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ORIGINAL ARTICLE

Serotypes and antibiotic resistance patterns of group B streptococci isolated from pregnant women at Urmia University Hospital, Iran

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ABSTRACT

Background and Objectives: Group B *Streptococcus* (GBS) is a common bacterium found in the gastrointestinal tract and genitalia of both humans and animals. GBS infections can lead to a range of conditions, including meningitis, pneumonia, and sepsis. The present study aimed to analyze the colonization rate, antibiotic susceptibility, and serotypes of GBS in pregnant women in Urmia, Iran.

Materials and Methods: Following GBS isolation from pregnant women and confirming its presence through PCR, antibiotic susceptibility testing was conducted to assess resistance patterns, followed by amplification of resistance genes (*mef*A, *erm*B, *erm*TR, *lin*B) and molecular serotyping to determine the genetic characteristics of the strains.

Results: Out of 400 samples, 31 (7.75%) were positive for GBS, with 22 (70.97%) showing multidrug resistance. Clindamycin had the highest resistance rate (80.65%), while penicillin showed the lowest (3.23%). Serotypes II and V were the most common (38.71% each), followed by Ia (19.35%) and III (3.23%). The *erm*B gene was detected in 4 strains, while *mef*A, *erm*TR, and *lin*B were not found.

Conclusion: Optimal management of GBS infections in pregnant women necessitates ongoing surveillance and antibiotic stewardship, considering penicillin resistance and observed resistance patterns.

Keywords: *Streptococcus agalactiae*; Penicillins; Pregnant women; Serotyping

INTRODUCTION

The human gastrointestinal and genitourinary tracts are often colonized by Group B *Streptococcus* (GBS), which acts as a commensal bacterium (1). This opportunistic pathogen can lead to several infections like bacteremia, urinary tract infection (UTI), and pneumonia. GBS is a significant concern during pregnancy (2), affecting approximately 10-30% of pregnant women worldwide (3). Maternal colonization with this bacterium poses risks to both mothers and the newborns, emphasizing the importance of early detection and appropriate intervention. Transmission of GBS from the mother to the infant commonly occurs during childbirth (4).

Screening pregnant women for GBS is crucial to identify carriers and take preventive measures. The most common approach involves obtaining vaginal and rectal swabs (5). Undetected or untreated GBS infection during pregnancy can cause various complications, including chorioamnionitis, endometritis, and postpartum sepsis (6). In newborns, GBS infection can result in early-onset sepsis, pneumonia, meningitis, and other life-threatening conditions (7).

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Commonly used antibiotics, including penicillin and ampicillin, play a key role in both managing maternal GBS infections and preventing neonatal exposure during delivery. In addition to antibiotics, implementing strategies like intrapartum antibiotic prophylaxis and maternal education can greatly decrease the risk of this bacterium transmission to the newborn (8). The use of erythromycin or clindamycin as second-line treatments is usually designated for women with penicillin allergy or those who cannot take the first-line treatment options. Although GBS is typically susceptible to penicillin, some isolates have been reported to exhibit resistance due to moderate susceptibility or a reduced minimum inhibitory concentration (MIC) (9). Macrolide resistance has also been reported in GBS in different regions around the world. The emergence of erythromycin and clindamycin resistance is linked to the Macrolide-Lincosamide-Streptogramin B resistance MLSB phenotype, driven by the acquisition and expression of *erm* genes (10).

Ten serotypes of GBS are identified by differences in capsular polysaccharide antigens encoded within the *cps* gene cluster. Among these, serotypes V, III, II, and Ia are most frequently associated with infections in pregnant women and newborns (11, 12).

GBS colonization and resistance have been studied in various regions worldwide, however, in Iran, research has been primarily focused on the central and southern areas, leaving a notable gap in data from the northwestern regions, including Urmia.

To address this deficiency, the present study was designated to investigate the carriage rate of GBS in pregnant women admitted to Urmia University Hospital, located in northwestern Iran. Additionally, the research aimed to estimate the prevalence of macrolide-resistant strains and characterize antimicrobial resistance patterns. It also sought to identify the presence of resistance genes and characterize the distribution of molecular serotypes. This study's outcomes are anticipated to contribute to the expansion of region-specific antibiotic stewardship policies and to enhance evidence-based strategies for intrapartum prophylaxis, particularly for patients with presumed β-lactam allergies.

