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Research Article

Frequency of Antibiotic Resistance and Virulence Factors in *Streptococcus agalactiae* Isolated from Vaginal and Urine Samples of Pregnant Women in Yazd, Iran: A Cross-sectional Study

Nastaran Azartoos [1], Maryam Sadeh [2,*], Mahmoud Vakili [3]

[1] Department of Microbiology, Faculty of Medicine, Shahid Sadoughi University of Medical Sciences, Yazd, Iran
[2] Department of Laboratory Sciences, School of Paramedicine, Shahid Sadoughi University of Medical Sciences, Yazd, Iran
[3] Health Monitoring Research Center, School of Medicine, Shahid Sadoughi University of Medical Sciences, Yazd, Iran

[*] ***Corresponding Author:*** Department of Laboratory Sciences, Faculty of Paramedical Sciences, Shahid Sadoughi University of Medical Sciences, Yazd, Iran. Email: sadeh_m20@yahoo.com

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Abstract

Background: *Streptococcus agalactiae*, or group B *Streptococcus* (GBS), is part of the normal flora of the female vagina and gastrointestinal tract. The capsular serotype, virulence factors, and antibiotic resistance determine the pathogenic properties of this bacterium.

Objectives: This cross-sectional study aims to determine the frequency of antibiotic resistance and virulence factor genes in GBS isolated from vaginal and urine samples of pregnant women in Yazd, Iran.

Methods: A cross-sectional study was conducted from January 2021 to May 2022 in Yazd, Iran. Ninety GBS isolates from pregnant women were subjected to analysis. The Kirby-Bauer method was performed to assess antibiotic resistance of GBS to penicillin, clindamycin, erythromycin, tetracycline, and levofloxacin. Genotypic investigation of resistance to erythromycin, tetracycline, and clindamycin genes, as well as adhesion and invasion protein genes, was also evaluated by molecular methods.

Results: The highest resistance was related to tetracycline (93.3%), and the resistance rates to clindamycin and erythromycin were 13.3% and 12.2%, respectively. The frequencies of *scpB*, laminin-binding protein (*lmb*), *fbsA*, and *fbsB* genes in vaginal samples were 97.8%, 93.3%, 96.7%, and 83.3%, respectively, and in urine samples were 100% for all genes; however, the *hvgA* gene was not found in any vaginal or urine isolates. The distribution of antibiotic resistance genes was *tetM* (87%), *int-Tn* (72.2%), *ermA* (42.2%), *ermB* (12.2%), *tetO* (7.8%), *tetK*, *linB* (1.1%), and *tetL* (0%).

Conclusions: This study revealed that most isolates had more than three virulence genes, and the frequency distribution of these genes in vaginal and urine samples was almost the same.

Keywords: *Streptococcus agalactiae*, Virulence Factor, Antibiotic Resistance, Pregnant Women, Urine Samples, Vaginal Samples

1. Background

Group B *Streptococcus* (GBS), originally a livestock pathogen, was linked to human infections in 1938 and became a major cause of infant illness in the United States after the 1970s (1). Though part of the normal flora, it can cause serious infections in newborns and vulnerable individuals, including neonatal meningitis, and is one of the most common causes of meningitis in neonates; however, it can rarely cause meningitis in adults (2). This bacterium uses various factors, including polysaccharide capsules and surface proteins, to attach to host cells, invade tissues, and avoid the immune system (3). The capsular polysaccharide (CPS) is a major factor in its virulence and determines its serotype — ten of which have been identified. Infection involves several complex steps, often driven by these surface proteins, that lead to tissue damage (4).

Key surface proteins involved in GBS attachment and invasion include C5a peptidase, fibronectin-binding proteins (*fbsA* and *fbsB*), laminin-binding protein (*lmb*), and hypervirulent adhesin (*hvgA*). C5a peptidase,

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encoded by the *scpB* gene, inactivates the human C5a protein, which impairs neutrophil recruitment and dampens the host's inflammatory response (1). The GBS binds to human fibrinogen to aid in colonization and invasion. Fibronectin-binding proteins *fbsA* and *fbsB* play key roles — *fbsA* promotes adhesion to epithelial and endothelial cells, while *fbsB* facilitates bacterial invasion. The GBS also binds to laminin, a major basement membrane protein, through the *lmb* lipoprotein, which is commonly expressed and helps the bacteria colonize and invade damaged tissue (5).

