

## Recurrent Generalized Lichen Nitidus Associated with Amenorrhea

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**A case of recurrent generalized lichen nitidus is reported. The eruptions developed in the proliferative phase of the menstrual cycle, but there has been a marked improvement after administration of estrogen and progesterone for the treatment of amenorrhea. Hormonal factors may play a role in the development of lichenoid tissue reaction in the lesions of this disease. Key word: ovarian failure.**

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Lichen nitidus is an uncommon chronic papulosquamous eruption of unknown etiology, first reported by Pinkus (1) in 1907. The disorder shows little or no response to therapy.

An estrogen-progesterone combination is prescribed for the cyclic treatment of amenorrhea and dysmenorrhea. We here report the case of a patient with generalized lichen nitidus whose lesions subsided after administration of estrogen and progesterone.

### CASE REPORT

A 19-year-old single female (gravida 0, para 0) presented in September 1992 for evaluation of recurrent asymptomatic eruptions of 4 months' duration. Menarche had occurred at 14 years. Her menstrual cycle had been irregular and associated with moderate dysmenorrhea for 6 months. She was otherwise healthy and her family history was not contributory. Physical examination revealed numerous 1–2 mm uniform, flesh-colored, discrete papules over the entire body except the face (Fig. 1). The eruption was initially noted near the elbows but slowly progressed to involve the entire body. The eruptions followed a cyclical pattern, improving during the latter half of the menstrual cycle, with a premenstrual peak and a tendency to become aggravated with the onset of blood flow. Fig. 2 shows the relationship between the severity of eruptions, serum progesterone and menses. The eruptions were graded on a scale from 1 to 10 and charted daily by the patient over several months before and after administration of estrogen and progesterone. The area involved in the disease was mainly used to evaluate skin manifestations.

Laboratory findings suggested that the patient had moderate anemia (red blood cell count  $330 \times 10^9/\text{mm}^3$ ). Hormonal analysis revealed low serum progesterone (1.5 ng/ml, reference value 6.0–30.0 ng/ml) and elevated estrogen (estradiol 220 pg/ml, reference value 50–200 pg/ml; estrion 50 pg/ml, reference value 20 pg/ml or less) on the 24th day of her cycle. Serum gonadotropin level was normal (luteinizing hormone 20.0 mIU/ml, reference value 3.0–30.0 mIU/ml; follicle-stimulating hormone 12.6 mIU/ml, reference value 4.0–30.0 mIU/ml; prolactin 12.8 ng/ml, reference value 5.0–40.0 ng/ml). No abnormalities were detected during general and gynecologic examinations, including pelvic sonogram.

A skin biopsy specimen from the upper back exhibited a well-circumscribed lymphohistiocytic infiltrate adjacent to the epidermis. The infiltrate was surrounded by rete ridges. The epidermis showed liquefaction degeneration of the basal cell layer.

The patient was initially treated with topical corticosteroid ointment, which failed to suppress the eruptions. At the beginning of this year, she

missed three menstrual cycles and received oral estrogen and progesterone for the cyclic treatment of amenorrhea. The eruptions cleared completely a few days after administration of estrogen and progesterone. She experienced regular cyclic bleeding without aggravation of the eruptions over the next 6 months (Fig. 2).

### DISCUSSION

Lichen nitidus is characterized by multiple tiny, discrete and confluent, flesh-colored papules that are usually localized on the upper extremities, genitalia, chest and abdomen. Its occurrence has infrequently been reported on the palms and soles, on mucous membranes and over the entire body (generalized form) (2, 3). In rare cases, the nails may exhibit pitting, thickening and ridging (4).

The etiology of lichen nitidus remains unknown. Over the years, many authors have discussed the possibility of lichen nitidus being a variant of lichen planus because of similar histologic patterns. Recently, using immunohistochemical techniques, Smoller & Flynn (5) have stressed that lichen nitidus is not a localized papular variant of lichen planus. The lack of symptoms, the low incidence of the disease and its occasional spontaneous resolution have made the evaluation of therapy difficult (3, 6). Treatments that may induce regression of cutaneous lesions include potent topical steroids, intralesional corticosteroids.



Fig. 1. Discrete papules on the upper back of patient with lichen nitidus.

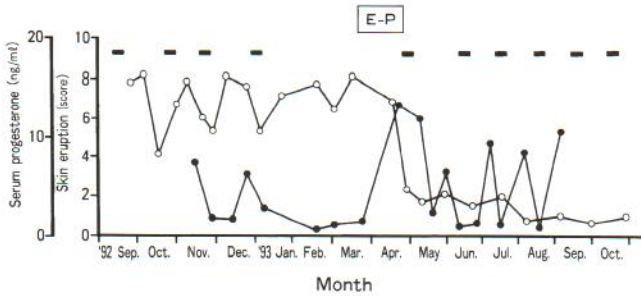


Fig. 2. Relationship of severity of eruptions (○) to serum progesterone levels (●) and menses (—). E-P = oral estrogen-progesterone combination therapy.

teroids, antihistaminic drugs, short courses of systemic steroids and photochemotherapy.

Hormonal variations are often believed to be the cause of exacerbation in skin diseases. Acne, psoriasis, atopic dermatitis, lichen planus, lupus erythematosus, herpes simplex, dermatitis herpetiformis and autoimmune progesterone dermatitis are often cited as examples (7–9). Despite this abundance of clinical knowledge, little is known concerning the underlying mechanism of such hormonally induced fluctuations.

In this case of lichen nitidus, the patient had recurrent asymptomatic papules during the week after menstruation and through the first few days of the secretory phase. Each episode was characterized by improvement during the latter half of the menstrual cycle, a time when progesterone levels are on the rise. Furthermore, cutaneous lesions dramatically improved by administration of estrogen and progesterone for the treatment of amenorrhea.

The combined administration of progesterones and estrogens involves the inhibition of ovulation by an interference with hypothalamic-pituitary mechanisms and induces regular epi-

sodes of the withdrawal of bleeding (10). These hormones may also have additional sites of action.

In our case, there were suspicious signs of ovarian failure such as persistent amenorrhea. Autoimmune processes involving the ovary have recently been suggested as one of the causes of premature ovarian failure (11). Autoallergic reaction due to ovarian dysfunction may cause or modulate intermittent eruptions of the disease.

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