TITLE: Prevalence, antimicrobial resistance and serotype distribution of Group B *Streptococcus* isolated among pregnant women and newborns in Rabat, Morocco

Running title: Group B *Streptococcus* from Rabat, Morocco

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Text word count: 3300

Abstract word count: 250

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Abstract

Introduction

Group B *streptococcus* (GBS) is an important cause of neonatal sepsis worldwide. Data on the prevalence of maternal GBS colonisation, risk factors for carriage, antibiotic susceptibility, and circulating serotypes are necessary to tailor adequate locally relevant public health policies.

Methods

A prospective study including pregnant women and their newborns was conducted between March and July 2013 in Morocco. We collected clinical data and vagino-rectal and urine samples of recruited pregnant women, together with clinical characteristics and body surface samples of their newborns. Additionally, the first three newborns admitted every day with suspected invasive infection were recruited for a thorough screening of neonatal sepsis. Serotypes were characterised by molecular testing.

Results

A total of 350 pregnant women and 139 of their newborns were recruited. The prevalence of pregnant women colonised by GBS was 24%. In 5/160 additional sick newborns recruited with suspected sepsis, blood cultures were positive for GBS. Gestational hypertension and vaginal pruritus were significantly associated with a vagino-rectal GBS colonisation in univariate analyses. All the strains were susceptible to penicillin, while 7% were resistant to clindamycin and 12% to erythromycin. Most common GBS serotypes detected included V, II and III.

Conclusions

In Morocco, maternal GBS colonisation is high. Penicillin can continue being the cornerstone of intrapartum antibiotic prophylaxis. A pentavalent GBS vaccine (Ia, Ib, II, III, and V) would have been effective against the majority of the colonising cases in this setting, but the trivalent one (Ia, Ib and III) would only prevent 28% of the cases.

Keywords: *Streptococcus agalactiae*, Group B Streptococcus, Morocco, antimicrobial resistance, risk factors, serotypes

Abbreviations

GBS: Group B *streptococcus*

HER*: Hôpital d'Enfants de Rabat*

ANC: Antenatal consultations

NBS: Newborn body surface

CHIS*: Centre Hospitalier Universitaire Ibn Sina*

MIC: Minimal inhibitory concentration

OR: Odds ratio

CI: confidence interval

MLST: multilocus sequence typing

Introduction

Group B streptococcus (GBS) is an important cause of neonatal sepsis worldwide and is associated with high mortality in low- and middle-income countries [1-3]. Maternal vagino-rectal GBS colonisation can lead to vertical transmission *in utero* or during childbirth, causing life-threatening neonatal infections [4]. To prevent vertical transmission of GBS, intrapartum antibiotics are recommended for women identified as GBS carriers through active screening conducted during the last trimester of pregnancy [5]. The incidence of early-onset GBS sepsis has notably decreased with this measure, but such a strategy is not effective against late-onset sepsis [6]. In certain countries, such as Morocco, GBS is not routinely screened in all pregnant women and intrapartum antibiotic prophylaxis is restricted to the recognition of determined risk factors as maternal fever, prolonged labour or prolonged rupture of membranes. For any of those strategies to work effectively, functional health systems and laboratory facilities are needed, something seldom available in the busy maternities of many middle- and low-income countries.

An additional strategy to prevent early and late neonatal GBS disease is based on the concept of “vertical vaccination” of pregnant women, under the assumption that the antibodies generated after vaccination would be passively transferred to the newborn. Various GBS conjugate vaccines against some GBS capsular polysaccharides are currently being developed [7]. These polysaccharides have chemical and antigenic differences that define ten GBS capsular types (serotypes) and play a role as virulence factors being involved in different mechanisms for evading the host immune system. As an example,

serotype III, and specifically its particular hypervirulent clone ST17, has been associated with a substantial proportion of invasive cases in infants [8, 9].

In this paper, we aimed to investigate a) the prevalence of GBS colonisation in pregnant women, b) the risk factors for GBS carriage, c) the antibiotic susceptibility of GBS isolates and d) the circulating serotypes in Morocco. In spite of the paucity of GBS data from Morocco, this pathogen is believed to cause a significant amount of morbidity and mortality in this country; however, preventive strategies are often inexistent.

Methods

Study setting and recruitment

A prospective study was conducted in the months of March to July 2013 in two different Moroccan hospitals: 1) the *Maternité des Orangers*, a public maternal tertiary level hospital in Rabat (Morocco); and 2) the neonatal unit of the *Hôpital d'Enfants de Rabat* (HER), the reference paediatric hospital for the country.

