering an autoimmune response against both skin and amnion hemidesmosomal proteins, mainly BP180, but also BP230 and type VII collagen. An association with HLA-DR3 and HLA-DR4 has been described (2).



Figure 1. Numerous round urticarial plaques on the abdomen.



Figure 2. Numerous round urticarial plaques on the thighs.

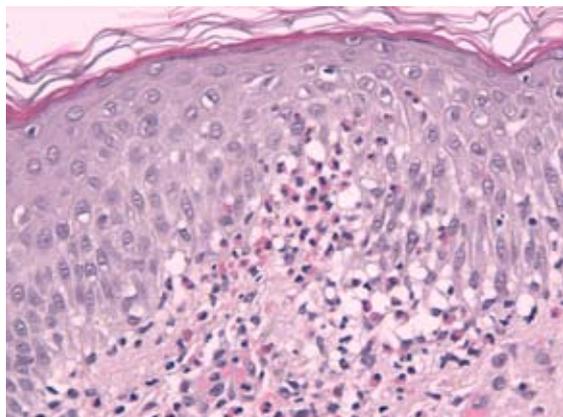


Figure 3. Cutaneous biopsy revealed eosinophilic spongiosis (hematoxylin and eosin, original magnification $\times 40$).

GP usually occurs during the second or third month of pregnancy, but it may appear at any time during pregnancy or puerperium. In the vast majority of cases, symptoms alleviate a few weeks before delivery, but they reemerge at the time of delivery. Recurrences are frequent in following pregnancies, with an earlier onset and more severe symptoms, and may occur during subsequent menstruations or hormonal contraceptive use (1).

GP clinically consists of the development of either early-onset urticarial lesions or late-onset subepidermal blisters that may linger for weeks or even months. They generally appear on the abdomen, specifically in the periumbilical area, with posterior widespread extension to proximal limbs. Facial and mucosal lesions are uncommon (1).

Histopathological studies are necessary to establish the diagnosis. These findings vary depending on the stage and severity of the disease and include subepidermal blisters, papillary dermal edema, eosinophilic spongiosis, and a polymorphous perivascular inflammatory cell infiltrate with a predominance of eosinophils. Direct immunofluorescence of perilesional skin shows a linear deposition of C3 along the basement membrane zone in all cases. IgG deposits can also be seen (3). These deposits are located within the lamina lucida and localized to the proximal part of anchoring filaments of the epidermal fragment of salt-split skin (4). Moreover, immunoblot and ELISA of the NC16a domain of BP180 RP are highly sensitive diagnostic methods in GP (5).

The aim of treatment is to alleviate the pruritus and prevent formation of new blisters. Topical corticosteroids and oral antihistamines may be used in mild cases. Systemic corticosteroids are the treatment of choice in moderate to severe cases. Other treatments that have been used are cyclophosphamide, dapsone, gold, methotrexate, and plasmapheresis (5).

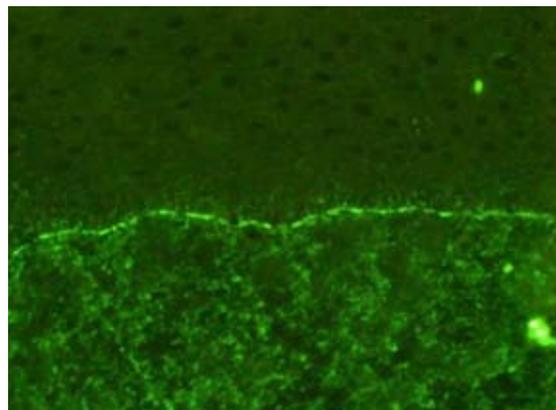


Figure 4. Direct immunofluorescence examination of perilesional skin showing linear staining with C3 at the basement membrane.

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