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Macrolide Resistance Among Group B Streptococcus Isolated from Clinical Samples In and Around Kanchipuram

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Abstract

Streptococcus agalactiae (Group B Streptococcus, GBS) remains a significant cause of invasive infections, particularly in pregnant women, neonates, and immunocompromised individuals. Rising resistance to macrolides and lincosamides, especially among penicillin-allergic patients, is a growing concern. This study aimed to determine the prevalence and patterns of macrolide resistance in clinical GBS isolates, evaluate inducible clindamycin resistance, and assess associated demographic and clinical variables in a tertiary care setting in South India. A cross-sectional study was conducted at Meenakshi Medical College Hospital and Research Institute, Kanchipuram, from January 2023 to June 2024. A total of 600 clinical samples—including high vaginal swabs, urine, pus, blood, and cerebrospinal fluid—were processed. GBS isolates were identified using standard microbiological and automated methods. Antimicrobial susceptibility testing was performed according to CLSI 2023 guidelines, and inducible clindamycin resistance was detected using the D-zone test. GBS was isolated from 130 samples (21.7%), predominantly from females (78.5%) and high vaginal swabs (32.4%). All isolates were susceptible to penicillin, vancomycin, and linezolid. Resistance to erythromycin and clindamycin was observed in 43.1% and 37.7% of isolates, respectively. Among erythromycin-resistant strains, the M phenotype was most prevalent (39.3%), followed by inducible MLSB (32.1%) and constitutive MLSB (25.0%). No significant associations were found with age, sex, or specimen type. These findings underscore the need for routine susceptibility testing and support penicillin as the first-line therapy.

Keywords: *Antimicrobial Susceptibility, Group B Streptococcus, Inducible Clindamycin Resistance, Macrolide Resistance, South India, Streptococcus Agalactiae.*

Introduction

Group B *Streptococcus* (*Streptococcus agalactiae*) is a β-hemolytic, Gram-positive bacterium that has emerged as a significant cause of morbidity and mortality, particularly in neonates, pregnant women, and immunocompromised individuals. While often part of the commensal flora of the gastrointestinal and genitourinary tracts, GBS can transition into a pathogen under certain conditions, causing invasive diseases such as sepsis, meningitis, endometritis, and urinary tract infections [1, 2]. Notably, the vertical transmission of GBS from colonized mothers to

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neonates during delivery remains a major public health concern globally.

Penicillin remains the antibiotic of choice for prophylaxis and treatment of GBS infections. However, in penicillin-allergic individuals—especially those at high risk of anaphylaxis—macrolides such as erythromycin and lincosamides like clindamycin are commonly used as alternative agents [3, 4]. This shift has led to the increasing reliance on macrolides and subsequent reports of rising macrolide resistance in clinical GBS isolates. The trend poses serious implications for the effective management of GBS infections, particularly in resource-limited settings where sensitivity-guided therapy may not be routinely available [5].

Global surveillance has shown significant geographical variation in macrolide and clindamycin resistance among GBS strains. Studies from North America and Europe have documented macrolide resistance rates ranging from 10% to over 50% depending on the population and healthcare setting [6]. In Asian countries, including India, resistance rates are also on the rise, with several recent investigations highlighting clindamycin and erythromycin resistance levels between 30% and 60% among GBS isolates from pregnant women and neonates [7–9]. Such trends threaten the efficacy of second-line therapeutic options and complicate the prophylactic strategies currently recommended by the CDC and other health bodies [10].

In addition to intrinsic resistance mechanisms, the phenomenon of **inducible clindamycin resistance**—detectable only via the D-test—further complicates the antimicrobial landscape. Isolates may appear susceptible to clindamycin during routine testing but demonstrate resistance when exposed to erythromycin, leading to potential treatment failure if not properly detected [11]. Therefore, D-zone testing has become an essential component of clinical microbiology workflows when evaluating GBS susceptibility patterns.