During the study, study staff asked pregnant women at gestational age between 35 and 37 weeks attending the general or high-risk antenatal consultations (ANC) whether they would be willing to participate in the study. "High risk pregnancy" was defined in this study as any given risk for the mother or foetus, identified by the obstetrician in charge. These included pre-existing chronic conditions or diseases of the mother, a previous unfavourable obstetric history, or other maternal, foetal or placental factors identified as potential risks to the outcome of the pregnancy. As some pregnant women may have failed to attend ANC during those weeks, an additional group of women (irrespective of

gestational age upon arrival) were directly enrolled at the time of delivery if they arrived in labour without both rupture of membranes and visible haemorrhage. For all groups, women above 18 years, or with permission of a legal guardian if younger, who signed an informed consent and without known allergy to material used for obtaining samples, underwent the standardised procedures. Due to the limited capacity of the laboratory, only the first three pregnant women who met inclusion criteria were recruited at each site every day.

Demographic, socio-economic and clinical data were collected into standardised questionnaires. One vagino-rectal and one vaginal swab were collected into Stuart transport medium according to standard methodologies [5] together with a urine sample and a vaginal smear.

Follow-up

Pregnant women recruited at the ANC were encouraged to attend the hospital for delivery. A unique identification number and a study card were given to them to facilitate the laboratory's feedback at the time of delivery. Pregnant women identified as GBS carriers were treated following the CDC guidelines [5]. The outcome of their delivery and basic clinical characteristics of their newborns were recorded using standardised forms. Newborn body surface (NBS) samples (ear and pharyngeal) were additionally collected into Stuart transport media within the first 6 hours of life. Newborns who showed any sign of infection were transferred and admitted to the neonatology unit of the HER. All children were offered a follow-up paediatric consultation after the 10^{th} day of life. Mothers who did not bring their children to these consultations were contacted by phone during the first month after the birth of their child. Mothers were also

encouraged to take their babies to the HER and identify themselves as study participants if the infants had any symptoms in the first 28 days of life. All infants born of participating pregnant women with suspected neonatal invasive infection were registered and their clinical data collected. Blood samples for basic sepsis screening (including 1 mL for blood culture), NBS (if they were younger than 6 hours) and cerebrospinal fluid were collected whenever indicated and possible. Clinical management of such patients was done according to national recommendations.

In addition, and with the objective of obtaining a complementary picture of the aetiology of neonatal sepsis in the same study area during the recruitment period, the first three newborns (irrespective of their mothers being or not part of the study) admitted every day with suspected invasive infection according to the admitting neonatologist (if maternal or guardian's written consent was obtained) were also recruited at HER's neonatology department with the same procedures.

Laboratory methods

Samples were initially analysed in the medical research laboratory of the *Centre Hospitalier Universitaire Ibn Sina* (CHIS). Vagino-rectal swabs were spread onto Granada agar specific for GBS isolation. The isolates were identified based on colony appearance, Gram stain and standard biochemical tests, using BD Phoenix (Becton-Dickinson, USA) whenever necessary. Blood from newborns was cultured using BACTEC 9050 (Becton-Dickinson, USA) culture system. Minimal inhibitory concentrations (MIC) (mg/L) of erythromycin, clindamycin, azythromycin, ampicillin, penicillin G, ceftriaxone, ciprofloxacin, meropenem,

and vancomycin were determined by E-test (BioMèrieux, France), according to CSLI guidelines [10]. *Streptococcus pneumoniae ATCC 49619* was used as a control strain for disk diffusion antibiotic sensitivity testing. Evaluation of potential vaginosis was conducted using Nugent's score [11]. GBS isolates were molecularly characterised at Hospital Clínic, in Barcelona, Spain. Capsular typing of GBS isolates was performed by multiplex-PCR [12, 13].

Data management and statistical analysis

All study questionnaires were manually verified for completeness, with errors edited where necessary, and subsequently doubly entered using a program written in Filemaker Pro 12 (Filemaker inc., Santa Clara, CA, USA). Statistical analyses were done with Stata 14.1 (Stata Corp., College Station, TX, USA). Study variables were counted and summarised in frequency tables. Data were analysed using the Fisher exact and Chi-square tests. Multivariate analyses were conducted using logistic regression methods. A probability of <0.05 was considered statistically significant.

