

Management of post-menopausal vaginal atrophy and atrophic vaginitis

Camil Castelo-Branco*, Maria Jesús Cancelo, Jose Villero,
Francisco Nohales, Maria Dolores Juliá

*Institut Clínic de Ginecologia, Obstetrícia i Neonatologia, Hospital Clínic, Facultat de Medicina,
University of Barcelona, Villarroel 170, 08036 Barcelona, Spain*

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Abstract

The involution of the female genital tract seems to reflect a built-in biological life expectancy, inter-related with the hypothalamic-hypophyseal-ovarian axis. Lower levels of oestradiol have a number of adverse effects, including on the lower urinary tract. The major universal change is vaginal atrophy. The vaginal mucosa becomes thinner and dry, which can produce vaginal discomfort, dryness, burning, itching, and dyspareunia. The vaginal epithelium may become inflamed, contributing to urinary symptoms such as frequency, urgency, dysuria, incontinence, and/or recurrent infections. Moreover, it has been suggested that reduced oestrogen levels may affect periurethral tissues and contribute to pelvic laxity and stress incontinence. In association with hypoestrogenemia, changes in vaginal pH and vaginal flora may predispose post-menopausal women to urinary tract infection.

Treatment to date has been based on local hormonal therapy, in the form of vaginal creams, tablets or suppositories. Other routes of hormone administration have also proved to be successful. Both local and systemic administration are both effective in maturation of the vaginal epithelium. However, despite the fact that the benefits of oestrogen replacement in preventing vaginal atrophy and reducing the incidence of related symptoms are well established, such therapy is contraindicated in some women and is not an acceptable option for others. Furthermore, the optimal HT administration route, the dosage regimen, and non-hormonal alternatives for improving symptoms and quality of life of the post-menopausal female population, have not been well studied. This review focuses on the changes involved in vaginal aging and efforts to present a synopsis of the pathophysiology and therapy of atrophic vaginitis and vaginal atrophy.

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1. Introduction

As the number of post-menopausal women grows, interest in the effects of oestrogen increase. The influence of oestrogen on certain body systems such as bone

* Corresponding author.

E-mail address: castelo@medicina.ub.es (C. Castelo-Branco).

or cardiovascular has been well documented. However, one specific area that has not been emphasised is the effects on urogenital tract, and a major problem related to menopause is the development of urogenital atrophy [1]. Oestrogen may be related to urogenital aging in several ways: oestrogen prevents a decrease in collagen in post-menopausal women. Topical and systemic oestrogen therapy increases the skin collagen content and maintains skin thickness. In addition, oestrogen maintains skin and urogenital territory moisture by increasing acid mucopolysaccharides and hyaluronic acid and maintaining the epithelial barrier function. Vaginal integrity also may depend on oestrogen levels as a result of the effects of the hormone on the elastic fibres and collagen. The vagina, vulva, urethra, and trigone of the bladder all contain oestrogen receptors and undergo atrophy when oestrogen levels decrease. The vulva and the vaginal walls also become pale and thin and lose their elasticity. This results in decreased vaginal secretion and susceptibility to trauma and pain. In addition, the oestrogen-deficient vagina develops a less acid pH level, ranging from 5.5 to 6.8 [1], which increases the likelihood of urinary tract infections. Fifty to seventy percent of breast cancer survivors indicate in surveys that they experience one or more symptoms of urogenital atrophy [2]. Symptoms include vaginal dryness, dyspareunia, urinary frequency, repetitive urinary tract infections, or urinary incontinence. Dyspareunia leads to decreased interest in coitus. As the frequency of coitus diminishes, vaginal lubrication declines further [3]. However, urogenital aging occurs because of a combination of many factors, not only as a result of oestrogen deprivation.

The age of spontaneous menopause in European countries is between 46.9 and 50.1 years [4]. Women's life expectancy has increased significantly during the past century, and nowadays a female can easily expect to live until the eighth or ninth decade of her life. Most women will spend in the order of one-third of their life in the post-menopausal period, a hypoeestrogenemic state. Fifteen percent of pre-menopausal women, 10–40% of post-menopausal women, and 10–25% of women receiving systemic hormone therapy experience urogenital atrophy [5]. Considering the proportions of this problem, more attention must be focused on the problems faced by women during the post-menopausal period.

The involution of the female genital tract seems to reflect a built-in biological life expectancy, inter-related with the hypothalamic-hypophyseal-ovarian axis. The major universal change is vaginal atrophy. Vaginal dryness, burning, itching, and dyspareunia are frequent complaints, along with dysuria, urinary frequency, and recurrent infections. Treatment to date has been based on local hormonal therapy, in the form of vaginal creams, tablets, or suppositories. Other routes of hormone administration have also proved to be successful. However, despite the fact that the benefits of oestrogen replacement in preventing vaginal atrophy and reducing the incidence of related symptoms are well established, such therapy is contraindicated in some women and is not an acceptable option for others. Approximately one-fifth of the 75–85% of post-menopausal women in whom symptoms of vaginal atrophy and atrophic vaginitis develop will actually go to a physician [6]. This review focuses on the changes involved in vaginal aging and attempts to present a synopsis of the pathophysiology and therapy for atrophic vaginitis, an inflammatory process, and vaginal atrophy, an involutive process.

2. What are we talking about?

Atrophic vaginitis is an inflammation of the vagina that develops when there is a significant decrease in levels of the female hormone oestrogen. Oestradiol, the main oestrogen, which is produced by the ovaries, plays a vital role in keeping vaginal tissues lubricated and healthy. When levels of oestradiol are decreased, vaginal tissue becomes atrophic—thin, dry, and shrunken. Common conditions from low oestrogen levels that result in atrophic vaginitis include menopause, breastfeeding, surgical removal of the ovaries before the age of natural menopause, which can be performed at the same time as a hysterectomy, and medication used to decrease oestrogen levels in women who have conditions such as uterine fibroids or endometriosis. It is clear that prolonged periods of transitional hypoeestrogenism, such as during long-term breast-feeding or GnRH analogue therapy, may induce severe vaginal atrophy and atrophic vaginitis, and that therapy for these situations is essential.

In a recent review, double-blind randomised controlled studies of oestrogen and/or testosterone on sex-

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