*S Afr Gen Pract*. 2025;6(2):51-56
https://doi.org/10.36303/SAGP.3483


S Afr Gen Pract
ISSN 2706-9613 EISSN 2706-9621

REVIEW

# Genitourinary syndrome of menopause

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Genitourinary syndrome of menopause (GSM) encompasses a spectrum of genital, urinary, and sexual symptoms resulting from oestrogen deficiency during and after menopause. Previously termed vulvovaginal or urogenital atrophy, the condition affects 40–54% of postmenopausal women. Oestrogen normally maintains the vaginal epithelium, its elasticity, lubrication, and a protective microbiome. Its decline leads to progressive atrophy, dryness, dyspareunia, higher vaginal pH, and increased infection risk. Urinary symptoms such as urgency, frequency, dysuria, and recurrent urinary tract infections are also common. GSM is underdiagnosed, with many women reluctant to discuss symptoms. Diagnosis is based on clinical features, supported by measures such as vaginal pH or maturation index. First-line management includes lifestyle modification, vaginal moisturisers, and lubricants, while low-dose vaginal oestrogen remains the most effective therapy, improving epithelial health and urinary outcomes. Novel options, such as ultra-low-dose oestriol combined with lactobacilli, show promising efficacy and tolerability.

## Introduction

Genitourinary syndrome of menopause (GSM) is a term used to describe the spectrum of symptoms and physical changes resulting from declining oestrogen levels in the female genitourinary tract during and after menopause.[1] Various terms have previously been used to describe this condition, such as vulvovaginal atrophy, urogenital atrophy, vaginal atrophy, or atrophic vaginitis.[2] These terms, however, did not encompass the urinary symptoms associated with GSM such as incontinence, urgency, and discomfort, and did not allude to the hypo-oestrogenic state.[2]

The term genitourinary syndrome of menopause was introduced in 2014 by the International Society for the Study of Women's Sexual Health (ISSSWH) and the North American Menopause Society (NAMS).[3] The term encompasses all the atrophic symptoms women may experience in the vulvovaginal and bladder-urethral areas from the loss of oestrogen that occurs with menopause.[3]

Around 40–54 % of postmenopausal women and 15% of premenopausal women are affected by GSM.[2]

Other than menopause, there are also other risk factors associated with the development of GSM.[2] These include bilateral oophorectomy, hypothalamic amenorrhoea, alcohol abuse, ovarian failure, radiation therapy, treatment with anti-oestrogens e.g. for breast cancer, smoking, as well as other induced hypo-oestrogenic states not associated with menopause.[2]

## Premenopausal physiology

In premenopausal women, oestradiol is the main circulating oestrogen responsible for maintaining a well-epithelialised vagina during the reproductive years.[4] Oestrogen acts on its receptors in the vagina, vulva, urethra, and trigone of the bladder to maintain:[4]

- Collagen content of the vaginal epithelium, which affects its thickness and elasticity
- Glycosaminoglycan and hyaluronic acid content of the vaginal epithelium, which keeps epithelial surfaces moist
- Optimal genital blood flow
- A healthy vaginal microbiome

As a result, the epithelium of the vagina in response to oestrogen is thick, rugated, and rich in glycogen.[4] Glycogen from sloughed cells is the substrate for the Döderlein lactobacilli, which convert glucose into lactic acid, creating the acidic vaginal environment.[4] The acidity of the vagina helps maintain the normal vaginal flora and protects against vaginal and urinary tract infections.[4]

## Effects of hypo-oestrogenism

Menopause leads to a marked reduction in oestrogen production, with an approximate 95% decline in oestradiol levels from the premenopausal to the postmenopausal state.[4] This dramatic decrease in serum oestrogen levels, exacerbated by the normal ageing process, is responsible for the vaginal atrophy associated with a hypo-oestrogenic state.[4] These changes, unlike vasomotor symptoms of menopause such as hot flashes and/or night sweats, are progressive and usually develop gradually over a period of years.[1,2,4] Hypo-oestrogenic vaginal changes may include:[2,4]