Results

During the study, a total of 350 pregnant women and 139 neonates born of these women were recruited (Fig. 1). Many of the recruited mothers delivered outside of the hospital where they had conducted their antenatal follow-up. Additionally, 160 different newborns with suspected clinical sepsis were also investigated. For the principal maternal analysis, 349 vagino-rectal and vaginal swabs, 347 vaginal smears and urine samples were available. In addition, 136

ear and pharyngeal cultures from the infants born of these pregnant women were available.

Pregnant women's outcomes

Mean age of recruited pregnant women was 27 years old [Standard deviation (SD) 6.15; range 17-45]. Almost half of them, 47% (165/350), were primiparous. Almost 10% (33/350) referred an infection during the current pregnancy, 73% (24/33) genital and 27% (9/33) of the urinary tract.

Nearly a quarter of the pregnant women were GBS colonised (82/349; 24%) according to the results of the vagino-rectal swabs. As expected, the positivity of GBS in vaginal (only) samples was lower, 8% (26/349), although all women who had a positive vaginal sample had also a positive vagino-rectal one.

Risk factors significantly associated in a univariate analysis with a vagino-rectal colonisation with GBS are shown in Table 1. Pregnant women with a history of hypertension during the current pregnancy had a significantly higher risk of being GBS colonised [Odds ratio (OR) 17.34; 95% confidence interval (CI) (1.91,157.07); p<0.001], similarly to pregnant women with vaginal pruritus [OR 2.30; 95%CI (1.22,4.31); p=0.008].

No association of Nugent's score and vaginal pruritus was found (p=0.873), or between GBS colonisation and Nugent's score (Table 1). The observed association between vaginal pruritus and GBS colonisation remained statistically significant in the group with normal Nugent´s score (p=0.004), but not in the groups with intermediate results or overt vaginosis (p=0.745 and p=0.533, respectively).

Among the 82 GBS strains isolated from pregnant women, 67 (82%) were available for molecular characterisations. Serotypes V, II and III were the most frequently isolated, representing 36% (24/67), 25% (17/67) and 18% (12/67) of the cases, respectively. The remaining detected serotypes included Ia (6/67, 9%), IV (5/67, 7%) and IX (3/67, 5%) (Table 2).

Only 6/67 (9%) of the strains were erythromycin-resistant (MIC >256 mg/L), and 4/67 (6%) clindamycin-resistant (MIC >256 mg/L). All strains were fully (100%) susceptible to penicillin.

When specifically looking at the two more frequent serotypes (II and V), strains belonging to serotype II appeared to be susceptible to all the antimicrobial agents routinely administered as part of antenatal prophylaxis. In contrast, strains belonging to serotype V were more resistant to antimicrobial agents [13% (3/24) resistant to clindamycin and erythromycin].

Outcomes from newborns born of recruited pregnant women

A total of 139 newborns of the recruited 350 pregnant women (40%) were born in the study maternity wards and in 136 (97%) NBS swabs were performed (Table 3). Recruited babies were born vaginally in 76% (106/139) of the cases, through vaginally instrumented delivery in 13% (18/139) and by Caesarean section in 9% (13/139) of the cases. For two of the newborns, no information on mode of delivery was available. No differences in newborns' weight, length, or Apgar score at birth were observed in relation to GBS colonisation (data not shown). Ten of 136 children (7.4%) had at least one NBS culture positive for GBS and eight of them (80%) where available for molecular analysis (Tables 2

and 3). Two colonised newborns were born of pregnant women with a vagino-rectal culture negative for GBS. Colonisation of the pregnant women with GBS was significantly associated with newborn superficial colonisation ($p<0.001$). A borderline association was observed between colonisation of the pregnant woman by *E. coli* and GBS positive cultures in the newborn ($p=0.055$). Two of the three mothers colonised by *E. coli* who had a child with GBS colonisation were also colonised by GBS. The borderline association between maternal *E. coli* colonisation and GBS colonisation in the newborn must be interpreted with caution due to the low numbers, and the confounding effect of simultaneous maternal GBS colonisation. All strains were fully susceptible to penicillin, erythromycin and clindamycin.

