# CHRONIC VULVAR DYSTROPHIES

## Surgical therapy

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SUMMARY

The Authors take up a proposal made in 1976 by the International Society for the Study of Vulvar Dystrophies (I.S.S.V.D.) and, with the help of clinical cases, consider the problem of vulvar dystrophic pathology from the point of view of clinico-histologic classification and terminology.

The schematic analysis of the hetiopathogenetical hypothesis (still uncertain) and the consequent not unfrequent failures of clinical therapy suggest to consider indications for surgical treatment. This is particularly true for young patients because in these cases the treatment must also aim at an acceptable aesthetic result and the recovery of satisfactory sexual functionality.

To show the possibility of meeting these requirements, the Authors report the results of surgico-plastic treatment on a 28 year patient suffering from chronic vulvar dystrophy after many failures of medical therapy.

### INTRODUCTION AND CLASSIFICATION OF VULVAR DYSTROPHIES

Terms like atrophic and hypertrophic leukoplakia, lichen sclerosus and atrophicus, leukoplastic vulvitis, primary atrophy, sclerotic dermatosis, kraurosis etc. are often indiscriminately used by Gynecologists and Dermatologists referring to different clinical affections lacking correct classifications and histopathologic definitions.

As early as in 1961, Jeffcoate and Woodcock ([1]) suggested the general definition of 'chronic vulvar dystrophies', the most appropriate from the clinical point of view.

A further description is up to the histopathologist, who must take into account the most important characteristic shared by all these lesions: their potential capability to evolve into invasive and preinvasive malignant forms, related to their degree of cellular atypia.

Subsequently, the International Society for the Study of Vulvar Dystrophies ([2]) suggested a new classification taking into account not only the histopathologic characteristics of dystrophic diseases, but also their malignant potential, which is essential to decide the right treatment.

This classification includes:

1) Hyperplastic dystrophy:
   a) without cell atypia;
   b) with cell atypia.
2) Lichen sclerosus.
3) Mixed dystrophy (Lichen sclerosus with foci of epithelial hyperplasia):
   a) without cell atypia;
   b) with cell atypia.

The lesion commonly referred to as leukoplakia (etym. 'white plate') – characterized by whitish, thickened, irregular cutaneous patches – should therefore be classified among hyperplastic dystrophies.

From the histologic point of view, they are characterized by hyperkeratosis of the



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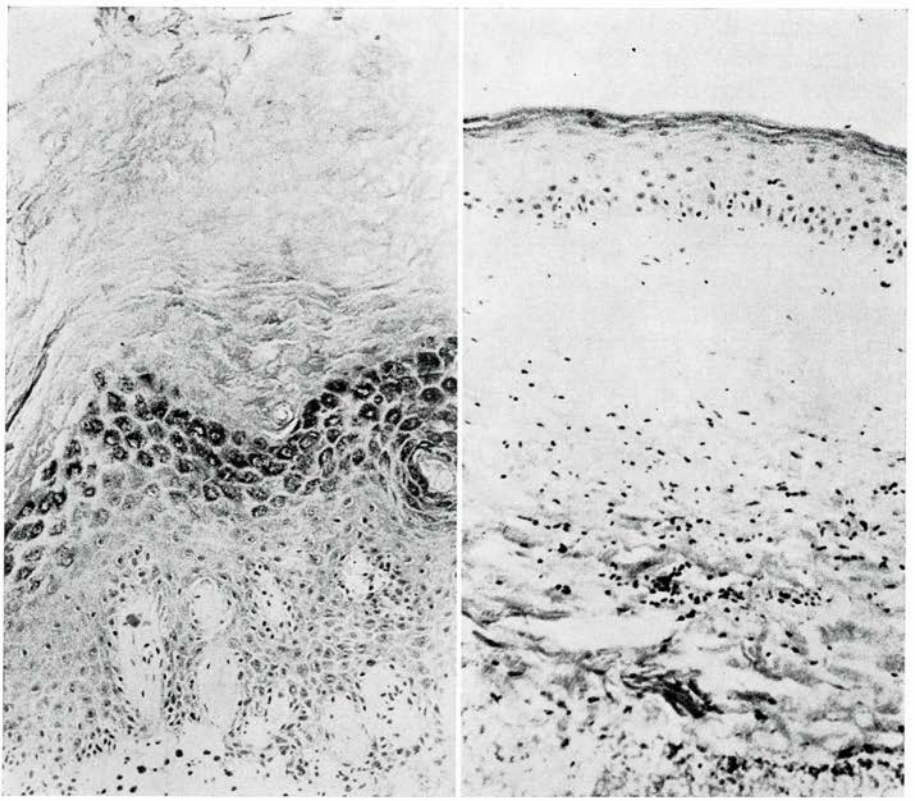