- Loss of elasticity and thinning of the vaginal epithelium
- Loss of vaginal rugae
- Shortening and narrowing of the vaginal canal, with loss of distensibility
- Reduction in vaginal secretions and an increase in vaginal pH to ≥ 5

Thinning of the vaginal epithelium increases susceptibility to trauma, resulting in bleeding, petechia, and ulceration with

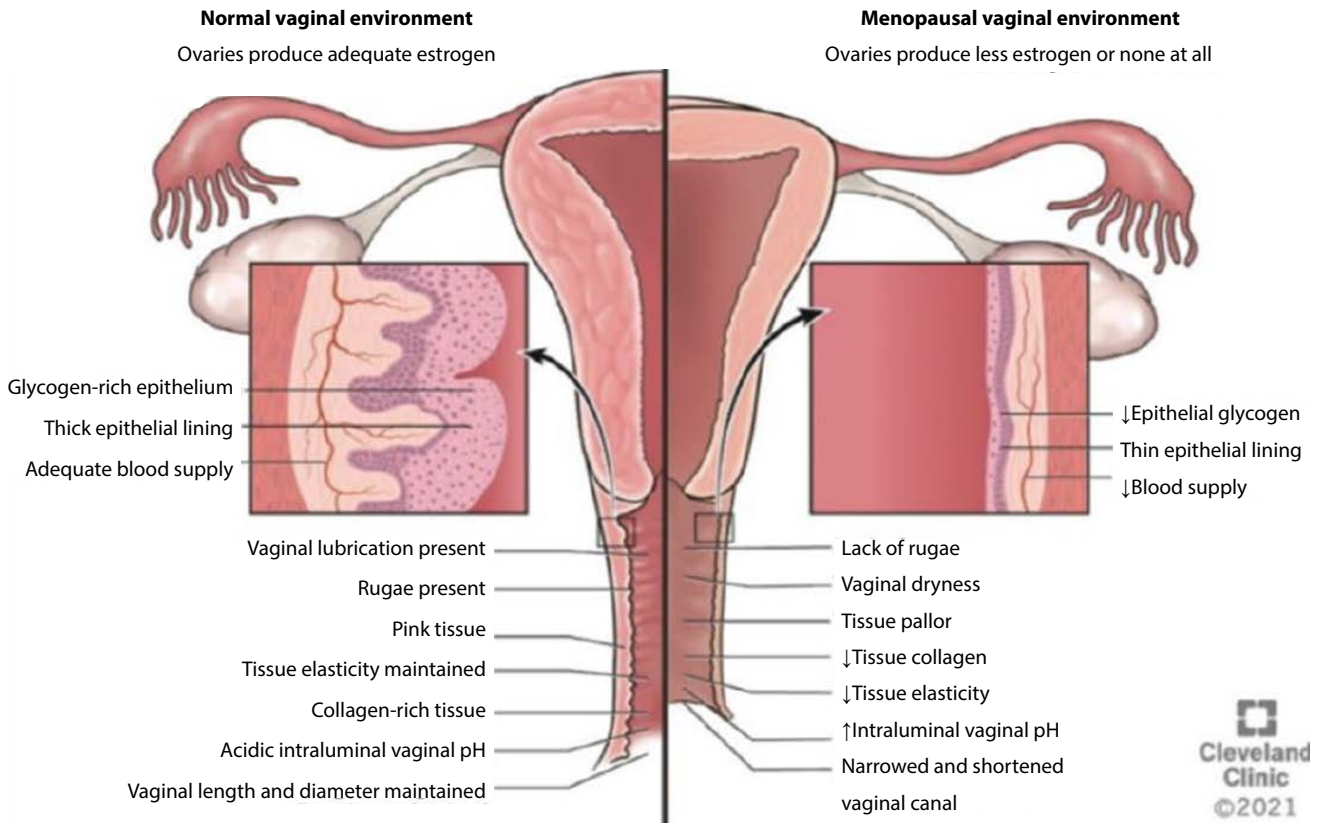


**Figure 1:** An illustration of vaginal atrophy

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pressure, including with sexual activity or the performing of a Pap smear.[4]

