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Spontaneous Regression of Atopic Dermatitis following delivery: Case Report

Rushulo Khing[1], N Romita Devi[2], R K Praneshwari Devi[3],
L Ranjit Singh[4], L Somen Kumar Singh[5]
Department of obstetrics and Gynaecology, RIMS, Imphal, Manipur, India
[1]Postgraduate student, [2]Senior Resident, [3]Assistant Professor, [4]Professor, [5]Senior Resident

***Abstract**: Atopic eruption of pregnancy (AEP) or prurigo of pregnancy (PP) may be very distressing to some mothers because of intense itching, aggressive in nature and difficulty in managing the symptoms. However it usually regresses very well following delivery without any known adverse maternal or foetal outcome. Here we present a typical case of atopic dermatitis in pregnancy that flared up extensively which is rare and spread to the whole body during pregnancy that resolved spontaneously following delivery.*

***Keywords**: Atopic, delivery, dermatitis, pregnancy, prurigo.*

I. Introduction

Pregnancy has many complex endocrinological, immunological, metabolic and vascular changes, which may influence the skin in various ways [1-3]. Skin changess in pregnancy can be classified as physiologic skin changes, alterations in pre-existing skin diseases and specific dermatoses of pregnancy [1]. Atopic Eruption of Pregnancy (AEP) has recently been introduced as a new disease complex in a recent reclassification of dermatoses of pregnancy [1] and; it remains the most common pregnancy dermatosis [1-3]. It is a benign pruritic disorder of pregnancy which includes eczematous and /or papular lesions in patients with a personal and/or family history of atopy and/or elevated IgE levels the diagnosis being made after exclusion of other dermatoses of pregnancy [2,4]. It may represent a gestational variant of atopic dermatitis (AD) [5]. Hypotheses as regards the cause of AD include epidermal barrier defects, as well as immune dysregulation of both the innate and adaptive immune systems [6]. AEP encompasses Atopic Eczema (AE) during pregnancy, Prurigo of Pregnancy (PP) and Pruritic Folliculitis in Pregnancy (PFP) [1].

II. Case Report

A 31 year old primigravida was admitted at 36 weeks of gestation with multiple itchy nodular skin eruptions all over the body. Excoriations and crusting of the lesions due to scratching were observed; some with serous like discharges. The first manifestation was at around 12 weeks of pregnancy in the extensor aspect of extremities. These were small, multiple, discrete, erythematous and pruritic lesions which then flared up spreading all over the whole body including face in few weeks. The patient was first treated with oral antihistaminics with no significant relief from symptom and lesions. At 30 weeks period of gestation she was treated with oral steroid (prednisolone 20 mg daily) with the working diagnosis of atopic dermatitis (prurigo of pregnancy) with extensive and severe lesions all over her body. At 33 weeks period of gestation, there was still no significant improvement in her symptoms. The treating physician continued the steroid in tapering dose and added oral antihistaminic (fexofenadine), moisturiser creams along with topical antibiotic and steroid preparations (Nadifloxacin with clobetasol). A course of oral antibiotic (Azithromycin) was given. Oral calcium and vitamin-D were also prescribed. She continued to suffer with the lesions that did not regress on medications except some symptomatic relief. Oral steroid was tapered and stopped a week before delivery. There was history of mild skin eruptions in the past at around 16 years of age but was treated successfully by the treating physician otherwise there is no other significant history in the past. There is no similar history or history of asthma, hay fever in the family or near relatives. Routine blood and urine examinations were within normal limits. Thyroid profile was normal and serological tests for syphilis and HIV were negative. Total vitamin-D level was 17ng/ml(less). Ultrasound at 35 weeks 2day of gestation showed a single live intrauterine fetus in cephalic presentation with expected foetal weight of 2.3 kg with adequate liquor. Histopathological examination of the lesions showed nonspecific chronic inflammatory cells. Direct and indirect immunofluorescence test were negative. Laboratory test showed normal Ig E level. The diagnosis of prurigo of pregnancy was made. She delivered a live female baby of 2.4 kg by caesarean section at 38 weeks period of gestation for intrauterine growth restriction (diagnosed clinically and by ultrasonography) with non reassuring FHR pattern by non stress test (NST). The amniotic fluid was adequate, straw coloured and turbid but the baby looked healthy and cried well with normal apgar score. Following delivery there was dramatic improvement in

her symptoms with 50% spontaneous regression of the lesions in a week without any treatment and only scar tissues remain after four weeks postpartum without any symptom.

