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Review Article

Scoping review of common bacterial organisms causing vaginal discharge and their antibiotic susceptibility profile in Indian subcontinent

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ABSTRACT

Vaginal discharge (VD) is a major public health issue in the Indian subcontinent, where rising antimicrobial resistance (AMR) compromises syndromic case management (SCM). This scoping review maps the bacterial etiologies of VD and their antibiotic susceptibility across the Indian subcontinent. Based on the CoCoPop framework, a systematic search was conducted on PubMed, Scopus, Embase, Cochrane Library and Google Scholar up to 15 March 2025. Data was extracted from 107 eligible studies involving 37,846 women. Majority of women were from low socioeconomic status. Pathogens isolated amongst symptomatic women included *Gardnerella vaginalis* (0.38-74%), *Staphylococcus sp*. (0.9-52.5%), *Mobiluncus sp.* (4.29-42.13%) and *Neisseria gonorrhoeae* (0.14-40.34%). In asymptomatic women, *Peptostreptococcus sp.* (66.67%), *Bacteroides sp*. (33.33%), *Streptococci sp.* (25.64%) were identified. Diagnosis relied primarily on microscopy and culture over molecular methods. The key pathogens demonstrated moderate to high sensitivity to Penicillins, Tetracyclines, Macrolides, Sulfonamides and Fluoroquinolones. High sensitivity was reported for Cephalosporins against *N. gonorrhoeae* (89.5%) and Metronidazole (71%) against *G. vaginalis*. Aminoglycosides, Beta-Lactam/Beta-Lactamase Inhibitor combinations and Carbapenems demonstrated >70% sensitivity against resistant bacteria including *S. aureus, E. coli, Pseudomonas sp*. Antibiotic data on anaerobes and MIC data was scarce. High resistance rates challenge the efficacy of standard treatment kits. A strategic shift integrating affordable diagnostics with evidence-based regimens tailored to regional microbial resistance data optimizing treatment and combat antimicrobial resistance is essential.

Keywords: Vaginal discharge, Bacterial vaginosis, Indian subcontinent, Antibiotic susceptibility, Antimicrobial resistance

INTRODUCTION

Overview of vaginal discharge and infectious aetiologies

Pathological vaginal discharge (VD) is a leading cause of gynecological and sexually transmitted infection (STI) clinic visits worldwide.[1] While common pathogens include *Chlamydia trachomatis, Trichomonas vaginalis*, and *Candida spp*., this review focuses on bacterial causes and their antimicrobial susceptibility in the Indian subcontinent.[2]

Key bacterial causes of vaginal discharge worldwide, include Neisseria gonorrhoeae and bacterial vaginosis (BV), characterized by a shift from protective Lactobacillus to anaerobes such as Gardnerella, Prevotella, and Mobiluncus.[3,4] Aerobic vaginitis (AV) affects up to 23% of non-pregnant women, typically caused by *Enterococcus faecalis, E. coli, Group B Streptococcus*, and *S. aureus*.[5] Both conditions cause significant morbidity including pelvic inflammatory disease (PID) and adverse pregnancy outcomes, while emerging pathogens like *Mycoplasma genitalium* present growing resistance concerns.[6,7]

Bacterial colonization is also reported in vaginal discharge of asymptomatic women, demonstrating that microbial presence alone does not indicate infection.[8]

Burden of the problem: global and Indian

An estimated 1 million STIs occur worldwide, with 374 million new infections annually.[9] In India, symptomatic VD prevalence ranges from 14.5% to 78.5%.[10] Common causative bacteria include *S. aureus, E. coli, Klebsiella, Enterococcus, Pseudomonas* and GBS.[11] BV prevalence varies by region and population ranging from 13% in East India, 32.8% in Delhi, and higher in human immunodeficiency virus (HIV)-positive women.[12-14] AV prevalence ranges from 5.3% to 22.9%.[15] Approximately 6% of adults have one or more STIs in India.[16]

Limited diagnostic availability and affordability in low-middle income countries (LMICs) has prompted strategies like syndromic case management (SCM) (India).[17] SCM relies on identifying consistent symptom patterns to guide empirical treatment without waiting for laboratory confirmation, ensuring timely care. However, growing prevalence of antimicrobial resistance necessitates pathogen-directed approaches. World Health Organization (WHO) recommends laboratory-based diagnosis with quality-assured NAATs to improve targeted therapy.[18]

Prevalence of symptomatic VD during pregnancy varies widely ranging from 5% in Nigeria to 48% in India, 89% in Pakistan, with global estimates indicating 23–29%.[9,19,20] In a study from India 11.3% of menopausal women reported VD, later diagnosed due to BV and PID.[21] At menopause, VD is frequently linked to vulvovaginal atrophy (VVA) from estrogen deficiency. Rare causes like cervical tuberculosis require differential diagnosis.[22]

Antibiotic susceptibility testing

The Clinical Laboratory Standards Institute (CLSI) and European Committee on Antimicrobial Susceptibility Testing (EUCAST) have established internationally recognized protocols to ensure reliable and comparable antibiotic susceptibility testing (AST) results across different laboratories and geographical regions.[23] These standardization efforts are critical for generating clinically meaningful resistance data that can guide therapeutic decisions and inform surveillance programs.

Minimum inhibitory concentration (MIC) determines the lowest concentration of antibiotics required to inhibit pathogen growth. CLSI defined breakpoints guide therapeutic decisions.[23] Organisms are categorized as susceptible (S), intermediate (I), and resistant (R) as per CLSI breakpoints to assist drug choice and optimize outcomes.[23]

Challenges in testing pathogens associated with vaginal discharge particularly for bacterial vaginosis-associated organisms including *Gardnerella vaginalis, Prevotella sp*., and other anaerobes is the requirement of specialized culture conditions.[24] Fastidious organisms such as *Mycoplasma sp*. and *Chlamydia sp*. necessitate molecular detection methods rather than conventional culture-based approaches, making standard AST methods inadequate.[25]

Rationale

To guide therapy, regional susceptibility data are necessary due to the wide range of antibiotic resistance patterns observed across pathogens. This scoping review identifies the prevalence and antibiotic susceptibility profiles of bacteria associated with vaginal discharge in the Indian subcontinent, allowing clinicians to move beyond empirical regimens towards evidence-based targeted treatment.

Evidence gap map

This review includes an evidence gap map (EGM) to map existing evidence to present the antibiotic use for bacterial vaginal discharge in the Indian subcontinent.[26]

METHODS

Protocol and registration

The scoping review protocol was developed based on PRISMA-ScR extension for scoping reviews and registered on 07th November 2024 with Open Science Framework (OSF) database.[27]

The standard steps for scoping review were: identifying relevant studies, study screening and selection, data extraction. This scoping review was aimed to systematically map the existing literature without assessing the methodological quality of included studies, report writing, summarizing data and charting the data as an evidence gap map (EGM).

To structure this scoping review, the population, condition, context (CoCoPop) framework was adopted. This framework is well-suited for scoping reviews aiming to map evidence on a specific health condition within a particular population and setting.