The GBS expresses the lipoprotein *lmb*, which binds laminin, a major glycoprotein component of the basement membrane. This interaction facilitates bacterial colonization and invasion, particularly in damaged host tissues. The *lmb* gene is conserved across most clinical GBS isolates, underscoring its importance in pathogenesis (6). Another surface protein, *hvgA*, promotes intestinal colonization in infants and enables the bacteria to cross intestinal and blood-brain barriers, potentially leading to meningitis (7). Since 1990, the incidence of GBS diseases has decreased significantly due to the use of antibiotics during pregnancy (8). The GBS is sensitive to beta-lactams, but in recent years, strains resistant to erythromycin, tetracycline, and clindamycin have increased in several countries (9)

2. Objectives

This study aims to identify virulence factors (adhesion-aggressiveness) and antibiotic resistance genes in pregnant women's urine and vaginal samples and investigate the relationship between different genes found in GBS isolates.

3. Methods

3.1. Samples

Ninety archived GBS isolates (57 vaginal swabs and 33 midstream urine samples), previously serotyped using multiplex PCR, were stored at -70°C and used in this study. All 90 available archived GBS isolates collected during the study period were included, ensuring an adequate sample size for descriptive analysis. The recommendation to screen all pregnant women for GBS colonization between 35 and 37 weeks of gestation was first released in 1996 by the American College of Obstetricians and Gynecologists (ACOG), and was quickly followed by the Centers for Disease Control and Prevention (CDC) and the American Academy of Pediatrics (AAP). All isolates were handled under standardized conditions, and laboratory assessments were performed blinded to patient information to minimize potential sources of bias.

3.2. Group B Streptococcus Isolates Confirmation

The GBS isolates were recultured on blood agar (Liofilchem, Italy) and incubated at 37°C for 24 hours. Colonies showing β-hemolysis, gram-positive cocci, and a negative catalase test were identified as GBS. Final confirmation of *Streptococcus agalactiae* was done using the CAMP test and hippurate hydrolysis (10).

3.3. Antimicrobial Susceptibility Testing

Antimicrobial susceptibility testing was performed on 90 GBS isolates using the Kirby-Bauer disc diffusion method on Mueller-Hinton agar with 5% sheep blood. The antibiotics tested included clindamycin (2 µg), penicillin (10 µg), levofloxacin (5 µg), erythromycin (15 µg), and tetracycline (30 µg) (MAST, England), with results interpreted according to CLSI 2022 guidelines (11). Bacterial suspensions were prepared by adjusting pure GBS colonies in saline to match a 0.5 McFarland standard. The medium was inoculated with a sterile swab in three directions, antibiotic discs were placed, and plates were incubated at 37°C for 24 hours. *Streptococcus pneumoniae* ATCC 49619 was used as a control strain.

3.4. Double Disc Diffusion

The detection of inducible clindamycin resistance was done using the D test method. Briefly, erythromycin (15 µg) and clindamycin (2 µg) were placed 12 mm apart edge to edge (12).

3.5. DNA Extraction and Molecular Detection of Group B Streptococcus and Serotyping

Genomic DNA was extracted using a streamlined boiling-lysis method (thermal lysis), wherein bacterial cells were lysed by heating at ~100°C for 10 minutes (13). Molecular identification targeted the 952-bp *dltS* gene using specific primers (14). For each sample, a PCR reaction mixture of 20 µL was prepared, consisting of 4.5 µL sterile distilled water, 10 µL 2X PCR master mix (Amplicon, Denmark), 2.5 µL primers (with a final concentration of 10 pmol), and 3 µL template DNA. Amplification was processed as follows: First denaturation at 94°C for 300 seconds, followed by 35 denaturation cycles at 94°C for 60 seconds, annealing of cycles at 55°C for 60 seconds, and an extension cycle at 72°C for 60 seconds. The final extension cycles were done at 72°C for 300 seconds. Then, the amplicons were visualized using 1% agarose gel and a Gel Documentation

System (Life Technologies, USA). Serotyping of the initial 80 isolates had been previously performed, while the 10 replacement isolates were serotyped in this study using multiplex PCR as described by Poyart et al. (15).