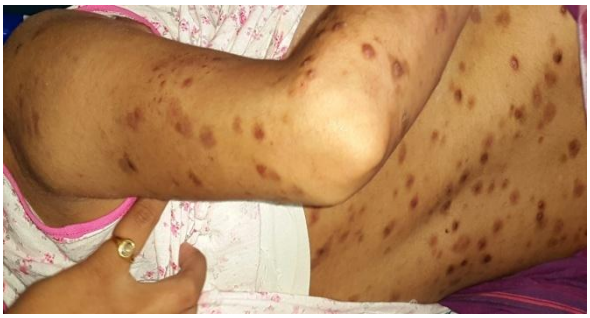

Fig 1: Lesions on extensor and back

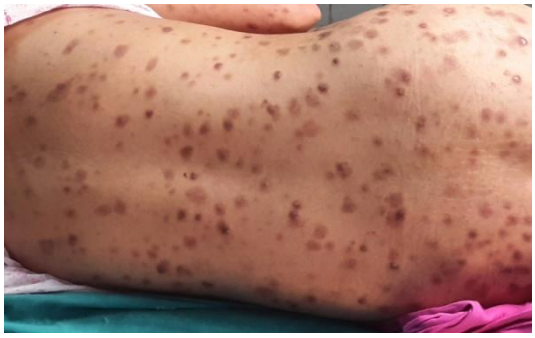

Fig 2: Lesions on trunk

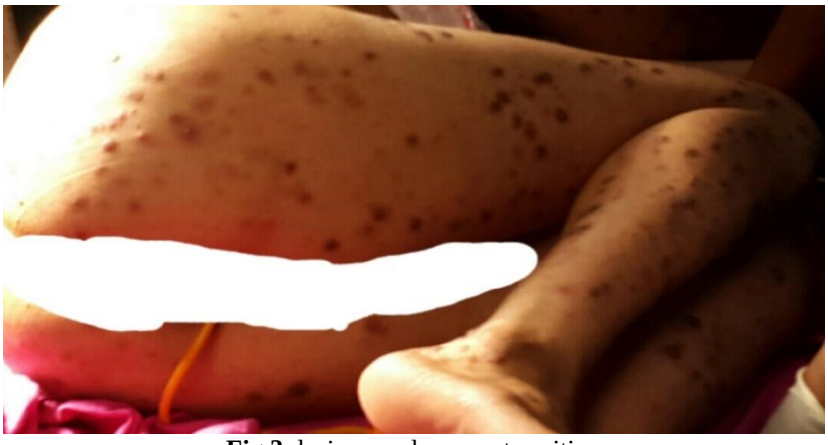

Fig 3: lesions on lower extremities

III. Discussion

Although, the aetiology of AEP is not yet fully elucidated [1,3,7,8], a Th2 response during pregnancy worsens the imbalance that already exist in most atopic patients [1]. A recent study indicated that it is more the intrinsic (‘non allergic’) than the extrinsic (IgE-associated) eczema that is affected by pregnancy [1]. Patients with intrinsic AE, as opposed to those with the extrinsic type, have no associated respiratory disease, showing normal total serum IgE levels, no specific IgE and negative skin prick tests to aeroallergens or foods [1].

Emollients remain an integral part of eczema management in all patients [1]. Basic treatment together with topical corticosteroids for several days will usually lead to quick improvement of skin lesions. Urea (3–10 %) and anti pruritic additives (menthol, polidocanol) can be safely employed during pregnancy [1,2,4]. Severe cases may require a short course of systemic corticosteroids and antihistamines [2]. Systemic corticosteroids have a greater bio disponibility than topical corticosteroids, but they also have more potential for fetal toxicity than topical corticosteroids, namely reduction in fetal birth weight and an increase in preterm delivery [1]. Phototherapy (UVB) is a safe additional tool, particularly for severe cases in early pregnancy [1,2,4]. Patients with AD might benefit from supplementation of vitamin D, particularly if they have a documented low level or low vitamin D intake [6]. If systemic agents other than oral steroids are needed for several/ recalcitrant AD, cyclosporine is the safest option and this should be used for the shortest possible duration, usually less than 6

months, in order to avoid the increasing risk of renal impairment in the mother [1]. Another second line drug is azathioprine, which readily crosses the placenta and its uses during pregnancy has been associated with miscarriage, preterm delivery and fetal growth restriction. However, the fetus seems to be protected from teratogenic effects [1].

Pregnancy seems to have an effect on the prognosis of eczema in most women. Improvement is observed in 25% and in more than 50% there is deterioration at any stage of pregnancy and it is still higher in the second trimester. There are approximately 10% of flares in the postpartum period [1].

IV. Conclusion

It is important to differentiate AEP from other specific dermatoses of pregnancy or other dermatoses coinciding by chance with pregnancy. There is a necessity to clarify the aetiology and the relationship between PP, PFP and AE. Dermatologists, Obstetricians and primary care physicians should be well aware of the clinical characteristics of these disorders and the potential maternal/foetal risks and necessary treatment. Effective treatment for refractory cases is a challenge faced by physicians looking after pregnancy.

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