Virulence Factors of *Streptococcus agalactiae* in Neonatal Sepsis

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ABSTRACT

Background: *Streptococcus agalactiae or group B streptococcus (GBS) is a normal flora of the vagina of healthy women. It emerged as the leading cause of neonatal invasive infections. The purpose of this study was to detect the magnitude of GBS neonatal infection and colonization and to compare between invasive and colonizing strains as regard antibiotic susceptibility patterns, serotypes and virulence factors.* ***Methods:*** *A total of 145 neonatal blood samples and 95 vaginal swabs from pregnant women were collected in the present study. Invasive GBS were isolated from neonates by blood cultures. Colonizing GBS isolates were identified by vaginal swabbing of pregnant women using Todd-Hewitt selective broth medium, supplemented with gentamicin (8µg/mL) and nalidixic acid (15µg/mL). Antibiotic sensitivities and serotyping by latex agglutination were done. GBS virulence factors were studied including detection of β-haemolysin production, CAMP test on blood agar plates, C5a peptidase production encoded by scpB gene and presence of highly virulent GBS ST-17 clone.* ***Results:*** *GBS were isolated from 11.7% of neonates (17/145) and from 18.9% (18/95) of vaginal swabs. Resistance patterns of isolated invasive GBS were 29.4 %, and 17.6% for erythromycin and clindamycin respectively. Among invasive and colonizing GBS isolates, serotype III was the most common. GBS neonatal sepsis was significantly associated with respiratory distress, pneumonia, use of mechanical ventilation and use of nasal continuous positive airway pressure. All of GBS isolates were CAMP test positive. Hemolytic GBS were 91.4% (32/35) of isolates. The scpB gene was detected in 88.2% and 88.9% of invasive and colonizing GBS isolates respectively while presence of ST-17 clone was significantly associated with invasive GBS isolates with P value of 0.002.*

INTRODUCTION

Group B streptococcus, known as *Streptococcus agalactiae*, is a facultative Gram-positive coccus originally known for causing bovine mastitis[(1)]. In the 1970s, GBS emerged as the leading cause of neonatal morbidity and mortality, with a frequency of 2–3 cases per 1000 live births and case-fatality ratios as high as 50% [(2)].

Group B streptococcus is an important pathogen with the potential to cause disease in pregnant women, adults with underlying chronic medical conditions, but, above all, in newborns[(3)]. GBS have remained the leading cause of bacterial sepsis in neonates for the last two decades, despite a broadly implemented screen-and-treat policy. The incidence of invasive neonatal GBS infection is reported to range from 0.5 to 3 per 1000 live births [(4)].

The gastrointestinal tract is the natural human reservoir for GBS and is the likely source of vaginal colonization. GBS carriage in pregnant women may be chronic, intermittent, or transient [(5)]. GBS colonization rates up to 35% in healthy pregnant women have been reported [(3)].

Group B streptococcus is an important cause of neonatal sepsis in developed countries[(6)]. Neonatal sepsis occurring within the first week of life are designated early-onset neonatal sepsis (EONS) while late-onset neonatal sepsis (LONS) infections occur in infants aged >1 week[(4)]. GBS neonatal disease is acquired intrapartum from mothers with vaginal/ rectal colonization with GBS[(6,7)]. Besides vertical transmission, other sources of GBS colonization in neonates have been established. Cross-contamination from infected to uninfected neonates can occur from the hands of nursery personnel [(8)].

Group B streptococci virulence factors, including the polysaccharide capsule and several gene regulation mechanisms, have been identified, and may all contribute to the complex pathogenesis of GBS disease. Most known GBS virulence genes are clustered into 14 genetic islands and are considered to be potential GBS pathogenicity islands [(3)].

Ten antigenically and partially chemically distinct type-specific capsular polysaccharides of GBS are associated with human infection. Sialic acid residues of capsular polysaccharides contribute to GBS virulence by inhibiting C3 opsonisation and activation of the alternative complement pathway. Furthermore, sialic acid

mimics the human Lewis X antigen, thus making GBS capsular polysaccharide a poor antigen [9].

Among known GBS capsular serotypes, serotype III GBS strains are of particular importance, as they are responsible for a substantial proportion of neonatal infections. Various molecular typing methods suggested that a large majority of neonatal invasive diseases, and almost all meningitis, are caused by a limited number of strains of serotype III, defined as ''highly virulent clones'' or ST-17 clones [10].

