with urticaria, Stevens-Johnson syndrome, toxic epidermal
necrolysis, pruritus, or vasculitis.10

Patch testing with drugs has been demonstrated to
be useful in determining the cause of drug-related skin
reactions.9,11 The main advantages of patch testing over
other diagnostic procedures are that serious adverse
reactions are rare and testing can be performed with any
commercial form of the drug.

Given the absence of a gold standard for determining
the causal agent and the importance of reaching a specific
diagnosis in these patients, other tests, such as prick,
intradermal, and oral challenge tests, should be performed
if the results of the patch tests are negative.

Conflict of Interest

The authors declare they have no conflict of interest.

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lesions recurred, but cleared rapidly. One month later, the lesions had disappeared completely and at follow-up 8 months later, the patient remained asymptomatic.

EAC can occur at any time during life but it is most common in patients older than 50 years. It affects men and women equally and the lesions generally resolve within weeks when the trigger is eliminated. The most commonly affected sites are the buttocks, the thighs, and the upper arms.

EAC is currently considered to be a skin reaction pattern with nonspecific histology, as in its original description.\(^4\) Classically, it was categorized as either superficial or deep. According to Ziemer et al,\(^5\) the term EAC should only be used to refer to the superficial variant. According to those authors, clinical and histologic differential diagnosis should include 3 groups of disease: a) lupus erythematosus tumidus; b) spongiotic dermatitis (subacute or chronic) and severe forms of pityriasis rosea or stasis dermatitis; and, less frequently c) pseudolymphoma, particularly in association with \textit{Borrelia} infection.

Other conditions that should also be considered in the differential diagnosis include diseases characterized by erythematous annular lesions such as pityriasis rosea, tinea corporis, subacute cutaneous lupus erythematosus, seborrheic dermatitis, pustular psoriasis, granuloma annulare, syphilis, and linear immunoglobulin A dermatosis. When EAC is detected in pregnancy, as occurred in our case, the differential diagnosis should also include all eczematous eruptions that come under the umbrella of atopic eruption of pregnancy. These dermatoses are early-onset diseases (occurring in the first and second terms of pregnancy) and include prurigo gestationis (Besnier prurigo), Nurse early prurigo of pregnancy, papular dermatitis of pregnancy, and pruritic folliculitis of pregnancy. All of these conditions have nonspecific histologic features and findings that overlap with those observed in our patient.\(^6\)

It is believed that EAC may be a hypersensitivity reaction to different antigens. Among the triggers that have been suggested are tinea pedis, candidiasis, molluscum

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**Figure 1** Polycyclic plaques with advancing arcuate lesions with raised borders and clear centers on the patient’s thigh.

**Figure 2** Superficial perivascular infiltrate with foci of epidermal spongiosis and parakeratosis (hematoxilin-eosin, original magnification, ×40).

**Figure 3** Hypopigmented residual lesions on the patient’s thighs 2 days postpartum.
contagiosum, Epstein-Barr virus, and parasites. EAC has also been associated, albeit less frequently, with drugs (eg, diuretics, antimalarials, and gold salts), food consumption, rheumatic disorders, sarcoidosis, liver disease, neoplasms (eg, lymphomas), endocrine disorders, and pregnancy.7

The main treatment strategy consists of eliminating the trigger and using topical corticosteroids.

Our review of the literature of EAC in pregnancy revealed just 3 cases that followed the same course as in our patient, ie, with immediate resolution of lesions postpartum (Table 1). There is evidence that the menstrual cycle and estrogenic substances may act as triggers. Dogan3 was the first to establish a causal link between EAC onset and human chorionic gonadotropin hormone, whose levels peak in the 12th week of pregnancy. This hormone might also explain the lesions in our case given that they appeared when the patient was 12 weeks pregnant. One particularly noteworthy observation in our study, not previously reported, was the fact that the lesions returned when, as it is known colloquially, the mother's milk came in. When this happens, the breasts become very distended and the suckling of the infant on the nipples stimulates the release of oxytocin and prolactin, 2 key hormones that regulate the onset of lactation.

As has occurred in other dermatoses of pregnancy, the reporting of new cases of EAC in pregnant women may help to shed light on the role played by hormonal and immunologic changes in the onset of lesions.

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<table>
<thead>
<tr>
<th>Publication</th>
<th>Patient Age, y</th>
<th>Week of Pregnancy at Onset of Lesions</th>
<th>Treatment</th>
<th>Disappearance of Lesions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Choonakarn et al1</td>
<td>27</td>
<td>33 (first pregnancy)</td>
<td>Unknown</td>
<td>Gradually in first month postpartum</td>
</tr>
<tr>
<td>Rosina et al2</td>
<td>28</td>
<td>31 (first pregnancy)</td>
<td>Vaseline</td>
<td>36th week of pregnancy</td>
</tr>
<tr>
<td>Dogan3</td>
<td>28</td>
<td>12 (first pregnancy)</td>
<td>Low-potency topical corticosteroid</td>
<td>3 days postpartum</td>
</tr>
<tr>
<td>Present case</td>
<td>34</td>
<td>12 (first pregnancy)</td>
<td>Low-potency topical corticosteroid</td>
<td>3 days postpartum</td>
</tr>
</tbody>
</table>