3.6. Detection of Resistance and Virulence Genes

Amplification of all virulence factor genes, including *scpB*, *lmb*, *fbsA*, *fbsB*, and *hvgA*, was done by the PCR method, and molecular investigation of antibiotic resistance genes (*ermB*, *tetL*, *tetK*, *tetO*, *linB*, *ermA*, *tetM*, and *int-Tn*) was done by PCR and multiplex PCR methods. The primers used for detecting resistance and virulence genes are listed in Table 1. For each DNA isolate, a 20 µL reaction mixture was prepared, containing 5 µL of nuclease-free water, 10 µL of 2X PCR master mix (Amplicon, Denmark), 2 µL of the primer (with a final concentration of 10 pmol), and 3 µL of DNA template.

The PCR program was performed with an initial denaturation at 94°C for 300 seconds, followed by 35 denaturation cycles at 94°C for 60 seconds, annealing of cycles at 58°C for 30 seconds for *lmb*, *fbsA*, *fbsB*, *hvgA*, *tetL*, *tetK*, *linB*, and *tetO* genes, and 60°C for 30 seconds for the *scpB* gene, and 53°C for 30 seconds for the *ermB* gene, with an elongation cycle at 72°C for 60 seconds. The final elongation cycle was done at 72°C for 300 seconds. The amplicons were then visualized using 2% agarose gel and a Gel Documentation System (Life Technologies, USA). Additionally, multiplex PCR was used to detect *ermA*, *tetM*, and *int-Tn* genes as previously described by Poyart et al. (16).

For internal positive control, seven samples containing *lmb*, *fbsA*, *fbsB*, *scpB*, *tetK*, *tetO*, and *linB* genes were sequenced, and the results were analyzed. As for *tetM*, *ermB*, *ermA*, and *int-Tn* genes, previously sequenced isolates were used as positive control samples.

3.7. Statistical Analysis

The chi-square test was conducted using SPSS version 16 software for evaluation.

4. Results

4.1. Participant Characteristics

In this study, out of 90 GBS isolates, eighty isolates presented as GBS strains according to phenotyping tests. Among the 90 collected samples, 80 (88.9%) were identified as *S. agalactiae* by phenotypic methods. All were confirmed by PCR targeting the *dltS* gene, showing 100% concordance between culture and molecular identification. Ten GBS isolates collected from the same pregnant women were replaced due to contamination. The predominant serotype was serotype III (54.4%), followed by serotype II, Ia, Ib, and V, at 25.6%, 14.4%, 3.3%, and 2.2%, respectively. This study did not observe serotypes IV, VI, VII, or VIII.

4.2. Antibiotic Susceptibility

Table 2 shows that 100% of vaginal and urine isolates were sensitive to penicillin and levofloxacin, while over 90% of both vaginal and urine isolates were resistant to tetracycline. No significant difference was observed between the antibiotic resistance of vaginal and urinary isolates.

4.3. Virulence Genes

In the 90 strains studied, the frequencies of *scpB*, *lmb*, *fbsA*, and *fbsB* genes in vaginal samples were 97.8%, 93.3%, 96.7%, and 83.3%, respectively, and in urine samples, 100% of the samples had these four genes. However, the *hvgA* gene was not found in any of the vaginal and urine isolates. The frequency of the *fbsB* gene in vaginal samples is less compared to other genes (Table 3 , Figure 1).

4.4. Antibiotic Resistance Genes

The highest frequency in antibiotic resistance genes is related to the *tetM* gene (87%), and the frequency of other resistance genes, in order from high to low, is related to *int-Tn* (72.2%), *ermA* (42.2%), *ermB* (12.2%), *tetO* (7.8%), *tetK* and *linB* (1.1%), and *tetL* (0%). As Table 3 shows, the abundance of these genes in vaginal and urine samples was almost equally distributed (Figure 2).

4.5. Association of Virulence and Resistance Genes with Capsular Serotypes

As Table 4 shows, no significant difference was observed in the presence of virulence genes in different serotypes ($P > 0.05$). Also, capsular serotype III was associated with the most resistant *S. agalactiae* isolates with *tetM*, *int-Tn*, and *ermA* genes.