CAMP factor is an extracellular protein produced by GBS which oligomerizes in the target membrane to form discrete pores and trigger cell lysis [11]. Another extracellular GBS product is β-haemolysin, which is encoded by *cylE* gene. Expression of β-haemolysin is associated with bacterial invasion, cytolytic injury and resistance to oxidative killing of GBS[9].

C5a peptidase, encoded by *scpB* gene, plays an important role in GBS attachment and invasion of host tissues. C5a peptidase also targets the chemotactic complement component C5a which enables GBS immune evasion by inhibition of chemotaxis and phagocytosis [9].

In the present study, we aimed to determine the magnitude of GBS neonatal infection and colonization, to study virulence factors of invasive GBS and to help designing strategies for prevention of neonatal GBS infection.

PATIENTS & METHODS

I- Study design:

This study was conducted over a period of 18 months from April 2010 to September 2011. Two groups were enrolled in this study, newborn infants showing signs of sepsis and pregnant women.

II- Study population:

Participants in this study included 145 newborn infants admitted to Neonatal Intensive Care Unit of Mansoura University Children's Hospital and 95 pregnant women between 35 to 37 weeks of gestation attending the Antenatal Outpatient Clinic of Obstetrics and Gynecology Department of Mansoura University Hospitals. Full history and clinical data of participants were collected. Consents were taken from parents of neonates and pregnant women included in the present study.

III- Sample collection and processing:

Blood samples were collected from 145 neonates showing clinical signs of septicemia suspecting GBS infection as fever, tachypnea and cyanosis [12]**.** After proper disinfection, 1 ml of venous blood was collected from each neonate through an umbilical venous catheter.

Vaginal swabs were collected from pregnant women according to Centers for Disease Control and Prevention (CDC) recommendations [4]**.** Swabs from the mucosal secretions of the lower-third part of the vagina were placed into Amies transport media.

All collected samples were transported to the Microbiology Diagnostics and Infection Control Unit (MDICU) for further processing. Blood samples were inoculated into pediatric blood culture bottles and incubated at 37˚C for 24 hrs. Blood culture bottles were subcultured on blood agar plates for 7 successive days aerobically at 37°C [13]. Any isolated GBS were considered invasive GBS isolates.

Vaginal swabs were inoculated into Todd-Hewitt selective broth medium (Oxoid, UK), supplemented with gentamicin (8µg/mL) and nalidixic acid (15 µg/mL). Inoculated broth was incubated aerobically for 24 hours at 37°C. The broth was then subcultured on a blood agar plate which was incubated aerobically for 24 hours at 37°C [4]**.** Any isolated GBS were considered colonizing GBS isolates.

IV- Identification of GBS:

- Colonies of GBS were grey, mucoid with production of narrow zone of β-hemolysis on blood agar,
- Microscopical examination showed Gram-positive cocci arranged in pairs and chains,
- API 20 Strep test (BioMérieux SA, France),
- SLIDEX® Strepto Plus B latex agglutination test for streptococcal grouping (bioMérieux SA, France).

V- Antibiotic sensitivity testing:

The sensitivity patterns of the isolated GBS were detected by disc diffusion method [14], using Mueller-Hinton agar with 5% sheep's blood and group of commercially prepared antibiotic discs (penicillin, ampicillin, cefazolin, cefotaxime, ofloxacin, gentamicin, erythromycin, clindamycin**,** imipenem**.** vancomycin and tetracycline).

VI- Detection of some virulence factors of GBS:

A- Detection of β-haemolysin production by GBS: Colonies of β–haemolysin producing GBS were identified by being β hemolytic on blood agar plates.

B-Detection of CAMP factor by CAMP test: A beta-hemolysin producing strain of *S. aureus* was inoculated as a streak across the centre of sheep blood agar plate. A single streak of

suspected GBS was inoculated perpendicular to that of the *S. aureus* with taking care that the two streaks do not intersect.The plate was incubated aerobically for 24 hours at 37 °C. Positive CAMP test appeared as an arrow-head shaped area of increased lysis at the junction of the two streaks [15].

C-Detection of type specific capsular polysaccharides: Type specific capsular polysaccharides were detected by GBS Typing Antisera (Denka Seiken, UK) against most common 6 capsular polysaccharides (Ia, Ib, II, III, IV, and V). It is a slide agglutination tests using rabbit monovalent antibodies against capsular polysaccharides.