MATERIALS AND METHODS

Study design and bacterial isolation. During this cross-sectional study, a total of 400 rectal and high vaginal swabs were taken from women attending the gynecological clinic in Kosar Hospital, Urmia. Sample collection was carried out at 35 to 37 weeks of gestation, after obtaining their informed consent. This study received ethical approval from the Urmia University of Medical Sciences Ethics Committee (IR.UMSU.REC.1395.27). Women who had symptoms of UTI, had used antibiotics, or had experienced bleeding in the two weeks prior to sampling were excluded from this study.

The vaginal-rectal swabs taken from each woman were transferred to Todd-Hewitt-broth (THB) (Hi Media, India) supplemented with gentamicin (8 µg/mL) along with nalidixic acid (15 µg/mL) (MAST, UK). All samples were obtained aseptically and immediately sent to the lab. In the lab, the swabs were inoculated onto sheep blood (5%) agar medium (SBA) (Merck, Germany), subsequently, incubation was done at 35°C for 24 h in a 5% CO_2 atmosphere.

GBS identification and confirmation. Colonies with expected hemolytic and morphological patterns (the whitish-grey with β-hemolytic colonies) on SBA plates were picked and assessed by using other methods such as Gram staining, bacitracin and trimethoprim plus sulfamethoxazole (SXT) susceptibility, Christie-Atkins-Munch-Petersen (CAMP) reaction, hippurate hydrolysis, and catalase. Confirmation was performed by polymerase chain reaction (PCR) targeting the *dltS* gene using primers described by (13). Genomic DNA from *S. agalactiae* ATCC 12386 was used as the positive control, whereas nuclease-free water, devoid of DNA, served as the negative control.

Analysis of antimicrobial susceptibility. The confirmed GBS isolates were investigated for susceptibility to antibiotics (MAST, UK), including tetracycline (T, 30 µg), erythromycin (E, 15 µg), quinupristin-dalfopristin (Synercid, 15 µg), penicillin G (PG, 10 units), ofloxacin (OFX, 5 µg), chloramphenicol (C, 30 µg), clindamycin (CD, 2 µg), azithromycin (AZM, 15 µg), and ciprofloxacin (CIP, 5 µg) through Kirby-Bauer method using the CLSI criteria. The control strain used was *Streptococcus pneumoniae* (ATCC 49619). Eventually, the diameter of the inhibition zone around antibiotic disks was measured.

Disk induction test. According to the CLSI guidelines, a bacterial culture suspension made to the level of 0.5 McFarland's standard was used to create a lawn

culture on a MHA plate supplemented with 5% sheep blood. After placing for CD (2 μg) and E (15 μg) discs on this plate 12 millimeters apart from one another edge-to-edge, the plates were incubated for 24 hours at 37°C. Using the disk induction test (D-test), four phenotypes were identified (14). Diminishing of the CD inhibition zone proximal to the E disk was recognized as an inducible Macrolide-lincosamide streptogramin B ($iMLS_B$) phenotype. Resistance to both showed constitutive MLS_B methylation (cMLSB) phenotype.

Detection of resistance genes. Primarily, Bacterial DNA extraction was conducted using the Geno Plus™ Genomic DNA Extraction Mini Prep System Kit (VIOGENE, Taiwan) following the manufacturer's protocols. Using the PCR technique, the *S. agalactiae* isolates were examined for the presence of resistance genes such as *erm*B, *mef*A, *erm*TR, and *lin*B. The particular primers displayed in Table 1 were synthesized at Takapouzist, Tehran, Iran. For PCR, 5 μL of extracted DNA was used as a template and the steps were as mentioned in (15). PCR products were investigated by gel electrophoresis in agarose (approximately 1%) in the 1X TAE buffer, stained with a safe stain. Finally, DNA bands were visualized by a UV transilluminator.

Molecular serotyping. Screening of capsular determinants of the GBS isolates for serotyping was done through multiplex PCR (mPCR) as described in (17) and primers used in this study were also adopted from the same reference. Furthermore, the amplicons were investigated using agarose gel (approximately 1%) and visualized by means of a UV transilluminator.

