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Position of the Spanish Menopause Society regarding vaginal health care in postmenopausal women

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ABSTRACT

Vaginal health, defined as the vaginal state in which the physiological condition remains stable, being protected from the onset of symptoms and facilitating a satisfying sex life, is one of the most common and less valued concerns in postmenopausal women. Many of the conditions that affect the vagina are related to its trophism and susceptibility to infection by unusual germs, which are phenomena strongly influenced by estrogen impregnation and the microbiota composition, ultimately affecting sexuality and the quality of life. An expert panel of the Spanish Menopause Society met to establish criteria for diagnosing and treating the processes that affect overall vaginal health and to decide the optimal timing and methods based on the best evidence available.

1. Introduction

We define "vaginal health" as the vaginal state in which the physiological condition remains stable, being protected from the onset of symptoms and facilitating a satisfying sex life. The vagina is a target organ for estrogens, which affect its moisture, pH, blood flow, and bacterial microbiota. This fact explains why postmenopausal hypoestrogenism causes the vagina to become thin, losing elasticity and rendering it more vulnerable to pain and infection, which affects sexuality and the quality of life [1].

Because a high percentage of women report disorders related to vaginal health during menopause, a panel of experts from the Spanish Menopause Society (SMS) met to review the conditions that determine vaginal pathophysiology and the recommendations for preventing or treating the possible diseases. The SMS considers it appropriate to develop its own recommendations based on the GRADE (*Grading of Recommendations Assessment, Development and Evaluation*) system to elaborate clinical practice guidelines and to classify the quality of the evidence and the strength of the recommendations [2].

2. Vaginal microbiota

Vaginal microbiota refers to the community of microorganisms that colonize the vagina in healthy women. We differentiate indigenous or resident microbiota from transient microbiota. The former is composed of microorganisms that colonize the vagina over a long period and participate in its physiological functions, although their balance depends on factors such as age; phase of the ovarian cycle; sexual activity; pregnancy; and the use of contraceptives,

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antibiotics, or hygiene products [3]. During the reproductive years, the indigenous microbiota is mainly composed of various species of *Lactobacillus*, accompanied to a lesser extent by bacilli and anaerobic Gram-positive cocci, streptococci, enterococci, staphylococci, anaerobic actinomycetes, *Ureaplasma*, and *Mycoplasma hominis*. Isolated in much smaller quantities are bacilli and anaerobic Gram-negative cocci, anaerobes of the genus *Mobiluncus*, *Gardnerella vaginalis*, and *Escherichia coli* [4].

The main components responsible for maintaining the balance of the vaginal microbiota are lactobacilli, which constitute a genus of Gram-positive, anaerobic, and aerotolerant bacteria known for their ability to convert lactose and other monosaccharides into lactic acid. This characteristic causes the vaginal habitat to have an acidic pH, slowing the growth of other potentially pathogenic microorganisms [5]. Some Lactobacillus species isolated in the vagina are also found in our digestive flora, in the digestive flora of other animals, and in various foods (e.g., meat, fish, fresh produce, beer, or wine). The predominant species in the indigenous vaginal microbiota are *L. crispatus*, *L. jensenii*, and *L. gasseri*, followed by *L. iners*, *L. salivarius*, and *L. vaginalis* and to a lesser extent by *L. rhamnosus*, *L. casei*, and *L. plantarum*. In a smaller percentage of women, other bacteria (*Atopobium*) are predominant and are responsible for the microbiota balance, involving the production of antimicrobial substances (e.g., organic acids, hydrogen peroxide, surfactants, or bacteriocins) [6].

Although it is not entirely clear whether the reduction or disappearance of these protective organisms, mainly lactobacilli, is the cause or the effect of the proliferation of other pathogenic microorganisms, we now know that the disruption of the balance of the indigenous microbiota is the pathophysiological basis of vaginitis and vaginosis [7].

As we have discussed, one of the factors that regulate microbiota balance is the pH of the vagina, which is hostile to invasion by other germs when it is acid and thwarts the growth of lactobacilli when it is alkaline. Thus, *Gardnerella* and *Trichomonas vaginalis* are some of the opportunistic pathogens isolated in most cases of vaginitis, displaying a potent enzymatic activity that raises the pH, which favors their own expansion and inhibits the growth of lactobacilli. The same pathophysiological reasoning applies to infections caused by other opportunistic pathogens such as *Candida* spp. or those of the urinary or digestive tract [8].