Three assessors (AGR, CG, YKP) independently screened titles and abstracts and appraised full texts after duplicate removal. (PP) built search strategies for major medical databases and compiled studies selected for full-text screening. Data extraction from the final 107 selected studies was performed using a structured sheet on Microsoft Excel 2007 capturing details such as study design, sample size, participant socioeconomic details, bacteria prevalence, antibiotic susceptibility/resistance, minimum inhibitory concentration (MICs).[28] Data entry onto the CADIMA platform as was conducted by AS, CG and YKP. RKM, AGR and KS oversaw data synthesis and report writing. The data were analyzed for three specified outcomes: pooled prevalence of bacteria organisms associated with vaginal discharge, antibiotic sensitivity and resistance for bacterial pathogens and MICs of relevant antibiotics.

Search strategy

We conducted a comprehensive search of the following electronic databases: PubMed, Scopus, Embase, and the Cochrane Library. The search was limited to English-language studies and was conducted without a date restriction to capture the full scope of available evidence. All publications up to 28 February 2025 were included in the initial search and supplementary search in google scholar included studies up to 15 March 2025. A combination of keywords and controlled vocabulary (MeSH terms where applicable) was used.

The search was executed with the following filters applied across all databases: Language (English only) and Population (Human studies). No date restrictions were applied to ensure a comprehensive historical scope. The results reported in the PRISMA-ScR flowchart (Figure 1) reflect the number of citations retrieved after these filters were applied. All publications up to 15 March 2025 were included in the search on Google Scholar.

Study selection

Across indexed databases in initial search, 4170 studies were found. In supplementary search on Google Scholar, 65 additional studies were found (Figure 1). EndNote version 21 was used to remove 562 duplicate records. CADIMA was used to screen 3673 titles and abstracts, of which 247 were selected for full text screening as per inclusion and exclusion criteria.[2,29,30] During the screening process, non-primary research articles such as editorials, letters, and narrative reviews were excluded.

A total of 247 articles were assessed for full-text eligibility. From these, 140 articles were excluded for the following reasons: full-text not available (n=27), no relevant bacterial data provided (n=68), irrelevant population group (n=22), study not from the Indian subcontinent (n=12), conference abstracts with insufficient data (n=9) and narrative reviews (n=2). This resulted in a final inclusion of 107 studies for data extraction (Figure 1). Although studies reporting on mixed etiologies (e.g., bacterial and fungal) were considered for inclusion, data extraction for this review was strictly limited to bacterial pathogens and their related findings to adhere to the primary research question.

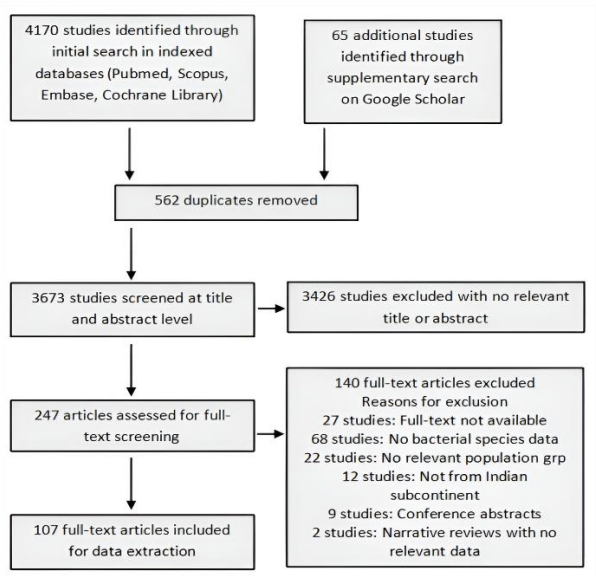

Figure 1: PRISMA-ScR flow chart.

Data extraction

A standardized data extraction sheet was developed and piloted using Microsoft Excel (version 2007).[28] Three reviewers independently extracted the data (CG, KS, YP). Discrepancies were resolved through discussion and consensus. In accordance with the PRISMA-ScR guidelines for scoping reviews, a formal risk of bias assessment of the included studies was not performed, as the aim was to map the extent of the available evidence regardless of its quality. GRADE assessment was not conducted. An evidence gap map (EGM) was developed using EPPI-Mapper, affiliated with EPPI-Reviewer, to visually synthesize evidence on antibiotic use across the Indian subcontinent for treating vaginal discharge.[26] Of the 107 studies included, only 43 reported data on antibiotic use.

A narrative synthesis was undertaken to summarize findings. Data were categorized by bacterial species, antibiotic susceptibility, and population subgroup. Descriptive statistics (frequencies, proportions) were used to summarize organism distribution and resistance patterns.

RESULTS

Overview of included studies

This review included 107 studies comprising data from 37,846 women across the Indian subcontinent. The geographical distribution of the studies, with most research

originating from public healthcare facilities in India (n=71).

While the review focused on bacterial pathogens, *Vulvovaginal candidiasis* (5-72.4%) and *Trichomoniasis* (0.4-16.7%) were also cited as important causes of vaginitis.

Evidence gap map

An evidence gap map (EGM) was developed from the 43 studies that reported on antibiotic use to visualize research density across different populations and interventions. Cephalosporins were the most frequently studied antibiotic class, while "non-pregnant healthy women" was the most investigated population. Research was heavily concentrated in India, with significant evidence gaps for other Indian subcontinent countries.

Each colored block within the map represents number of studies conducted in the specific population subgroup; pregnant women (pink), non-pregnant adolescent (yellow), non-pregnant healthy women (green), non-pregnant women with comorbidities (red) and non-pregnant menopausal women (blue).

Demography

21 out of 107 included studies reported data on socioeconomic status (SES). Majority (50-71%) of the women belonged to low socioeconomic strata.[20,21,31]

Key socioeconomic factors and significant associations

India

A significant association was found between vaginal discharge and low socioeconomic status in women of reproductive age in North India (p=0.01).[22,32] Low socioeconomic status was also associated with bacterial vaginosis (BV) in pregnant women in India (35.7% versus 10.5%, p=0.041); higher bacterial culture positivity compared to high income group women with VD in South India (16% versus 4.66%).[33,34] Co-morbidities and vaginal infection were significantly correlated in rural Tamil Nadu, providing a striking contrast between the two conditions: 72.4% of diabetics and 66.6% of HIV-positive women were diagnosed with vulvovaginal candidiasis, whereas bacterial vaginal infection was only detected in 6.9% of diabetics and none of HIV-positive women.[34]

Pakistan

Low SES was significantly associated with STI prevalence (p=0.001) among non-pregnant women.[35] Further, Pakistani women from low socioeconomic backgrounds (68.3%) were 1.27 times more likely to contract Trichomoniasis (TV) and 3 times more likely to contract BV.[36] However, a study from Karachi, Pakistan, found no difference in chlamydia prevalence (2%) between asymptomatic pregnant women from low or higher SES.

Nepal

Infectious vaginitis was significantly higher among women with low family income (p<0.001).[37]

Bangladesh

In a study of low income 439 female sex workers in Bangladesh, the prevalence of gonorrhoea/chlamydia cervical infections was not statistically significant (OR 0.9, 95% CI: 0.6-1.5).[38]

Bhutan

No relevant socioeconomic data reported in two included studies from Bhutan.