Compounding this issue is the emergence of **multi-drug resistance (MDR)** among GBS strains. While resistance to penicillin remains rare, concurrent resistance to macrolides, tetracyclines, and fluoroquinolones has been increasingly reported, especially in isolates from hospital environments and high-risk populations [12]. These patterns underscore the necessity for localized antimicrobial stewardship strategies and periodic antibiogram updates to inform empiric therapy, particularly in regions with limited access to molecular diagnostic tools [13-15].

Despite these concerns, few studies have focused on the **epidemiological and clinical correlates** of macrolide resistance in GBS from Indian settings. Most available data are either limited to colonization studies in antenatal women or derive from isolated case series, thereby failing to capture the broader spectrum of GBS infections and resistance trends across age groups and clinical syndromes. A comprehensive understanding of demographic distribution, resistance prevalence, and co-resistance patterns is urgently needed to inform both preventive and therapeutic strategies.

Against this backdrop, the present study was undertaken with the following objectives: (i) to determine the prevalence of macrolide resistance among GBS isolates from various clinical samples; (ii) to evaluate the susceptibility pattern of GBS to commonly used antibiotics including erythromycin and clindamycin; (iii) to assess the incidence of inducible clindamycin resistance through D-test; and (iv) to analyze demographic and clinical characteristics associated with macrolide-resistant GBS infections. In addition, we sought to examine co-resistance trends, multi-drug resistance profiles, and temporal patterns of resistance, thereby generating data critical for revising local antimicrobial guidelines [16].

Understanding the prevalence and clinical implications of macrolide resistance in GBS is essential for guiding empiric therapy, especially in vulnerable populations such as pregnant women and neonates where treatment choices are limited. This study aims to fill critical gaps in regional surveillance data, promote awareness of emerging resistance trends, and contribute to the development of effective antimicrobial policies tailored to the needs of the local healthcare ecosystem.

Materials and Methods

Study Design and Setting

A cross-sectional observational study was conducted over a period of 18 months (January 2023 to June 2024) at the Central Research Laboratory, Meenakshi medical college hospital and research institute, in the Department of Microbiology. The study was approved by the Institutional Ethics Committee (Ref. No: CRL/IEC/2023/09), and all procedures were carried out following ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments.

Sample Collection and Inclusion Criteria

Clinical samples were collected from both inpatients and outpatients presenting to the hospital with suspected bacterial infections. The study included a variety of sample types such as high vaginal swabs (HVS), urine, pus, blood, and cerebrospinal fluid (CSF). All patients with confirmed *Streptococcus agalactiae* infections were included, irrespective of age and sex. Repeat isolates from the same patient and those with incomplete clinical data were excluded.

Samples from pregnant women were specifically collected between 35–37 weeks of gestation as part of routine antenatal screening, following CDC guidelines for GBS colonization [1].

Isolation and Identification of GBS

Samples were processed using standard microbiological techniques. High vaginal and rectal swabs were inoculated onto selective media—5% sheep blood agar and Columbia CNA agar—and incubated at 35–37°C for 24–48 hours under 5% CO_2 (2). Colonies showing β-hemolysis were subjected to Gram staining, catalase test, and confirmed using the CAMP test and latex agglutination for Group B antigen (3). Identification was further validated using automated systems such as VITEK 2 Compact (bioMérieux, France), where applicable [13].

Antimicrobial Susceptibility Testing (AST)

Antibiotic susceptibility testing of GBS isolates was performed by the **Kirby-Bauer disk diffusion method** on Mueller–Hinton agar supplemented with 5% defibrinated sheep blood, following **CLSI guidelines** (2023) (4). The antibiotics tested included: Penicillin (10 units); Erythromycin (15 µg); Clindamycin (2 µg); Tetracycline (30 µg); Levofloxacin (5 µg); Linezolid (30 µg); Vancomycin (30 µg) [3-5, 14].

The minimum inhibitory concentrations (MICs) of penicillin, erythromycin, and clindamycin were determined using the **MIC gradient strip method** (Lingam Microbiological Laboratory) for selected resistant isolates [13].