The most common serotypes presented in pregnant women (II and V) were similarly present in infants (Table 2). However serotype IV was significantly more frequently isolated in children than among pregnant women ($p=0.035$) perhaps suggesting an association with transmission (Table 2). Other comparisons of serotype distribution among the pregnant women group vs. newborns with positive blood culture and this group of newborns vs. all newborns with any positive peripheral culture were performed, but no significant differences were observed (Table 4). None of the children born of pregnant women recruited at delivery developed clinical sepsis in the first 48 hours, irrespective of NBS results. Only 37 children born of the 82 colonised pregnant women were born in the study maternity wards, and 16 of them (43%) were followed up during their first month of life with only one being admitted with clinical sepsis (no microorganisms were isolated in samples obtained as part of the sepsis workup, and evolution was good).

Neonatal outcomes in recruited sick newborns with suspected sepsis

Of the 160 additional recruited newborns with sepsis suspicion in HER, 120 (75%) were aged less than 24 hours, and overall 154 (96%), were younger than seven days.

Among 160 newborns admitted with clinical sepsis, 158 had blood culture results available, and 31 (19%) had cerebrospinal fluid samples for meningitis investigation. Among the 158 blood cultures, 15 samples (9%) were positive. The isolated bacteria in blood included GBS (5, 3.2%; one case being also positive in CSF), *S. aureus* (6, 3.8%), *E. coli* (1, 0.6%), *K. pneumoniae* (1, 0.6%) and *Enterococcus* (2, 1.3%). One of these GBS-infected newborns died, yielding a case fatality rate for GBS infection of 20%. Among the 78 infants with available peripheral cultures, six (8%) infants presented at least one peripheral culture with GBS growth. All GBS detected in those infants were penicillin susceptible. Information of molecular analysis of the GBS isolated is summarised in Table 2.

Discussion

This study presents the first comprehensive effort to determine the prevalence of GBS colonisation, serotype distribution, and associated risk factors for this infection during pregnancy in Morocco, a country for which no clear preventive measures have been implemented to decrease the burden and morbidity associated with vertical GBS transmission. The prevalence of GBS colonisation in pregnant women of the study was 24%, similar to previous smaller studies in other areas of the country including pregnant women directly in the delivery

room (20.5% Marrakech; 23.3% Fez) [14, 15]. Such carriage rates were also in line with those described in other countries of the Northern African/Middle Eastern region (22%), or to more recent data from Sub-Saharan Africa [16-19], but generally higher than the overall regional estimate of 18% [18, 19]. Importantly, GBS was confirmed as an important cause of neonatal invasive bacterial disease, as it was detected in 3% (5/160) of the additional studied neonates with suspected neonatal sepsis.

In this study, GBS colonisation was significantly associated with hypertension during the current pregnancy. This association has not been reported in similar studies conducted elsewhere [15, 16, 20-23]. The association between pregnancy-related hypertension and asymptomatic bacteriuria has been known [24]. *E. coli* associated gastrointestinal infections during pregnancy have also been linked with maternal hypertension and infection and inflammation may be involved in the pathogenesis of preeclampsia [25-28]. The mechanistic of this association and the true impact of such a finding need to be confirmed and further explored.

Our study also confirmed an association between GBS carriage and vaginal pruritus, an easily detectable clinical symptom. Classically, GBS colonisation has been considered asymptomatic [29]. Co-infections could be the cause of the pruritus acting as potentially confounding factors, but it seems unlikely because no association between GBS and *E. coli* co-infection or between presence of vaginal *E. coli* and pruritus could be found. Similarly, the association persisted even in the group with a normal Nugent's score, ruling out

bacterial vaginosis as an alternative explanation. The true meaning of this finding, and its potential public health implications, also warrant further research. Other reported risk factors of GBS colonisation, such as race, smoking, age, diabetes, multiple partners, *Candida* colonisation, intravaginal cleaning, anaemia and seasonality among others were not identified in this study.[16, 17, 20, 30-34]. This limitation could be partly explained by the low prevalence of some conditions in our study (i.e. differences in race) or the heterogeneity of the populations evaluated in other studies, which often included non-pregnant women, HIV-infected women or pregnant women at other gestational ages [16, 17, 20, 30-34].

GBS colonisation in newborns was significantly associated with colonisation in their mothers ($p<0.001$). A positive maternal vaginal culture is the strongest risk factor for neonatal infection, which is in concordance with the results of this study [29]. It is, however, important to note that two colonised newborns were born of pregnant women with a negative vagino-rectal culture.