Prevalence of bacteria organisms associated with vaginal discharge (VD)

As shown in Tables 4 and 5, the prevalence of bacteria associated with vaginal discharge varies by region within the Indian subcontinent and within India. Figure 4 illustrates the wide range and variability of findings across 107 studies. Due to the limited number of studies for specific pathogens, a formal meta-analysis could not be conducted to calculate a pooled prevalence. Furthermore, significant heterogeneity was observed among the included studies in terms of study populations, diagnostic methodologies, regional settings, and timeframes. Therefore, we have presented the prevalence range of pathogens.[39,40]

Most common species in Indian subcontinent

In India; *G. vaginalis* (0.38-74%) and *Staphylococcus sp.* (0.9-52.5%), Pakistan; *G. vaginalis* (2.9-59.67%) and *E. coli* (5.2-29.62%), Nepal; *Mobiluncus sp.* (42.13%), *G. vaginalis* (BV) (15.65-49.25%), Bangladesh; *G. vaginalis* (22.35-48.06%), Sri Lanka; *Streptococci sp.* (28%) and Bhutan; *G. Vaginalis* (22.75%) were reported (Table 1).

Overall, anaerobic bacteria and Gram-negative organisms were the most frequently reported categories of pathogens associated with vaginal discharge across the included studies (Tables 1 and 2).

Diagnostic methods

Pathogen identification predominantly relied on traditional culture-based methods (61.7%) and microscopy/Gram staining (51.4%), with modern molecular methods (PCR/NAATs) used in only 11.2% of studies (Table 3). Most studies reported using a combination of these diagnostic approaches.

Study design and sample size distribution of included studies

Among the included studies, 67 (62.6%) were cross-sectional studies, and 20 (18.7%) were prospective cohort studies. A critical finding from 12 studies was that pathogenic bacteria were often present in asymptomatic women, indicating that microbial presence does not always indicate clinical infection (Table 4).

Figure 3 depicts the bacterial profile across pregnant, non-pregnant and menopausal women. Pregnant women (23.83%) and menopausal women (6.23%) were most likely to have *Gardnerella vaginalis* (BV), while non-pregnant women (36%) had *Peptostreptococcus spp.*

Clinical symptoms

Only 50 of the 107 studies provided data on symptoms and signs. The most frequently reported clinical symptom was pruritus followed by lower abdominal pain, dysuria and dyspareunia while the clinical characteristics of vaginal discharge was mostly described as white, curdy white, or greyish in colour. The volume of discharge varied across studies, with reports ranging from scanty to copious or profuse. The consistency was observed to be thick or homogeneous with a foul or fishy odor which was a prominent and commonly reported symptom. These findings underscore the varied clinical presentations and highlight the importance of correlating symptoms with microbiological evaluation for accurate diagnosis and management.

Table 1: Country-wise distribution of bacteria associated with VD in Indian subcontinent.

Pathogen	India (%)	Pakistan (%)	Nepal (%)	Bangla-desh (%)	Sri Lanka (%)	Bhutan (%)	Prevalence range (%)	Number of studies
Anaerobes								
Peptostreptococcus spp.	36	*	*	*	*	*	36	1
Mobiluncus spp.	4.29-5.33	*	42.13	*	*	*	4.29-42.13	3
Lactobacillus spp.	7.14	*	10.61-21.88	*	*	*	7.14-21.88	3
Bacteroides spp.	4-13	*	*	*	*	*	4-13	3
Gram-negative lactobacilli	7.33	*	*	*	*	*	7.33	1
Prevotella spp.	6	*	*	*	*	*	6	1
Clostridium spp.	2.83-5.33	*	*	*	*	*	2.83-5.33	2
Fusobacterium spp.	2	*	*	*	*	*	2	1
STD causing pathogens								
Ureaplasma urealyticum	20.63	*	*	2.01	*	*	2.01-20.63	3
Mycoplasma spp.	1.22-10.33	*	*	*	*	*	1.22-10.33	5
Chlamydia trachomatis	0.21-14.93	5.33-12.25	0.7-15.49	5.39-6.45	*	*	0.21-15.49	17
Neisseria gonorrhoeae	0.14-11	*	40.34	7.19-13.63	*	13.64	0.14-40.34	25
Treponema pallidum	0.42-4.22	0.75	2.5	*	*	*	0.42-4.22	4
Gram-positive organisms								
Staphylococcus spp.†	0.9-52.5	4-17.52	2.59-3.11	17.50	*	*	0.9-52.5	33
Streptococci spp.‡	2.26-25.85	3.33-6.12	1.55-2.01	12.12	28	*	1.55-28	21
Enterococcus spp.	1-19.66	3.43-17	1.04-1.98	*	*	*	1-19.66	20
Bacillus subtilis	*	5	*	*	*	*	5	1
Diptheroids	0.5-5.6	*	*	*	*	*	0.5-5.6	2
Eubacterium	3	*	*	*	*	*	3	1
Gram-negative organisms								
Gardnerella vaginalis	0.38-74	2.9-59.67	15.65-49.25	22.35-48.06	*	22.75	0.38-74	50
Gram-negative coccobacilli	7.33-46	*	*	*	*	*	7.33-46	2
Escherichia coli	3.21-43.54	5.2-29.62	5-39.25	17.5	*	*	3.21-43.54	14

Continued.

Pathogen	India (%)	Pakistan (%)	Nepal (%)	Bangla-desh (%)	Sri Lanka (%)	Bhutan (%)	Prevalence range (%)	Number of studies
Klebsiella spp.	2-21.77	3.27-21.82	1.04-28.5	11.33	*	*	1.04-28.5	12
Pseudomonas spp.	0.85-10	1.36-7	1.55-6.25	19.50	*	*	0.85-19.5	10
Citrobacter spp.	0.47-17.69	1-5.91	0.63	*	*	*	0.47-17.69	4
Enterobacter spp.	0.47-4	0.05-4	0.63-21.5	19.50	*	*	0.46-21.5	6
Acinetobacter spp.	1.38-3.5	1.6	5	*	*	*	1.38-5	6
Proteus spp.	0.97-4	0.52-5	2.07-3.13	*	*	*	0.52-5	4
Providencia spp.	0.32-0.85	0.26	*	*	*	*	0.26-0.85	3
Morganella spp.	*	0.05	*	*	*	*	0.05	1

†*Staphylococcus sp.*=Coagulase negative Staphylococci (CoNS) + *Staphylococcus aureus* + *Staphylococcus epidermis* + *Staphylococcus albus*. ‡*Streptococcus sp.*=*Beta-hemolytic streptococci* + Group B streptococci (GBS)+ Group D streptococci + other Streptococci species.
*Data not available

Table 2: Region-wise distribution of bacteria associated with vaginal discharge in India.