The lower glycogen content of a thinner epithelium leads to a reduction in lactic acid production by lactobacilli, resulting in an increase in vaginal pH.[4] The higher pH encourages overgrowth of non-acidophilic coliforms, predisposing women to infection by skin and rectal microflora (e.g. *Gardnerella vaginalis*, streptococci, staphylococci, coliforms, as well as Trichomonas species).[4]

Urinary tract structures in women also contain oestrogen receptors.[4] Therefore, the bladder, urethra and pelvic floor musculature are affected by a hypo-oestrogenic state. Possible consequences of atrophy of the female urinary tract include urethral discomfort, urinary frequency, haematuria, dysuria, and an increased frequency of urinary tract infections.[4]

## Clinical features

The duration of hypo-oestrogenism is a major factor in the development and severity of GSM.[4] By definition, women with GSM are symptomatic.[4] However, not all patients with atrophic changes on pelvic examination are symptomatic.[4]

Symptoms are progressive and worsen with the duration of hypo-oestrogenism.[4] Early in the menopause transition, women may notice a slight decrease in vaginal lubrication upon sexual arousal, which is often one of the first signs of oestrogen insufficiency.[4] As the hypo-oestrogenic state persists, additional symptoms may be reported.[4] Symptoms of GSM can be divided into genital, urinary and sexual symptoms. See Table I.

Atrophic effects usually become clinically apparent 4 to 5 years after the menopause.[5]

- Despite its high prevalence, GSM remains extremely underdiagnosed and untreated.[2,6]
- Up to 70% of women with vaginal atrophy do not discuss their symptoms with a healthcare provider.[4]

It may be of value for clinicians to ask patients if they have pain, itching or sexual symptoms during routine clinical checkups.[4]

**Table I:** Clinical features of GSM[2,3,4,6]

| Genital symptoms | Urinary tract symptoms | Sexual symptoms |
|---|---|---|
| Vulvovaginal dryness<br>Vulvovaginal irritation, itching, or burning<br>Abnormal vaginal discharge, bleeding or spotting | Urinary frequency, urinary urgency, dysuria<br>Stress/urgency incontinence<br>Urethral discomfort<br>Haematuria<br>Recurrent urinary tract infections<br>Urethral prolapse/caruncle | Dyspareunia<br>Decreased lubrication during sexual activity<br>Decreased arousal, orgasm, or sexual desire |
| **Signs** | | |
| Loss of vaginal mucosal folds (rugae)<br>Vaginal pallor<br>Decreased elasticity of the vaginal tissues<br>Higher vaginal pH | Labial atrophy<br>Thinning/greying of pubic hair<br>Pelvic organ prolapse | |

## Evaluation

Clinicians diagnose GSM based on symptoms in a post-menopausal woman, with or without related physical findings, and after ruling out other aetiologies or co-occurring pathologies (e.g. infectious vaginitis, an active urinary tract infection).[1]

Objective measures of postmenopausal vaginal changes include the Vaginal Maturation Index (VMI) and vaginal pH.[1,4]

- The maturation index is the proportion of parabasal, intermediate, and superficial cells in each 100 cells counted on a smear of the upper two-thirds of the vagina. In premenopausal women with adequate oestrogen levels, intermediate and superficial cells predominate.[4] The VMI for these patients is typically 40–70 intermediate cells, 30–60 superficial cells, and 0 parabasal cells.[4] In patients with vaginal atrophy, the VMI demonstrates a shift from superficial cells to parabasal cells as the vaginal epithelium thins.[1] Patients in early menopause typically have a maturation index of 30 intermediate cells, 5 superficial cells and 65 parabasal cells.[4] As patients age, parabasal cells continue to increase, and the VMI may eventually consist entirely of basal cells.[4]
- The vaginal pH increases because fewer epithelial cells exfoliate and break down to release glycogen and glucose, which would typically be broken down into lactic acid by lactobacilli in an oestrogenised vagina.[1] The pH of an oestrogenised vagina is acidic, in the range of 4 to 4.5.[4] Vaginal pH may reach levels of 5.5 to 6.8 or higher in postmenopausal women, especially in those who are not on oestrogen therapy.[4] Therefore, a pH ≥ 5 in the absence of other causes (e.g. infection) can be an indicator of vaginal atrophy due to oestrogen deficiency.[4]

