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Factors Altering the Genitourinary Tract Microbiota: Clinical and Therapeutic Implications

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Abstract

This article examines the composition and function of the genitourinary tract microbiota and its relationship with various urogenital pathologies, highlighting the clinical and therapeutic implications of microbiota imbalance. Under normal conditions, the vaginal and urinary microbiota is dominated by Lactobacillus genus, which play a protective role through the production of lactic acid, hydrogen peroxide, and bacteriocins. However, several factors such as indiscriminate antibiotic use, hormonal changes, intimate hygiene practices, aging, and immune conditions promote microbial dysbiosis. This imbalance favors the proliferation of pathogens such as Escherichia coli, Gardnerella vaginalis, Candida albicans, and Trichomonas vaginalis, increasing susceptibility to recurrent urinary tract infections (UTIs), bacterial vaginosis, candidiasis, and sexually transmitted infections (STIs). The aim of this study was to identify the main factors that alter the microbial balance of the genitourinary tract and analyze their clinical consequences. To this end, a literature review was conducted using recent national and international scientific sources. The study examines pathogenic mechanisms of uropathogenic bacteria, their immune evasion strategies, biofilm formation, and their role in infection recurrence are addressed. Additionally, emerging therapeutic strategies are discussed, including the use of probiotics, personalized treatments, and the importance of a syndromic approach in STI management. We conclude that preserving the eubiosis of the urogenital microbiome is essential to prevent infections, improve reproductive health, and optimize targeted medical interventions.

Keywords: Urogenital Microbiota, Dysbiosis, Urinary Tract Infections, Escherichia Coli, Vaginal Health, Probiotics, Sexually Transmitted Infections, Eubiosis.

Introduction

In 2023, the Mexican Ministry of Health reported that urinary tract infections (UTIs) represented the second most common cause of morbidity in the country, with more than three million three hundred sixty-five thousand affected individuals. Furthermore, recent data from the Global Burden of Disease Study 2019 provide a global overview of the incidence, mortality, and disability-adjusted life years attributable to UTIs across 204 countries between 1990 and 2019 [1].

Under normal physiological conditions, urine and the urinary tract remain sterile, except for the microbial flora of the distal urethra, which includes lactobacilli, corynebacteria, and streptococci. The pathogenesis of UTIs begins when intestinal flora—particularly Escherichia coli—colonize the vaginal and periurethral regions, either persistently or transiently, subsequently ascending to the bladder and, in less frequent cases, to the renal parenchyma [2]. The urogenital microbiota, once considered a sterile environment, has emerged as a complex and dynamic ecosystem playing a central role in urinary tract health and disease. It is composed of bacteria, fungi, and viruses, with Lactobacillus, Gardnerella, Corynebacterium, and Streptococcus being among the most abundant genera. This microbial community performs essential protective functions such as preventing col-

onization by pathogenic microorganisms, modulating immune responses, and maintaining optimal pH levels. The balance of this microbiota can be altered by factors such as age, hormones, sexual activity, intimate hygiene, and antibiotic treatments, which favor the colonization of pathogenic microorganisms. In particular, the impact of these alterations on the development of UTIs, recurrent cystitis, and chronic inflammatory diseases has generated significant clinical and scientific interest [3].

The current understanding of UTIs has evolved considerably, considering not only the presence of common pathogens such as Escherichia coli but also the fundamental role of the urogenital microbiota in health and disease. Recent studies have demonstrated that, far from being sterile, the urogenital tract harbors complex microbial communities, particularly in the vagina, where genera such as Lactobacillus, Gardnerella, and Corynebacterium predominate [4-9]. These communities perform essential protective roles through pH regulation, inhibition of pathogens, and immune modulation. Their imbalance is associated with an increased risk of UTIs, recurrent cystitis, and other gyneco-urological conditions.