Pathogen	North India prevalence range (%)	No. of studies in North India	South India prevalence range (%)	No. of studies in South India	West India prevalence range (%)	No. of studies in West India	East India prevalence range (%)	No. of studies in East India
Anaerobes								
Bacteroides spp.	12.5-13.5	2	4	1	*	*	6.6	1
Mobiluncus spp.	*	*	4.28	1	*	*	*	*
Lactobacillus spp.	*	*	6.9-7.4	2	*	*	*	*
Peptostreptococcus spp.	36	1	*	*	*	*	*	*
Prevotella spp.	6	1	*	*	*	*	*	*
Clostridium spp.	5-7	2	*	*	*	*	2.83	1
Fusobacterium spp.	2	1	*	*	*	*	*	*
Gram negative lactobacilli	*	*	7.33	1	*	*	*	*
STD causing pathogens								
Treponema pallidum	2.32	1	*	*	*	*	*	*
Neisseria gonorrhoeae	3.5-4.5	2	6.05	1	0.97	1	*	*
Chlamydia trachomatis	6.67	1	1.42	1	0.98	1	*	*
Mycoplasma spp.	6.22	1	2-2.5	2	6.04	1	*	*
Ureaplasma urealyticum	15.24	1	*	*	20-21.8	2	*	*
Gram-positive organisms								
Staphylococcus spp.†	7-7.5	2	7.8-8.2	2	3.28	1	5.18	1
Streptococci spp.‡	4.74	1	4	1	4.15	1	2.59	1
Enterococcus spp.	8.79	1	8.51	1	7.05	1	1	1
Diptheroids	*	*	6	1	0.5	1	5.6	1
Eubacterium	3	1	*	*	*	*	*	*
Gram negative organisms								
Gardnerella vaginalis	0.38-62.39	10	4-74	15	4.4-53.6	12	20-58.52	3
E. coli	16.5-30	5	3.21-43.54	9	11.95-41.75	3	7.5-8.49	2
Klebsiella spp.	7.5-9.03	2	2-21.77	8	12-19	2	17.92	1
Proteus spp.	0.97-3.75	2	1.5-4	2	1.2	1	2.83	*

Continued.

Pathogen	North India prevalen-ce range (%)	No. of studies in North India	South India prevale-nce range (%)	No. of studies in South India	West India prevalen -ce range (%)	No. of studies in West India	East India prevalenc e range (%)	No. of studies in East India
Pseudomonas spp.	2.26	1	0.85-5	6	10	1	1.6-5.66	2
Enterobacter spp.	*	*	0.47-4	2	*	*	*	*
Citrobacter spp.	*	*	0.47-17.69	4	*	*	*	*
Providencia spp.	0.32	1	0.85	1	*	*	*	*
Acinetobacter spp.	*	*	1.92	1	*	*	*	*
Gram-negative coccobacilli	*	*	22.8	1	*	*	*	*

*Data not available, †*Staphylococcus sp=Coagulase negative Staphylococci+Staphylococcus aureus+Staphylococcus epidermis+Staphylococcus albus*, ‡*Streptococcus sp=beta-hemolytic streptococci*+Group B streptococci+Group D streptococci+Streptococci species

Table 3: Distribution of diagnostic methods used among 107 included studies.

Diagnostic method category	Number of studies	Percentage (%)
Culture-based methods	66	61.68
Wet mount microscopy and gram staining	55	51.4
Clinical criteria (Amsel's, Nugent sinitial)	35	32.7
Molecular methods (PCR, NAAT)	12	11.2
Serology/antigen-detection	8	7.4
Method not specified	5	4.6

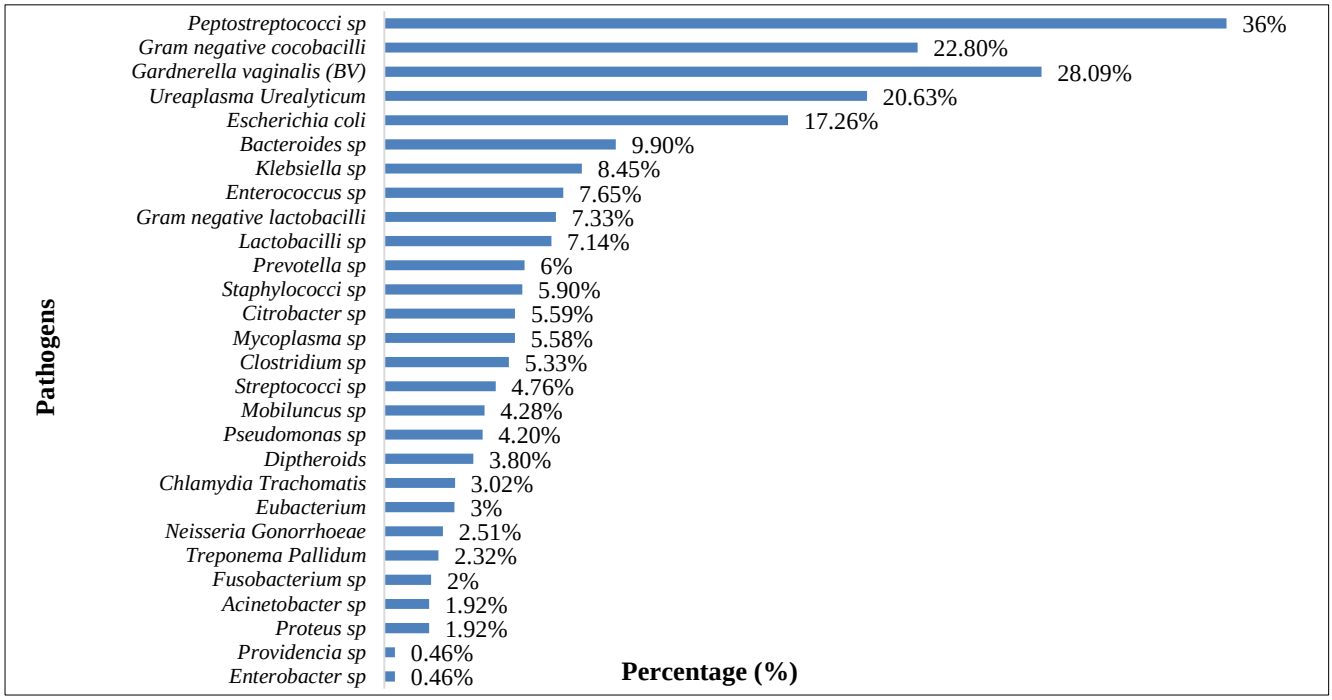

Figure 2: Prevalence of bacterial organisms associated with vaginal discharge across the Indian subcontinent (n=107).

Table 4: Comparison of bacteria prevalence in symptomatic women and bacterial colonization in asymptomatic women (n=12).

Bacteria organisms	Asymptomatic women (%)	Symptomatic women (%)
Anaerobes		
Peptostreptococcus species	66.67	33.33

Continued.