GBS strains isolated in this Moroccan setting were fully susceptible to penicillin and ampicillin, justifying that these two antibiotics remain the first-line treatment and prophylaxis option. However, the observed resistance to clindamycin (6%), or erythromycin (9%) could constitute a clinical problem in cases of allergy to penicillin, as these are the recommended alternatives [5].

Importantly, the previously described association between maternal colonisation with GBS and *E. coli* [16] was not observed in this study. This may be due to the lower prevalence of *E. coli* colonisation, although a borderline association

was observed between colonisation of the pregnant woman by *E. coli* and GBS-positive cultures in the newborn, suggesting that a relationship between both bacteria is plausible. This finding, if further confirmed, would have important public health implications, as treating GBS/*E. coli* co-infections with penicillin/ampicillin would therefore appear inadequate, as up to 20% of circulating *E. coli* isolates may be resistant to such antimicrobials [35].

Geographical diversity of GBS serotype distribution has been reported over time [36-38], with the capsular polysaccharide type III as the major culprit for GBS disease, causing more than 60% of all global cases [39]. A pentavalent conjugate vaccine including serotypes Ia, Ib, II, III and V could thus prevent 85%-98% of the global GBS neonatal disease [1, 19]. In Rabat, the two most common serotypes were V and II, both clearly related to invasive infection [40], closely followed by serotype III. In spite of the differences observed in this series, it is important to highlight that such a vaccine, currently under development, would have been effective against almost 90% of the colonising cases found in this setting, with the other candidate trivalent vaccine being able to prevent only 28% of the cases. In this study, serotype IV was observed in 6% of colonised pregnant women, similar to data from other countries as Turkey, Brazil and United States and lower than in other Middle Eastern countries, such as the United Arab Emirates [41-45]. This could suggest that serotype IV is an emergent serotype as it was significantly related to transmission and identified as cause of invasive disease, suggesting that this non-vaccine serotype could play a role in neonatal sepsis in this area. It is important to highlight, however, that GBS vaccines have been designed to prevent invasive neonatal disease

secondary to GBS, rather than to eradicate maternal GBS colonisation. Therefore, it is the serotype distribution of invasive neonatal GBS disease that should guide the design of any future GBS vaccine. Our findings predominantly reflect the distribution of circulating serotypes carried by Moroccan mothers or their children but not necessarily that of those responsible for invasive neonatal disease, as numbers in this particular group were very small. Larger studies better characterising septic infants with confirmed GBS disease in this setting are required to confirm the validity of the role of serotype IV and the potential repercussions of adding it to any new candidate GBS vaccine.

A study limitation is the absence of the molecular characterisation of the isolates with an analysis by multilocus sequence typing (MLST) that would significantly improve the results, as it would allow the comparison of the GBS genetic lineages circulating in Morocco with those in other countries. Additionally, it would be a valuable indicator for the analysis of vertical transmission when comparing the pregnant woman/neonate pairs.

Conclusions

GBS maternal colonisation in Rabat occurs in almost a quarter of pregnancies, and vertical transmission of this pathogen can therefore be a real but preventable threat to their offspring. Considering the high prevalence of GBS colonisation, the adequate acceptability of the screening procedures, the absence of easily identifiable risk factors and the insufficiency of the current intrapartum risk factor policy, universal screening would be an acceptable option in Morocco. A cost-effectiveness and feasibility study should be conducted in order to evaluate whether universal screening of GBS during

pregnancy is warranted, mirroring what is done in other parts of the world. Such a strategy could be a temporary solution in anticipation of the much-awaited pentavalent GBS maternal vaccine.

Funding information

The research leading to these results has received funding from the People Programme (Marie Curie Actions) of the European Union's seventh Framework Programme FP7/2007-2013 under REA grant agreement n° 612216. Sara M. Soto has a fellowship from the program I3, of the ISCIII. This material is based upon work supported by Grants PI10/01579 and PI13/00127 integrated in the Plan Nacional de I+D+I and cofunding from the "ISCIII Subdirección General de Evaluación", the "Fondo Europeo de Desarrollo Regional (FEDER)", and Miguel Servet contract (CP11/00039). Quique Bassat had during the duration of the study a fellowship from the program Miguel Servet of the ISCIII (Plan Nacional de I+D+I 2008-2011, grant number: CP11/00269). Fundació Clínic per a la Recerca Biomèdica receives core funding from the Spanish Agency for International Cooperation and Development (AECID) (Convenio de Fortalecimiento del sistema nacional de salud, con especial atención a la salud materno-infantil, Marruecos, 2008-2012). The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript.