Research such as that of Andreu et al. (2005) indicates that UTI pathogenesis depends on host factors, environmental conditions, and specific uropathogenic strains that possess well-defined virulence genes. Likewise, Gilbert et al. (2022) emphasize the need to understand how vaginal and urinary dysbiosis influences the recurrence and severity of these infections. In this regard, France et al., studying the diversity and stability of the vaginal microbiota, suggest that new sequencing technologies allow for the identification of microbial patterns relevant to the prevention and personalized treatment of UTIs. Collectively, these studies reinforce the idea that a holistic approach—integrating the study of microbiota and its interaction with host factors—is essential to advance the diagnosis, management, and prevention of urogenital infections [2, 3, 10].

Infections in the Genitourinary Tract

Factors That Promote Infections

Urinary tract infections (UTIs) are among the most frequent pathologies in gynecological and urological clinical practice, particularly among women, due to a combination of anatomical, physiological, and hormonal conditions that increase their susceptibility. The female urethra, significantly shorter than that of males and located near the perineal area, facilitates the ascent of pathogenic microorganisms, with Escherichia coli being the predominant etiological agent in more than 80% of uncomplicated cases [11]. However, this etiological profile may vary depending on age, hormonal status, and clinical history, which necessitates the consideration of specific contexts in therapeutic management. For example, in postmenopausal women, decreased estrogen levels cause alterations in the urogenital epithelium, reducing defense capacity and modifying the composition of the vaginal microbiota, leading to the loss of protective genera such as Lactobacillus, thereby facilitating the colonization of uropathogenic bacteria [12].

Recent analyses of the role of the vaginal microbiota in female urogenital health have highlighted the influence of lifestyle and intimate hygiene practices on microbial composition. Factors such as the use of vaginal douches, perfumed soaps, intimate deodorants, and excessive washing have been associated with a reduction in dominant species like Lactobacillus crispatus and an increase in dysbiotic bacterial communities, predisposing women to bacterial vaginosis, vaginitis, and a higher risk of recurrent UTIs [13]. Similarly, Morsli et al. (2024) reported that variables such as smoking, diet, sexual activity, hormonal contraceptive use, and stress are closely linked to alterations in the stability of the vaginal microbiota. These practices and conditions can disrupt local homeostasis, lower vaginal pH, and create a favorable environment for pathogenic microbial colonization, which in turn facilitates bacterial migration to the urinary tract [14].

Baimakhanova et al. (2025) provided a global perspective on the clinical, epidemiological, and economic burden of UTIs in women, emphasizing the high recurrence rates, frequent self-medication, and inappropriate use of antibiotics as factors contributing to antimicrobial resistance. They underscore the need for personalized approaches to UTI clinical management, considering not only the causative agent but also the host's physiological conditions and local microbial ecology [15].

From an anatomical and functional perspective, the urinary tract possesses multiple defense mechanisms against microbial colonization, including the production of uromodulin (a protein produced in the kidneys that plays an important role in preventing urinary tract infections), constant urinary flow, and the activity of the urothelial epithelium. However, these defenses can be overcome in the presence of dysbiosis, hormonal imbalances, or compromised immune conditions, highlighting the close relationship between anatomical integrity, microbial homeostasis, and susceptibility to developing UTIs [13].

Pathogenesis of UTIs

Urinary tract infections caused by uropathogenic Escherichia coli (UPEC) represent a paradigmatic model of bacterial infection, combining efficient epithelial colonization with sophisticated immune evasion mechanisms. The pathogenesis of these infections begins with the adhesion of UPEC to the urothelium through type 1 and P fimbriae, which specifically recognize mannose and glycosphingolipid receptors present on the epithelial cells of the urinary tract. This step is essential to resist urinary flow and establish initial colonization [16]. Once adhered, UPEC can invade urothelial cells and form intracellular bacterial communities (IBCs), structures that provide protection from the innate immune response, allowing survival within macrophages and evasion of phagocytosis. Furthermore, this pathogen can modulate the host's inflammatory response by inhibiting key pathways such as inflammasome activation and the production of proinflammatory cytokines, facilitating bacterial persistence and promoting recurrent infections. This immunomodulatory mechanism not only protects the bacterium but also contributes to subclinical inflammation that can damage the epithelium and disrupt the local microbiota, creating a favorable environment for reinfection [16].