Bacteria organisms	Asymptomatic women (%)	Symptomatic women (%)
Bacteroides species	33.33	18.97
Gram -ve bacilli anaerobes	3.29	*
Gram +ve bacilli anaerobes	2.47	*
Gram positive		
Streptococci spp. (Beta strep + Group B Strep)	25.64	14.43
Staphylococcus aureus	1.10	*
Gram negative		
Gardnerella vaginalis	5.76	5.92
STDs causing pathogens		
Ureaplasma urealyticum	22	15.84
Mycoplasma spp.	5.08	8.30
Treponema pallidum	4.76	5.18
Chlamydia trachomatis	1.64	4.98
Neisseria gonorrhoea	0.16	*
Bacterial vaginosis (BV), aerobic vaginitis (AV), mixed (BV+yeast vaginitis (YV))		
BV	9.32	38.92
AV	33.33	10

*Data not available

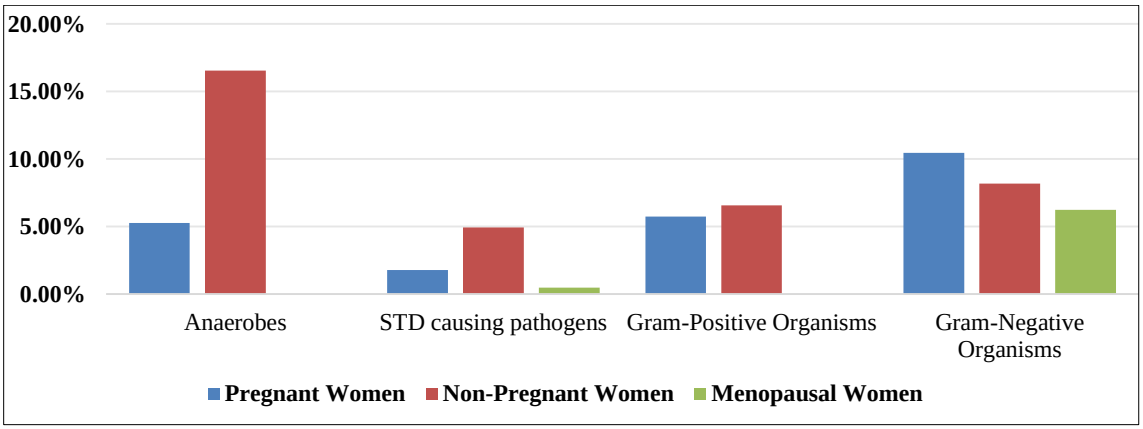

Figure 3: Prevalence of pathogens in specific population groups.

Clinical signs

Only 11 out of the 107 studies reported specific clinical signs. The studies from Nepal, Sri Lanka, and Bhutan did not report any clinical signs. Most observed clinical signs were vaginitis (28.3%), cervical erosion/friability (23.9%), adnexal/uterine tenderness (16.8%), and inguinal lymphadenopathy (15.5%), genital ulcers (8%), Condylomata lata, (6.4%), genital warts (5.7%), Nabothian follicles (3.45%) and endocervical polyps (0.6%) were also reported.

Amsel criteria for diagnosis of BV

Amsel criteria for diagnosis of BV includes vaginal pH >4.5 (20-40%), clue cells (30-54%) and whiff/amine test positivity in 49-85%.

Antibiotic susceptibility (sensitivity and resistance)

An analysis of susceptibility data from 43 studies revealed that antimicrobial resistance is widespread and significant throughout the Indian subcontinent. The heat maps in Figures 4 and 5 illustrate the profiles of sensitivity and resistance. As studies reported only one metric, sensitivity and resistance data were treated as separate outcomes.[41-44]

STD causing pathogens

While susceptibility to cephalosporins (94%) BL-BLI combinations (93%), macrolides (79%), and spectinomycin (99%) remained high (Figure 4), *Neisseria gonorrhoeae* were found to be resistant to penicillin (88%), tetracycline (74%), and fluoroquinolone (70%), making them unsuitable for empirical treatment (Figure 5).

Aerobic gram-positive organisms

Pathogens like *Staphylococcus aureus* and *Enterococcus spp.* demonstrated varied susceptibility. They were highly sensitive to BL-BLI combinations (69-85% sensitivity), aminoglycosides (73-76% sensitivity), and lincosamides such as clindamycin (69.5% sensitivity for *S. aureus*) (Figure 4). Several reserve antibiotics, such as

glycopeptides/oxazolidinones (vancomycin/linezolid) (60-62% sensitivity) and carbapenems (63-91% sensitivity), were also effective (Figure 4). However, resistance was critically high for common antibiotics against *S. aureus*, including penicillins (66.4% resistance), macrolides (71.4% resistance), and sulfonamides (80.7% resistance) (Figure 5).

Aerobic gram-negative organisms

E. coli and *Klebsiella spp.* showed excellent sensitivity to carbapenems (84% sensitivity) and good activity with aminoglycosides (69-71% sensitivity). For *E. coli*, urinary antiseptics (nitrofurantoin/fosfomycin) and newer broad-spectrum agents (tigecycline/colistin) were also highly effective, with sensitivity exceeding 90%. In contrast, there were high resistance rates reported for penicillins (85.2% for *E. coli*) and sulfonamides (81-86%). Efficacy of cephalosporins (59% sensitivity) and fluoroquinolones (53% sensitivity) was moderate and inconsistent, limiting their reliability for empirical use (Figure 5). As shown in Figure 4, *Gardnerella vaginalis* is highly susceptible to BL-BLI combinations (92.5% sensitivity) and cephalosporins (86.8% sensitivity). Some studies also reported good sensitivity to Metronidazole (71.1%); however, the high resistance rate of 51.2% observed in other studies raises significant clinical concerns (Figure 4). The sensitivity to macrolides (32.3%) and sulfonamides (40.1%) was poor, limiting their utility (Figure 5).