Acknowledgements

We would like to thank all the staff from the different maternities/hospitals in Rabat and other members of the team who made this work possible, mainly Pascal Andignac, Eva López, Younes Ben Azzouz, Maria José López, Roberto Álvarez and the rest of the team at Fundació Clínic Maroc, and the technical personnel at the research laboratory of CHIS. We thank Julie Chaccour for help revising the manuscript. Finally, we are also thankful to the pregnant women, guardians and study participants. ISGlobal is a member of the CERCA Programme, Generalitat de Catalunya.

Conflicts of interest

The authors have no conflicts of interest to disclose. The authors have no financial relationships relevant to this article to disclose.

Ethical statement

The study was approved by the clinical Research Ethics Committee of the Hospital Clinic of Barcelona (Spain), and by the Institutional Review Board (C*omité d'Éthique de la recherché Biomédicale, Départ* Nº 153-21Fev2012) of the Faculty of Medicine in Rabat, Morocco.

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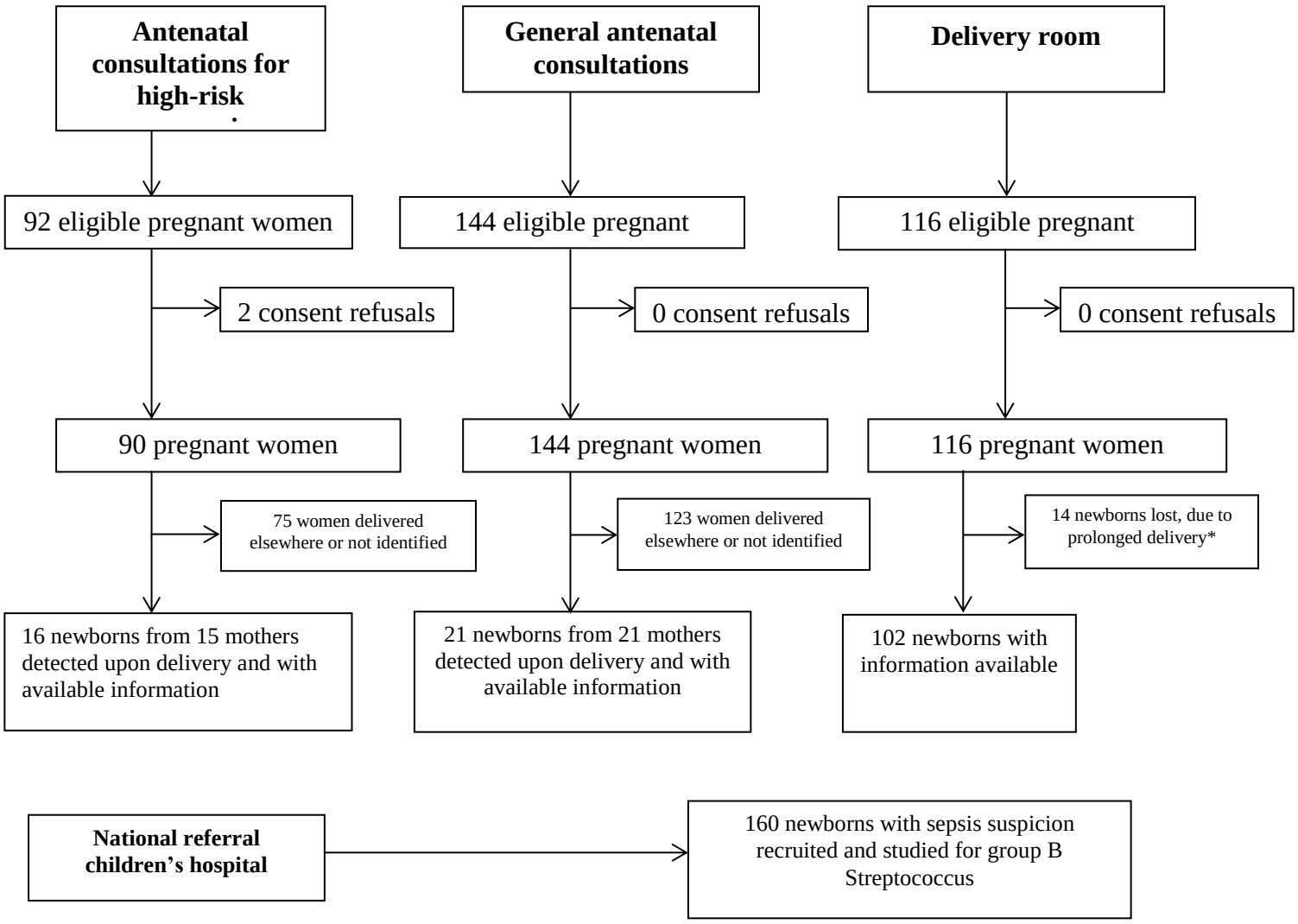
Figure 1: Study flow-chart