Additionally, the genomic study by Sung et al. (2024) provides a comprehensive view of the molecular determinants underlying this pathogenic capacity. The analysis of clinical UPEC strains revealed a broad presence of virulence genes, including adhesins such as fimH and papG; siderophores such as ent and iutA for iron acquisition; hemolysins; and biofilm formation factors

[17]. These structures not only facilitate colonization and tissue damage but also act as physical barriers that hinder antimicrobial penetration. Moreover, UPEC displays a high capacity to express different phenotypes, evidenced by the presence of plasmids, pathogenicity islands, integrons, and transposons that enable the horizontal acquisition of antimicrobial resistance genes, giving rise to multidrug-resistant strains. This combination of factors makes UPEC particularly difficult to eradicate, as it can survive conventional therapy, form intracellular reservoirs, and reemerge after treatment—significantly contributing to the clinical burden and costs associated with recurrent UTIs. Both studies agree that understanding these molecular mechanisms is crucial for developing new targeted therapeutic strategies, including vaccines, adhesin inhibitors, and immunomodulatory therapies [17].

The etiology of UTIs in women is closely linked not only to colonization by common pathogens such as UPEC but also to imbalances in the vaginal microbiota that increase susceptibility to infection. Vaginitis, defined as inflammation of the vaginal mucosa, frequently results from dysbiosis characterized by a decrease in protective Lactobacillus species, which are responsible for maintaining an acidic vaginal pH (approximately 3.5–4.5) and producing antimicrobial compounds such as hydrogen peroxide, lactic acid, and bacteriocins. The loss of this protective barrier facilitates the proliferation and colonization of opportunistic microorganisms such as Gardnerella vaginalis, Candida spp., and Trichomonas vaginalis, which disrupt vaginal homeostasis and promote a local inflammatory environment. This alteration of the vaginal ecosystem not only causes local symptoms but can also facilitate the ascension of uropathogenic bacteria into the urinary tract, increasing the risk of recurrent UTIs. Changes in microbial composition and elevated vaginal pH reduce natural competition against pathogens and promote adhesion and biofilm formation by bacteria such as UPEC. Therefore, vaginitis and its effects on the vaginal microbiota constitute a key etiological factor that contributes to the susceptibility and persistence of urinary infections in women [18].

Microorganisms Causing UTIs

The main microorganisms responsible for urinary tract infections (UTIs), classified by type of pathogenic agent, are listed below (see Table 1).

Table 1: Main Etiological Agents of UTIs

Type of Microorganism	Representative Examples	Brief Description
Bacteria	Escherichia coli, Klebsiella pneumoniae, Staphylococcus saprophyticus, Enterococcus spp.	Gram-negative and Gram-positive bacilli, with E. coli being the most frequent cause of UTIs. (Nicolle, 2008)
Viruses	Herpes simplex virus (HSV), Human papillomavirus (HPV), Cytomegalovirus (CMV), Human immunodeficiency virus (HIV)	Viruses that can affect the urinary tract, particularly in immunocompromised individuals. (Heidegger et al., 2015)
Parasites	Trichomonas vaginalis	Protozoan that causes urethritis and vaginitis, sexually transmitted. (Workowski et al., 2010)
Yeasts	Candida albicans	Opportunistic yeast that can cause UTIs, especially in immunocompromised patients. (McLaughlin & Carson, 2004)

Note: Table created by the author based on the articles by Nicolle (2008), Heidegger et al. (2015), Workowski et al. (2010), and McLaughlin & Carson (2004), which provide an updated overview of the main etiological agents involved in urinary tract infections. The table summarizes the principal groups of pathogens bacteria, viruses, parasites, and yeasts their representative species, microbiological characteristics, and clinical relevance, emphasizing their role in both uncomplicated and recurrent infections, particularly in immunocompromised or predisposed individuals [19-22].