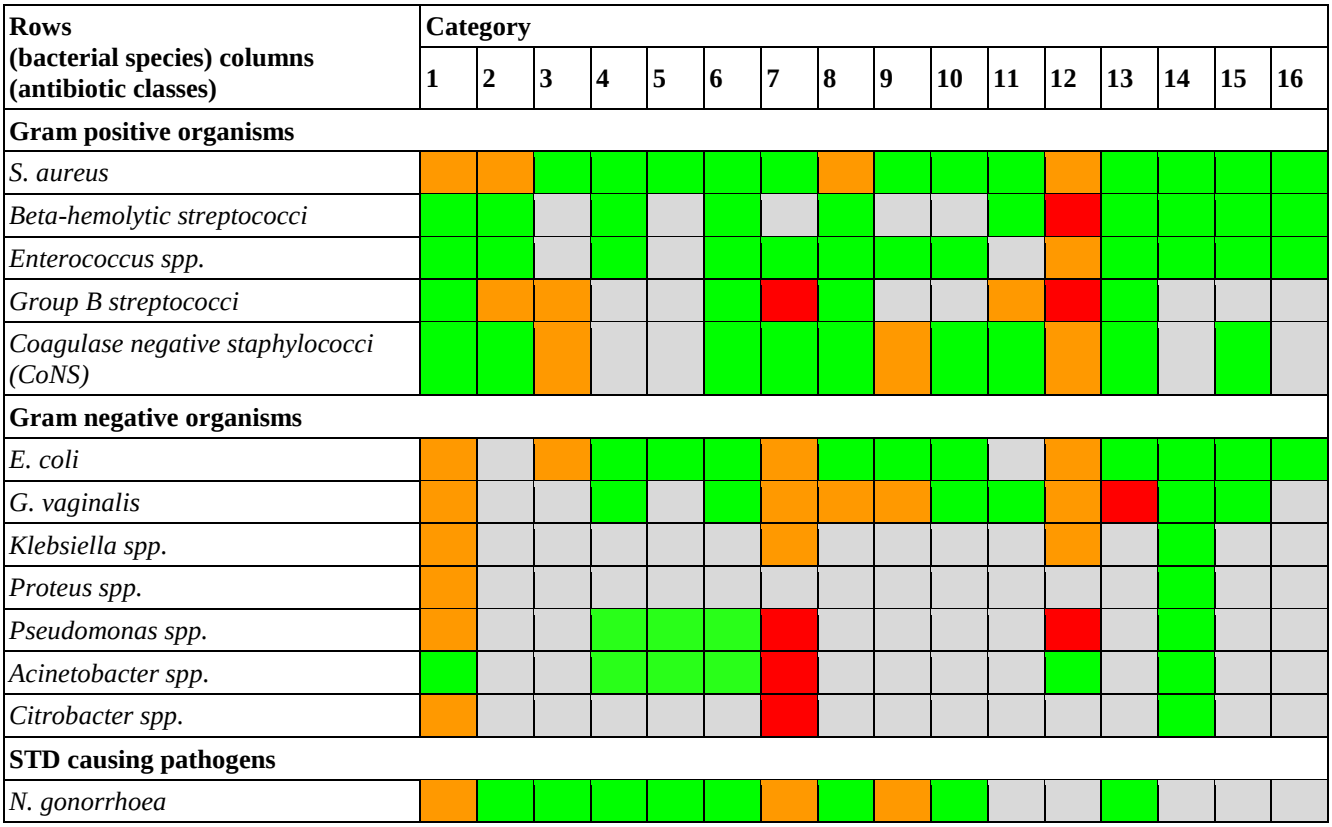

Figure 4: Heat map of antibiotic sensitivity in Indian subcontinent.

>50%: Green, 25 to <50%: orange, <25%: red, no data available: grey; no antibiotic sensitivity data reported in anaerobes among the included studies; category 1: Penicillins (Ampicillin, Amoxicillin, Cloxacillin, Oxacillin, Penicillin, Piperacillin); category 2: 1st generation Cephalosporins (Cephalexin, Cephradine); category 3: 2nd generation Cephalosporins (Cefaclor, Cefuroxime); category 4: 3rd generation Cephalosporins (Cefixime, Cefotaxime, Cefoperazone, Cefpodoxime, Ceftazidime, Ceftriaxone); category 5: 4th generation Cephalosporins (Cefepime); category 6: beta-lactam/beta-lactamase inhibitor combinations (BL-BLI) (Amoxicillin/clavulanic acid, Ampicillin/sulbactam, Piperacillin/Tazobactam, Cefoperazone/Sulbactam, Ceftriaxone/Tazobactam, Ticarcillin/clavulanate); category 7: Tetracyclines (Tetracycline, Doxycycline); category 8: Macrolides (Erythromycin, Azithromycin, Josamycin); category 9: Fluoroquinolones and Quinolones (Ciprofloxacin, Ofloxacin, Levofloxacin, Norfloxacin, Nalidixic Acid, Gatifloxacin, Sparfloxacin); category 10: Aminoglycosides (Gentamicin, Amikacin, Tobramycin, Netilmicin, Spectinomycin, Streptomycin, high-level gentamicin); category 11: Lincosamides and Nitroimidazoles (Clindamycin, Lincomycin, Metronidazole); category 12: Sulfonamides (Cotrimoxazole/Trimethoprim-Sulfamethoxazole); category 13: Glycopeptides and Oxazolidinones (Vancomycin, Teicoplanin, Linezolid); category 14: Carbapenems (Imipenem, Meropenem, Ertapenem); category 15: broad-spectrum antibiotics (Chloramphenicol, Tigecycline, Colistin, Polymyxin B, Fusidic acid, Aztreonam); and category 16: urinary antiseptics (Fosfomycin, Nitrofurantoin)

Rows (bacterial species) columns (antibiotic classes)	Category													
	1	2	3	4	5	6	7	8	9	10	11	12	13	14
Gram positive organisms														
S. aureus														
Beta-hemolytic streptococci														
Enterococcus spp.														
Group B streptococci														
Coagulase negative staphylococci (CoNS)														
Gram negative organisms														
E. coli														
G. vaginalis														
Klebsiella spp.														
Proteus spp.														
Pseudomonas spp.														
Acinetobacter spp.														
Citrobacter spp.														
STD causing pathogens														
N. gonorrhoea														

Figure 5: Heat map of antibiotic resistance in Indian subcontinent.

>50%: red, 25 to <50%: orange, <25%: green, no data available: grey; no antibiotic sensitivity data reported in anaerobes among the included studies

Minimum inhibitory concentrations

A critical scarcity of MIC data was identified, with only four of the 107 included studies (3.7%) providing this information.[45-48] Limited data from Pakistan, Bangladesh, Nepal, and Sri Lanka, interpreted using CLSI 2019–20 breakpoints (which were applied in this review to align with the timeline of included MIC data), revealed significant resistance among common pathogens (Table 5).

Notably, *Streptococcus pyogenes* and *Staphylococcus aureus* were resistant to multiple antibiotic classes, including Cephalosporins (Cephalexin, Cefepime), Macrolides (Azithromycin), Tetracyclines (Tetracycline, Doxycycline) and Aminoglycosides (Gentamicin, Amikacin) (Table 5). *Escherichia coli* was resistant to Cefepime and Tetracycline, but sensitive to Gentamicin, Amkicin and Azithromycin (Table 5). Group B Streptococcus (GBS) showed intermediate susceptibility to Clindamycin. Furthermore, Neisseria gonorrhoeae was resistant to Ciprofloxacin. For other antibiotics tested against *Neisseria gonorrhoeae*, a conclusive interpretation was limited by the lack of corresponding CLSI breakpoints (Table 5).

DISCUSSION

WHO estimates 357 million new STI/RTI cases annually, with bacterial vaginosis (BV) a leading cause.[9] This scoping review of 107 studies identified substantial variability in study populations and diagnostic methods, from microscopy and culture to molecular assays. Such heterogeneity complicates data analysis and reflects evolving diagnostic practices. Molecular methods offer greater sensitivity for targeted pathogens, while culture can detect non-target organisms. The prevalence of specific bacteria depends on both the diagnostic method and time of the study. Aerobic vaginitis (AV), though less recognized than BV or STIs, is an important and underdiagnosed cause of VD. In low- and middle-income countries, reliance on syndromic management despite rising antimicrobial resistance highlights the need for region-specific antibiotic susceptibility data.