Clinical trials in women with GSM may limit inclusion to women with at least moderate to severe symptoms, 5% or fewer superficial cells on VMI, and a vaginal pH > 5.[1]

However, these measures are not required nor commonly used for clinical diagnosis and treatment of GSM.[1]

## Treatment of GSM

GSM is a chronic and progressive condition.[2] Symptoms of GSM tend to deteriorate if left untreated and rarely resolve spontaneously.[6] The physical changes, signs, and symptoms can negatively affect sexual function, interpersonal relationships, and quality of life among postmenopausal women.[2] Therefore, management of GSM is important.[2]

The main aim of treatment for GSM is to manage the symptoms.[2] Recommended lifestyle modifications include cessation of smoking, weight loss if obese, and regular physical exercise.[2]

### *Local non-hormonal therapy*

First-line therapy for mild to moderate symptoms of GSM includes non-hormonal vaginal moisturisers and lubricants.[2,3] Symptoms of vaginal dryness can be managed by the regular use of vaginal moisturising agents with supplemental use of vaginal lubricants during sexual intercourse.[3] These therapies increase vaginal moisture and improve comfort during sexual intercourse, but do not reverse atrophic vaginal changes.[3]

Patients should be informed about the differences between vaginal moisturisers and lubricants and how to use them.[3]

- Vaginal moisturisers are intended for use routinely, typically two or three times a week, independent of sexual activity.[5] Moisturisers are an option for women who experience ongoing discomfort due to vaginal dryness.[3,6] These products are typically bioadhesives and adhere to the superficial cells of the vagina.[3] They retain moisture, which is then released locally, mimicking physiological vaginal secretions.[5] Hyaluronic acid is often a key ingredient found in vaginal moisturisers.[3]
- Lubricants are used only at the time of sexual activity.[3] They are fast-acting and provide temporary relief of symptoms of vaginal dryness and dyspareunia.[6] Lubricants may be water-based, silicone-based or oil-based.[3] However, oil-based lubricants may cause breakdown of latex condoms.[3] Lubricants differ to moisturisers in that they are not absorbed into the vaginal epithelium and are specifically designed to reduce friction-related irritation to atrophic tissues during sexual intercourse.[6]

### *Vaginal oestrogen therapy*

Low-dose vaginal oestrogen is an effective treatment option for GSM that does not respond adequately to vaginal moisturisers and lubricants.[2,3] It allows for a lower dose of oestrogen than that used in systemic therapy for vasomotor symptoms.[6]

The typical administration schedule consists of an initial loading dose of daily applications for e.g. two weeks, followed by a maintenance regimen (e.g. two to three times a week) for as long as needed to manage symptoms.[6] The rationale behind this administration regimen is that the absorption of oestrogens is highest in the first few days of treatment when the vaginal epithelium is atrophic and has a thinner superficial epithelial layer.[6] Once the epithelium has matured, the local absorption of oestrogen decreases and smaller doses of oestrogen are sufficient to prevent recurring atrophy.[6]

Use of vaginal oestrogen therapy is appropriate for patients with symptoms of vaginal atrophy in the setting of low oestrogen levels, provided there are no contraindications to this therapy (e.g. patients with oestrogen-dependent tumours).[3]

Vaginal oestrogen is absorbed through the vaginal mucosa, improving the blood supply, restoring the normal vaginal acidic pH and microflora, thickening the vaginal epithelium, and improving vaginal lubrication.[2] In addition, vaginal oestrogen therapy is associated with urinary tract benefits.[3] These include a reduction in the incidence of urinary tract infections and overactive bladder symptoms.[3] Urgency and stress urinary incontinence, however, do not improve with oestrogen therapy alone.[3]

