Bacterial Etiologic Agents of Genitourinary Tract Infections Among Female Patients Attending a Hospital in Port Harcourt, Nigeria

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https://doi.org/10.56201/ijhpr.vol.11.no10.2026.pg1.7

Abstract
This review outlines the microbiological landscape, clinical complications, and molecular resistance mechanisms of key bacterial pathogens specifically Neisseria gonorrhoeae and Staphylococcus aureus associated with genitourinary tract infections (GUTIs) and dysbiosis among female patients attending a tertiary hospital in Port Harcourt, Nigeria. In resource-limited settings such as this, the convergence of N. gonorrhoeae and dysbiosis-associated S. aureus poses severe threats to female reproductive health, escalating clinical morbidity and complicating empirical treatment. By integrating epidemiological data and molecular frameworks, we highlight how horizontal gene transfer, chromosomal mutations, and vaginal microbiota disruption facilitate both pathogen acquisition and multidrug resistance. Notably, N. gonorrhoeae exhibits widespread resistance to fluoroquinolones, beta-lactams, and macrolides through target gene mutations and plasmid-mediated determinants, while S. aureus presents formidable challenges via methicillin resistance (MRSA carrying the mecA gene) and emerging vancomycin-intermediate (VISA) phenotypes. Furthermore, S. aureus colonization and associated dysbiosis disrupt the protective vaginal microenvironment, increasing susceptibility to gonococcal and ascending infections like pelvic inflammatory disease (PID). Ultimately, the high burden of antimicrobial resistance (AMR) in this region underscores the critical limitations of empirical monotherapies, highlighting an urgent need for localized, molecularly informed surveillance, routine susceptibility testing, and robust stewardship programs to safeguard female reproductive health.

Keywords: *Genitourinary Tract Infections, Neisseria gonorrhoeae, Staphylococcus aureus, Antimicrobial Resistance, Vaginal Dysbiosis, Port Harcourt.*

Introduction

Antimicrobial resistance (AMR) is increasingly recognized as a major and urgent public health threat because it reduces the effectiveness of antibiotics used for prevention, treatment, and control of bacterial infections. Antibiotic resistance emerges when bacterial populations acquire and accumulate genetic changes that render drugs less effective, often amplified by antibiotic overuse, misuse, and insufficient development of new antimicrobials (Gould & Bal, 2013; CDC, 2013). Sexually transmitted infections (STIs) are particularly relevant to AMR dynamics because they sustain ongoing transmission at the population level and frequently require antimicrobial therapy. Among bacterial STIs, *Neisseria gonorrhoeae* (gonorrhea)

remains a major global concern, both due to its high incidence and its demonstrated propensity to develop resistance to multiple antibiotic classes (WHO, 2014; Unemo & Shafer, 2014). In parallel, *Staphylococcus aureus* is clinically important in women because it can colonize the vagina and contribute to dysbiosis-associated conditions, including bacterial vaginosis (BV), thereby potentially increasing susceptibility to other infections (Mumtaz et al., 2008; Wondemagegn et al., 2015). Together, AMR in *N. gonorrhoeae* and *S. aureus* can contribute to increased morbidity, prolonged infection, and escalated clinical and economic burden especially in resource-limited settings where diagnostic and treatment options may be constrained (CDC, 2013). This review outlines the microbiological landscape and resistance mechanisms of key bacterial pathogens, specifically *N. gonorrhoeae* and *S. aureus*, associated with genitourinary tract infections and dysbiosis among female patients attending a tertiary hospital in Port Harcourt, Nigeria.

Epidemiology

Neisseria gonorrhoeae

Global estimates indicate substantial ongoing transmission of gonorrhea, with the number of new infections measured in tens of millions annually. WHO-supported surveillance has reported large burdens and continues to document geographic and sex-specific variation in prevalence and incidence (Kirkcaldy et al., 2019). In the United States, routine surveillance has highlighted the scale of resistant infections and the associated health impacts (CDC, 2013). Regional data also suggest that prevalence can vary considerably across populations and settings (Abbai et al., 2013; Mlisana et al., 2012; Pourabbas et al., 2018).

Staphylococcus aureus and vaginal dysbiosis

Staphylococcus aureus colonization in females of reproductive age has been documented across a wide age range, and it has been implicated in aerobic vaginitis and dysbiosis-associated conditions (Mumtaz et al., 2008; Wondemagegn et al., 2015). BV is common among women of reproductive age and varies by country, with reported estimates spanning a broad range (Murta et al., 2005). Importantly, BV and vaginal microbiota disruption are linked to increased acquisition and spread of STIs, which may create a context in which resistant organisms gain epidemiological advantage (Myer et al., 2005; Roberta et al., 2005).