Bacterial etiologies of VD

Bacterial vaginosis

BV affects about a quarter of reproductive-age women globally.[9] *Gardnerella vaginalis* remains the primary pathogen (95% of BV cases) due to biofilm formation.[49] Although *Prevotella sp.* are globally linked to BV recurrence, their prevalence was relatively low in this review, possibly due to regional microbiota differences or diagnostic variability.[50] *Mobiluncus* species had higher prevalence (36%) followed by Lactobacilli (11%) and Bacteroides (9%). Notably, *Fannyhessea vaginae*, associated with metronidazole resistance, was not found in this review, indicating a gap compared to global findings.[51]

Table 5: Minimum inhibitory concentrations (MIC) data reported from 4 included studies and CLSI 2019-20 breakpoints and interpretation.

Study ID (location)	Bacterial species and antibiotics	Antibiotic class	Reported MIC (µg/ml)	CLSI 2019-20 breakpoints (µg/ml) sensitive (S), intermediate (I), resistant (R)	Interpretation based on CLSI 2019-20
Ali et al, (2019) (Pakistan)	***Streptococcus pyogenes***				
	Azithromycin	Macrolides	23.5	S=0.5, I=1, R=2	Resistant
	Cephalexin	Cephalosporins	55.5	S=0.5, I=not given, R=not given	Resistant
	Cefepime	Cephalosporins	10	S=1, I=2, R=4	Resistant
	Tetracycline	Tetracyclines	46.5	S=2, I=4, R=8	Resistant
	Staphylococcus aureus				
	Azithromycin	Macrolides	15	S=2, I=4, R=8	Resistant
	Tetracycline	Tetracyclines	46	S=4, I=8, R=16	Resistant
	Doxycycline	Tetracyclines	29.3	S=4, I=8, R=16	Resistant
	Gentamicin	Aminoglycosides	38	S=4, I=8, R=16	Resistant
	Amikacin	Aminoglycosides	23.1	S=4, I=8, R=16	Resistant
	Escherichia coli				
	Cefepime	Cephalosporins	32	S=2, I=8, R=16	Resistant
	Gentamicin	Aminoglycosides	2	S=4, I=8, R=16	Sensitive
	Amikacin	Aminoglycosides	2	S=16, I=32, R=64	Sensitive
	Tetracycline	Tetracyclines	23	S=4, I=8, R=16	Resistant
	Azithromycin	Macrolides	5	S=16, I=not given, R=32	Sensitive
	Neisseria gonorrhoeae				
	Cefepime	Cephalosporins	2	S, I, R=not given	No MIC breakpoints available for comparison
	Tetracycline	Tetracyclines	15	S, I, R=not given	
	Doxycycline	Tetracyclines	30	S, I, R=not given	
	Azithromycin	Macrolides	12.5	S, I, R=not given	
	Gentamicin	Aminoglycosides	16	S, I, R=not given	
	Amikacin	Aminoglycosides	16	S, I, R=not given	
Mahbub et al, (2020) (Bangladesh)	Ciprofloxacin	Fluoroquinolones	1-4*	S=0.06, I=0.12-0.5, R=1	Resistant
Chaudhary et al, (2005) (Nepal)	Ceftriaxone	Cephalosporins	0.004-0.03 ¶	S, I, R=not given	No MIC break-points available for comparison
	Tetracycline	Tetracyclines	0.25-32 ‡	S, I, R=not given	
	Ciprofloxacin	Fluoroquinolones	0.06 - 8 #	S=0.06, I=0.12-0.5, R=1	Intermediate (Spans S, I & R)
	Azithromycin	Macrolides	0.06-16 §	S, I, R=not given	No MIC break-points available for comparison
	Penicillin	Penicillins	0.008-0.5 †	S, I, R=not given	
	Spectinomycin	Broad spectrum antibiotic	8-16 **	S, I, R=not given	
Dilrukshi et al, (2020) (Sri Lanka)	***Group B strep (GBS)***				
	Clindamcyin	Lincosamides	0.5	S=0.25, I=0.5, R≥1	Intermediate

*Range from 3 distinct MICs (1, 2, 4 µg/ml) reported for 13 isolates (Mahbub 2020); **range from 2 distinct MICs (8, 16 µg/ml) reported for 16 isolates (Chaudhary 2005); †range from 5 distinct MICs (0.008, 0.03, 0.12, 0.25, 0.5 µg/ml) reported for 16 isolates (Chaudhary 2005); ‡range from 8 distinct MICs (0.25, 0.5, 1, 2, 4, 8, 16, 32 µg/ml) reported for 16 isolates (Chaudhary 2005); §range from 5 distinct MICs (0.06, 2, 4, 8, 16 µg/ml) reported for 16 isolates (Chaudhary 2005); ¶range from 4 distinct MICs (0.004, 0.008, 0.015, 0.03 µg/ml) reported for 16 isolates (Chaudhary 2005); all reported MICs measured in same unit (i.e. µg/ml); #range from 7 distinct MICs (0.06, 0.12, 0.25, 0.5, 1, 2, 8 µg/ml) reported for 16 isolates (Chaudhary 2005)

Aerobic vaginitis

AV pathogens included GBS (0.7–58.7%), *Staphylococcus aureus* (6–37.4%), *Coagulase-negative Staphylococci* (0.2–41.7%), *E. coli* (4–23%), *Enterococcus* (0.3–2.4%), among others.[52] Most are gastrointestinal/genitourinary commensals but may cause infection. *E. coli* (16%), *Klebsiella* (8%), GBS and *S. aureus* (9% each), and *Enterococcus* (6%) were most common, less frequent were Citrobacter, Acinetobacter, *Pseudomonas* and *Enterobacter* (2-4%); *Haemophilus influenzae* was not detected.

STI pathogens

C. trachomatis (3.7%) and *N. gonorrhoeae* (3.5%) were predominant, similar to global figures.[53] *T. pallidum* was 1.6%—higher than previously reported 0.2%.[54] *Ureaplasma sp.* and *Mycoplasma hominis*, frequently colonize asymptomatically, with reported prevalence rates 50-80% among sexually active women. *Mycoplasma genitalium*, linked to cervicitis and PID, had a low VD-related prevalence (5%) in our review, versus 18% in other studies.[55] Ureaplasma prevalence was 19%, lower than the 50% previously reported.[54]

Antimicrobial susceptibility trends

AST data remain sparse, especially outside India. Gardnerella vaginalis showed high resistance to penicillins (50.6%) but retained sensitivity to Metronidazole (71.1%) and Amoxicillin-cluvulanate (92.5%), although one included study from India reported metronidazole resistance up to 68%, suggesting clindamycin may be more reliable.[56,57] *Prevotella spp.* remain largely metronidazole-sensitive but can resist clindamycin. *Neisseria gonorrhoea* exhibited low sensitivity to tetracyclines (39%) and ciprofloxacin (29.2%), yet maintained high susceptibility to cephalosporins (89.5%) and azithromycin (91.7%), contrasting with global WHO resistance data.[54] Gram-positives such as *S. aureus* and *Enterococcus sp.*, retain 75–100% susceptibility to β-lactamase inhibitors and glycopeptides, with 76% sensitivity of *S. aureus* to aminoglycosides; however, methicillin resistant *S. aureus* susceptibility varied (5–62.5%).[58] *S. aureus* showed moderate sensitivity to tetracyclines (60.2%) and reduced sensitivity to azithromycin (49.3%) alongside group B *Streptococcus* (71.8%). Decreasing azithromycin susceptibility calls for reviewing its role in syndromic management.