Vaginal oestrogen therapy in South Africa is available as vaginal creams or vaginal tablets.[7] The preparations appear to be equally effective in managing GSM and patient preferences should

be considered when deciding on which product to use.[2] Low-dose vaginal oestrogen is defined as ≤ 0.05 mg oestradiol or ≤ 0.3 mg conjugated oestrogens (in ≤ 0.5 g cream).[3] Other doses of conjugated oestrogens (≥ 0.625 mg in 1 g of cream) are considered higher dose preparations.[3] The recommendation is to administer the lowest local dose of oestrogen therapy.[5]

A vaginal cream may be preferred for patients who have symptomatic external atrophy (e.g. vulvar fissures), so that the cream may be applied directly to these areas.[3] When the vulvar atrophy improves, patients may be switched to a vaginal tablet, depending on patient preference.[3]

For patients with GSM treated with low doses of vaginal oestrogen, a progestogen is generally not indicated to protect against endometrial hyperplasia or carcinoma.[3] However, clinical trial data supporting endometrial safety beyond one year are lacking.[3]

Patients treated with low-dose vaginal oestrogen usually have improvement in symptoms after two to four weeks of treatment.[3] Low-dose vaginal oestrogen may be used indefinitely, based on the low risk of adverse effects.[3] However, clinical trials to date have not followed patients beyond one year.[3]

Low-dose vaginal oestrogen is preferred over systemic oestrogen (oral or transdermal administration) for patients who only report vaginal atrophy symptoms.[3] This is consistent with Menopause Society guidelines that state that for urogenital atrophy alone, local vaginal oestrogen therapy is generally recommended over systemic therapy.[3,8,9] For patients receiving systemic oestrogen therapy for other menopausal symptoms (e.g. vasomotor symptoms), low-dose vaginal oestrogen therapy may be added if relief of atrophic symptoms is insufficient.[3]

While topical vaginal oestrogen is associated with a change in microbiome composition (e.g. increased dominance of *Lactobacillus*), a combination of ultra-low-dose oestriol (0.03 mg oestriol (E3) and *Lactobacillus acidophilus* may be an effective option for the treatment of GSM.[5]

- The dose of E3 is substantially lower than that of conventional local vaginal preparations and generates a sufficient local response without having a relevant effect on the endometrium.[5]
- The *Lactobacillus acidophilus* strain has been shown to possess the essential, beneficial *in-vitro* properties for vaginal use.[5]

Combining E3 with lactobacilli has demonstrated synergistic effects.[5,10]

- The 0.03 mg oestriol/lactobacilli combination was considerably more effective in reducing urogenital atrophy, the frequency of urinary tract infections and stress incontinence compared to a 1 mg E3 preparation.[5,10]

Overall, the clinical efficacy of 0.03 mg E3/lactobacilli combination has been demonstrated in various clinical studies.[9] More than 600 women have been evaluated.[5]

- The vaginal 0.03 mg E3/lactobacilli combination leads to similar efficacy compared to the 16-fold higher E3 dose of 0.5 mg.[5]
- Relevant improvements in signs and symptoms such as vaginal dryness, soreness, and dyspareunia have been demonstrated vs. placebo and vs. comparators.[5]

The safety of 0.03 mg E3/lactobacilli combination has been evaluated in more than 4 000 women.[5] The incidence of adverse drug reactions in these studies was 1.7 %, with most side-effects (> 80 %) being local reactions.[5] There was no evidence of typical side-effects of systemic oestrogen treatment, such as thromboembolic events.[5]

### *Other medications*

Dehydroepiandrosterone (DHEA), also referred to as prasterone, is a precursor to oestrogen.[2] Its local application causes increased conversion to oestrogen without any effect on the endometrium.[2] Vaginal DHEA has been shown to improve symptoms of GSM.[2] Daily intravaginal application of DHEA may improve the VMI, vaginal pH and dyspareunia.[2,3] Some data suggest an improvement in libido with vaginal DHEA, but the effect, if present, is modest.[3]

Oral ospemifene, a selective oestrogen receptor modulator (SERM), acts as an oestrogen agonist in the vagina and appears to have no clinically significant oestrogenic effects on the endometrium or breast.[3] The disadvantages of ospemifene compared with vaginal oestrogen are systemic side-effects (hot flashes, potential risk of thromboembolism).[3]