*Recruitment started during working hours but because delivery was prolonged, children were born during the weekend when the laboratory was not available and some information was lost.

Table 1: Risk factors for vagino-rectal GBS colonisation in pregnant women, according to the univariate analysis

Variables	GBS (+) n (%)	GBS (-) n (%)	p-value*
Recruitment place			
High-risk antenatal consultation	25 (30.49)	65 (24.34)	0.226[#]
General antenatal consultation	27 (32.93)	116 (43.45)	
Delivery room	30 (36.59)	86 (32.21)	
Medical insurance			
Yes	61 (74.39)	210 (78.65)	0.418[#]
No	21 (25.61)	57 (21.35)	
Pregnant women education			
None	12 (14.81)	38 (14.34)	
Primary	22 (27.16)	70 (26.42)	0.989[#]
Secondary	36 (44.44)	117 (44.15)	
University	11 (13.58)	40 (15.09)	
Partner's education			
None	4 (5.00)	25 (9.62)	
Primary	22 (27.50)	59 (22.69)	0.516[#]
Secondary	44 (55.00)	139 (53.46)	
University	10 (12.50)	37 (14.23)	
Pregnant women age (years)			
< 20	4 (4.88)	6 (2.25)	0.392[#]
20-29	39 (47.56)	140 (52.43)	
≥ 30	39 (47.56)	121 (45.32)	
Parity			
Primiparous	43 (52.44)	121 (45.32)	0.258[#]
Multiparous	39 (47.56)	146 (54.68)	
Use of antibiotics during pregnancy			
Yes	3 (3.66)	14 (5.24)	0.560[#]
No	79 (96.34)	253 (94.76)	
Vaginal cleansing			
Yes	16 (19.51)	48 (17.98)	0.753[#]
No	66 (80.49)	219 (82.02)	
Number of antenatal consultations			
0	2 (2.44)	10 (3.75)	0.847[#]
1-3	35 (42.68)	114 (42.70)	
>3	45 (53.56)	143 (53.56)	
Anaemia			
Yes	14(17.07)	52 (19.48)	0.627[#]
No	68 (82.93)	215 (80.52)	
Gestational diabetes			
Yes	5 (6.10)	13 (4.87)	0.660[#]
No	77 (93.90)	254 (95.13)	
Hypertension during pregnancy			
Yes	5 (6.10)	1 (0.37)	**0.003**[§]
No	77 (93.90)	266 (99.63)	
Infection during pregnancy			
Yes	8 (9.76)	25 (9.36)	0.915[#]
No	74 (90.24)	242 (90.64)	
Leucorrhoea			
Yes	20 (24.39)	64 (23.97)	0.938[#]
No	62 (75.61)	203 (76.03)	
Vaginal pruritus			
Yes	20 (24.39)	33 (12.36)	**0.008**[#]
No	62 (75.61)	234 (87.64)	
Dysuria			
Yes	10 (12.20)	27 (10.11)	0.592[#]
No	72 (87.80)	240 (89.89)	
E. coli colonisation			

Yes	5 (6.10)	34 (12.73)	0.095[#]
No	77 (93.90)	233 (87.27)	
Bacterial vaginosis			
Normal	63 (79.75)	219 (82.95)	
Intermediate	14 (17.72)	33 (12.50)	0.389[#]
Vaginosis	2 (2.53)	12 (4.55)	
GBS in newborn			
At least one positive culture	8 (9.8)	2 (0.75)	**< 0.001[§]**
Negative	28 (34.2)	94 (35.6)	
No culture available	46 (56)	168 (63.6)	

*: p value of the comparison between the first two columns; #: Pearson chi-square; §: Fisher's exact test; ł: 3 additional children with negative culture did not have mother's result available. **Bold characters**: statistically significant. GBS: Group B streptococcus