*a* *b*

TABLE 1. — *a*) Histologic picture concerning the case shown in tab. 2 fig. *a*): pronounced epithelial hyperplasia *in toto* and hyperkeratosis. *b*) Histologic picture concerning the case shown in tab. 2 fig. *b*): epithelial atrophy with disappearance of interpapillary plugs; sclerosis of the derma that has been replaced by omogeneous collagenous tissue; moderate superficial hyperkeratosis.

lining epithelium, cellular increase in the granular layer, acanthosis that is thickening of the whole malpighian layer, and increase in the base-layer cells (fig. 1*a*, 2*a*).

Lichen sclerosus includes atrophic lesions, often referred to as primary atrophia or kraurosis (etym. 'dryness'). In these cases, the atrophia affects the whole of the vulva, resulting in drying and shriveling of its mucosa. It can go as far as producing typical thinning and levelling of the labia minora and stenosis of the vaginal ostium.

The histologic characteristics of this clinical picture are: thin epithelial layer, with minimal nuclear component; hyalin degeneration and phlogistic dermoinfiltration (fig. 2*b*, 1*b*).

Finally, mixed dystrophies include clinico-histologic affections simultaneously presenting lichen sclerosus, atrophic lesions and highly hyperplastic areas. Therefore, the various macro and microscopic aspects of vulvar dystrophies are likely to indicate different responses to a single group of patogenous and still unidentified agents, rather than different diseases.