Existing data provide some support for vaginal testosterone to improve symptoms of vaginal atrophy.[3] A reason to consider vaginal testosterone for GSM appears to be for patients who also desire treatment with testosterone for low libido.[3]

Complementary medicines include oral and vaginal herbal supplements (such as the phyto-oestrogens), as well as vitamins and probiotics.[1]

### *Laser or radiofrequency devices*

Laser and other energy-based devices have been used to relieve symptoms of GSM, but there are limited data regarding the safety and efficacy of these devices.[3] These devices may stimulate collagen formation, angiogenesis, and epithelial thickening by causing microtrauma or heating superficial tissue layers.[1]

## Conclusion

Low-dose vaginal oestrogen, when not contraindicated, remains the most effective option for the treatment of GSM and has the fewest side-effects.[6] The use of ultra-low-dose E3 in combination with lactobacilli demonstrates synergistic effects by stimulating the vaginal epithelium and supporting the development of physiological flora.[5] The combination is well tolerated with a low overall incidence of side effects and negligible E3 absorption.[5]

While patient preferences for a particular low-dose vaginal oestrogen product need to be considered, the 0.03 mg E3/ lactobacilli combination is a suitable option for the treatment of symptomatic GSM.[5]

## References


1. Agency for Healthcare Research and Quality (AHRQ). Genitourinary syndrome of menopause. 2024. Available from https://www.pcori.org/research-results/2022/genitourinary-syndrome-menopause-systematic-review. Accessed 27 August 2025.
2. Wasnik VB, Acharya N, Mohammed S. Genitourinary syndrome of menopause; A narrative review focusing on its effects on the sexual health and quality of life of women. Cureus. 2023;15(11):e48143. https://doi.org/10.7759/cureus.48143.
3. Bachmann G, Pinkerton JV. Genitourinary syndrome of menopause (vulvovaginal atrophy): Treatment. Uptodate®. Apr 21, 2025. Available from https://www.uptodate.com/contents/genitourinary-syndrome-of-menopause-vulvovaginal-atrophy-treatment. Accessed 1 September 2025.
4. Bachmann G, Pinkerton JV. Genitourinary syndrome of menopause (vulvovaginal atrophy): Clinical manifestations and diagnosis. Uptodate®. Mar 26, 2025. Available from https://www.uptodate.com/contents/genitourinary-syndrome-of-menopause-vulvovaginal-atrophy-clinical-manifestations-and-diagnosis. Accessed 1 September 2025.
5. Mueck AO, Ruan X, Prasauskas V, Grob P, Ortman O. Treatment of vaginal atrophy with estriol and lactobacilli combination: a clinical review. Climacteric. 2018;21(2):140-7. https://doi.org/10.1080/13697137.2017.1421923.
6. Da Silva AS, Baines G, Araklitis G, Robinson D, Cardozo L. Modern management of genitourinary syndrome of menopause. Faculty Reviews. 2021;10:25. https://doi.org/10.12703/r/10-25.
7. Monthly Index of Medical Specialities (MIMS). 2025;65:6.
8. Guidozzi F, Alperstein A, Bagratee JS, et al.; on behalf of the Council of the South African Menopause Society. South African Menopause Society (SAMS) revised consensus position statement on menopausal hormone therapy 2014. S Afr Med J. 2014;104(8):537-43. https://doi.org/10.7196/SAMJ.8423.
9. North American Menopause Society (NAMS). NAMS Position Statement. The 2022 hormone therapy position statement of the North American Menopause Society. Menopause. 2022;29(7):767-94. https://doi.org/10.1097/GME.0000000000002028.
10. Capobianco G, Wenger JM, Meloni GB, Dessole M. Triple therapy with Lactobacilli acidophili, estriol plus pelvic floor rehabilitation for symptoms of urogenital aging in postmenopausal women. Arch Gynecol Obstet. 2014;289(3):601-8. https://doi.org/10.1007/s00404-013-3030-6.