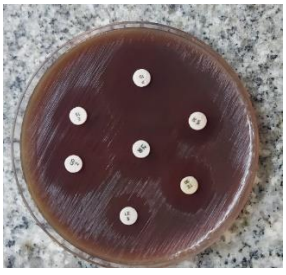

Figure 1. Antibiotic susceptibility testing by disk diffusion method [14]

Detection of Inducible Clindamycin Resistance (D-test)

Inducible clindamycin resistance was identified in erythromycin-resistant, clindamycin-sensitive isolates using the D-zone test. Erythromycin and clindamycin discs were placed 15–20 mm apart on a blood agar plate inoculated with the test organism. Flattening of the clindamycin inhibition zone adjacent to the erythromycin disc (D-shaped zone) was interpreted as inducible resistance (iMLSB phenotype) (5).

Phenotypic Classification of Resistance Patterns

Based on the resistance profiles, isolates were categorized into the following phenotypes:

1. **M phenotype**: Resistance to erythromycin only, clindamycin sensitive, D-test negative.
2. **iMLS^{B}phenotype**: Resistance to erythromycin, susceptible to clindamycin, D-test positive.
3. **cMLS^{B}phenotype**: Resistance to both erythromycin and clindamycin, D-test positive or negative.
4. **L phenotype**: Isolated clindamycin resistance, erythromycin sensitive (rare).

Data Collection and Statistical Analysis

Clinical data, including patient demographics (age, sex), clinical diagnosis, inpatient/outpatient status, and comorbidities, were recorded. Data were compiled in Microsoft Excel and analyzed using **SPSS version 26.0** (IBM Corp., Armonk, NY).

1. Prevalence of macrolide resistance was calculated as a percentage of total GBS isolates.
2. Chi-square or Fisher's exact test was used to determine the association between resistance and clinical variables.
3. Trends in resistance over the study period were assessed using linear regression analysis.
4. A p-value <0.05 was considered statistically significant.

Quality Control

The reference strain *Streptococcus agalactiae* ATCC 12386 was used as a control for AST procedures. All media, reagents, and antibiotic discs were quality-checked according to the manufacturer's instructions.

Results

A total of 600 clinical specimens were processed for the isolation of Group B Streptococcus (GBS). The distribution of GBS isolates across various specimen types is presented in Table 1. Among the specimen types, high vaginal swabs (HVS) yielded the highest number of GBS isolates (n = 68), representing an isolation rate of 32.4%. Urine samples followed with a rate of 19.4% (n = 32), while pus, blood, and cerebrospinal fluid (CSF) samples showed lower rates of 15.5% (n = 17), 12.6% (n = 11), and 7.1% (n = 2), respectively. The overall isolation rate of GBS across all specimens was 21.7%, indicating a notable burden of colonization and infection, particularly in genitourinary and obstetric settings.

Table 1. Distribution of Clinical Specimens from Which Group B Streptococcus (GBS) Was Isolated.

Specimen Type	No. of Samples Collected (n)	No. of GBS Isolates (n)	Isolation Rate (%)
High Vaginal Swabs	210	68	32.4
Urine	165	32	19.4
Pus	110	17	15.5

Blood	87	11	12.6
CSF	28	2	7.1
Total	**600**	**130**	**21.7**

The highest isolation rate of GBS was observed in high vaginal swabs, followed by urine samples. This reflects the prevalence in pregnant women and urinary tract infections.

The demographic characteristics of patients from whom GBS was isolated are summarized in Table-2. Of the 130 patients, 78.5% were female (n = 102), and the majority of isolates were obtained from the 21–30 year (32.3%) and 31–40-year (27.7%) age groups, aligning with the reproductive age range typically targeted for antenatal screening. In terms of patient type, 63.1% were inpatients and 36.9% were outpatients, indicating a greater frequency of GBS detection in hospitalized individuals, potentially due to increased sampling and clinical indications.

Table 2. Demographic Distribution of Patients with GBS Isolates.