Mechanisms of Antimicrobial Resistance

Resistance mechanisms in *Neisseria gonorrhoeae*

AMR in *N. gonorrhoeae* can arise through (i) spontaneous genetic mutation and (ii) horizontal acquisition of resistance determinants. Reported mechanisms include alteration of antibiotic targets, inactivation of antimicrobials, enhanced efflux, and decreased antimicrobial uptake (Unemo, 2015). Resistance to fluoroquinolones is associated with amino acid changes in chromosomal targets, particularly topoisomerase and DNA gyrase functions (Tanaka et al., 1998). For β-lactams, resistance is linked to modifications in penicillin-binding proteins (PBP1 and PBP2) and/or recombination and non-synonymous mutations that impair drug binding (Ropp et al., 2002). Macrolide and spectinomycin resistance have been associated with nucleotide changes in rRNA targets, while altered porin proteins and upregulated efflux systems can reduce intracellular antibiotic concentrations (Galimand et al., 2000; Gill et al., 1998; Zarantonelli et al., 1999; Veal et al., 2002). Additional reports describe plasmid-associated β-lactamases and *tetM*-associated determinants relevant to tetracycline and penicillin resistance (Muhammad et al., 2014).

Resistance mechanisms in *Staphylococcus aureus*

Staphylococcal resistance is frequently multidrug and can be mediated by mobile genetic elements that facilitate spread between strains and sometimes species (Todar, 2011). Methicillin resistance is a key example: MRSA sustains cell wall synthesis under β-lactam pressure through altered penicillin-binding protein function, notably PBP2 linked to the *mecA* gene contained within the mec region (Hiramatsu, 1995; Hiramatsu et al., 1997).

Vancomycin has traditionally been used against MRSA, but intermediate and resistant phenotypes (VISA/hetero-VISA and VRSA) have been described, reflecting progressive adaptation and narrowing therapeutic options (Hiramatsu et al., 1997; Sievert et al., 2002). This progression supports the need for ongoing molecular characterization of resistance determinants rather than reliance solely on phenotypic susceptibility patterns.

Clinical implications

Untreated gonorrhea in women

Gonococcal cervicitis is often asymptomatic or minimally symptomatic in women, enabling persistence and ascending infection. Untreated infection can lead to pelvic inflammatory disease (PID), which is associated with long-term reproductive sequelae including infertility and ectopic pregnancy. Reported estimates indicate that a substantial proportion of infected women may experience complications, and gonorrhea has been implicated as an important etiologic contributor to PID (Gerbase et al., 1998; Kristen et al., 2017).

In pregnancy, gonococcal infection has also been linked to adverse outcomes such as chorioamnionitis, intrauterine growth restriction, preterm membrane rupture, and preterm birth (Liu et al., 2013; Christine et al., 2017). Neonatal transmission during vaginal delivery may cause severe neonatal disease, including ocular complications (Johnson et al., 2011).

BV/aerobic dysbiosis and STI susceptibility

BV is characterized by a shift away from lactobacilli-dominated vaginal microbiota toward higher abundance of opportunistic and pathogenic taxa, which can change vaginal pH and immune context. This dysbiosis is associated with increased risk of acquiring STIs, including gonorrhea and other sexually transmitted pathogens (Murta et al., 2005; Roberta et al., 2005).

In addition, *S. aureus* colonization and toxin-producing strains may contribute to inflammatory vaginal conditions and may increase the likelihood of complications including reproductive adverse outcomes and postpartum conditions (Myer et al., 2005; Hay et al., 1994; CDC, 2006).

Treatment and stewardship challenges

International guidance has moved toward combination therapy strategies in response to treatment failures associated with monotherapy, particularly in settings where ceftriaxone effectiveness is threatened by rising resistance (WHO, 2016). However, the continued evolution of resistance along with variable resistance levels to key agents means dual therapy does not guarantee long-term efficacy. This underscores the importance of local susceptibility testing, antimicrobial stewardship, and surveillance to inform treatment algorithms (Unemo & Shafer, 2014; WHO, 2016).

Knowledge gaps

Despite extensive documentation of AMR mechanisms and resistance evolution, several gaps remain relevant for region-specific microbiology and translational public health:

1. **Local incidence and resistance patterns:** Resistance profiles and organism prevalence can vary substantially across geography and patient subgroups, yet many settings particularly those with limited microbiology infrastructure lack longitudinal and molecularly informed surveillance.

2. **Molecular correlation with clinical resistance:** Phenotypic susceptibility patterns do not always fully explain clinical treatment outcomes; integrative datasets linking MIC/susceptibility results with resistance gene profiles remain underutilized in many local studies.

3. **Interrelationship of dysbiosis and STI acquisition:** The microbiological pathways connecting BV/aerobic vaginitis, *S. aureus* colonization density, and subsequent gonococcal acquisition and transmission require further mechanistic and epidemiological clarification.

4. **Implementation of surveillance and stewardship:** Even when guidelines exist, real-world implementation including antibiotic prescribing practices and diagnostic access can differ markedly by setting, affecting the effectiveness of interventions.

Conclusions

AMR in *Neisseria gonorrhoeae* and *Staphylococcus aureus* represents a converging threat at the intersection of reproductive health, STI transmission, and antimicrobial stewardship. In women, gonococcal infection can progress to severe reproductive sequelae, while vaginal dysbiosis involving *S. aureus* may further heighten STI susceptibility and clinical complications. Current evidence supports the need for integrated surveillance combining microbiological culture, antimicrobial susceptibility testing, and molecular characterization of resistance determinants. Such evidence is essential to guide empirical therapy, reduce treatment failures, and inform targeted public health interventions especially in settings where the burden of infection and the risk of resistant pathogen spread are disproportionately high.

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