D- Detection of C5a peptidase production: Detection of the *scpB* gene, encoding C5a peptidase, was performed using polymerase chain reaction (PCR). GeneJET genomic DNA purification kit, K0721 (Fermentas, Germany) was used for GBS DNA extraction**.** Nucleic acid amplification was done using primers *specific* for *scpB* gene[16]**:** forward primer 5' ACAACGGAAGGCGCTACTGTTC 3', reverse primer 5' ACCTGGTGTTTGACCTGAACTA 3' . PCR reaction was performed using the following steps: initial denaturation step for 5 minutes at 94 ºC, three step cycling x 45 times (denaturation for 30 secs at 94 ºC, annealing for 30 secs at 55 ºC and DNA extension for 30 secs at 72º C) and final extension for 2 mins at 72º C. After PCR analysis the size of GBS amplification fragment was found to be 255 base pair (bp) for *scpB* gene.

E-Detection of highly virulent GBS ST-17 clone: PCR was done by using primers specific for detection of ST-17 clone [10]: ST- 17S 5' ATACAAATTCTGCTGACTACCG 3', ST-17AS 5' TTAAATCCTTCCTGACCATTCC 3'. The expected size of GBS amplification fragment was 210-bp for ST-17 clone.

VII- Statistical analysis:

Data were analyzed using the statistical package for social science (SPSS version 17). Qualitative data were represented in the form of number and frequency, while quantitative data were represented in the form of mean ± standard deviation (mean ± SD). The analysis of variance among the group means is compared using one-way ANOVA test. All tests were considered significant when P value ≤0.05.

RESULTS

During the period of this study, 35 GBS isolates were isolated from collected samples, of them 17 invasive (11.7%) and 18 colonizing (18.9 %). Of these invasive isolates, 10 isolates caused EONS and 7 isolates caused LONS. Isolated bacteria from neonatal blood are shown in table (1).

Table (1): Types of bacteria isolated from neonatal blood.

Bacteria isolated from neonatal blood (n=145)	***Number (%)***
Gram-positive bacteria:	
- *Staphylococcus aureus*	47 (32.4)
- *GBS*	17 (11.7)
- *Coagulase negative staphylococci*	15 (10.3)
- *Enterococci*	5 (3.4)
Gram-negative bacteria:	
- *E. coli*	42 (29)
- *Klebsiella species*	15 (10.3)
- *Pseudomonas species*	2 (1.4)
- *Proteus species*	2 (1.4)

Table (2) and (3) demonstrate risk factors associated with GBS neonatal infection and vaginal colonization in pregnant women. It was found that premature rupture of membranes, intrapartum fever and young maternal age were highly significantly associated with invasive GBS infections in neonates. Low gestational age and vaginal mode of delivery were significantly associated with invasive GBS infections. On the other hand, young maternal age and bad obstetric history were the significant risk factors associated with GBS vaginal colonization in pregnant women.

Table (2): Risk factors associated with neonatal GBS infection.

Variable	***GBS positive*** *(n=17)*	***GBS negative*** *(n=128)*	***P value***
Body weight *grams±SD*	2980.0 ± 484.1	2971.2 ±585.5	0.953
Gestational age *weeks±SD*	36.75± 3.0	34.11 ± 3.53	**0.048***
Premature rupture of membranes	12 (70.6%)	29 (22.6%)	**0.001****
Intrapartum fever	10 (58.8%)	32 (25.0%)	**0.004****
Mode of delivery • ***Vaginal*** • ***C.S***	 14 (82.3%) 3 (17.7)	 71 (55.4%) 57 (44.6%)	**0.035***
Parity • ***Primipara*** • ***Multipara***	 7 (41.2%) 10 (58.8%)	 55 (42.9%) 73 (57.1%)	0.675
Sex • ***Male*** • ***female***	 8 (47.1%) 9 (52.9%)	 61 (47.6%) 67 (52.4%)	0.916
Maternal age *Years±SD*	22.2 ± 5.8	29.5 ± 6.3	**0.001****

* low significance, ** high significance

Table (3): Risk factors associated with GBS vaginal colonization in pregnant women.

