

A young woman with autoimmune progesterone dermatitis

Commentary by

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Progesterone dermatitis is a distressing condition and requires management. Antihistamines and topical corticosteroid creams rarely control the condition.

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CASE SCENARIO

Jude is 22 years old and for several years has been having monthly flares of an irritating dermatitis that erupts on her trunk and proximal limbs. She says that each outbreak starts at about the time she is in the middle of her menstrual cycle, and only subsides when her next period arrives. Her skin becomes red and itchy, papules develop, which she frequently scratches and infects, and she is left with residual scars. Jude says that no standard treatment for dermatitis has helped her – not even a course of oral corticosteroids.

Could she be allergic to her own progesterone? How should she be managed?

COMMENTARY

Progesterone dermatitis presents as a recurrent cyclical premenstrual eruption

typically commencing six to eight days before a menstrual period and subsiding soon after its onset. This recurrent pattern is the major clue to the diagnosis. Although a number of chronic skin conditions such as acne, psoriasis, lupus and dermatitis herpetiformis may flare premenstrually, these are variable flares and do not usually present with such a precise relation to the menstrual cycle.

The skin eruptions may present in a variety of forms including papulovesicular lesions, urticarial lesions, eczema or erythema multiforme. Lesions may be present on the oral mucosa, lips, palms, trunk or feet. Patients generally describe marked pruritus and, if oral lesions are present, these may be quite painful.

Once the diagnosis is suspected, it may be confirmed by performing an

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DIAGNOSTIC FEATURES OF PROGESTERONE DERMATITIS

- Recurrent premenstrual flare
- Positive intradermal skin test with progesterone
- Recurrence of eruption following intramuscular or intravaginal progesterone
- Resolution of rash with inhibition of ovulation

intradermal skin test with progesterone that will cause a weal and flare response within 15 to 30 minutes. Intramuscular injection of progesterone or intravaginal progesterone given as a 'challenge' should reproduce the rash.¹ Inhibition of ovulation prevents the onset of the rash (see the box on this page listing the diagnostic features of progesterone dermatitis).

Progesterone dermatitis is a rare autoimmune hypersensitivity to endogenous progesterone. The mechanisms involved in its evolution are not entirely understood. Some patients have had previous exposure to external progesterone in the form of the oral contraceptive pill and this may sensitise them to react against their own internal progesterone, but not all affected patients have been exposed to previous hormone therapy. Another theory is that patients with autoimmune progesterone dermatitis produce an altered form of progesterone that incites an immunological response against it. A third hypothesis is that progesterone heightens the individual's hypersensitivity to another unknown allergen.

Progesterone dermatitis is a distressing condition and requires management. As is the case with Jude, use of antihistamines and topical corticosteroid creams rarely controls the condition. There are reports of control with systemic corticosteroids but this is an undesirable option in the long term.

Definitive treatment requires anovulatory agents that suppress progesterone secretion. The 17- α -alkylated steroids such as danazol have been used to suppress ovulation; however, long-term side effects such as virilisation, abnormal liver function and hyperlipidaemia make it a difficult choice for many women. Case reports suggest that it may be effective when commenced before the expected onset of menses and continued three days after the period has ended. By using it in this way most of the long-term side effects may be avoided.

Tamoxifen, a nonsteroidal anti-estrogen agent, has been used to induce remission in patients with progesterone dermatitis. Different gonadotropin-releasing hormone agonists have also been used to induce remission by causing ovarian suppression. Problems with this approach include the expense of these agents and the frequent need for oestrogen supplementation. A successful progesterone desensitisation protocol has been described in a woman with progesterone dermatitis undergoing in vitro fertilisation.²

Finally, for patients with intractable symptoms that have not been controlled by medical management, definitive treatment is ultimately surgical management with oophorectomy.³ MT

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