The Genitourinary Tract Microbiome

Anatomy and Physiology as Barriers Against Infections

The urinary tract, under normal physiological conditions, remains sterile despite its proximity to body regions with a high microbial load. This condition is maintained through anatomical and physiological defense mechanisms such as the constant flow of urine, the integrity of the urothelial epithelium, protective mucosal layers, and proteins like uromodulin, which prevent bacterial adhesion to the urinary tract walls. Functional anatomy also plays a crucial role in preventing infections. Frequent urination, the presence of anti-reflux valves, and complete bladder emptying reduce the risk of bacterial proliferation. When these structures are compromised by conditions such as vesicoureteral reflux, prolonged catheter use, or urinary retention, susceptibility to genitourinary tract infections increases considerably [13].

Urinary Microbiome in Health and Disease

Recent studies have refuted the notion that the urinary tract is sterile. Through molecular sequencing techniques, a specific, low-biomass microbiota has been identified as resident in the bladder, urethra, and urine of healthy individuals. This microbiota plays an essential role in defending against infections, mainly through mechanisms of competitive exclusion and local regulation of the urothelial environment. Within this microbiota, genera such as Lactobacillus crispatus have been documented as key to maintaining a healthy environment. These bacteria produce lactic acid, lower urinary pH, and strengthen the mucosal barriers of the epithelium, thereby inhibiting the proliferation of pathogenic microorganisms [7].

The intestinal origin of many urinary tract infections is well established; however, the role of the vaginal microbiota and its relationship to infection susceptibility is also recognized. A decrease in the abundance of Lactobacillus spp. can facilitate colonization of the periurethral area by uropathogenic bacteria, promoting their ascent toward the bladder [23]. When vaginal

balance is disrupted, local parameters such as pH, cervical mucus viscosity, and defensin expression are altered. This weakens the system's ability to block external bacterial invasion, increasing the risk of recurrent genitourinary tract infections [24].

Virulence Factors and Pathogen Colonization

Uropathogenic E. coli (UPEC) strains, responsible for most urinary tract infections, have developed specialized colonization mechanisms. Among these, P-type and type 1 fimbriae stand out for their ability to adhere strongly to the urothelial epithelium, resist the mechanical flow of urine, and facilitate persistence in the bladder [12, 25]. Additionally, these strains can modulate their metabolism according to the urinary microenvironment. This metabolic plasticity enables them to efficiently utilize available nutrients, increasing their viability and replication capacity in an environment that is normally hostile to other microorganisms [25].

Urinary Microbiome

The urinary system including the kidneys, ureters, bladder, and urethra harbors a diverse microbiota that previously went unnoticed due to the limitations of conventional culture techniques. Thanks to molecular detection methods, a wide variety of microorganisms have been identified, particularly in the urethra and bladder, where bacteria such as Lactobacillus, Staphylococcus, and Gardnerella predominate, many of which play protective roles. Current research seeks to understand how these microorganisms influence urinary tract balance and their involvement in diseases such as urinary tract infections, incontinence, and cancer [26].

Table 2:

Genus / Species	Type of Bacteria	Characteristics	Clinical Implications
Lactobacillus spp.	Facultative anaerobe, Gram-positive	Produces lactic acid and hydrogen peroxide; commonly found in healthy women	Protects against uropathogens such as Escherichia coli and Klebsiella pneumoniae; its decrease favors infections
Gardnerella vaginalis	Facultative anaerobe, Gram-variable	Second most abundant after Lactobacillus in women; forms biofilms	Associated with urinary infections in women; increases during dysbiosis
Streptococcus spp.	Facultative anaerobe, Gram-positive	Present in the healthy urinary tract of both sexes	Linked to incontinence in women; contributes to the degradation of beneficial flora in dysbiosis
Prevotella spp.	Strict anaerobe, Gram-negative	Common in young and prepubertal individuals; also present in adults	More frequent in dysbiosis; found in urinary incontinence and neurogenic bladder
Corynebacterium spp.	Aerobe or anaerobe, Gram-positive	More prevalent in men, but also found in women	Can be commensal, but associated with infections during dysbiosis
Escherichia coli (UPEC)	Facultative anaerobe, Gram-negative	Main pathogen in UTIs; expresses P-fimbriae adhesins and forms biofilms	Causes ~80–90 % of UTIs; resistant to host defenses and spreads under dysbiotic conditions
Enterococcus spp.	Facultative anaerobe, Gram-positive	Part of the normal flora but increases during dysbiosis	Associated with urinary tract infections and antibiotic resistance
Klebsiella pneumoniae	Facultative anaerobe, Gram-negative	Nosocomial uropathogen, frequent in elderly patients with catheters	Second leading cause of UTIs; capable of biofilm formation and antimicrobial resistance
Aerococcus urinae	Aerobe, Gram-positive	Forms biofilms; can cause bacteremia and endocarditis	Invasive infections in the elderly; proper diagnosis and antimicrobial sensitivity testing are essential