Variable	***GBS positive*** *(n=18)*	***GBS negative*** *(n=77)*	***P value***
Age *Years±SD*	22.9 ± 7.1	30.2 ± 4.7	**0.01****
Parity • ***Primipara*** • ***Multipara***	 10 (55.5%) 8 (44.5%)	 40 (51.9%) 37 (48.1%)	0.939
Bad obstetric history (abortion or stillbirth)	12 (66.6%)	30 (38.9%)	**0.043***

While comparison of neonates with GBS and other bacterial infections, the following clinical findings were significant as demonstrated in table (4): respiratory distress, pneumonia, use of mechanical ventilation and use of nasal continuous positive airway pressure.

Table (4): Comparison of clinical picture between newborn infants with and without GBS infection.

Clinical findings	*GBS Positive Sepsis (n=17)*	*GBS Negative Sepsis (n=128)*	*P*
Apgar Score • **1 minute** • **5 minute**	 7.2 (±0.85) 8.5 (±0.61)	 7.3 (±0.75) 8.1 (±0.55)	 0.85 0.65
Respiratory Distress	12 (70.6%)	52 (40.6%)	**0.02***
Hypotension	9 (53.0%)	65 (50.8%)	0.71
Fever	5 (29.4%)	42 (32.8%)	0.66
Leucocytosis	11(64.7%)	86 (67.1%)	0.74
Thrombocytopenia	12 (70.6%)	88 (68.7%)	0.69
Pneumonia	9 (53.0%)	37 (28.9%)	**0.03***
Meningitis	1 (5.9%)	6 (4.7%)	0.59
Sepsis	12 (70.1%)	92 (71.8%)	0.78
PPHN	1 (5.9%)	8 (6.2%)	0.80
Use of Dopamine	8 (47.0%)	58 (45.3%)	0.73
Mechanical Ventilation	7 (41.2%)	31 (24.2%)	**0.02***
N-CPAP	4 (23.5%)	15 (11.7%)	**0.04***
Mortality	1 (5.9%)	8 (6.2%)	0.67

PPHN= primary pulmonary hypertension, N-CPAP= nasal continuous positive airway pressure, Apgar score is used to assess intrauterine environment (1 minute) and extrauterine environment (5 minutes), Dopamine is used to treat hypotension in sepsis.

Out of isolated invasive GBS isolates, 29.4%, 17.6%, 88.2% and 100% were resistant to erythromycin**,** clindamycin**,** tetracycline and gentamicin respectively as shown in table (5). As regarding serotypes distribution, all invasive GBS isolates belonged to serotypes III, Ia and V in descending manner. Serotype III was the most common among GBS isolates causing EONS and LONS. Colonizing GBS isolates belonged to serotypes III, V, Ia, II and IV in descending manner as shown in table (6).

Table (5): Resistant GBS isolates causing EONS and LONS.

Antibiotic	*Resistant GBS isolates No=17 (%)*	*Resistant GBS isolates causing EONS No=10 (%)*	*Resistant GBS isolates causing LONS No=7 (%)*
Erythromycin	5 (29.4%)	2 (20%)	3 (42.8%)
Clindamycin	3 (17.6%)	1 (10%)	2 (28.6%)
Tetracycline	15 (88.2%)	8 (80%)	7 (100%)
Gentamicin	17 (100%)	10 (100%)	7 (100%)

Table (6): Different serotypes among invasive and colonizing GBS isolates.

Serotype	*Invasive GBS isolates No=17*		*Colonizing GBS isolates No=18 (%)*
	Invasive GBS isolates causing EONS No=10 (%)	Invasive GBS isolates causing LONS No=7 (%)	
Ia	3 (30)	1 (14.3)	4 (22.2)
Ib	0 (0)	0 (0)	0 (0)
II	0 (0)	0 (0)	1 (5.6)
III	6 (60)	5 (71.4)	7 (38.9)
IV	0 (0)	0 (0)	1 (5.6)
V	1 (10)	1 (14.3)	5 (27.7)
VI	0 (0)	0 (0)	0 (0)

Comparison of invasive and colonizing GBS isolates as regarding antibiotic resistance, serotype distribution, virulence factors and virulence genes among invasive and colonizing GBS isolates are demonstrated in table (7). Presence of ST-17 clone was significantly associated with invasive GBS isolates.

Table (7): Comparison between invasive and colonizing GBS isolates.

