IJMS
Vol 48, No 6, November 2023

Original Article

Molecular Serotyping and Antibiotic Resistance Profile of Group B *Streptococcus* Strains Isolated from Iranian Pregnant Women with Urinary Tract Infection

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Received: 08 August 2022
Revised: 15 October 2022
Accepted: 22 November 2022

Abstract

Background: Group B *Streptococcus* (GBS) can cause serious infections in neonates and pregnant women. GBS may cause urinary tract infections (UTIs). However, molecular epidemiology of such infections is rarely reported. The present study aimed to determine drug resistance patterns and molecular serotyping of GBS isolates in a population of pregnant Iranian women with UTIs.

Methods: A cross-sectional study was conducted during the first half of 2021 in the Department of Biology, East Tehran Branch, Islamic Azad University (Tehran, Iran). Sixty GBS strains isolated from the urine and placenta samples of pregnant women with UTIs were evaluated. The women were aged 19-46 years old at 35 to 37 weeks of gestation. The molecular serotype of GBS isolates was determined using a multiplex polymerase chain reaction, and the disc diffusion method was used to determine the antibiotic susceptibility pattern of isolates for different antibiotics. The association of the GBS serotype with the phenotype of antibiotic resistance was statistically analyzed using SPSS software (version 22.0) with a Chi square test and Cramer's V test. $P<0.05$ was considered statistically significant.

Results: GBS capsular serotype II was most prevalent (66.7%) followed by serotypes Ib (21.7%), Ia (3.3%), and III (1.7%). The prevalence of non-typeable isolates was significantly low (6.6%). Of the 60 GBS isolates, 18.3% were resistant to penicillin, 81.6% to ampicillin, 23.3% to clindamycin, and 30% to vancomycin; indicating the need for treatment alternatives.

Conclusion: Region-specific information on antibiotic resistance and molecular characteristics of GBS is essential for epidemiological investigations, effective treatment, and vaccine development.

Please cite this article as: Gharabeigi N, Tabatabaee Bafroee AS, Amini K. Molecular Serotyping and Antibiotic Resistance Profile of Group B *Streptococcus* Strains Isolated from Iranian Pregnant Women with Urinary Tract Infection. Iran J Med Sci. 2023;48(6):542-550. doi: 10.30476/ijms.2023.96346.2787.

Keywords ● *Streptococcus agalactiae* ● Pregnant women ● Urinary tract infection ● Serotyping ● Drug resistance

What's Known

- Group B *Streptococcus* (GBS) capsular serotype III is highly prevalent in Iran.
- GBS isolates are predominantly resistant to erythromycin and clindamycin.

What's New

- Capsular serotype II and drug resistance to ampicillin and tetracycline were identified as prominent. A possible association between drug resistance patterns and serotypes was also determined.
- Region-specific information on antibiotic resistance and molecular characteristics of GBS is essential for epidemiological investigations, effective treatment, and vaccine development.

Introduction

Streptococcus agalactiae, also known as Lancefield group B *streptococcus* (GBS), is the primary risk factor for neonatal infections with high morbidity and mortality. It was reported

that GBS can be found in the genitourinary tract of 11-30% of women worldwide, which may lead to birth canal infection and potentially severe infections in newborns.[1] According to 2018 reports, the prevalence of GBS among Iranian pregnant women was 11.9%, comprising colonization levels of 12.9%, 9.7%, 18.5%, and 3.7% in vaginal, recto-vaginal, rectal, and endocervical, respectively.[2, 3] The main issue associated with this bacterium is mother-to-child transmission during pregnancy.[4] It is estimated that approximately 50-75% of neonates are subjected to GBS during fetal development.[5] GBS infection is classified into early-onset disease (EOD) within six days of birth and late-onset disease (LOD) between seven and 89 days of life. Sepsis is prevalent in EOD due to direct transmission from mother to neonate, whereas in LOD, infants get meningitis from the mother or the environment.[6]