5. Discussion

In this study on 90 GBS isolates from vaginal (57) and urine (33) samples of pregnant women in Yazd hospitals, resistance was lowest to penicillin (0%) and levofloxacin (0%), followed by erythromycin (12.2%), clindamycin (13.7%), and highest to tetracycline (92.85%). The frequency of five virulence genes — *scpB*, *lmb*, *fbsA*, *fbsB*, and *hvgA* — and eight resistance genes — *ermA*, *ermB*, *tetL*, *tetK*, *tetM*, *tetO*, *linB*, and *int-Tn* — was assessed. Vaginal isolates had *scpB* (97.8%), *fbsA* (96.7%), *lmb* (93.3%), and

Table 1. Sequences of Primers and Amplicon Size of PCR Products Expected for Each *Streptococcus agalactiae* Antibiotic Resistance and Virulence Gene

Primers	Forward 5′-3′	Reverse 5′-3′	Amplicon (bp)
int-Tn (16)	GATGGTATTGATGTTGTAGG	GGTCTATATTGACAAGACG	528
ermA (17)	TCAGGAAAAGGACATTTTACC	ATACTTTTTGTAGTCCTTCTT	423
ermB (17)	GGTAAAGGGCATTTAACGAC	CGATATTCTCGATTGACCCA	454
tetM (17)	GTGGAGTACTACATTTACGAG	GAAGCGGATCACTATCTGAG	359
tetL (18)	TGAACGTCTCATTACCTG	ACGAAAGCCCACCTAAAA	993
tetO (18)	AACTTAGGCATTCTGGCTCAC	TCCCACTGTTCCATATCGTCA	538
tetK (18)	TCCTGGAACCATGAGTGT	AGATAATCCGCCCATAAC	552
linB (18)	CCTACCTATTGTTTGTGGAA	ATAACGTTACTCTCCTATTC	944
l mb (19)	GACGCAACACACGGCAT	TGATAGAGCACTTCCAAATTTG	300
hvgA (20)	ATACAAATTCTGCTGACTACCG	TTAAATCCTTCCTGACCATTCC	210
fbsA (19)	TGTAGCTAATGGACCGATGTT	TTTTCATTGCGTCTCAAACC	156
fbsB (19)	ACAACTGCGGAAATGACCTC	ACGAGCGACGTTGAATTCTT	186
scpB (21)	ACAACGGAAGGCGCTACTGTTC	ACCTGGTGTTTGACCTGAACTA	250

Abbreviation: *Lmb*, laminin-binding protein.

Table 2. Antibiotic Susceptibility of 90 Group B *Streptococcus* Isolates Obtained from Pregnant Women's Vaginal and Urine Samples [a,b]

Antibiotic	Resistant	Intermediate	Susceptible
Penicillin	0/0	0/0	57 (100)/33 (100)
Levofloxacin	0/0	0/0	57 (100)/33 (100)
Tetracycline	54 (94.18)/30 (90.9)	1 (1.17)/1 (3)	2 (3.15)/2 (6.1)
Clindamycin	7 (12.3)/5 (15.1)	2 (3.5)/1 (3)	48 (84.2)/27 (81.9)
Erythromycin	7 (12.3)/4 (12.1)	12 (21)/6 (18.2)	38 (66.7)/23 (69.7)

[a] Values are expressed as No. (%) vagina/urine.

[b] $P > 0.05$.

Table 3. Frequency of Virulence and Antibiotic Resistance Genes in Vaginal and Urine Samples (N = 90) [a]

Gene	Gene Type	Total (N = 90)	Vaginal (N = 57)	Urine (N = 33)
scpB	Virulence	88 (97.8)	55 (96.5)	33 (100)
lmb	Virulence	84 (93.3)	51 (89.5)	33 (100)
fbsA	Virulence	87 (96.7)	54 (94.7)	33 (100)
f bsB	Virulence	75 (83.3)	42 (73.7)	33 (100)
hvgA	Virulence	0 (0)	0 (0)	0 (0)
ermA	Antibiotic resistance	38 (42.2)	23 (40.4)	15 (45.5)
ermB	Antibiotic resistance	11 (12.2)	7 (12.3)	4 (12.1)
tetM	Antibiotic resistance	79 (87.8)	50 (87.7)	29 (87.9)
tetO	Antibiotic resistance	7 (7.8)	4 (7.0)	3 (9.1)
tetK	Antibiotic resistance	1 (1.1)	0 (0)	1 (3.0)
tetL	Antibiotic resistance	0 (0)	0 (0)	0 (0)
linB	Antibiotic resistance	1 (1.1)	1 (1.8)	0 (0)
int-Tn	Antibiotic resistance	65 (72.2)	42 (73.7)	23 (69.7)

Abbreviation: *Lmb*, laminin-binding protein.