Statistical analysis. The SPSS statistical analysis tool (version SPSS 16) was used to examine the study's findings, and chi-square tests were used to evaluate the relationships between the variables. A significant threshold of $p < 0.05$ was established.

RESULTS

Among the 400 samples analyzed, 31 (7.75%) tested positive for GBS based on phenotypic methods. PCR analysis confirmed all phenotypically identified isolates, demonstrating full agreement between phenotypic and genotypic identification techniques. The antibiotic susceptibility test results are shown in Table 2. Among the 31 isolates, 22 (70.97%) were multidrug-resistant. MDR is defined as resistance to at least one antimicrobial drug in three or more antimicrobial categories. The highest resistance rate was reported to clindamycin and quinupristin-dalfopristin with 80.65% and 77.42%, respectively. Furthermore, according to our observations, resistance to penicillin G was also seen (3.23%). Of note, all chloramphenicol-resistant isolates exhibited co-resistance to clindamycin, another antibiotic used in the management of

Table 2. Antibiotic susceptibility patterns of 31 GBS strains.

Antibiotic susceptibilities of GBS isolates			
Agent	**NO. (%) of isolates**		
	Susceptible	**Intermediate**	**Resistant**
PG	25 (80.65)	5 (16.13)	1 (3.23)
E	9 (29.03)	12 (38.71)	10 (32.26)
T	4 (12.9)	4 (12.9)	23 (74.2)
OFX	25 (80.65)	4 (12.9)	2 (6.45)
C	7 (22.58)	13 (41.94)	11 (35.48)
CD	5 (16.13)	1 (3.23)	25 (80.65)
AZH	5 (16.13)	5 (16.13)	21 (67.74)
CIP	8 (25.81)	20 (64.52)	3 (9.68)
Quinupristin-dalfopristin	6 (19.35)	1 (3.23)	24 (77.42)

Table 1. Primer sequences used for amplification of resistance genes (16).

Target gene	Forward and reverse primers (5′ to 3′)	Amplicon size (bp)
*mef*A	AGTATCATTAATCACTAGTGC	348
	TTCTTCTGGTACTAAAAGTGG	
*Lin*B	CCTACCTATTGTTTGTGGAA	925
	ATAACGTTACTCTCCTATTC	
*erm*TR	GAAGTTTAGCTTTCCTAA	400
	GCTTCAGCACCTGTCTTAATTGAT	
*erm*B	GAAAAGGTACTCAACCAAATA	640
	AGTAACGGTACTTAAATTGTTTAC	

bacterial infections.

All 31 GBS isolates were phenotypically evaluated for resistance. Of these, 2 (6.45%) exhibited the $iMLS_B$ phenotype and 9 (29.03%) showed the $cMLS_B$ phenotype. We analyzed the isolates for resistance genes by the PCR method. Among the 31 examined isolates, only 4 harbored the *erm*B gene, while no other resistance genes (such as *mef*A, *erm*TR, or *lin*B) were detected. The serotypes distribution was as follows: II (38.71%), V (38.71%), Ia (19.35%), and III (3.23%). Other serotypes were not seen and all of the isolates were typeable. Importantly, serotype V isolates exhibited resistance to all tested antibiotics.

DISCUSSION

As mentioned earlier, GBS screening in pregnant women is essential, however, unfortunately in our country especially in our city (Urmia) little is known about it. The GBS colonization rate in Urmia (7.75%) was similar to that reported in Kashan (6.7%) (18). This relatively low prevalence is consistent with findings from other countries such as Argentina (19) and India (20). However, it is notably lower than the rates reported by Zakerifar et al. and Rostami et al. in other regions of Iran (21, 22). The reason for these different frequencies can be explained based on the differences in the sampling procedure, identification methods, and geographical variations. Importantly, all 31 isolates detected by phenotypic methods were true positives and confirmed with PCR, which was also seen in a study by HajiAhmadi et al. (23). Therefore, both phenotypic and genotypic methods seem to be appropriate approaches for GBS screening in pregnant women.