Similarly, the role of estrogens is relevant to the maintenance of vaginal microbiota, as these hormones regulate vaginal trophism and the composition of its exudate, which is rich in glycogen and other monosaccharides that cause the pH to become acidic, by being metabolized by lactobacilli. Before puberty, the vaginal microbiota is composed of microorganisms from the skin and gastrointestinal tract and is colonized by lactobacilli or other protective species when estrogens begin to exert their effects on the vagina. The parallel between estrogen levels and the vaginal microbiota composition explains why the lactobacilli density is greater in the first phase of the cycle and descends to lower levels during menstruation, when alkalinization of the vaginal microbial habitat occurs due to bleeding and the "drag" effect of hygienic products. In an analogous manner, during intercourse, apart from introducing other germs, semen also increases the vaginal pH, temporarily modifying the microbiota [9].

Furthermore, postmenopausal hypoestrogenism causes thinning of the vaginal epithelium, and there is a smaller contribution of glycogen; consequently, the lactobacilli population is reduced, and the pH rises, favoring colonization by other opportunistic microorganisms. While the microbiota of these women change to consist of intestinal and skin bacteria, as occurred during childhood, almost one-half of these women retain a substantial population of lactobacilli, particularly if they use any type of estrogen treatment [10,11].

3. Atrophic vaginitis

The vagina is the organ with the highest expression of estrogen receptors, which explains why the postmenopausal depletion of this hormone causes progressive hypotrophism, and why this process is often accompanied by some discomfort. The pathognomonic, clinical picture of this period is atrophic vaginitis (AV) (also called vaginal or urogenital atrophy), and unlike other symptoms of menopause that tend to diminish or disappear with the passage of time, AV worsens with age and compromises sexuality and the quality of life of the sufferer. AV affects up to 40% of postmenopausal women, although only a small percentage seek medical attention [12].

In AV, thinning of the mucous membranes of the genital area is observed, with loss of vaginal roughness and elasticity, as well as decreased blood flow, sensory perception, and responsiveness to sexual stimulation. All these factors combined cause itching, dryness, bleeding, vaginal discharge, dyspareunia, inflammation, and urinary symptoms (e.g., dysuria and incontinence), as well as microbiota composition changes that predispose to infection [13]. The reduction of collagen secondary to hypoestrogenism affects the support mechanisms of the pelvic floor and the occurrence of genital prolapsed [14].

Although the diagnosis of AV is confirmed by cytology, it is readily detected during inspection of the external genitalia and vagina by the lack of pubic hair; decreased elasticity, turgor, and moisture of the vagina; scratch marks; thinning of the labia majora, and disappearance of the labia minora and clitoral hood. Sometimes tissue appears prominently in the urethral meatus (urethral caruncle), in addition to polyps and urethral prolapse. Therefore, it can be argued that symptoms of AV are common and can adversely affect quality of life.

Recently a questionnaire for measuring vulvovaginal symptoms in postmenopausal women has been developed: the Vulvovaginal Symptoms Questionnaire is a 21-item written questionnaire with four scales: symptoms, emotions, life impact, and sexual impact [15].

4. Treatment of AV

Conventionally, to relieve the symptoms of AV or dyspareunia, it is recommended to begin treatment with lubricant/moisturizer creams, using topical estrogen therapy only when lubricants provide no relief or do not meet the expectations of the patient, reserving systemic hormonal therapy (HT) for women with other menopausal symptoms [16,17]. However, between 10 and 20% of HT users complain of vaginal dryness [18]. It is also recommended to maintain sexual activity or the use of vaginal dilators because in both cases, the vaginal blood flow is increased, improving the elasticity and lubrication [19].

In addition to other advantages for overall health, it is recommended to quit smoking because this practice worsens vaginal health by reducing the blood perfusion and the bioavailability of estrogens [20].

5. Topical estrogen therapy (TET)

TET is the most effective measure for relieving symptoms arising from AV by normalizing the pH, enhancing vascularization/lubrication, and improving the sexual response. The TETs that are available in our setting are listed in Table 1. In 2012, an EMAS clinical guide stated that the effectiveness of these TETs is similar regardless of the application form (creams, pessaries, ring or tablet) [21]. This guide updates the Cochrane review of 2006 [22] and adds recommendations on TETs use in breast cancer survivors.