Different virulence factors may contribute to GBS, of which capsular polysaccharide (CPS) predominates and therefore the main target of antibody-mediated killing.[2] Based on unique serological characteristics and significant biochemical structures of sialic acid-rich CPS, GBS has been divided into ten different serotypes, namely Ia, Ib, II, III, IV, V, VI, VII, VIII, and IX. Of these, type III is the primary cause of meningitis in LOD, and serotype Ia is most prevalent in EOD.[7] Given the variations in the prevalence of GBS colonization in both pregnant and non-pregnant women in different regions, capsular serotyping of GBS provides useful epidemiological information. Previous studies reported that serotypes Ia, Ib, III, and V were often involved in invasive GBS infections. In the past decade, conjugated multivalent CPS-based vaccines were developed and shown to induce effective immunogenic responses and increase protection against perinatal GBS disease through maternal immunization.[6, 8, 9] As a preventive measure, intrapartum antibiotics are recommended in high-risk pregnancies to prevent maternal colonization of GBS. Intravenous penicillin G and ampicillin are the two recommended beta-lactam antibiotics. However, in case of penicillin allergy, other types of antibiotics (e.g., cefazolin, clindamycin, tetracycline, and vancomycin) may serve as an alternative.[10-12]

Despite a 70% reduction in the number of cases with perinatal GBS infection, there is still a need for further research to address the resistance of GBS isolates to antibiotics, such as ampicillin, vancomycin, penicillin, and clindamycin.[10] Information on antibiotic resistance and molecular characteristics of GBS is essential for epidemiological research, effective treatment, and vaccine development. Therefore, the present study aimed to determine drug resistance patterns and molecular serotyping of GBS isolates in a population of pregnant Iranian women with urinary tract infections (UTIs).

Materials and Methods

A cross-sectional study was conducted during the first half of 2021 in the Department of Biology, East Tehran Branch, Islamic Azad University (Tehran, Iran). Sixty GBS strains isolated from 16 urine and 44 placenta samples of pregnant women aged 18-45 years with UTIs were obtained from the microbiology laboratory of a hospital in Tehran (Iran). Pregnant women with confirmed UTI infection using antibiotic drugs were included in the study. However, those not using antibiotic drugs were excluded. Target isolates were further confirmed using Gram staining, colony morphology, ß-hemolytic activity, biochemical tests (sodium hippurate hydrolysis, CAMP [Christie–Atkins–Munch-Peterson] test, and resistance to bacitracin), and molecular tests (amplification of the *dlts* gene).[7] Then, all isolates were preserved in Trypticase Soy Broth with 20% glycerol at -70 °C.

The study was approved by the Ethical Committee of East Tehran Branch, Islamic Azad University, Tehran, Iran (IR.IAU.ET.REC.1400.035). Initially, the participants were informed about the purpose of the study, and confidentiality of the provided information was guaranteed. Subsequently, written informed consent was obtained from all participants.

Molecular Serotyping Using CPS Genes

DNA extraction: Genomic DNA extraction of GBS isolates was performed using a commercial genomic DNA extraction kit (catalog number: DM05050, Gene Transfer Pioneer, Pishgaman Co., Tehran, Iran) according to the manufacturer's instructions.

Multiplex Polymerase Chain Reaction (PCR): Molecular serotyping of GBS isolates was performed using four primer pairs to detect common capsular types (Ia, Ib, II, and III). Furthermore, the *dlt*S gene encoding a histidine kinase specific to GBS was included as an internal positive control (table 1).[13, 14] The sequences of all primers used were validated by Basic Local Alignment Search Tool (BLAST) in the National Center for Biotechnology Information (NCBI) databases. Then, they were sent to Macrogen (Seoul, South Korea) for oligonucleotide DNA synthesis. Positive controls for sequencing were confirmed and used for the target CPS genes.

Table 1: Primer sequences used in the study

Gene group	Gene name	Primer sequence 5' to 3'	Size (bp)
CPS genes*	Serotype Ia (*cps1aH*)	F: GGTCAGACTGGATTAATGGTATGC R: GTAGAAATAGCCTATATACGTTGAATGC	521
	Serotype Ib (*cps1bJ*)	F: TAAACGAGAATGGAATATCACAAACC R: GAATTAACTTCAATCCCTAAACAATATCG	770
	Serotype II (*cps2K*)	F: GCTTCAGTAAGTATTGTAAGACGATAG R: TTCTCTAGGAAATCAAATAATTCTATAGGG	397
	Serotype III (*cps1a/2/3I*)	F: TCCGTACTACAACAGACTCATCC R: AGTAACCGTCCATACATTCTATAAGC	281
Histidine kinase specific to GBS**	*dltS*	F: AGGAATACCAGGCGATGAACCGAT R: TGCTCTAATTCTCCCCTTATGGC	199