Variable	**Category**	**No. of Patients (n = 130)**	**Percentage (%)**
Sex	Female	102	78.5
	Male	28	21.5
Age Group (years)	<20	9	6.9
	21–30	42	32.3
	31–40	36	27.7
	41–50	21	16.2
	>50	22	16.9
Patient Type	Inpatient	82	63.1
	Outpatient	48	36.9

Most GBS isolates were from females aged 21–40 years, correlating with antenatal screening data.

The antimicrobial susceptibility profile of the 130 GBS isolates is depicted in Table 3. All isolates (100%) were uniformly sensitive to penicillin, linezolid, and vancomycin, underscoring the continued efficacy of these agents for GBS infections. However, significant resistance was observed against tetracycline (70.8%), erythromycin (43.1%), and clindamycin (37.7%), suggesting substantial resistance to macrolides and lincosamides. Levofloxacin remained broadly effective, with a sensitivity rate of 96.2%, though 2 isolates showed intermediate susceptibility and 3 were resistant.

Table 3. Antimicrobial Susceptibility Profile of GBS Isolates (n = 130).

Antibiotic	**Sensitive n (%)**	**Intermediate n (%)**	**Resistant n (%)**
Penicillin	130 (100)	0 (0)	0 (0)
Erythromycin	74 (56.9)	–	56 (43.1)
Clindamycin	81 (62.3)	–	49 (37.7)

Tetracycline	38 (29.2)	–	92 (70.8)
Levofloxacin	125 (96.2)	2 (1.5)	3 (2.3)
Linezolid	130 (100)	0 (0)	0 (0)
Vancomycin	130 (100)	0 (0)	0 (0)

High resistance was observed against tetracycline and macrolides (erythromycin and clindamycin), while all isolates remained susceptible to penicillin, vancomycin, and linezolid.

Further characterization of erythromycin-resistant isolates (n = 56) revealed distinct resistance phenotypes as detailed in Table 4. The M phenotype, indicative of an active efflux mechanism, was predominant (39.3%), followed by the iMLSB phenotype (32.1%), which shows inducible clindamycin resistance confirmed via D-test. The constitutive MLSB (cMLSB) phenotype, denoting resistance to both erythromycin and clindamycin, accounted for 25.0%, while the rare L phenotype (3.6%) exhibited isolated clindamycin resistance. The distribution of these resistance phenotypes provides insight into underlying genetic mechanisms and guides empirical treatment decisions.

Table 4. Distribution of Resistance Phenotypes Among Erythromycin-Resistant GBS Isolates (n = 56).

Phenotypic Pattern	**Description**	**No. of Isolates**	**Percentage (%)**
iMLSB	Inducible clindamycin resistance (D-test positive)	18	32.1
cMLSB	Constitutive resistance to erythromycin and clindamycin	14	25.0
M phenotype	Erythromycin resistant, clindamycin susceptible, D–test negative	22	39.3
L phenotype	Clindamycin resistant, erythromycin susceptible	2	3.6

The M phenotype was the most prevalent among macrolide-resistant isolates, indicating the presence of active efflux mechanisms.

The association between macrolide resistance and selected clinical and demographic variables is explored in Table 5. No statistically significant differences were observed when comparing resistance rates across sex, patient type (inpatient vs. outpatient), or sample source categories (HVS, urine, others), as determined by Chi-square analysis ($p > 0.05$ in all comparisons). This suggests that macrolide resistance is widespread and not confined to a specific demographic or clinical group within this study population.

Table 5. Association Between Resistance to Macrolides and Clinical Parameters.

Variable	**Category**	**Macrolide-Resistant (n = 56)**	**Macrolide-Sensitive (n = 74)**	**p-value**
Sex	Female	42	60	0.735
	Male	14	14	

Patient Type	Inpatient	37	45	0.812
	Outpatient	19	29	
Sample Type	HVS	31	37	0.401
	Urine	12	20	
	Others	13	17	

Statistical analysis using the Chi-square test revealed no significant association between macrolide resistance and the demographic or clinical parameters analyzed ($p > 0.05$).

