Lichen Sclerosus in Pregnancy: Presentation of Two Cases

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Abstract: Numerous skin diseases occurring in the pregnant patient have been reported. Some of these diseases are unique to pregnancy and some, including vulvar varicosities, vulvar edema, postpartum labial adhesions, and hematomas, are a result of physiological changes of pregnancy or the birth process. In addition, a variety of viral and bacterial infectious diseases of the vulva may occur during pregnancy. Vulvar neoplasms may also be found in pregnancy. In two patients, ages 27 and 31, lichen sclerosus first was diagnosed during their initial prenatal visits. Only one of the patients was symptomatic. The symptomatic patient used topical steroids for relief of vulvar itching. Two patients with lichen sclerosus of the vulva in pregnancy are reported, with emphasis on the diagnosis and treatment of this condition.

Key Words: lichen sclerosus, pregnancy, vulvar diseases

CASE REPORTS

Case 1

A 27-year-old gravida 3 para 0 presented to her obstetrician for her initial evaluation at 11 weeks’ gestation. Her vulvar appearance is demonstrated in Figure 1. She complained of diffuse vulvar pruritus. A biopsy was taken from a whitened area on the lateral aspect of the left labium majus (Fig. 2). She declined therapy for her lichen sclerosus at that time. She delivered a 3,380-g infant by a normal spontaneous vaginal delivery at 39 weeks’ gestation. Ten weeks after delivery, her vulvar pruritus was unabated, and she was examined at the University of Michigan Center for Vulvar Diseases. Examination of the vulva revealed extensive bilateral whitening and loss of the labia minora, consistent with the previously diagnosed lichen sclerosus. She was treated with topical steroids (clobetasol propionate, 0.05% ointment daily for 3 months, followed by hydrocortisone valerate, 0.2% ointment daily) with good relief of her symptoms and a gradual resolution of the lichen sclerosus.

Case 2

A 31-year-old gravida 2 para 1 presented at 11 weeks’ gestation for her initial obstetrical examination. At that visit, whitening in a single area was noted, and biopsy was performed at the edge of the lesion on her right interlabial sulcus (Fig. 3). The biopsy revealed lichen sclerosus (Figs. 4 and 5). The patient was asymptomatic throughout her pregnancy. She delivered a 2,720-g infant by a normal spontaneous vaginal delivery. She is currently on topical steroids (triamcinolone acetonide, 0.1% ointment daily) for her vulvar lichen sclerosus.
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DISCUSSION

Lichen sclerosus is a skin disorder that may occur on the vulva. The gross lesions consist of white patches that are thin and may be parchmentlike in appearance. Lichen sclerosus constitutes 40% of the nonneoplastic epithelial conditions of the vulva. The female-male ratio is 10:1. Postmenopausal women are the age group most often affected, but the disorder can be seen in all age groups.

Vulvar lichen sclerosus appears with flat, angular papules that are firm, white, pink, or translucent. Atrophy of the skin is a prominent finding late in the course of the disease. A fine wrinkly “cigarette paper” appearance is characteristic (see Fig. 1). Often a symmetrical involvement about the vulva and complete involvement of the anal orifice are noted, producing an “hourglass” pattern.

The exact cause of lichen sclerosus is unknown. Approximately 16 cases of familial lichen sclerosus have been reported; therefore, it seems likely that heredity plays a role in some cases [1]. Other investigators have proposed an autoimmune pathogenesis for lichen sclerosus [2].

The incidence of lichen sclerosus in pregnancy is unknown. Dalziel [3] analyzed questionnaires from 45 women who had lichen sclerosus and were attending a dermatological vulvar clinic. Eight women reported that they had been pregnant since developing symptoms of lichen sclerosus. Five thought that the disease had been no different during pregnancy. Three of the women stated that it had improved. One woman noted that there had been a worsening of symptoms after each of three pregnancies. A total of 13 deliveries were reported, of which 10 were vaginal deliveries with episiotomy, 2 were vaginal deliveries with episiotomy and forceps, and 1 was a cesarean section. Two women thought that lichen sclerosus had affected their deliveries; one woman stated that a cesarean section was necessary because of lichen sclerosus.

Figure 1. Case 1: A gross representation of lichen sclerosus.

Figure 2. Skin section showing loss of rete ridges. The subepithelial dermis is edematous and hypocellular and contains a small vessel. These features are consistent with lichen sclerosus. (H&E, ×400)
TREATMENT

Lichen sclerosus was first reported by Cinberg in 1945 [4]. Traditionally, it was treated with topical testosterone. Side effects of testosterone may include acne, hirsutism, clitoromegaly, and altered libido. Symptomatic relief often does not occur for 2 to 3 months with testosterone. Testosterone treatment may also be problematic during pregnancy, particularly if the parturient is carrying a female fetus. It can cause labial fusion and clitoromegaly in the female infant. Testosterone has been assigned pregnancy risk factor X by the US Food and Drug Administration.

Currently, the recommended treatment for lichen sclerosus is short-term topical clobetasol propionate, 0.05% (Temovate) [5–7], which often is followed by long-term use of lower-dose topical steroids. There are various methods for the topical steroid dosages. One method consists of clobetasol propionate, 0.05% ointment or cream applied to the vulva twice daily for 1 month, at bedtime for 2 months, and then twice weekly for a period of 3 months. Another approach is the use of clobetasol propionate 0.05% twice daily for 1 month, then daily for 2 months (not to exceed 30 g in 3 months). This is then followed with a midpotency steroid for several months and a gradual decrease to low-potency steroid ointments or creams for long-term use.

The risks and benefits of a high-dose topical steroid must be thoroughly evaluated prior to its use in pregnancy. Clobetasol propionate is absorbed systemically. Conditions that augment systemic absorption include
the application of the more potent corticosteroids, use over large surface areas, prolonged use, and the addition of occlusive dressings. Adrenal suppression may be seen in a patient on topical steroids, but generally that is with doses much higher than those recommended for the treatment of lichen sclerosus. While general glucocorticoid toxicity, such as cataract formation and immunosuppression, have infrequently been described in children born to women on systemic therapy, these adverse effects have not been reported with topical steroids [8]. It is a pregnancy category C medication and has not been tested for teratogenicity when applied topically. As with any category C drug, the potential benefit must justify the potential risk to the fetus. Glucocorticoids have been associated with palatal clefting in mice and rats; however, there is no evidence that human congenital anomalies are increased by the use of these agents during pregnancy [9]. However, gestational exposure to corticosteroids in both animal experiments and human pregnancies have been associated with retarded fetal growth and an increased incidence of low birth weight among offspring [10]. Even lower-dose topical steroids may have been associated with growth retardation in one patient [11]. There are no adequate and well-controlled studies of the teratogenic effects of topically applied corticosteroids, including clobetasol, in pregnant women.

Lichen sclerosus may occur in childhood, and treatment may have to be altered in the young. Pediatric patients may demonstrate greater susceptibility to topical corticosteroid-induced hypothalamic pituitary axis suppression and Cushing’s syndrome than do mature patients because of a larger ratio of skin surface area to body weight. Despite these concerns, potent topical corticosteroids have been used in young children without significant side effects [12]. Testosterone or its derivatives are contraindicated in female children, as they may cause masculinization of the genitalia [13].

REFERENCES