GBS: Group B *Streptococcus*; CPS: Capsular polysaccharide; *Teatero and colleagues,[14] **Poyart and colleagues[13]

The 20 µL multiplex PCR mixture contained 2 µL of DNA (50 ng), 10 µL of 2X Master Mix with standard buffer, and 0.8 µL of each four primer pairs. The thermal cycling program consisted of an initial denaturation at 94 °C for 3 min, followed by 30 cycles of denaturation at 94 °C for 30 sec, primer annealing at 58.5 °C for 30 sec, and primer extension at 72 °C for 1 min. To complete the chain extension, additional incubation at 72 °C for 5 min was considered after the last cycle. The obtained multiplex PCR products were visualized using 1.5% agarose gel (m/v) electrophoresis (at 110 V for 60 min) and an ultraviolet (UV) transilluminator (PoteinSimple SA-1000 Red Imager).

Antibiotic Susceptibility Test

Based on the Clinical and Laboratory Standards Institute (CLSI) 2017 guideline,[15] disc diffusion method was used to determine the antibiotic susceptibility pattern of isolates for different antibiotics. Antibiotic susceptibility testing (MAST group, Merseyside, UK) included clindamycin (2 µg), vancomycin (30 µg), erythromycin (15 µg), penicillin (10 µg), ampicillin (10 µg), tetracycline (30 µg), rifampin (5 µg), chloramphenicol (30 µg), and levofloxacin (5 µg). Resistance to at least three antibiotics of different classes was considered multidrug resistance (MDR). *Streptococcus agalactiae* (ATCC® 12386™) was used as quality control.

Statistical Analysis

Data were analyzed using SPSS software, version 22.0 (IBM SPSS Statistics, Armonk, NY, USA). The Chi square test was used to test the dependency between variables (CPS genes and resistance to antibiotics). Cramer's V test was used to calculate the degree of association between two nominal variables. $P<0.05$ was considered statistically significant.

Results

The 60 GBS isolates included 16 urine and 44 placenta samples. The samples were obtained from pregnant women aged 19-46 years old, most of whom (70%) were at 35 to 37 weeks of gestation. None of the participants had a history of spontaneous abortion and had not taken antibiotics two weeks prior to sample collection.

The Serotype Distribution of GBS Isolates

The molecular serotype of 60 GBS isolates was determined using multiplex PCR with primers specific to different serotypes and CPS loci (figure 1). A total of four capsular types were identified, and all but two GBS isolates were typeable. The prevalence of serotypes was 3.3% (Ia), 21.7% (Ib), 66.7% (II), and 1.7% (III) (table 2).

Antimicrobial Resistance Pattern

All studied GBS isolates were individually assessed for susceptibility to the nine common clinically relevant classes of antibiotics (table 3). Of the 60 GBS isolates, 18.3% were resistant to penicillin, 81.6% to ampicillin, 23.3% to clindamycin, and 30% to vancomycin. In addition, 16.7% and 5% of the isolates exhibited resistance and intermediate susceptibility to erythromycin, respectively. In terms of tetracycline, 86.6% were identified as resistant, and only 5% showed intermediate susceptibility. Furthermore, 1.6%, 8.3%, and 5% exhibited resistance, and 6%, 5%, and 11.6% exhibited intermediate susceptibility to levofloxacin, chloramphenicol, and rifampin, respectively. In addition, of the 60 GBS isolates, 26 (43.33%) were identified as MDR (table 4).

Clinical isolates were clustered by serotype to determine the association between the capsular serotype of GBS isolates and the phenotype of antibiotic resistance. Data on the two most prevalent serotypes (Ib and II) were analyzed using the Chi square test and Cramer's V test (table 5). The results showed that resistance to tetracycline and ampicillin was not associated with a specific serotype, and tetracycline- and ampicillin-resistant strains were highly prevalent serotypes.