Table 6 provides the Minimum Inhibitory Concentration (MIC) values for penicillin, erythromycin, and clindamycin. All isolates had low MICs for penicillin, with MIC50 and MIC90 values of 0.03 µg/mL and 0.06 µg/mL, respectively, consistent with full susceptibility. In contrast, erythromycin showed a broad MIC range (0.5–32 µg/mL), and an elevated MIC90 of 16 µg/mL, reflecting a substantial proportion of high-level resistance. Clindamycin displayed a MIC range of 0.06–8.0 µg/mL, with a MIC50 of 0.25 µg/mL and MIC90 of 4.0 µg/mL, consistent with moderate resistance levels.

Table 6. Summary of Minimum Inhibitory Concentration (MIC) Range and MIC50/MIC90 Values for Selected Antibiotics.

Antibiotic	**MIC Range (µg/mL)**	**MIC50 (µg/mL)**	**MIC90 (µg/mL)**
Penicillin	0.03 – 0.06	0.03	0.06
Erythromycin	0.5 – 32	2.0	16.0
Clindamycin	0.06 – 8.0	0.25	4.0

MIC50 and MIC90 represent the minimum inhibitory concentration required to inhibit 50% and 90% of isolates, respectively. Resistance to erythromycin was reflected by elevated MIC90 values.

Taken together, the findings highlight a concerning prevalence of resistance to macrolide and tetracycline antibiotics among GBS isolates, particularly in women of reproductive age, while maintaining full susceptibility to penicillin, vancomycin, and linezolid. The detection of multiple resistance phenotypes among erythromycin-resistant isolates emphasizes the need for routine phenotypic characterization and susceptibility testing to inform optimal treatment regimens.

This study provides critical insight into the antimicrobial susceptibility patterns and macrolide resistance phenotypes of Group B Streptococcus (GBS) isolates recovered from clinical specimens in and around a tertiary care setting in South India. The results align with global observations in some respects while diverging significantly in others, particularly regarding resistance to alternative antibiotics like erythromycin and clindamycin. These discrepancies highlight the importance of local epidemiological surveillance to guide empirical therapy and prophylactic protocols.

Discussion

GBS isolates in this investigation retained complete susceptibility to beta-lactam antibiotics such as penicillin, as well as to vancomycin and linezolid. This mirrors long-standing global findings; penicillin has remained universally effective against GBS for decades. For example, a large-scale, longitudinal study conducted by the CDC (Centers for Disease Control and Prevention) reported no penicillin resistance among GBS

isolates over a 20-year period [1-5]. Likewise, more recent surveillance from a tertiary care hospital in South India confirmed that all invasive GBS isolates were penicillin susceptible [1]. These consistent findings reinforce current guidelines, which continue to recommend penicillin or ampicillin as the first-line treatment for GBS infections [6].

However, emerging reports of penicillin non-susceptibility are not entirely absent. For instance, one Indian study by McGuire et al. (2025) cited up to 27% penicillin resistance based on altered penicillin-binding protein profiles. Although our study did not encounter any such resistant strains, the existence of these anomalies should serve as a cautionary note. They emphasize the need for vigilant monitoring and confirmatory testing using MIC methods, especially in high-risk or treatment-failure cases [10].

Our findings indicate an alarming rate of resistance to erythromycin (43.1%) and clindamycin (37.7%) [17, 18]. These figures are significantly elevated compared to earlier Indian studies, which reported erythromycin resistance in the range of 20–30% [19, 20]. In global contexts, studies conducted in Saudi Arabia [21] and China [22] have reported erythromycin resistance rates ranging from 25% to over 75%, reflecting wide geographical variation in antibiotic usage patterns and selective pressure.

The implications of this resistance are particularly severe for penicillin-allergic individuals, for whom erythromycin and clindamycin have traditionally served as alternative prophylactic and therapeutic options. The current findings suggest that reliance on these agents without routine susceptibility testing may lead to treatment failure, especially in intrapartum prophylaxis scenarios.