Variable	*Invasive GBS isolates (No=17) No (%)*	*Colonizing GBS isolates (No =18) No (%)*
Antibiotic resistance:		
- Erythromycin resistance	5 (29.4)	3 (16.7)
- Clindamycin resistance	3 (17.6)	2 (11.1)
- Tetracyclin resistance	15 (88.2)	15 (83.3)
- Gentamycin resistance	17 (100)	18 (100)
Serotypes:		
- Serotype Ia	4 (23.5)	4 (22.2)
- Serotype Ib	0 (0)	0 (0)
- Serotype II	0 (0)	1 (5.6)
- Serotype III	11 (64.7)	7 (38.9)
- Serotype IV	0 (0)	1 (5.6)
- Serotype V	2 (11.8)	5 (27.7)
- Serotype VI	0 (0)	0(0)
Virulence factors:		
- Hemolytic GBS	15 (88.2)	17 (94.4)
- Non hemolytic GBS	2 (11.8)	1 (5.6)
- CMP test positive	17 (100)	18 (100)
- CMP test negative	0 (0)	0 (0)
Virulence genes:		
- *scpB* gene positive	17 (100)	18 (100)
- *scpB* gene negative	0 (0)	0 (0)
- ST-17 clone positive	8 (47.1)	1 (5.6)
- ST-17 clone negative	9 (52.9)	17 (94.4)

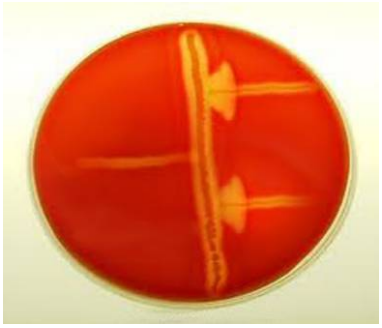

Figure (1): Blood agar plate showing positive CAMP test with an arrow-head shaped area of increased hemolysis on the right side and negative CAMP test on the left side.

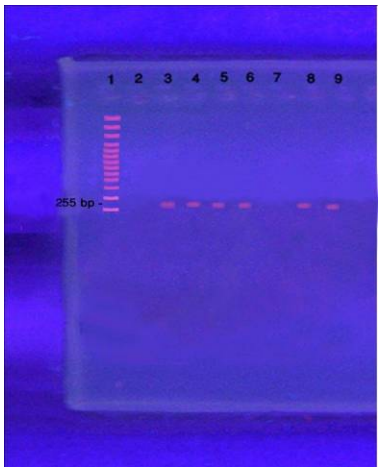

Figure (2): Polymerase chain reaction results with primers for *scpB* gene. Lane 1: molecular size marker # SM0323, Lane 2: negative control, Lane 3, 4, 5, 6, 8 and 9: positive results of 255 bp amplification fragments.

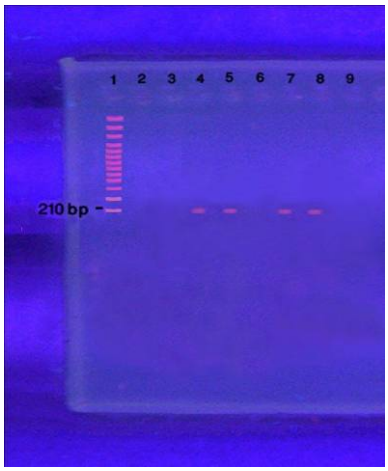

Figure (3): Polymerase chain reaction results with primers specific for ST-17 clone. Lane 1: molecular size marker # SM0323, Lane 2: negative control, Lane 4, 5, 7 and 8: positive results of 210 bp amplification fragments**.**

DISCUSSION

Group B streptococcus remains an important organism in both early onset and late onset neonatal sepsis for several reasons. First, GBS has the capacity to affect healthy newborn infants. Second, the clinical course of GBS sepsis and meningitis is dramatic with a high morbidity and mortality due to an excessive inflammatory response [(9)]. The present study included 145 newborn infants and 95 pregnant women between 35 to 37 weeks of gestation. Regarding the contribution of GBS among all cases of neonatal sepsis, invasive GBS isolates were identified in 11.7% (17/145) of blood cultures. GBS were isolated from EONS in a higher percentage 13.5 % (10/74) than from LONS 9.8% (7/71).

Our results are similar to those of ***Bindayna et al.*** [(17)] ***& Lukacs and Schrag*** [(18)] who investigated the organisms causing neonatal sepsis and reported that GBS caused 7.8 % and 9% of neonatal sepsis respectively.