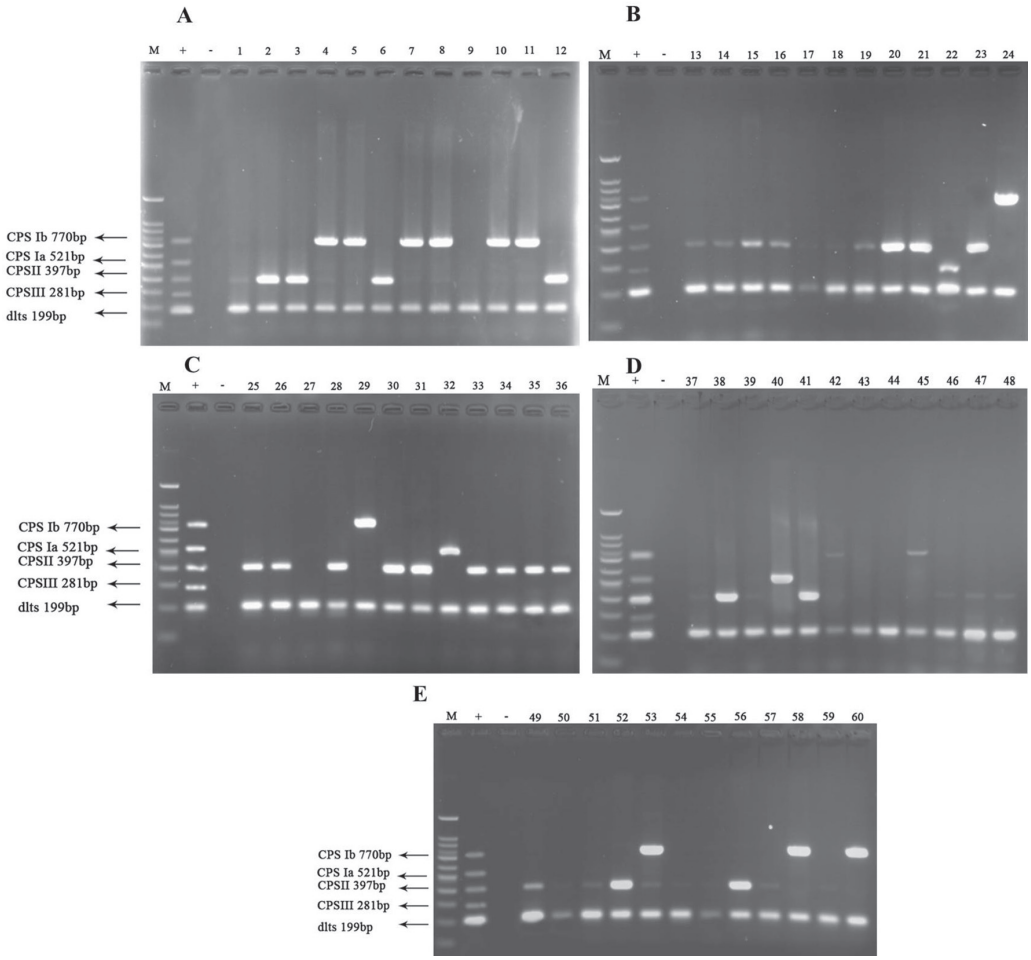

Figure 1: The figure shows specific multiplex PCR for the identification of capsular polysaccharide genes (*cps1aH*, *cps1bJ*, *cps2K*, and *cps1a/2/3I*) in 60 group B *streptococcus* isolates. M: DNA size marker 100 bp; +: Positive control; -: Negative control (distilled water); Wells 1 to 60: GBS isolates from pregnant women with urinary tract infection.

Table 2: Serotype distribution of group B streptococcus isolates in the study

Serotype	Ia	Ib	II	III	Non-typeable
Distribution (n/N)	2/60	13/60	42/60	1/60	2/60
Strain codes	32, 40	4, 5, 7, 8, 10, 11, 24, 29, 42, 45, 53, 58, 60	1, 2, 3, 6, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 23, 25, 26, 28, 30, 31, 33, 34, 35, 36, 37, 38, 39, 41, 43, 44, 46, 47, 48, 49, 50, 51, 52, 54, 55, 56, 57, 59	22	9, 27

n: Number of serotypes; N: Total number of isolates; Ia: *cps1aH*; Ib: *cps1bJ*; II: *cps2K*; III: *cps1a/2/3I*

Table 3: Antimicrobial susceptibility patterns of the 60 group B streptococcus (GBS) isolates in terms of commonly prescribed antibiotics

Antimicrobial agents (breakpoint in mm)	Frequency of GBS antibiotics		
	Sensitive	Intermediate	Resistant
Clindamycin (15-19 mm)	46 (76.6%)	0	14 (23.3%)
Vancomycin (R≤16 mm, S≥17 mm)	42 (70%)	na	18 (30%)
Erythromycin (15-21 mm)	47 (78.3%)	3 (5%)	10 (16.7%)
Penicillin (R≤23 mm, S≥24 mm)	49 (81.6%)	na	11 (18.3%)
Ampicillin (R≤23 mm, S≥24 mm)	11 (18.3%)	na	49 (81.6%)
Tetracycline (18-23 mm)	5 (8.33%)	3 (5%)	52 (86.6%)
Rifampin (14-21 mm)	50 (83.3%)	7 (11.6%)	3 (5%)
Chloramphenicol (17-21 mm)	56 (93.3%)	3 (5%)	1 (1.6%)
Levofloxacin (17-21 mm)	54 (90%)	1 (1.6%)	5 (8.3%)

na: Not available. Data are expressed as numbers and percentages.

