

What is your diagnosis?

A 25-year-old primigravida presented at 35 weeks of gestation with progressive, severe, bilateral pitting, lower limb edema with excessive weight gain. This was associated with itching, scaling, weeping, crusting, ulceration, and discoloration. She had no history of varicose veins or treatment for venous disorders. Her general examination findings were unremarkable. The skin of both lower limbs showed eczematous scales, oozing, crusts, and brownish pigmentation that involved the dorsum of both feet and ankles up to the knees with scattered areas of ulceration (Figure 1). All her prenatal investigation results were within normal limits, and a color duplex examination of the lower limbs ruled out any vascular disorder. Treatment included elevation of the legs, intermittent compression therapy, and application of mid-potency steroids and systemic and topical antibiotics. However, the skin lesions progressed to mimic cellulitis. At 38 weeks of gestation, a decision of pregnancy termination was taken, which was followed by a dramatic improvement within 1 week after cesarean section (Figure 2).



Figure 1. The skin of both lower limbs shows the typical picture of severe stasis dermatitis (eczematous, crusts, and brownish discoloration) that involved the dorsum of both feet and ankles up to the knees with scattered areas of ulceration.



Figure 2. One week after cesarean section, both lower limbs showed dramatic improvement of stasis dermatitis with few residual areas of brownish discoloration due to hemosiderin deposition.



Answer

Lower extremity gestational edema (GE) is an almost universal finding in late pregnancy (i.e., physiologic), occurring secondary to increased venous pressure in the legs, the obstruction of lymphatic flow, and reduced plasma colloid osmotic pressure (1). Stasis dermatitis/eczema is an inflammatory dermatosis, commonly affecting the lower extremities; it occurs in patients with chronic venous insufficiency, often in association with varicose veins and dependent chronic edema. Clinical features include erythema, scaling, weeping, crusting, hyperpigmentation, lipodermatosclerosis, and ulcerations (2). Thus, as a diagnosis of exclusion, our patient was managed as a case of "idiopathic eczema/dermatitis."

The mechanism by which venous stasis causes eczema/dermatitis is yet to be elicited. The hypoxia/stasis theory has suggested that increased hydrostatic pressure leads to decreased oxygen content of pooled blood, resulting in hypoxic damage of the overlying skin (3). This may, in part, explain the rapid development of stasis dermatitis with severe GE. In spite of intensive treatment, the skin lesions exhibited progressive acute inflammation and exudation that mimicked severe cellulitis. Diagnostic skin biopsies are rarely indicated (4); thus, it was not recommended. At this stage, a decision of pregnancy termination was taken to eliminate the venous congestion in the legs caused by the pressure exerted mechanically by the uterus onto the inferior vena cava and iliac veins (5). Surprisingly, the patient showed a dramatic improvement in her condition within 1 week after cesarean section.

In conclusion, our presented case suggests that the rapid development of this type of "stasis dermatitis" and its dramatic improvement after pregnancy termination supports our speculation to be as a new subtype termed "gestational stasis dermatitis" induced by severe GE. Pregnancy termination is indicated in such cases.

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