In contrast to our findings, several studies from developed countries reported higher contribution of GBS in EONS in a percentage up to 45%[(19,20,21)]. The apparent lower incidence of invasive GBS disease in developing is possibly caused by inadequate culture techniques and microbiological methods, small blood volumes available or unrecognized/ undetermined causes of early neonatal or premature deaths and stillbirths [(22)].

In the present study, GBS vaginal colonization rate was18.9% among pregnant women. Our results are concordant with reported rates of maternal GBS carriage of 16% in Canada and Germany [(23,24)]. In Italy and Uruguay, vaginal GBS colonization rate were reported to be 18% and 17.3% respectively[(25,26)]. In one Egyptian study carried in Alfayom, the GBS carriage rate was reported to be 17.89% which is near to our results [(27)].

Regarding risk factors, it was found that preterm (gestational age <37 weeks), prolonged rupture of membranes ≥18hrs, presence of intrapartum fever, vaginal delivery and young maternal age below 20 years were significant risk factors for neonatal infection with GBS. Our results are in coordinate with the other reports of GBS neonatal disease risk factors[(28,29,30,31)].

We reported that previous history of abortion or stillbirth is a risk factor for GBS colonization among pregnant women. In consistent with our results, ***Rocchetti et al.*** [(32)] and ***Sharmila et al.*** [(33)] revealed that occurrence

of previous spontaneous abortion was associated with GBS colonization.

As regard clinical manifestations of GBS neonatal sepsis, we found that respiratory distress, pneumonia, use of mechanical ventilation and use of nasal continuous positive airway pressure were significantly associated with GBS neonatal sepsis. Our results run in parallel with the work of [31], who reported that respiratory failure, pneumonia and ventilator use occurred more often in EONS caused by GBS.

In the present study, all invasive and colonizing GBS isolates were sensitive to penicillin, ampicillin, cefuroxime, cefotaxime, imepenem, vancomycin and ofloxacin. The resistance rates for erythromycin and clindamycin were 29.4 % and 17.6% among invasive isolates and 16.7 % and 11.1% among colonizing isolates respectively.

Similar to ours, the prevalence of resistance among invasive GBS isolates in the United States ranged from 25% to 32% for erythromycin and from 13% to 20% for clindamycin[34,35]. Compared to reports from other countries with erythromycin resistance rates of up to 36% and clindamycin resistance rates of up to 18% [36, 37].

No trend toward reduced penicillin or ampicillin susceptibility of GBS isolates was observed in present study. Our findings demonstrate that routine susceptibility testing for β-lactams resistance of GBS strains isolated appears unnecessary.

As regard serotype distribution, we found that invasive isolates belonged to serotypes III, Ia and V while colonizing isolates belonged to serotypes III, V, Ia, II and IV in descending manner. This serotype distribution was similar to what has been found in other countries[25,38,39,40].

We studied many virulence factors of GBS including β-haemolysin, CAMP factor, *scpB* gene encoding for C5a peptidase production and presence of highly virulent GBS ST-17 clone. Among GBS isolates, 91.4% (32/35) were hemolytic. On the other side, all of GBS isolates were CAMP test positive. The *scpB* gene was detected in 88.2% isolates (15/17) and 88.9% isolates (16/18) of invasive and colonizing GBS isolates respectively.

In one study, 5.6% of GBS were found to be non hemolytic which is slightly lower than ours of 8.6% (3/35) [41]. As regard detection of *scpB* gene, our results are concordant with other worker who concluded that *scpB* gene is found in all invasive and colonizing GBS isolates[16,27].

Detection of highly virulent GBS ST-17 clone in vaginal samples or in neonates should permit the identification of a neonate population presenting a high risk for GBS infection [10]. In this study, we reported that 47.1% of invasive GBS isolates (8/17) were ST-17 clone positive confirming the high prevalence of this clone in invasive disease. On the other side, only 5.5% (1/18) of colonizing isolates were ST-17 clone positive. The ST-17 clone was significantly associated with invasive GBS isolates with P value of 0.002.

As regard detection of ST-17 clone, higher results were obtained by ***Lamy et al.*** [10] and ***Poyart et al.*** [42] who detected ST-17 clone percentage among 67.3% and 68.8% of invasive GBS isolates respectively. ***Poyart et al.*** [42] reported nearly similar results to ours as regarding colonizing GBS isolates. They reported that 7.7% of colonizing isolates were identified as ST-17 clone positive. ***Jones et al.***[43] reported that ST-17 clone was identified significantly more frequently among invasive isolates than among colonizing isolates.