Table 4: Antibiotic resistance pattern of multidrug resistance group B streptococcus (GBS) isolates from pregnant women with urinary tract infections

Resistance pattern of GBS isolates (n=60)								Antibiotic (n)	Total number of MDR isolates (n, %)
Amp	Tet	Van						3	9 (15%)
Amp	Tet	Van							
Amp	Tet	Van							
Amp	Tet	Van							
Amp	Tet	Lvx							
Amp	Tet	Ery							
Amp	Tet	Ery							
Amp	Tet	Ery							
Amp	Tet	Pne							
Amp	Tet	Van	Cli					4	7 (11.66%)
Amp	Tet	Ery	Cli						
Amp	Tet	Ery	Cli						
Amp	Pne	Ery	Rif						
Amp	Pne	Van	Rif						
Amp	Tet	Van	Lvx						
Amp	Tet	Van	Lvx						
Amp	Tet	Ery	Cli	Van				5	2 (3.33%)
Pne	Tet	Ery	Cli	Van					
Amp	Tet	Ery	Cli	Van	Lvx			6	4 (6.66%)
Amp	Tet	Ery	Cli	Van	Pne				
Amp	Tet	Pne	Cli	Van	C				
Amp	Tet	Pne	Cli	Van	RIF				
Amp	Tet	Ery	Cli	Van	RIF	Pne		7	2 (3.33%)
Amp	Lvx	Ery	Cli	Van	RIF	C			
Amp	Tet	Ery	Cli	Van	RIF	C	Pne	8	2 (3.33%)
Amp	Tet	Lvx	Cli	Van	RIF	C	Pne		

Cli: Clindamycin; Van: Vancomycin; Ery: Erythromycin; Pne: Penicillin; Amp: Ampicillin; Tet: Tetracycline; RIF: Rifampin; C: Chloramphenicol; Lvx: Levofloxacin; MDR: Multidrug resistance (resistance to at least three antibiotics of different classes)

Table 5: Antibiotic sensitivity distribution of the most prevalent capsular serotypes (Ib and II) in the study

Antibiotics		Serotype Ib (n=13)			Serotype II (n=42)		
		S	R	P value	S	R	P value
Antibiotic resistance pattern	Clindamycin	8 (61.5%)	5 (38.4%)	0.14	34 (80.9%)	8 (19.04%)	0.38
	Vancomycin	5 (38.4%)	8 (61.5%)	0.02*	33 (78.5%)	9 (21.4%)	0.02*
	Erythromycin	9 (69.2%)	2 (15.3%)	0.66	34 (80.9%)	7 (16.6%)	0.45
	Penicillin	10 (76.9%)	3 (23.07%)	0.61	35 (83.3%)	7 (16.6%)	0.34
	Ampicillin	0	13 (100%)	0.05*	10 (76.9%)	32 (76.1%)	0.05*
	Tetracycline	0	13 (100%)	0.27	7 (16.6%)	3 3 (78.5%)	0.34
	Rifampin	12 (92.3%)	0	0.54	33 (78.5%)	4 (9.52%)	0.40
	Chloramphenicol	11 (84.6%)	1 (7.6%)	0.13	40 (95.2%)	0	0.36
	Levofloxacin	10 (76.9%)	3 (23.07%)	0.08	39 (92.8%)	3 (7.1%)	0.33

*Significant association between serotype and antibiotic resistance (P<0.05); S: Sensitive to antibiotic; R: Resistance to antibiotic. Chi square test was used to test the dependency between variables. Cramer's V test was used to calculate the degree of association between two nominal variables. P<0.05 was considered statistically significant.

The highest percentage of vancomycin- and erythromycin-resistant phenotypes was observed in serotype II. Furthermore, a significant association was found between vancomycin resistance phenotype and serotypes Ib and II (P=0.02).

