Resolution of autoimmune progesterone dermatitis after treatment with oral contraceptives

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Key words: autoimmune progesterone dermatitis; menstruation; oral contraceptives.

CASE REPORT
A 20-year-old woman presented with a 6-year history of recurrent erythematous papules and plaques on the trunk. The eruption recurred cyclically, beginning 5 days before menses and resolving 1 to 2 days after the menstrual period. The lesions were asymptomatic, and there were no associated systemic symptoms. She had not previously sought treatment. Her medical history was unremarkable, and she was not taking any medications.

Examination found widespread erythematous and edematous papules and nonscaly plaques, on the chest, back, and lower neck (Fig 1). Lesions were uniform in color, ranging in size from 3 mm to 4 cm in diameter. A biopsy specimen was taken for histopathologic evaluation (Fig 2).

Results of a punch biopsy of the skin showed dermal perivascular lymphohistiocytic inflammation, with rare eosinophils. An intradermal progesterone test read at 20 minutes was positive (progesterone, 50 mg/mL at a dilution of 1:10 in aqueous solution elicited a 10-mm wheal vs a 4-mm wheal for the glycerine control).

The patient was started on levonorgestrel/ethinyl estradiol, 0.1 mg to 20 μg daily. There was a marked reduction in the number of lesions during her next menstrual period. By the third menstrual cycle, after beginning oral contraceptive pills (OCPs), no lesions appeared perimenstrually. The patient remained clear for another 4 months while taking Levonorgestrel/Ethinyl Estradiol 0.1 mg to 20 μg. She then discontinued treatment because of the loss of health insurance. Despite discontinuing OCPs, lesions have not returned in the 8 months since the medication was stopped.

DISCUSSION
Autoimmune progesterone dermatitis is a rare cyclic reaction to progesterone that occurs during the luteal phase of the menstrual cycle.1 The most

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Funding sources: None.
Conflicts of interest: None declared.
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JAAD Case Reports 2015;1:319-20.
2352-5126
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http://dx.doi.org/10.1016/j.jdcr.2015.07.011

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common cutaneous manifestations are urticaria, eczematous eruptions, and erythema multiformelike lesions, although morbilliform, vesiculobullous, and oral lesions have also been reported. There are no distinguishing histologic features of autoimmune progesterone dermatitis.

The earliest cases of autoimmune progesterone dermatitis appear at menarche, and some patients have autoimmune progesterone dermatitis that appears to be triggered by hormonal therapy or pregnancy. Although pregnancy can trigger the eruption or exacerbate the dermatosis, a subset of patients with autoimmune progesterone dermatitis improves during pregnancy, possibly because of desensitization resulting from persistently elevated progesterone levels.

Although the diagnosis can be made clinically, an intradermal progesterone test can confirm the diagnosis. With regard to the intradermal injection, progesterone in an aqueous suspension is the preferred modality, as the oil suspension can irritate the skin and lead to a false-positive result.

Various treatments are used for autoimmune progesterone dermatitis, and most are aimed at suppressing ovulation. OCPs are the most commonly used therapy. This case report suggests that even a short course of OCPs can provide long-lasting clearance of lesions, possibly because of the desensitization to progesterone. Although this patient remains clear of lesions 8 months after discontinuing OCPs, we will continue to monitor her to determine if clearance is lasting. Danazol (prophylactic monthly dosing) has also been reported to be effective in some patients. Although gonadotropin-releasing hormone analogs have been used to treat autoimmune progesterone dermatitis, as administration inhibits ovulation and decreases sex hormone production, they should not be used for longer than 6 months because of negative effects on bone metabolism and the cardiovascular system. In refractory cases, bilateral oophorectomy has been used as a last resort and provides a definitive treatment.

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