Phenotypic characterization revealed that among erythromycin-resistant isolates, the most prevalent resistance phenotype was M-type (39.3%), followed by inducible MLSB (iMLSB, 32.1%) and constitutive MLSB (cMLSB, 25%). These findings align with those of Aldawsari et al. (2023), who also reported a predominance of the M phenotype among erythromycin-resistant GBS isolates. The M phenotype, typically associated with the *mefA/E* genes, is mediated by efflux pumps and generally does not confer resistance to clindamycin. However, iMLSB phenotypes, caused by *erm* genes, can result in therapeutic failure due to the ability to induce resistance during treatment.

The identification of iMLSB phenotypes underscores the necessity of performing D-zone testing alongside routine susceptibility profiling. Studies from Korea and Japan have emphasized that failure to detect inducible clindamycin resistance can lead to serious clinical consequences, particularly in neonatal sepsis and postpartum infections [20, 21]. Therefore, D-zone testing should be integrated into routine laboratory practice, especially in regions where macrolide resistance is rising.

The exceptionally high resistance to tetracycline (70.8%) observed in our study aligns with both national and international findings. Tetracycline resistance in GBS is often stable and genetically mediated through the *tetM*, *tetO*, or *tetL* genes, which encode ribosomal protection proteins or efflux pumps [10]. A pan-European study found tetracycline resistance rates exceeding 80%, reinforcing the notion that this antibiotic class has limited clinical relevance in GBS management today [15]. While not commonly used for GBS treatment, tetracycline resistance serves as a proxy for horizontal gene transfer and overall resistance gene burden within the GBS genome. It may also signal increased selective pressure in community-acquired strains. Thus, tetracycline resistance data, although perhaps not directly impacting therapeutic choice, remains crucial from an epidemiological and surveillance perspective.

The elevated MIC90 values for erythromycin (16 µg/mL) and clindamycin (4

µg/mL) observed in this study support phenotypic findings. These high MIC values are indicative of clinically significant resistance and correlate with reduced efficacy, especially in systemic or deep-seated infections. A Chinese multicenter study similarly reported MIC90 values >8 µg/mL for erythromycin, which were associated with higher rates of treatment failure in neonatal meningitis.

The MIC data substantiate the clinical imperative for routine susceptibility testing rather than reliance on empirical macrolide therapy, particularly in penicillin-allergic cases.

Statistical analysis showed no significant association between resistance phenotypes and patient demographics such as sex, age, or sample type. This finding supports the hypothesis that macrolide resistance is widespread and likely due to community-level antibiotic exposure rather than localized nosocomial transmission. A study from Brazil [21] reached similar conclusions, suggesting that clonal expansion of resistant GBS strains in the community may be a primary driver of observed patterns.

These findings have several important clinical and public health implications. First, they emphasize the critical need to maintain penicillin as the cornerstone of GBS prophylaxis and therapy, supported by susceptibility confirmation in high-risk scenarios. Second, they advocate for the incorporation of D-zone testing and routine erythromycin/clindamycin susceptibility profiling, especially for obstetric patients. Third, they underscore the importance of local epidemiological surveillance to monitor evolving resistance trends.

In the future, whole-genome sequencing (WGS) and molecular resistance gene profiling should be considered to unravel the clonal relationships and resistance mechanisms in GBS isolates. Moreover, with vaccine candidates against GBS advancing to late-stage trials, such as the hexavalent GBS polysaccharide conjugate vaccine [21], there is hope for long-term preventive strategies, particularly for pregnant women and neonates.

Conclusion

Our study highlights the importance of routine antibiotic susceptibility testing for beta hemolytic streptococci as well as detection of inducible resistance to prevent therapeutic failure. Inducible clindamycin resistance should be included in the routine antimicrobial susceptibility testing. Extensive use and over usage of antibiotics has been the reason for emergence of antibiotic resistance which is a global concern and this has now emphasized the importance of antibiotic resistance monitoring.

Conflicts of Interest

There isn’t any conflicts of interest in this study.

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