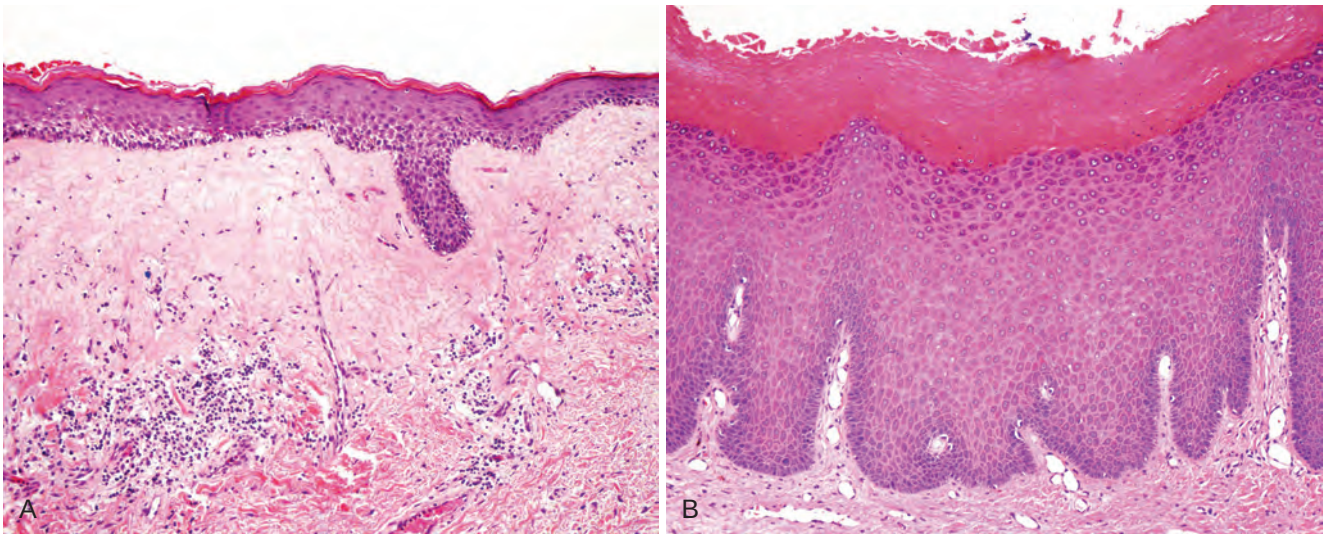

Figure 22-5 Nonneoplastic epithelial vulvar disorders. **A,** Lichen sclerosus. There is marked thinning of the epidermis, sclerosis of the superficial dermis and chronic inflammatory cells in deeper dermis. **B,** Squamous cell hyperplasia, displaying thickened epidermis and hyperkeratosis.

Bartholin Cyst

Infection of the Bartholin gland produces an acute inflammation (adenitis) and may result in an abscess. Bartholin duct cysts are relatively common, occur at all ages, and result from obstruction of the duct by an inflammatory process. These cysts are usually lined by transitional or squamous epithelium. They may become large, up to 3 to 5 cm in diameter, and produce pain and local discomfort. Bartholin duct cysts are either excised or opened permanently (marsupialization).

Nonneoplastic Epithelial Disorders

Leukoplakia is a descriptive clinical term for opaque, white, plaquelike epithelial thickening that may produce pruritus and scaling. Leukoplakia (literally, *white plaques*) may be caused by a variety of benign, premalignant, or malignant disorders, including:

- Inflammatory dermatoses (e.g., psoriasis, chronic dermatitis)
- Lichen sclerosus and squamous cell hyperplasia
- Neoplasias, such as vulvar intraepithelial neoplasia (VIN), Paget disease, and invasive carcinoma

Inflammatory dermatoses associated with leukoplakia are described in Chapter 25, while neoplastic disorders are discussed later in this chapter. Here the major nonneoplastic causes of leukoplakia—lichen sclerosis and squamous cell hyperplasia—are briefly discussed.

Lichen Sclerosus

Lichen sclerosis presents as smooth, white plaques or macules that in time may enlarge and coalesce, producing a surface that resembles porcelain or parchment. When the entire vulva is affected, the labia become atrophic and agglutinated, and the vaginal orifice constricts. Histologically the lesion is characterized by marked thinning of the epidermis (Fig. 22-5*A*); degeneration of the basal cells; excessive keratinization (hyperkeratosis); sclerotic changes of the superficial dermis; and a bandlike lymphocytic infiltrate in the underlying dermis. The disease occurs in all age groups but is most common in postmenopausal women. It may also be encountered elsewhere on the skin. Its pathogenesis is uncertain, but the presence of activated T cells in the subepithelial inflammatory infiltrate and the increased frequency of autoimmune disorders in affected women suggest that an autoimmune reaction is involved. Although lichen sclerosus is not itself a premalignant lesion, women with symptomatic lichen sclerosus have a slightly increased chance of developing squamous cell carcinoma of the vulva.

Squamous Cell Hyperplasia

Previously called hyperplastic dystrophy or *lichen simplex chronicus*, squamous cell hyperplasia is a nonspecific condition resulting from rubbing or scratching of the skin to relieve pruritus. Clinically it presents as leukoplakia and histologic examination reveals thickening of the epidermis (acanthosis), and hyperkeratosis (Fig. 22-5*B*). Lymphocytic infiltration of the dermis is sometimes present. The hyperplastic epithelium may show mitotic activity but lacks cellular atypia. While squamous cell hyperplasia is not considered premalignant, it is sometimes present at the margins of vulvar cancers.

Benign Exophytic Lesions

Benign raised (exophytic) or wartlike lesions of the vulva may be caused by infection or may be reactive conditions of unknown etiology. *Condyloma acuminatum*, a papillomavirus-induced lesion, also called a *genital wart*, and syphilitic *condyloma latum* (described in Chapter 21) are consequences of sexually transmitted infections. Vulvar