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Case Report

Recurrent herpes gestationis - A case report

Upadya M Gatha, Pandya Nitin

Department of Skin and STD, KMC Hospital, Attavar, Mangalore (Karnataka).

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Introduction

Herpes gestationis is an intensely pruritic bullous eruption that may develop in association with pregnancy trophoblastic tumor, hydatidiform mole, and choriocarcinoma [1]. The term herpes is used because of the frequent presence of grouped or herpetiform lesions in these women [2]. It is a rare disorder which may affect 1 in 1700 to 50000 pregnancies [3]. It occurs only with pregnancy, hydatidiform mole and choriocarcinoma [1]. However clinical course may be modulated by changes in estrogen and progesterone levels. Exacerbations may occur with oral contraceptives or pregnancy and the severity may vary during the menstrual cycle [1].

Case report

A 35 year $G_2 P_1 L_1$ was admitted on 28th July, 2002 in early third trimester with the complaints of intensely pruritic erythematous papules and urticarial plaques of 4 weeks duration. They started as itchy red papules and plaques over the abdomen and slowly spread to involve the limbs, back, palms, and soles over a period of 2 weeks. She noticed tense blisters on few of those reddish plaques a week back. There was no oral involvement. There was no history of drug intake prior to the onset. During her last pregnancy she had similar itchy reddish plaques one week before delivery which subsided a week after delivery. Systemic examination revealed no abormality. Obstetric examination showed 26 weeks pregnancy. On examination there were multiple erythematous papules, urticarial plaques, and target lesions seen on the abdomen (Figure 1), back, upper and lower limbs including palms and soles. There were tense blisters with clear fluid on few of those plaques on the limbs. No oral involvement was seen even though there were few erosions in the nasal mucosa. Sonography revealed a single normal fetus at 30 weeks gestation. Routine investigations like complete blood counts and urine examination were within normal limits. Random blood sugar was 157 mg/dL. Tzanck test was negative. A skin biopsy taken from a vesicle showed a subepidermal bulla with eosinophils in the bullous cavity (Figure 2), perivascular eosinophilic infiltrate and dermal edema which confirmed the diagnosis of herpes. Direct immunofluorescesce study (Figure 3) revealed the deposition of strong continuous basement membrane zone (BMZ) band of C_3. There was a weak discontinuous BMZ band of fibrinogen. She was managed with systemic steroids and antihistaminics. Initially clobetasol propionate was used locally for 5

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Correspondence :
Dr. Gatha M Upadya
Associatge Professor,
Department of Skin and STD,
KMC Hospital, Attavar, Mangalore (Karnataka).
Tel. 0824 2445858 (Hosp), 0824-5256891/0824-2426891 (R)
Mobile : 9845314028, Email : gathaupadya@hotmail.com

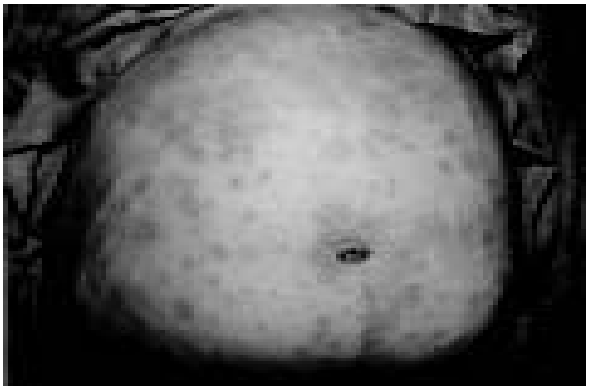

Figure 1. Herpes gestationis showing urticarial plaques

Figure 2. Skin biopsy showing subepidermal bulla with eosinophilic infiltrate (40 x)

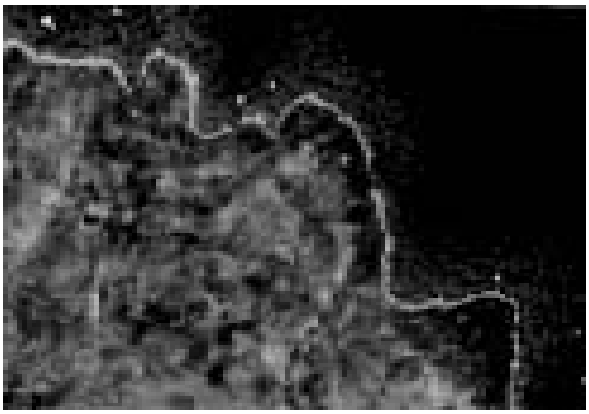

Figure 3. Immunofluorescence study of skin biopsy.

days but since there was no improvement she was changed over to prednisolone 10 mg three times a day for one month tapered to 5 mg three times a day. Simultaneously she was given chlorpheniramine maleate 4 mg three times a day for 1 week followed by once a day for 2 weeks and antihistaminics. As she showed marked improvement and prednisolone was increased to 30 mg a day. Her fasting blood sugar was 130 mg/dL. , she was discharged on 9th August, 2002. Routine antenatal examination revealed no abnormality. At 32 weeks she presented on 5th September, 2002 with exacerbation of the lesions, and her blood sugar level was high random 187 mg /dL, fasting 110 mg/dL, and 2 hours after 100 g oral glucose 160 mg/dL. Prednisolone was now increased to 5 mg four times a day and continued till delivery. Chlorpheniramine maleate 4 mg twice a day was also restarted and continued till delivery. She was given insulin – initially plain insulin and then mixtard subcutaneously till delivery. On 4th November, 2002 she was admitted in labor at 36 weeks and delivered a healthy male baby weighing 3.4 kg. The baby was normal and had no skin lesions. Mother's cutaneous lesions flared up after delivery for which prednisolone was increased to 30 mg aday. Her fasting blood sugar was 130 mg/dL. at 36 weeks. The cutaneous lesions flared up after delivery. She was discharged one week after. She never came for a follow up.

Discussion

Herpes gestationis is an autoimmune disease and may be associated with other autommune diseases such as Graves disease, vitiligo and alopecia areata [4]. Herpes gestationis could develop as a result of an immunological response to trophoblastic antigens. Anti-BMZ IgG antibodies are believed to be pathogenic. It may begin at any time between 4 weeks of gestation and 1 week postpartum, the average being 21-28 weeks of gestation [3]. In subsequent pregnancy the onset is likely to be earlier than in previous one. The eruption consists of urticarial papules and plaques, the target lesions being asscoaited with marked pruritus. Subsequently vesicles and large blisters appear. Based on the presence of classical target lesions a diagnosis of either bullous erythema multiforme or herpes gestationis was made in our case. However pruritic urticarial papules and plaques of pregnancy were ruled out as there were multiple tense blisters on those plaques. Biopsy showed the characteristic histopathological findings of herpes.

Herpes gestationis tends to improve after delviery but it may take few weeks to months. Rarely the disease may evolve into bullous pemphigoid. The

disease is associated with premature delivery with a risk of low birth weight [5,6]. In mild cases of herpes gestationis, topical steroids combined with antihistaminics are usually adequate. Once the bullous stage supervenes it is usually necessary to use systemic steroids. Our patient was also given potent topical steroid and was later started on systemic steroids as there was not much improvement with topical steroid. However the outcome of pregnancy was good in our case and she delivered a healthy baby with no skin lesions.

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