Note: Table developed by the author based on the article by Colella et al. (2023), which provides an updated review of the human urinary tract microbiota under health and disease conditions. The table summarizes the main bacterial genera and species identified, their classification, functional characteristics, and their relationship with various clinical states, highlighting their role in eubiosis as well as in dysbiotic conditions associated with urinary infections and other urogenital pathologies [26, 27].

Genitourinary Tract Microbiota

The urogenital microbiota comprises the community of microorganisms including bacteria, fungi, and others that naturally inhabit the urinary and genital tracts of both men and women. This microbial community plays essential roles in maintaining the health of the urogenital system. Among its main functions are protection against pathogenic agents through mechanisms such as competition for nutrients and adhesion sites, as well as the production of antimicrobial substances. It also contributes to local immune balance and helps maintain optimal physiological conditions, such as pH regulation. An imbalance in this microbiota, known as dysbiosis, can predispose individuals to urinary tract infections, inflammatory diseases, and other urogenital disorders. Therefore, studying the urogenital microbiota has become a key area for understanding its role in the prevention and treatment of various conditions [28, 29].

- **Urethral Microbiota**

In the study conducted by Chen et al. (2020), the microbial composition of the female urethra was investigated using advanced methods such as expanded culture and confocal microscopy to overcome the limitations of traditional techniques. These tools

enabled the identification of a significant bacterial diversity in the female urethra, including intracellular bacteria that are typically undetectable through conventional culturing. Among the microorganisms identified were Lactobacillus, Gardnerella, and other anaerobic genera commonly associated with the vaginal microbiota, suggesting microbial continuity between the vagina and the urethra. Moreover, intracellular bacteria were observed in urethral mucosa and asymptomatic urine samples, indicating that the urethra may harbor a stable and concealed microbiota potentially involved in persistent or recurrent urinary symptoms. These findings challenge the long-standing notion that the female urethra is sterile and open new perspectives for understanding the role of urethral microbiota in female urinary tract health and disease, emphasizing the importance of considering the urethra as a key component of the urinary ecosystem [30].

- **Vaginal Microbiota**

The vaginal microbiota is a dynamic microbial ecosystem characterized, under normal conditions, by the predominance of Lactobacillus species. Their protective function lies in maintaining an acidic pH, generating antimicrobial substances, and strengthening mucosal defenses. This microbial community plays a crucial role in infection prevention by acting as a barrier against opportunistic pathogens. However, when dysbiosis occurs such as a decrease in Lactobacillus and an increase in species like Gardnerella, Atopobium, or Prevotella conditions such as bacterial vaginosis, vaginitis, recurrent urinary tract infections, and pregnancy complications may develop. In addition, certain alterations in the microbiota have been associated with chronic inflammatory processes and increased susceptibility to sexually transmitted infections.

From a therapeutic perspective, characterization of the vaginal microbiota has driven the development of targeted probiotics, recolonization strategies, and microbiota-based therapies, as well as the use of new technologies (metagenomics, proteomics, metabolomics, culturomics, RNA sequencing, etc.) to design personalized approaches. This perspective opens promising pathways for the prevention, treatment, and clinical monitoring of vaginal dysbiosis and its consequences [28-31].