[a] Values are expressed as No. (%).

fbsB (83.3%), while all urine isolates carried these four genes; *hvgA* was absent in all.

Hannoun et al. (22) reported *lmb* (96.1%) and *scpB* (94.7%) in 76 isolates from pregnant women in Lebanon, aligning with our findings. Udo et al. (23) found *scpB* and

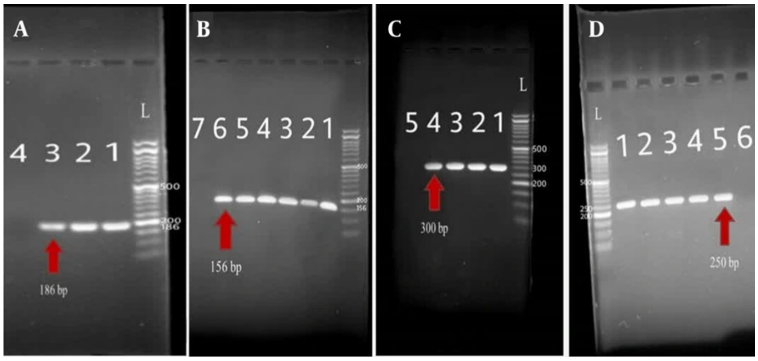

Figure 1. PCR amplification of virulence genes in group B *Streptococcus* (GBS) isolates: A, *fbsB*; B, *fbsA*; C, laminin-binding protein (*lmb)*; and D, *scpB*. Specific bands indicate gene presence.

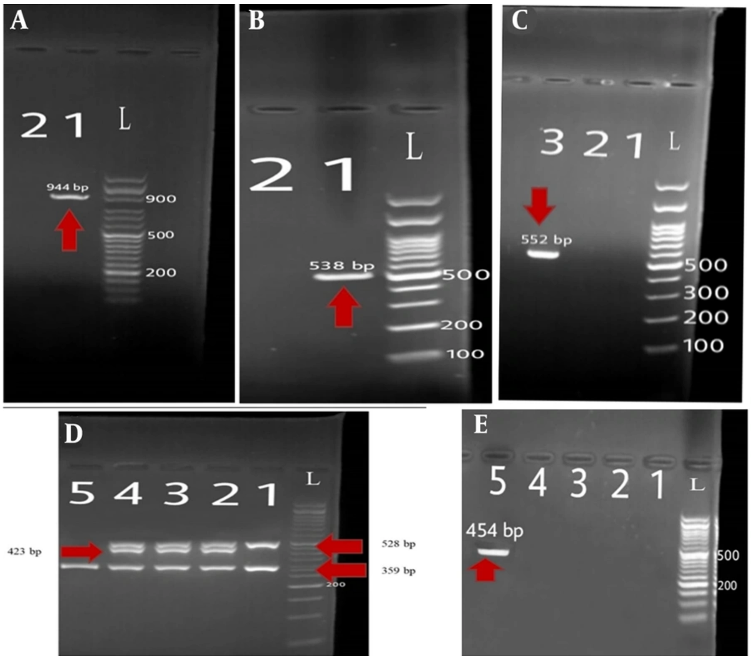

Figure 2. PCR amplification results of antibiotic resistance genes in vaginal and urine isolates: A, *linB*; B, *tetO*; C, *tetK*; D, multiplex PCR showing *ermA*, *tetM*; and E, *int-Tn ermB*.

lmb in 88.3%, and *fbsA* and *fbsB* in 49.5%. All urine isolates had *lmb*, *scpB*, *fbsA*, and *fbsB*; vaginal isolates had lower frequencies. Rosenau et al. (24) studied 111 isolates from vaginal and cerebrospinal samples, finding *fbsA* in 81.1%; 49.5% had both *fbsA* and *fbsB*, while 31.6% had only *fbsA*. They concluded *fbsB* is not expressed without *fbsA*, consistent with our data. The absence of *hvgA* in our isolates and its presence in infant GBS isolates, Li et al. (25) suggests it's infant-specific. In vaginal isolates, *fbsB* was less frequent and, due to its aggressive role, may be more common in neonatal isolates. Overall, *scpB*, *lmb*, and *fbsA* were dominant virulence genes in vaginal and urine samples.

