Maternal and Neonatal Group B *Streptococcus* colonisation in a cohort of Libyan women

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Abstract

Background: Group B streptococcus (GBS) may be vertically transmitted from mother to infant during labour and cause life threatening neonatal sepsis.
The objective of this study was to determine the risk and prevalence of GBS colonisation and vertical transmission and to establish an association between GBS colonisation and pregnancy outcomes.

Methods: One hundred samples were randomly collected from women at labour at Said hospital in Misrata, Libya. The study complied with the Declaration of Helsinki (2013). Maternal data was obtained through a questionnaire. Caesarean deliveries and women who had received antibiotic therapy within two weeks prior to sample collection were excluded from the study. Vaginal and rectal swabs were collected from each patient using sterile cotton-tipped swabs. Infant swabs were collected from the external ear canal at birth. GBS was detected using the Agilent AriaMx Real-Time PCR System.

Results: GBS was detected in 14 maternal rectal swabs and 12 vaginal swabs. GBS was detected in both vaginal and rectal swabs from only 8 mothers. Age, level of education, parity and gravidity showed no correlation with GBS colonisation nor pregnancy outcomes.

Of the 100 mothers who participated in the study, 93% delivered full term, while 7% delivered preterm. Normal birth weight was observed in 74% of the neonates with a significant association observed between birth weight of the neonate and GBS colonisation and between preterm delivery and GBS colonisation.

Conclusion: The prevalence of maternal GBS colonisation and risk for adverse pregnancy outcomes was low in this cohort of women, thus reducing the risk for neonatal sepsis.

Introduction

Group B *Streptococci* (GBS) are Gram positive bacteria found in 20% of normal microbiota in the gastrointestinal and genital tracts (1,2). GBS rarely causes illness in healthy individuals but it is pathogenic in cases of immune-compromised, elderly and pregnant women, infants and newborns (3). Usually women who carry GBS do not show any clinical symptoms. Sometimes GBS infections can cause urinary tract infections, chorioamnionitis, puerperal endometritis, especially following a caesarean delivery, and wound infections (4).

GBS infections in newborn babies is the most common cause of morbidity and mortality globally. It presents as early-onset disease (EOD) within 0–6 days of birth, or late-onset disease (LOD) after 7– 90 days. The most serious of these GBS infections is meningitis and pneumonia (5-7).

Materials and Methods

Isolation of DNA
Two hundred (200) µl of sample was transferred into a 2.0 ml collection tube and 200 µl of lysis buffer added. After adding 20 µl carrier RNA and 20 µl proteinase K, the sample was vortexed and incubated for 10 min at 65◦ C followed by 10 min at 95◦ C. After adding 260 µl binding buffer, the sample was mixed and incubated at room temperature for 5 min, and the lysate transferred to the Mini Spin Column and centrifuged for 1 min at 11.100 *g*. A volume of 600 µl wash buffer was added followed by centrifugation for 1 min at 11.100 *g*. The RTA collection tube with filtrate was discarded and the mini spin column placed into a new RTA collection tube. After the addition of 700 µl wash buffer II, the sample was centrifuged for another minute, the filtrate discarded and the mini spin column put back into the used RTA collection tube. This washing step was repeated once. After the addition of 100-200 µl of elution buffer, the sample was incubated at room temperature for 1 min, centrifuged for 1 min at 11.100 *g*, and the mini spin column discarded. The DNA/RNA sample was stored at -20◦ C to -80◦ C.

Real-Time PCR
All samples were analysed with the Agilent AriaMx Real-Time System. Normally the strip has eight holes, two holes for control and six holes for samples. Five (5) µl of sample was added to 15 µl rehydration mixture, while positive and negative controls consisted of 5 µl respectively added to 15 µl rehydration mixture. The system took 77 minutes to generate results.

Results

- Maternal medical history revealed that 15% of women had a vaginal discharge and 12% reported current urinary tract infections.
- Preterm deliveries accounted for 7% of deliveries (Table 2).
- 74% of the 100 births were normal birth weight infants; 3 infant delivered full term were GBS positive.
- 18% of mothers in the 18 - 25 & 26 - 35 years age groups were colonised with GBS.
- Multivariate regression analysis and the ANOVA statistical test were used to test the associations between GBS colonisation and pregnancy outcomes, with significant results being found for GBS colonisation and birth weight, GBS colonisation and term duration and between birth weight and term duration (Table 3).

Table 1. Prevalence of GBS from maternal rectal and vaginal swabs.

Rectal swabs	Vaginal swabs	Rectal and vaginal swabs
14 (14%)	12 (12%)	8 (8%)

Table 2. Associations between birth weight and preterm delivery

Birth weight	Preterm delivery
≤ 999g (n=0)	0%
1.000g -1.999g (n=3)	2%
2.000g -2.999g (n=19)	4%
3.000g -3.999g (n=74)	1%
4.000g -4.499g (n=4)	0%

Table 3. GBS colonisation and correlations with pregnancy outcomes

Paired sample correlations	Sig. *p* value
GBS colonisation and Birth weight	.000
GBS colonisation and Term	.000
Birth weight and Term	.000

Conclusions

The study was dominated by mothers who had a formal knowledge and understanding of maternal health issues even though level of education did not statistically influence the level of GBS colonisation, nor did age, parity and gravidity.

GBS colonisation significantly influenced the birth weight of the infants and was associated with PTD.

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References

1. McGee L, Beall B, de Filippis I, McKee ML. 2013. Molecular typing *Streptococcus* in bacterial infections. New York: Springer. 109-26.
2. Russell NJ, Seale AC, O'Driscoll M, *et al*. 2017. GBS Maternal Colonization Investigator Group. Maternal colonization with group B *Streptococcus* and serotype distribution worldwide: systematic review and meta-analyses. Clin Infect Dis. 65:100–11.
3. Gerolymatos G, Karlovasiti P, Sianou A. 2018. Antenatal group B *Streptococcus* detection in pregnant women: culture or PCR? J Infect Dev Ctries. 2018;12(8):631-5.
4. Morozumi M, Wajima T, Kuwata Y, Chiba N, Sunaosh K, Sugita K, Sakata H, Iwata S, Ubukata K. 2014. Associations between capsular serotype, multilocus sequence type, and macrolide resistance in *Streptococcus agalactiae* isolates from Japanese infants within invasive infections. Epidemiol Infect. 142:812-9
5. Lamagni TL, Keshishian C, Efstratiou A, *et al*. 2013. Emerging trends in the epidemiology of invasive group B streptococcal disease in England and Wales, 1991-2010. Clin Infect Dis 57:682–8.
6. Kohli-Lynch M, Russell NJ, Seale AC, *et al*. 2017. Neurodevelopmental impairment in children after group B streptococcal disease worldwide: systematic review and meta-analyses. Clin Infect Dis. 65:190–9.
7. Seale AC, Blencowe H, Manu AA, *et al*. 2014. Estimates of possible severe bacterial infection in neonates in sub-Saharan Africa, south Asia, and Latin America for 2012: a systematic review and meta- analysis. Lancet Infect Dis. 14:731–41.