Medical and Therapeutic Implications

Most Common Conditions

Under healthy conditions, the urinary tract hosts a community of protective microorganisms, mainly bacteria of the Lactobacillus genus, which play an essential role in inhibiting the growth of pathogens through the production of lactic acid, hydrogen peroxide, and other antimicrobial substances. However, when these beneficial bacteria decrease whether due to excessive antibiotic use, hormonal changes, aging, or other clinical conditions the proliferation of opportunistic microorganisms such as pathogenic Escherichia coli, Klebsiella pneumoniae, and Proteus mirabilis is promoted.

The dominance of these pathogens induces inflammatory processes, facilitates adhesion to the urothelial epithelium, and triggers recurrent infections, posing a significant public health challenge. In this context, preserving the stability and diversity of the urinary microbiota represents a key preventive strategy to reduce the incidence and recurrence of urinary tract infections (UTIs), highlighting the potential therapeutic value of interventions aimed at restoring eubiosis in this microbial ecosystem.

Cystitis, the most common form of UTI, can be complicated by antibiotic resistance. A recent study analyzed how the urinary microbiome changes during antimicrobial treatment in patients with cystitis. It was found that beneficial bacteria such as Lactobacillus protect against pathogens, but a standard seven-day cefalexin therapy reduced these protective bacteria, favoring recurrent infections. Moreover, just two days of treatment were sufficient to significantly reduce the pathogen load. These findings suggest the need to reassess treatment duration to optimize antibiotic use and preserve urinary microbiome balance [32].

Various gynecological diseases are closely linked to vaginal microbiota dysbiosis, highlighting how the imbalance within this microbial community contributes to the development and progression of multiple pathologies. Firstly, bacterial vaginosis is characterized by a significant decrease in beneficial Lactobacillus species and an increase in anaerobic bacteria such as Gardnerella vaginalis and Atopobium vaginae, which alters vaginal pH and promotes pathogen overgrowth. This condition not only causes discomfort but is also associated with an increased risk of ascending infections.

Another common infection is vaginal candidiasis, mainly caused by the proliferation of Candida albicans, an opportunistic yeast that takes advantage of microbial imbalance to colonize and cause inflammation. Likewise, trichomoniasis an infection caused by the protozoan Trichomonas vaginalis is favored in dysbiotic environments, leading to increased inflammation and epithelial damage.

Additionally, vaginal dysbiosis increases vulnerability to sexually transmitted infections (STIs) such as chlamydia and gonorrhea, facilitating the colonization and persistence of these pathogens. On a broader level, these microbial alterations are related to the development of pelvic inflammatory disease (PID), a serious complication that affects the reproductive organs and can result in infertility. Vaginal dysbiosis has also been associated with obstetric complications, such as preterm birth and premature rupture of membranes, which negatively impact maternal-fetal health.

Taken together, this evidence underscores the importance of maintaining vaginal eubiosis to prevent the onset and progression of gynecological diseases and to preserve women's reproductive health [28].

Complications in UTIs and Vaginal Infections

Complicated urinary tract infections (UTIs) represent a significant clinical challenge due to their association with anatomical, functional, or systemic factors that increase the risk of therapeutic failure, recurrence, and morbidity. These infections include cases in patients with structural abnormalities of the urinary tract such as obstruction, lithiasis, vesicoureteral reflux, or urological instrumentation, as well as in those with immunosuppression, renal failure, advanced age, or conditions such as diabetes.

In men, any episode of UTI is considered complicated due to anatomical factors and the potential for prostatic reservoirs. These infections are often caused by atypical or multidrug-resistant

pathogens, which require careful selection of antimicrobial therapy. Management involves the mandatory use of urine culture, empirical initiation of broad-spectrum antibiotics, and longer treatment durations, accompanied by complementary studies such as imaging and specialist evaluation.

The complexity of these infections demands a multidisciplinary approach involving physicians, pharmacists, and nursing staff, focusing not only on treatment but also on the prevention of recurrences and control of persistent infectious foci [19, 33].

