

Autoimmune Progesterone Dermatitis (APD) – a case report and review of the literature.

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Introduction

Autoimmune progesterone dermatitis (APD) is a rare cyclical cutaneous reaction to progesterone¹. The pathogenesis of APD remains to be elucidated. Current literature suggests that prior exposure to exogenous progesterone may cause sensitization. These patients may subsequently develop an inflammatory response to the physiological rise in progesterone during the luteal phase of the menstrual cycle, which occurs 3-10 days prior to menstruation².

Case

A 24-year-old female presented to the Dermatology clinic with recurrent pruritic periorbital oedema and associated erythematous, xerotic skin on the bilateral upper eyelids (Figure A-D). The symptoms were cyclical in nature, appearing every month, 3-5 days before menstruation. She was asymptomatic between outbreaks. The first episode occurred following a termination of pregnancy and subsequent commencement of the combined oral contraceptive pill (OCP). She had been trialled on multiple courses of oral and topical steroids without resolution. Laboratory investigations including thyroid function tests were unremarkable.

A diagnosis of APD was supported by the temporal relationship between symptom onset and the menstrual cycle, as well as a history of exogenous progesterone exposure. The OCP was discontinued and she was commenced on loratadine for symptomatic relief. She was referred for confirmatory testing with intradermal progesterone challenge (IDP). Discussion regarding treatment options and follow-up is ongoing.



Figure A-D: Patient presenting with pruritic periorbital oedema and erythematous, dry skin of upper eyelids.

Clinical Presentation & Diagnosis

A wide range of cutaneous presentations of APD have been described in the literature^{1,2} (Table 1). As a result, diagnosis may be challenging. A diagnosis of APD is made based on a clinical history of premenstrual exacerbation of skin lesions and confirmed with a progesterone challenge. However, progesterone testing has not been standardized³.

Table 1. Dermatological Presentations of APD¹

• Urticaria	• Purpura/petechiae
• Erythema multiforme	• Stomatitis
• Maculopapular eruptions	• Angioedema/oedema
• Vesicular eruptions	• Erythromelalgia
• Fixed drug eruption-like lesions	• Dermatitis herpetiformis-like lesions
• Eczematous dermatitis	• Erythema annulare-like lesions
• Nonspecific plaque-like rash	• Necrolytic migratory erythema

Treatment

Numerous treatment options have been reported with varying success¹ (Table 2). The main objective is to suppress ovulation and progesterone secretion. OCPs are often tried as initial therapy, but have limited success. As in our case, OCPs may contribute to exacerbations due to its progesterone component, further complicating treatment choice³.

Although antihistamines are the most commonly attempted treatment, they rarely provide disease control. The most successful treatment modality is TAH+BSO. However, the highly invasive nature and side effects of early menopause and infertility must be considered. Thus, this is not an acceptable treatment for some patients¹.

Table 2. APD Treatments Described in the Literature¹

Temporary symptom relief with minimal effect on disease control
• Antihistamines
• Systemic or topical corticosteroids
Suppression or elimination of ovulation / progesterone secretion
• OCP
• Conjugated estrogen therapy
• Danazol
• GnRH
• Tamoxifen
Progesterone desensitization
Definitive therapy / Surgery
• Total abdominal hysterectomy + bilateral salpingo-oophorectomy (TAH+BSO)

Conclusion

APD should be considered in women presenting with cyclical premenstrual cutaneous symptoms to facilitate early diagnosis and successful management.

References:

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