Special groups

HIV-positive women

The high BV prevalence range (23.75-70%) reported in six included studies is consistent with studies from Africa in HIV positive women, where BV prevalence ranged from 25.1-80.3%.[59]

Pregnancy

Among 26 studies on pregnant women, BV prevalence (up to 62.39%) was reported, exceeding the 4.9-49% range reported in a systematic review among pregnant women.[60]

Menopause

A single study among postmenopausal women in this review reported BV prevalence of 6.23%, which is lower than the pooled estimate of 16.9% (range 2-57%) reported in a systematic review of BV in postmenopausal women.

Over-treatment, antibiotic misuse and AMR

WHO emphasizes pathogen-specific diagnosis and AMR surveillance to enhance sexual and reproductive health.[62] SCM, widely used but poorly specific, drives antibiotic overuse and resistance. Kumari et al estimated 92.87% antibiotic overuse and 8.69% misuse with SCM, including substantial overuse of azithromycin-cefixime and doxycycline-metronidazole kits.[39] Similar overtreatment rates (78%) were reported in pregnant women, contributing to empirical treatment failures, economic burden, and AMR.[38]

Feasibility of laboratory-confirmed diagnosis

Laboratory diagnosis, particularly PCR-based methods, improves accuracy. Category II SCM incorporating PCR confirmed infections in 76.15% and reduced antibiotic overuse to 23.82%.[40] Even basic Gram staining enhances specificity.[52] GeneXpert-STI screening in antenatal care showed high uptake (86.4%) and rapid results (91%) in low-resource settings, capturing asymptomatic infections missed by SCM.[39]

Syndromic case management: limitations and future directions

SCM remains widely used in LMICs for cost-effectiveness but has low specificity missing asymptomatic and non-infectious cases.[63] It inadequately covers *Ureaplasma sp.* and omits clindamycin, recommended for aerobic pathogens by IUSTI/WHO.[63] Lack of diagnostics hampers adaptation to shifting microbial profiles. Scalable diagnostics—microscopy, culture, point-of-care molecular tests—are essential to improve targeting and combat AMR, with regular etiological surveillance every 2–3 years to update national protocols.[64]

Current SCM for VD regimens include metronidazole/secnidazole (BV/Trichomonas), fluconazole (Candidiasis), and cefixime plus azithromycin (*N. gonorrhoeae, Chlamydia*). Although metronidazole remains effective, emerging Azithromycin resistance is concerning, and SCM inadequately addresses AV, highlighting the need for etiological diagnosis when feasible.

BV recurrence and treatment outcomes

BV recurrence is high (over 50% within 6–12 months).[64] Risk factors include prior BV, regular partners, biofilm persistence, AMR and inadequate lactobacilli recolonization.[57] Though short-term cure rates reach 70–85%, long-term success is limited. Management includes therapy switching, suppressive metronidazole gel, adjunctive probiotics and biofilm disruptors.

Clinical implications of bacterial resistance

High resistance to common antibiotics challenges empirical SCM. *Streptococcus pyogenes* resists Azithromycin, Cephalexin, Cefepime, Tetracycline — questioning their empirical use in such infections. *Staphylococcus aureus* showed resistance to Azithromycin, Tetracycline, Doxycycline, Gentamicin, and Amikacin—limiting treatment options. *E. coli* resists Cefepime and Tetracycline, but remained sensitive to Gentamicin, Amikacin and Azithromycin, although CLSI 2025 removed Azithromycin breakpoint suggesting reduced utility.[23,65] *Neisseria gonorrhoeae* exhibits widespread Ciprofloxacin resistance, consistent globally.[65] GBS showed intermediate Clindamycin susceptibility, complicating treatment for penicillin-allergic patients.[48]

Clinicians should avoid empirical use of Azithromycin for *S. pyogenes*, *S. aureus*, and *E. coli*; Cephalexin/Cefepime for *S. pyogenes* and *E. coli*; Tetracycline/Doxycycline for *S. aureus* and *S. pyogenes*; and Clindamycin for GBS without test support. Empirical regimens must increasingly rely on local resistance profiles and accessible diagnostics.

Escherichia coli sensitivity and CLSI 2025 key updates

E. coli consistently remained sensitive to Gentamicin and Amikacin (Table 5), supporting their use in severe infections guided by local antibiograms. The azithromycin breakpoint removal in CLSI M100-Ed35 (2025), reflects evolving clinical evidence.[23]

Key CLSI 2025 updates include stable breakpoints for Tetracycline (*S. pyogenes*), Azithromycin/Tetracyclines/ Gentamicin (*S. aureus*), and Clindamycin (GBS); discontinued breakpoints for azithromycin (*E. coli*), amikacin (*S. aureus*), cefepime, and cephalexin (*S. pyogenes*); and new breakpoint additions for *N. gonorrhoeae*, aiding susceptibility interpretation. Regular consultation of updated CLSI standards is essential for treatment decisions.[23]

Study strengths

The study contributes to filling an important gap in the literature by evaluating bacterial prevalence and antibiotic susceptibility in vaginal discharge in low resource settings. PRISMA-ScR-compliant with a pre-registered OSF protocol. It synthesizes data from 107 studies involving 37,671 women across the Indian subcontinent, uses an EGM to visualize research density and gaps and highlights underrepresented causes like AV, often overlooked in STI- and BV-focused frameworks.

Limitations

The limitations of the study were; English-only inclusion may have excluded relevant non-English studies, heterogeneity in study design and populations may limit direct comparability and the CoCoPop framework centered the scoping review on bacterial pathogens, excluding fungal or parasitic etiologies.

CONCLUSION

In this review, bacterial causes of VD and antibiotic susceptibility in the Indian subcontinent are mapped, revealing a significant diversity of pathogens and alarming trends in antimicrobial resistance. Most vaginal discharge pathogens were anaerobic bacteria and Gram-negative organisms. The most prevalent bacteria were *Gardnerella vaginalis, E. coli and U. urealyticum*. The effectiveness of SCM is limited, resulting in over-treatment and failure to detect non-classical infections. The emergence of ciprofloxacin resistance in *Neisseria gonorrhoeae* and azithromycin resistance further raises questions about the effectiveness of current treatment kits. The lack of updated MIC data impedes the development of region-specific guidelines. As compared to laboratory-based diagnosis, SCM resulted in lower cure rates and a higher use of antibiotics. To inform regional health policy, further research is needed from countries in the Indian subcontinent.

Recommendations

Avoid empirical use of ciprofloxacin for *N. gonorrhoeae* and azithromycin for *E. coli* given high resistance. Prefer aminoglycosides (gentamicin, amikacin) for *E. coli* infections as per local antibiograms. A cautious use of aminoglycosides is advised for pregnant women in alignment with guidelines for treating infections in pregnancy. Supplement SCM with affordable diagnostics especially in high-risk or recurrent cases. Update national treatment guidelines regularly based on regional AMR data. Shift to affordable point-of-care diagnostics with evidence-based, locally tailored regimens to optimize patient care, reduce antibiotic misuse and combat AMR.

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