Table 2: Distribution of the different serotypes among Group B streptococcus strains isolated from pregnant women and newborns

Variables	Data from mother and newborns recruited at the maternity		p	Data from newborns admitted to the hospital with clinical sepsis suspicion		p
	Colonised mothers*	Colonised newborns#		Newborns with at least one positive NBS§	Newborns with positive GBS blood culture£	
Serotype	**N=67**	**N=8**		**N=6**	**N=5**	
Ia	6 (9%)	0 (0%)	1.000$^{\alpha}$	0 (0%)	0 (0%)	1.000$^{\alpha}$
II	17 (25%)	2(25%)	1.000$^{\alpha}$	2 (33%)	2 (40%)	1.000$^{\alpha}$
III	12 (18%)	0 (0%)	0.341$^{\alpha}$	0 (0%)	1 (20%)	0.455$^{\alpha}$
IV	5 (7%)	3 (38%)	**0.035**$^{\alpha}$	1 (17%)	1(20%)	1.000$^{\alpha}$
V	24 (36%)	3 (38%)	1.000$^{\alpha}$	3 (50%)	1 (20%)	0.545$^{\alpha}$
IX	3 (5%)	0 (0%)	1.000$^{\alpha}$	0 (0%)	0 (0%)	1.000$^{\alpha}$

*:Vagino-rectal or vaginal samples of pregnant women which strains were available for molecular analysis; #: Peripheral samples of colonised newborns born of recruited mothers which strains were available for molecular analysis; §: Infants recruited at the neonatal intensive care unit with clinical sepsis and with at least one newborn body surface (NBS) sample positive to Group B streptococcus and available for molecular analysis; ł: Infants recruited at the neonatal intensive care unit with clinical sepsis and blood culture positive to Group B streptococcus; α: Fisher's exact test; A probability of <0.05 was considered statistically significant. Bold characters: statistically significant.

Table 3: Body surface cultures results among infants with available samples, according to recruitment place

	High-Risk antenatal consultation	General antenatal consultation	Delivery room	Total	HER
Nº of recruited mothers	90	144	116	350	160*
Infants with available NBS swabs	15 (17%)	20 (14%)	101 (87%)	136 (39%)	78 (49%)
Results#					
GBS positive	3 (20%)	0 (0%)	7 (6.93%)	10 (7.35%)	6 (8%)
E. coli positive	0 (0%)	2 (10%)	12 (11.88%)	14 (10.29%)	7 (9%)
K. pneumoniae positive	0 (0%)	0 (0%)	3 (2.97%)	3 (2.20%)	1 (1%)
Negative	12 (80%)	18 (90%)	79 (78.22%)	109 (80.14%)	64 (82%)

Abbreviations: NBS: newborn body surface, GBS: Group B streptococcus HER: Rabat children's hospital; *: Total of children recruited with clinical sepsis. #: At least one out of two newborn body surfaces positive

Table 4: Comparison of serotype distribution among pregnant women group vs. newborns with positive blood culture and newborns with positive blood culture vs. all newborns with any positive peripheral culture.

	Mothers*	Newborns with positive blood culture ł	p	Newborns with positive blood culture ł	Newborns with any positive peripheral culture$	p
Serotype	**N=67**	**N=5**		**N=5**	**N=14**	
Ia	6 (9%)	0 (0%)	0.639α	0 (0%)	0 (0%)	--
II	17 (25%)	2 (40%)	0.397α	2 (40%)	4 (29%)	0.520α
III	12 (18%)	1 (20%)	0.642α	1 (20%)	0 (0%)	0.263α
IV	5 (7%)	1 (20%)	0.361α	1 (20%)	4 (29%)	0.603α
V	24 (36%)	1 (20%)	0.428α	1 (20%)	6 (43%)	0.366α
IX	3 (5%)	0 (0%)	0.803α	0 (0%)	0 (0%)	--

*:Vagino-rectal or vaginal samples from colonised pregnant women which strains were available for molecular analysis; ł: Infants recruited at the neonatal intensive care unit with clinical sepsis and blood culture positive to Group B streptococcus $: Peripheral samples Group B streptococcus positive from asymptomatic infants born of pregnant women and peripheral samples Group B streptococcus positive from infants recruited in the neonatal intensive care unit which strains were available for molecular analysis. α: Fisher's exact test; A probability of <0.05 was considered statistically significant.