Finally, a literature survey was conducted to determine variations in prevalent serotypes and the associated antibiotic resistance in different regions of Iran and some other countries (table 6). Overall, the results showed that serotype III was the most prevalent serotype.

Discussion

Maternal rectovaginal colonization is the main route for the transmission of GBS infection during the perinatal period. Women, the elderly, and immunocompromised patients are at increased risk of GBS infection. The prevalence of GBS due to UTI is reported at 1.1-2.3% of positive cultures in several countries.[27] In hospitalized patients

Table 6: The pattern of serotypes and associated antibiotic resistance in different regions

Articles	Region	Prevalent serotypes	Cli	Van	Ery	Pen	Amp	Tet	Rif	C	Lev
Current study	Tehran, Iran	*cps2K* (II), 42/60 (70%)	14/60 (23.3)	18/60 (30)	10/60 (16.7)	11/60 (18.3)	49/60 (81.6)	52/60 (86.6)	3/60 (5)	1/60 (1.6)	5/60 (8.3)
Sadeh et al.[5]	Yazd, Iran	*cps1a/2/3I* (III), 15/50 (30%)	-	-	-	-	-	-	-	-	-
Genovese et al.[11]	Eastern Sicily, Italy	*cps1a/2/3I* (III), 1,218/3,494 (34.9%) *cps*5O (V), 1,069/3,494 (30.6%)	1,090/3, 494 (31.2)	0/3,494 (0)	1,402/3, 494 (40.1)	6/3,494 (0.2)	5/3, 494 (0.1)	-	-	-	161/3, 494 (4.6)
Burcham et al.[16]	Michigan, USA	*cps*5O (V), 11/39 (28.2%)	12/39 (31)	12/39 (31)	17/39 (44)	6/39 (15)	-	37/39 (95)	-	-	-
Aboutorabi et al.[17]	Tehran, Iran	*cps1bJ* (Ib), 135/270 (50%)	90/ 270 (75)	135/ 270 (50)	90/ 270 (75)	90/ 270 (75)	118/ 270 (42.9)	90/ 270 (75)	-	90/ 270 (75)	-
Rostami et al.[18]	Isfahan, Iran	*cps1bJ* (Ib), 12/27 (44.4%) *cps1a/2/3I* (III), 11/27(40.7%)	8/27 (29.6)	0/27 (0)	12/27 (44.4)	5/27 (18.5)	5/27 (18.5)	-	-	-	3/27 (11.1)
Rahnama et al.[19]	Tehran, Iran	*cps1a/2/3I* (III), 25/50 (50%)	-	-	-	-	-	-	-	-	-
Aboutorabi et al.[20]	Hamadan, Iran	*cps1a/2/3I* (III), 35/62 (56.6%)	-	-	-	-	-	-	-	-	-
Beigverdi et al.[21]	Tehran, Iran	*cps1a/2/3I* (III), 27/41 (65.8%)	6/41 (14.6)	0/41 (0)	10/41 (24.4)	0/41 (0)	-	40/41 (97.6)	-	-	-
Nahaei et al.[22]	Tabriz, Iran	*cps*5O (V), 63/327 (19.5%)	-	-	-	-	-	-	-	-	-
Kimura et al.[23]	Kobe, Japan	*cps*6I (VI), 29/139 (20.8%) *cps*8J (VIII), 22/139 (15.8%)	-	-	-	-	-	-	-	-	-
Eskandarian et al.[24]	Selangor, Malaysia	*cps*6I (VI), 23/103 (22.3%)	18/103 (18)	-	24/103 (23)	0/103 (0)	-	74/103 (72)	-	-	-
Shabayek et al.[25]	Ismailia, Egypt	*cps*5O (V), 33/100 (33%)	7/139 (5.0)	-	14/139 (10.1)	0/134 (0)	0/134 (0)	-	-	-	-
Slotved et al.[26]	The Greater Accra Region, Southern Ghana	*cps*7M (VII), 44/108 (40.7)	-	-	-	-	-	-	-	-	-

Cli: Clindamycin; Van: Vancomycin; Ery: Erythromycin; Pne: Penicillin; Amp: Ampicillin; Tet: Tetracycline; RIF: Rifampin; C: Chloramphenicol; Lvx: Levofloxacin. Data are presented as the number of serotypes over the total number of isolates in percentages.