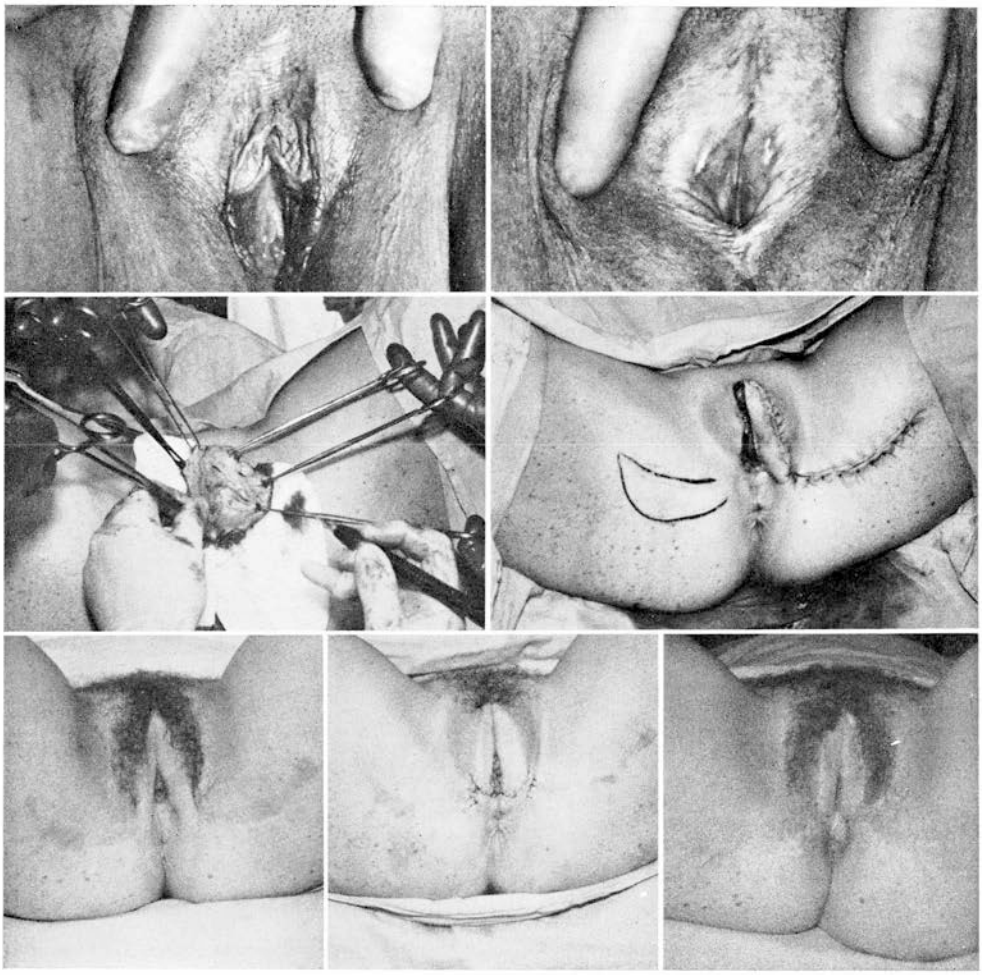

TABLE 2. — *a*) Luigia S. (age 50); hyperplastic dystrophy after toluidine blue test or Richard-Collins test with negative result. Thickened, irregular, whitish patches in the paraclitoral area. *b*) Augusta Z. (age 59); lichen sclerosus with complete disappearance of the labia minora and stenosis of the vaginal ostium. *c*) Simple vulvectomy modified by the exclusion of the side surface of the labia majora. *d*) Displacement of peduncles grafts (cutis and sub-cutis) from the gluteal regions to the removed vulvar region. *e*) Final picture after the first intervention. *f*) V-Y-shaped reconstructive plastic surgery of the vulvar fourchet to correct the result of the previous intervention. *g*) Final picture, a month after the last surgical intervention.

## ETIOPATHOGENETIC REMARKS

The unclear etiopathogenesis of vulvar dystrophies, together with the lack of broad and uniform case-studies, is certainly one of the causes of the terminologic confusion and disparity of therapeutical approach existing in the field of the non-neoplastic vulvar pathology.

Many Authors see the estrogenic deficiency as the one and only cause of these diseases, chiefly setting in after the menopause ([3, 4]).

But Miller and Coll. ([5]) retort that dystrophies can occur in young and even pregnant patients, presenting very high estrogen levels ([6]).

Moreover, many dystrophic patients have normal urinary and plasma estrogen levels, and even long, high-dose estrogen therapies have failed.

With regard to the possible involvement of allergic factors, the same Authors ([5]) point out that clear evidence of an allergic origin of vulvar dystrophies is still lacking, although Parks ([7]) tried to see a correlation between some allergic factors and vulvar itching.

However, they admit that foreign irritating substances can somehow facilitate, tought not cause, the occurrence of dystrophies.

Swift ([8]) was the first to suggest the 'deficiency hypothesis'.

These lesions might originate from a deficiency of vitamine A, due to its insufficient utilization because of gastric hypochloridria.

Other Authors ([9, 10]) confirm this hypothesis and stress the importance of vitamine A for the epithelial eutrophy.

However, Miller and Coll. ([5]) retort by recalling their failure to find vitamine A plasma deficits in 10 patients affected by leukoplakia.

Similarly, they found no noticeable hydrochloric acid deficiency in the gastric juice of 12 other patients affected by leukoplastic vulvopatiae.