Penicillin is the first line of treatment for GBS, except in cases of hypersensitivity and allergy. Today, the level of resistance to penicillin has become concerning. In this study, the sensitivity rate to penicillin was 80.65%, which is in agreement with the findings of Zakerifar et al., who reported a rate of 78.3% (21). Surprisingly, HajiAhmadi et al. also reported that all their detected isolates (n = 36) were resistant to penicillin (23). However, 100% sensitivity has also been reported in other studies (24-26). Clindamycin and erythromycin are commonly considered the second line of treatment for people who are allergic to penicillin. The sensitivity to E and CD was also estimated as 29.03% and 16.13%, respectively, which is concerning. In our country, unfortunately, excessive use of antibiotics has become one of the most important reasons for the increasing resistance rate. Given the increasing resistance to the first and second-line antibiotics, it seems necessary to perform antibiotic sensitivity testing to select the appropriate drug.

Our study showed that 22 isolates were MDR. The high rate of MDR in our study (70.97%) and the study conducted in Vietnam (60.66%) (24) shows the need to determine antibiotic sensitivity because this increase in MDR strains can lead to a widespread concern.

Most of the resistance mechanisms in our study are similar to previous studies which were related to $cMLS_B$ (29.03%) (21, 27). Additionally, $iMLS_B$ phenotype in our study had rates of 6.45%. The only resistance gene identified in this study was *erm*B, which encodes 23S rRNA methylases and modifies the antibiotic target site. Similar studies, such as those by Zakerifar et al. (21), have also found this mechanism to be the primary one. In our phenotypic analysis, 22 isolates showed reduced susceptibility to erythromycin, 12 were intermediate and 10 were resistant, but only four of them carried the resistance gene. This is probably because the gene coding for rRNA methylases has been mutated. This reason also applies to other isolates that were phenotypically resistant, but the resistance gene was not observed. Out of four strains that had the *erm*B gene, three of them showed $cMLS_B$ phenotype and the other one showed $iMLS_B$ phenotype. In the present study, contrary to what was described by Santana et al., two other genes namely *mef*A and *erm*TR were not observed. Similarly to our study, the *lin*B gene was absent (25).

Serotyping revealed four distinct serotypes among the isolates: II and V (38.71% each), Ia (19.35%), and III (3.23%). Other serotypes were not detected. In the present study, in agreement with Savoia et al., serotype V had a high frequency (28). On the other hand, there were other studies in which this serotype had a low frequency (13, 29).

Serotype II was similarly prevalent in this study, a pattern consistent with another research (26). Also, in a meta-analysis study done in Africa, the rate of this serotype was low, and more serotype V was seen there (30). These geographical variations in serotype distribution highlight the influence of regional factors and underscore the need for localized surveillance. As it is evident from Table 3, serotype V shows resistance to all antibiotics, while this situa-

Table 3. The antibiotic resistance and serotypes.

Antibiotic resistance (%) Serotype(n)	PG	E	T	OFX	C	CD	AZH	CIP	Quinupristin-dalfopristin
V (12)	8.33	41.66	75	16.66	50	83.33	58.33	16.66	75
II (12)	0	25	83.33	0	16.66	75	75	0	75
Ia (6)	0	33.33	66.66	0	50	83.33	83.33	16.66	83.33
III (1)	0	0	0	0	0	100	0	0	100

tion is not seen in other serotypes. Also, in a study by Wang et al., serotype Ib had this resistance condition (31). Another study also reported that serotype V had the highest resistance rates to azithromycin, clindamycin, and erythromycin (32). Serotype V is particularly important not only because of its high prevalence in our study but also due to its resistance to multiple antibiotics. Because of these concerns, ongoing surveillance and the inclusion of serotype V in vaccine strategies are important for effective control and prevention.

CONCLUSION

Effective management of Group B *Streptococcus* infections in pregnant women necessitates continual surveillance and prudent use of antibiotics. This involves careful consideration of penicillin resistance and the prevailing resistance patterns. Vigilant monitoring of GBS infections allows for timely interventions and tailored antibiotic regimens, optimizing maternal and neonatal outcomes. Additionally, implementing antibiotic stewardship programs ensures wise antibiotic usage, minimizing the risk of antimicrobial resistance emergence and preserving the effectiveness of these crucial medications for future generations. By integrating surveillance efforts and antibiotic stewardship practices, healthcare providers can mitigate the impact of GBS infections on pregnant women and their babies while combating the threat of antimicrobial resistance.

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