Table 4. Frequency of Virulence Genes and Antibiotic Resistance According to Capsular Serotypes [a]

Gene	Serotype (N)					Total
	Ia (13)	Ib (3)	II (23)	III (49)	V (2)	
Virulence genes						
scpB	12 (92.3)	3 (100)	23 (100)	48 (98)	2 (100)	88
lmb	12 (92.3)	3 (100)	19 (82.6)	48 (98)	2 (100)	84
fbsA	13 (100)	3 (100)	21 (91.3)	48 (98)	2 (100)	87
fbsB	11 (84.6)	3 (100)	15 (65.2)	44 (89.8)	2 (100)	75
hvgA	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
Antibiotic resistance genes						
ermA	26 (46)	1 (33.3)	2 (8.7)	28 (57.1)	1 (50)	38
ermB	3 (23.1)	2 (66.7)	3 (13)	2 (4.1)	1 (50)	11
tetM	11 (84.6)	2 (66.7)	3 (13)	2 (4.1)	1 (50)	79
tetO	1 (7.7)	2 (66.7)	2 (8.7)	2 (4.1)	0 (0)	7
tetK	0 (0)	1 (33.3)	0 (0)	0 (0)	0 (0)	1
tetL	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
linB	0 (0)	0 (0)	1 (4.3)	0 (0)	0 (0)	1
int-Tn	10 (76.9)	2 (66.7)	12 (52.2)	40 (81.6)	1 (50)	65

Abbreviation: lmb, laminin-binding protein.

[a] Values are expressed as No. (%).

Regarding resistance genes, *tetM* had the highest frequency (87%), followed by *int-Tn* (72.2%), *ermA* (42.2%), *ermB* (12.2%), *tetO* (7.8%), *tetK* and *linB* (1.1%), and *tetL* (0%), with similar distribution in both sample types. Boswihi et al. (26) reported tetracycline resistance in 89.5% of 128 samples, with *tetM* (94.5%), *tetO* (3.9%), *tetL* (2%), and *tetK* (1%), consistent with our findings. *TetM*'s ~88% prevalence in our isolates likely explains the high tetracycline resistance. Modzana et al. (9) found 97.7% tetracycline resistance with *tetM* (97.6%) and *tetO* (2.4%), consistent with our results. We found *linB* in one vaginal isolate; Modzana et al. (9) reported none. Clindamycin resistance (13.7%) correlates with *erm* gene presence; low *linB* frequency aligns with this. Poyart et al. (16) found *int-Tn* commonly co-occurred with *tetM* in erythromycin-resistant isolates; similarly, we found *int-Tn* (72.2%) and *tetM* (87.7%) frequently co-present. Li et al. (25) reported in neonatal isolates: *TetO* (75%), *tetM* (46%), *linB* (6.24%), *ermB* (7.85%), differing from our results due to sample type. Resistance patterns in neonatal and maternal isolates differ.

Most isolates had ≥ 3 virulence genes; virulence and resistance gene profiles were similar across vaginal and urine samples. *hvgA* was absent in all, suggesting infant-specificity. *fbsB* was less frequent in vaginal samples and may be more common in neonatal isolates due to its virulence. Penicillin and ampicillin remain first-line GBS treatments, but for allergic patients, alternatives require antibiogram guidance. High erythromycin and tetracycline resistance warrants antibiograms for all GBS isolates. Given similar gene profiles in urine and vaginal samples, urine may be suitable for screening, and antibiograms are essential for effective therapy.

5.1. Conclusions

Serotyping of *S. agalactiae* is an important epidemiological marker, as certain serotypes such as III and V are commonly associated with higher virulence and antibiotic resistance. In Iran, studies have shown that serotypes III, V, and Ia are the most prevalent among pregnant women, which is consistent with global patterns. These serotypes are particularly relevant for neonatal infections and should be considered in prevention strategies.

Footnotes

Authors' Contribution: Study concept and design: M. S.; Acquisition of data: N. A.; Analysis and interpretation of data: M. V.; Drafting of the manuscript: M. S.; Statistical analysis: M. V.

Conflict of Interests Statement: The authors declare no conflict of interest.

Data Availability: The dataset presented in the study is available on request from the corresponding author during submission or after publication. The data are not publicly available due to privacy concerns.

Ethical Approval: The study was approved by the Ethics Committee of Shahid Sadoughi University of Medical Sciences (IR.SSU.MEDICINE.REC.1401.082).

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Informed Consent: Written informed consent was obtained from patients during sample collection.

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