Jeffcoate ([11]), studying 269 cases of vulvar dystrophyes, tried to detect these or other possible etiologic agents. He found: glycosuria in 9 cases, despite normal tolerance of glucose load; gastric hypochloridria in 69 and deficiencies in 15 patients; macrocytic anemia in 6 cases; folic acid deficiency associated with bad absorbtion in 2 patients, Trichomonas vaginalis and Candida albicans in 33 patients' vaginae and moniliasis, esclusively in the vulvar and anal areas, in 26 cases.

No possible etiologic agent was found in the remaining 113 patients.

The possible correlation of chronic vulvo-vaginal infections, like candidiasis, and neurodermatites is only apparent, particularly in diabetic patients. In fact, itching, the most frequent symptom of this pathology, often leads patients to start a vicious circle producing scratching lesions or dermatites ([12]).

Friedrich ([13]) recently put forward a 'metabolic theory' to explain the development of vulvar dystrophies, though no clear evidence has so far been found.

According to him, in the normal cutis there is an equilibrium between the stimulus to prolipheration, coming from the sub-epidermic layer, and the production of an antimitotic factor, possibly a proteic hormon, by the epidermis.

In vulvar dystrophies, that equilibrium is broken: atrophic dystrophies present hyperproduction of the mitotic factor; hyperplastic affections show deficiencies of this factor and the dermal stimulus to prolipheration prevails.

These conflicting observations make it impossible to identify one or more essential etiopathogenetic factors in vulvar dystrophies.

The only possible conclusion, for the time being, is that more etiologic factors, though statistically not well identified yet, concur in producing dystrophies of the vulvar cutis.

They act for more or less longer periods, wether in close association or not, and alter the normal cell metabolism and trophism of vulvar cutis cells.

## SURGICAL THERAPY

This study does not aim at examining all the suggested therapies for the treatment of vulvar dystrophies. But we deem it right to report briefly the indications for the surgical exeresis of the dystrophic vulvar area, before examining our clinical

case. Surgical intervention should be performed when:

a) the biopsy of the dystrophic area reveals marked cell atypia suggesting a possible anaplastic evolution, particularly when the atypic dystrophic lesion is multifocal;

b) after the failure of repeated medical therapies, wether hormonal or antimetabolitic, or envisaging local applications of solutions acting through different mechanisms (gentian violet, Betadine, Burow solution etc.);

c) in the presence of chronic dystrophies – particularly in young patients – even if without cell atypia, resisting medical therapies and gradually causing anatomofunctional changes preventing normal sexual activity.

This is the case of a young patient that we observed recently.

## CLINICAL CASE

Maria T., born in 1950, 2 pregnancies ended by normal labours, a spontaneous abortion at the third month. No particular datum in the physiologic and pathologic anamnesis. Vulvar dystrophy was diagnosed in December 1977. She underwent various local medical therapies (testosteron, testosteron-cortisonic association) with neither subjective nor objective results.

The vaginal ostium stenosis worsened with cutaneous chappings of the fourchet. The patient reported itching and serious dyspareunia.

First hospitalization (20-10-1978, hospital file No. 1438/78).

The patient underwent simple vulvectomy, with the exclusion of the outer part of the labia majora and inclusion of clitoridectomy (fig. 2 *c*); dislocation of skin grafst with subcutis and peduncles in the seats of the removed vulvar parts (fig. 2 *d*).

Histologic examination (No. 4903): atrophic epidermic epithelial segments together with hyperplastic parts, with evident leukocytal infiltration into the underlying chorion. Atrophic segments are always associated with sclerotic chorion. Mixed vulvar dystrophy.

Second hospitalization (17-2-1979, hospital file No. 324/79).

Second surgical intervention of plastic correction: peduncles resection and widening of the vulvar ostium (fig. 2 *e*, 2 *f*).

During the following hospitalization (26-11-1979, hospital file No. 1961/79) the whole of the perineale plane cutis, also affected by dystrophy, is removed.

## CONCLUSION

The subsequent clinical controls confirmed the satisfactory surgical and aesthetic outcome of the intervention (fig. 2*g*). The patient reported normal vulvo-vaginal sensitivity and satisfactory sexual activity. In our opinion, no further comment is necessary.

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