Vaginal infections in women of reproductive age carry several clinical complications that affect both maternal and perinatal health. Vulvovaginal candidiasis is frequently associated with threatened abortion, cervical dysplasia, pelvic inflammatory disease, salpingitis, recurrent urinary tract infections, postpartum endometritis, and infertility, reflecting its impact on the integrity of the reproductive tract and long-term reproductive function.

Regarding trichomoniasis, this infection has been implicated in premature rupture of membranes, preterm labor, low birth weight, and atypical pelvic inflammatory disease, in addition to a high incidence of threatened miscarriage, underscoring its potential to cause severe obstetric complications.

Chlamydial infection is also associated with a broad range of obstetric and reproductive complications, including preterm birth, premature rupture of membranes, chorioamnionitis, postpartum endometritis, infertility, spontaneous abortion, and, in severe cases, fetal and maternal death. These complications highlight the clinical importance of early detection and proper management to prevent adverse outcomes in women's reproductive health and fetal viability [34].

Treatments

UTIs in adults continue to be a frequent and clinically significant problem that requires accurate diagnosis and appropriate treatment to avoid complications and antimicrobial resistance. In recent years, significant advances have been made in both diagnostic techniques and therapeutic options.

Among the innovations is the use of molecular methods that allow faster and more specific detection of causative pathogens, improving the ability to identify recurrent or complicated infections. Likewise, therapeutic management has evolved toward individualized strategies based on local resistance profiles and clinical severity, promoting the rational use of antibiotics and the incorporation of prophylactic treatments in selected cases. These innovations contribute to optimizing clinical management, reducing morbidity, and limiting the spread of resistant strains—key aspects for public health [35].

Effective prevention of sexually transmitted infections (STIs) requires a comprehensive approach combining educational strategies, correct and consistent use of barrier methods, and timely surveillance and treatment of diagnosed infections. In patients with HIV, coinfection can complicate the clinical course and increase transmission of both types of infections, making early detection and appropriate management essential. Treatment must also be adapted to account for drug interactions and the patient's immunological status.

Continuing medical education is fundamental to ensuring up-to-date knowledge of best diagnostic and therapeutic practices, as well as promoting risk reduction and improving quality of life in these vulnerable populations [36].

The treatment of STIs represents a major public health challenge due to their global morbidity impact, HIV transmission potential, and associated complications such as infertility, ectopic pregnancy, and neonatal diseases. The World Health Organization (WHO) guidelines emphasize a standardized, syndromic approach especially in resource-limited regions where etiological diagnosis may be inaccurate or inaccessible.

Syndromic management is based on the identification of clinical syndromes such as urethral discharge, genital ulcers, vaginal discharge, scrotal inflammation, or lower abdominal pain, offering empirical treatment against the most likely pathogens including Neisseria gonorrhoeae, Chlamydia trachomatis, Treponema pallidum, Herpes simplex virus, and Trichomonas vaginalis, among others.

The use of highly effective antimicrobials (≥95%), preferably in a single oral dose, is recommended, prioritizing regimens with low toxicity and good tolerance. Increasing antimicrobial resistance has limited the use of traditional drugs such as tetracyclines and sulfonamides, favoring third-generation cephalosporins, macrolides, and fluoroquinolones.

In addition to medical treatment, patient education, condom promotion, HIV counseling, and notification and treatment of sexual partners are emphasized. Respectful, confidential, and nonjudgmental care is essential to ensure access, therapeutic adherence, and effective control of STIs [37].

Conclusion

Current evidence confirms that the microbiota of the genitourinary tract plays a crucial role in preventing infections and maintaining urogenital health. Factors such as inappropriate antibiotic use, hormonal imbalances, and inadequate hygiene practices can disrupt this ecosystem, promoting dysbiosis and the emergence of opportunistic pathogens. Understanding the interactions among the microbiota, host, and pathogens enables the development of more effective diagnostic and therapeutic strategies.

In this context, the rational use of antimicrobials, the incorporation of probiotics, and personalized treatments emerge as complementary measures to preserve eubiosis and reduce infection recurrence. Finally, integrating this knowledge into clinical practice, prevention, and research is essential to optimize urogenital health and address the growing challenge of antimicrobial resistance.

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