Gestational Pityriasis Rosea: Suggestions for Approaching Affected Pregnant Women

Dear Editor,

Pityriasis rosea is a common, acute, and self-limiting dermatosis, which is associated with the endogenous systemic reactivation of human herpesvirus (HHV)-6 and/or HHV-7 (1). It predominantly affects individuals of both sexes in their second or third decade of life and is clinically characterized by the occurrence of an initial erythematosquamous plaque followed by the appearance of disseminated similar but smaller lesions one or two weeks later. Several patients develop systemic symptoms such as nausea, anorexia, malaise, headache, fever, arthralgia, and lymphadenopathy that may precede or accompany the eruption; the latter follows the cleavage lines of the trunk creating the configuration of a Christmas tree and spontaneously resolves within 4 to 8 weeks.

Mainly based on the nature of the underlying viral reactivation, pityriasis rosea is classified into five different forms (2): 1) Classic and 2) Relapsing (characterized by sporadic and relapsing HHV-6/7 systemic reactivation, respectively), 3) Persistent (persistence of HHV-6/7 viremia), 4) Pediatric (longer activity of HHV-6/7 infection; recent primary infection) and 5) Gestational (HHV-6/7 reactivation and possible intrauterine transmission).

Clearly, the inevitable impairment of immune response in pregnancy favors viral reactivation and possibly also the intrauterine transmission of HHV-6/7. Indeed, it is well known and documented that pityriasis rosea more frequently occurs in pregnant women (18%) as compared to the general population (6%) (3). However, the literature concerning the possible effect of pityriasis rosea on the outcome of pregnancy is surprisingly sparse. Only an Italian group, Drago et al (4,5), has systematically investigated the impact of this disorder on pregnant women. They found that 22 out of 61 women (36%) who developed pityriasis rosea during pregnancy had unfavorable outcomes, whereas 8 others miscarried (13%). None of the latter had any risk factors, other than pityriasis rosea, for intrauterine fetal death. All miscarrying women reportedly revealed an aggressive course of widespread eruption and severe constitutional symptoms; all of them had HHV-6 DNA in the plasma, placenta, skin lesions, and fetal tissues, whereas HHV-7 DNA was detected in the plasma and skin lesions in 3 out of 8 (37.5%) miscarrying women. HHV-6 DNA was found only in the plasma of 2 out of 31 women (6.45%) with normal pregnancy, whereas HHV-7 DNA was detected in the plasma of 3 (9.45%) and in the skin lesions of 2 women (6.45%) with normal pregnancy.

The total abortion rate in women who developed pityriasis rosea during their pregnancy (13%) does not differ from that observed in the general population. Nevertheless, it is markedly higher in cases affected during the first 15 gestational weeks (57%) (4,5). Surprisingly, this devastating impact of pityriasis rosea on the outcome of pregnancy is almost completely unknown not only to the public but also to many members of the medical community. It is also largely unknown that, particularly during the first 15 gestational weeks, all pregnant women should avoid any contact with patients known to have pityriasis rosea. Since we have received a considerable number of requests for consultation with pregnant women with pityriasis rosea over the last few years, our group has compiled suggestions approaching the affected patients:

1. If an eruption suggestive for pityriasis rosea occurs in a pregnant woman, the following factors should be excluded:

a. Exposure to drugs prior to the development of the rash (biologic agents, captopril, clonidine, hydrochlorothiazide, atenolol, lamotrigine, nortriptyline, barbiturates, metronidazole, terbinafine, omeprazole, non-steroidal anti-inflammatory drugs, and isotretinoin), which are capable of inducing a pityriasis rosea-like eruption (6) and

b. Disorders included in the differential diagnosis

(syphilis and infections due to parvovirus, herpes virus, cytomegalovirus, and Epstein-Barr virus).

2. The clinical diagnosis of pityriasis rosea should be made according to the morphological criteria (peripheral collarette scaling with central clearance on at least two lesions) put forth by Chuh (7).

3. Since specific anti-HHV-6 and -7 IgM antibodies are detected only in a low percentage of infected pregnant women (8), HHV-6 and -7 DNA should be assessed in plasma by nested PCR. Especially during the first 15 gestational weeks, pregnant women with positive PCR results deserve, apart from close monitoring, appropriate information about the existing risks in order to be able to make informed decisions.

4. Reliable and definite data from adequate and controlled human studies on the safety of acyclovir or valacyclovir in pregnant women and their efficacy in pityriasis rosea are lacking. Thus, the decision on whether these antiviral compounds will be administered should be tailored to each individual pregnant woman, subsequent to a meticulous assessment of the potential risks and their balancing against the potential benefits.

References:

- Watanabe T, Kawamura T, Jacob SE, Aquillino EA, Orenstein JM, Black JB, et al. Pityriasis rosea is associated with systemic active infection with both human herpes virus-6 and -7. J Invest Dermatol 2002;119:793-7.
- Drago F, Ciccarese G, Rebora A, Broccolo F, Parodi A. Pityriasis rosea: Comprehensive classification. Dermatology 2016;232:431-7.
- Corson EF, Luscombe HA. Coincidence of pityriasis rosea with pregnancy. AMA Arch Derm Syphilol 1950;62:562-4.
- 4. Drago F, Broccolo F, Zaccaria E, Malnati M, Cocuzza C, Lusso P, et al. Pregnancy outcome in patients with pityriasis rosea. J Am Acad Dermatol 2008;58: S78-S83.

- Drago F, Broccolo F, Javor S, Drago F, Rebora A, Parodi A. Evidence of human herpesvirus-6 and -7 reactivation in miscarrying women with pityriasis rosea. J Am Acad Dermatol 2014;71:198-9.
- 6. Eisman S, Sinclair R. Pityriasis rosea. BMJ 2015;Oct 29;351:h5233.
- Chuh AAT. Diagnostic criteria for pityriasis rosea-a prospective case control study for assessment of validity. J Eur Acad Dermatol Venereol 2003;17:101-3.
- Adams O, Krempe C, Kogler G, Wernet P, Scheid A. Congenital infections with human herpesvirus 6. J Infect Dis 1998;178:544-6.

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