Over presentation of ST-17 among invasive strains is recognized worldwide and highlights the fact that this clone is adapted to neonate pathogenesis and may possess specific virulence traits. Early detection of this clone among colonizing strains in pregnant women or in neonates at delivery may therefore constitute the basis for developing new prevention strategies[42].

CONCLUSION

Group B streptococcus is one of the important causes of neonatal sepsis in Egypt. Therefore, universal vaginal screening of GBS in near-term pregnant women with intrapartum antibiotic prophylaxis should be considered to reduce GBS neonatal infection. Also, detection of ST-17 clone by PCR can be used as a parameter to measure the invasiveness of GBS isolates in neonatal sepsis.

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عوامل الضراوة لميكروب المكورات السبحية الأجالكتية في حالات التسمم الدموي للأطفال حديثي الولادة

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بكتيريا المكورات السبحية الأجالكتية هي بكتيريا موجبة الجرام عرفت كمسبب في التهاب الضرع البقري. تعتبر بكتيريا المكورات السبحية الأجالكتية أحد المسببات الهامه لأمراض الأطفال حديثي الولادة و يمكن أن تسبب هذه البكتيريا الاصابه بالتسمم الدموي والالتهاب الرئوي و التهاب السحايا عند الأطفال حديثي الولادة.

تنتقل بكتيريا المكورات السبحية الأجالكتية من الأم إلى الطفل بشكل رئيسي أثناء الولادة المهبلية و تشمل عوامل الضراوة لميكروب المكورات السبحية الأجالكتية بيتا هيموليسين وعامل كامب وجين *scpB* المسؤل عن انتاج س ٥ بيبتيداس و ومستعمرة ST-17 . يمكن الوقاية من حوالي ٧٠٪ من حالات العدوى للأطفال حديثي الولادة عن طريق إعطاء المضادات الحيوية أثناء الولادة للنساء المعرضات لخطر انتقال بكتيريا المكورات السبحية الأجالكتية لاطفالهم اثناء الوضع.

و هدف البحث الي دراسة بكتيريا المكورات السبحية الأجالكتية بوصفها العامل المسبب للتسمم الدموي للأطفال حديثي الولادة والكشف عن نسبة التوطن المهبلي بهذه البكتيريا لدى النساء الحوامل والكشف عن عوامل الضراوة الخاصة بهذه البكتيريا .

شملت هذه الدراسة ١٤٥ طفل حديثي الولادة و٩٥ من النساء الحوامل . تم اخذ عينات من الدم من الأطفال حديثي الولادة الذين تظهر عليهم علامات سريرية للتسمم الدموي وتم تجميع مسحات مهبلية من النساء الحوامل . تم التعرف على بكتيريا المكورات السبحية الأجالكتية الموجودة فى العينات باستخدام المزارع واختبارالتفاعلات الكيميائية API واختبار التلزن لمعرفة المجموعات والانواع السيرولوجية المختلفة . كما تم الكشف على بعض عوامل الضراوة لميكروب المكورات السبحية الأجالكتية مثل بيتا هيموليسين وعامل كامب و س ٥ بيبتيداس ومستعمرة ST-١٧ .

وقد استنتجنا من هذا البحث ان بكتيريا المكورات السبحية الأجالكتية تشكل نسبة 11.7 % (١٧\١٤٥) من حالات التسمم الدموي لدى الأطفال حديثي الولادة بينما كان التوطن المهبلي بهذه البكتيريا لدى النساء الحوامل 18.9 % (١٨\٩٥). و قد وجدت عوامل الضراوة لميكروب المكورات السبحية الأجالكتية بنسب متقاربة فى البكتيريا المجتاحة و المتوطنة ما عدا مستعمرة ST-17 حيث وجدت بنسب اعلي احصائيا فى البكتيريا المجتاحة.

اظهر هذا البحث ان بكتيريا المكورات السبحية الأجالكتية هي احد المسببات الهامة في التسمم الدموي لدى الأطفال حديثي الولادة , لذا نوصى بالوقاية منها عن طريق إعطاء المضادات الحيوية أثناء الولادة للنساء المعرضات لخطر انتقال بكتيريا المكورات السبحية الأجالكتية لاطفالهم اثناء الوضع. كما يمكن استخدام مستعمرة ST-17 كاحد الدلالات القوية على ضراوة البكتيريا المجتاحة.