with bacteremia due to GBS, UTI caused by the same species were reported as the underlying condition in 39% of elderly patients.[28] The molecular epidemiology of such infections is not routinely investigated, although information on serotype patterns and antibiotic resistance is essential to develop effective vaccines. Identification of large capsular variants among GBS isolates indicates high genetic diversity of GBS UTIs.[29]

In the present study, we have evaluated 60 GBS isolates from the 16 urine and 44 placenta samples of pregnant women with UTIs to determine molecular serotyping and antibiotic resistance of the GBS strains. Based on the results, we identified four discrete serotypes with serotype II as the most prevalent, followed by serotypes Ib, Ia, and III. Unlike our study, several studies in Iran have reported serotype III as the most prevalent, followed by serotypes II, Ib, and Ia (table 6). For example, the population in Yazd and Hamadan showed the predominance of GBS serotype III.[5, 20] However, another study in Tehran reported serotype Ib as the most prevalent type, whereas serotype V was reported as the most common in Tabriz.[17, 22] The difference in the reported findings is primarily due to the unpredictable evolution of GBS strains, which in turn poses treatment challenges. We did not investigate GBS serotypes IV, VI, VII, and VIII, since they have rarely been isolated from patients in Iran. However, other countries such as Japan, Malaysia, Egypt, and Ghana reported an increased prevalence of these serotypes.[21, 24, 25]

Such geographical distribution of different serotypes necessitates the identification of GBS serotypes based on their capacity to cause disease. Therefore, region-specific epidemiological studies for the identification of predominant serotype(s) could pave the way for the development of appropriate prevention measures such as vaccines.[12]

Currently, intrapartum antibiotic prophylaxis (IAP) is recommended as a primary strategy to prevent early-onset GBS disease in neonates. Successful implementation of this strategy requires the selection of effective antibiotic therapy and prevention of bacterial resistance according to standard guidelines. Increased antibiotic resistance were reported in pathogens and normal flora.[16] The GBS isolates exhibited different levels of antibiotic resistance. Our results showed that 49 GBS isolates did not exhibit susceptibility to tetracycline, and 11 were resistant to penicillin. In contrast, a previous study reported a high rate of antibiotic susceptibility of GBS to tetracycline.[30] Currently, penicillin is the first-line antibiotic for pregnant women with a positive rectovaginal culture for GBS. In case of penicillin allergy, other types of antibiotics (clindamycin, erythromycin, vancomycin) are prescribed.[31] Most of the serotype II isolates were resistant to antibiotics, including penicillin. Other studies reported similar findings on the resistance of GBS isolates and specific serotypes to penicillin.[12, 31] Therefore, GBS serotyping is essential to efficiently treat GBS colonized pregnant women before delivery.

Our results showed that most isolates (n=49) exhibited phenotype resistance to ampicillin. Other recent studies also reported reduced susceptibility to β-lactams in GBS isolates from pregnant women.[12, 17, 31] We also found that 30%, 23.3%, and 16.7% of the isolates were resistant to vancomycin, clindamycin, and erythromycin, respectively. Our results showed that most of the GBS serotypes, particularly serotype II, were resistant to at least three antibiotics of different classes, the so-called MDR strain.[16, 18] In addition to efficient treatment strategies for pregnant women colonized by GBS before delivery, we underscore the importance of region-specific epidemiological studies for the identification of predominant serotypes and appropriate prevention measures (e.g., vaccines). The main limitation of the study was the collection of GBS isolates from a single center and at a specific period. Further studies in different regions and seasons are recommended.

Conclusion

Region-specific information on antibiotic resistance and molecular characteristics of GBS is essential for epidemiological research, effective treatment, and vaccine development. Molecular serotyping of GBS isolates showed that serotype II was highly prevalent in our patients. All GBS isolates were susceptible to commonly prescribed first-line antibiotics and exhibited MDR, particularly serotype II, indicating the need for treatment alternatives. Our findings contribute to the development of preventive strategies as well as narrow-spectrum antibiotics to effectively treat invasive GBS infections in pregnant women and neonates.

Acknowledgment

The authors would like to thank the staff at the Department of Biology, East Tehran Branch, Islamic Azad University (Tehran, Iran) for their technical support.

Authors' Contribution

All authors contributed to the study conception and design; data acquisition, analysis, and interpretation; and drafting and revising of the manuscript. They have read and approved the final manuscript and agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

Conflict of Interest: None declared.

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