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Urinary Tract and Vaginal Infections caused by Group B Streptococcus and the Macrolide-Inducible Resistance to Clindamycin in Non-pregnant Females

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A B S T R A C T

An increasing incidence of Group B Streptococcus (GBS) infection in non-pregnant women has been noted in recent years and its early diagnosis remains difficult, since symptoms are very nonspecific, its frequency has not been completely investigated in urinary tract and vaginal infections in non-pregnant women. The aim of this study to investigate the role of GBS in urinary and vaginal infections in non-pregnant adults from outpatients Healthcare Center and their antimicrobial susceptibility profiles. Also, to assess the Macrolide-inducible resistance to Clindamycin. A total of 46 GBS isolates were investigated; 28 urine and 19 HVS samples. The samples were processed by wet smear, direct gram, culture, identification by catalase, serotyping and VITEK 2 Compact and finally the susceptibility and inducible Clindamycin resistance by VITEK 2 Compact. We detected 3.8% and 9.5% GBS in the urine and HVS samples respectively. Most of UTI samples were in October and November (25%, 21.4%) respectively, however HVS samples were highest in June (21%). Both UTI and vaginal infections were common in autumn (50%, 36.8%) respectively. We found that 75% of UTI samples had high colony count (>10^5/CFU) and low WBCs. We detected 13/15 (86.7%) urine samples and 10/13 (76.9%) HVS samples which were Erythromycin resistance and inducible clindamycin resistance positive with highly statistically difference (P=0.00). GBS was highly susceptible to Ampicillin, Benzyl Penicillin, Cefotaxime, Ceftriaxone, Linezolid, TMP/SMZ and Vancomycin. Levofloxacin showed good susceptibility (82.1%, 78.9%) for UTI and vaginal infections respectively. However Erythromycin and Clindamycin showed moderate resistance for urinary and vaginal infections (53.6%, 68.4%) for Erythromycin and (57.1%, 63.2%) for Clindamycin respectively. Tetracycline showed very high resistance rate (96.4%, 94.7%) for urinary and vaginal infections respectively. Finally, the present study concluded that we must do culture if the patient complaining of manifestation of UTI even in presence of low WBCs count (absence of pyuria). Physicians can start empirically with Penicillin or Ampicillin for GBS but in case of allergy to Penicillin, we must do the antibiotic susceptibility due to the high resistance rate to Clindamycin and Erythromycin nowadays.

Introduction

Streptococcus agalactiae, also known as Group B Streptococcus (GBS) is considered a common commensal of the female urogenital tract and rectum (Schuchat, 1999). Its importance is referred to severe neonatal pathologies by perinatal transmission from women to new-born (Lambiase *et al*., 2012). Bacterial colonization is seen in about 10% of non-pregnant women and 25% of pregnant individuals (Mandell *et al*., 2000).

The number of cases of invasive infection caused by S. agalactiae in non-pregnant adults is increasing (Huang *et al*., 2006; Matsubara and Yamamoto, 2009) especially in patients that had underlying medical conditions. The mortality rate range is 3-47% being highest in elderly patients with underlying medical conditions (Phares *et al*., 2008).

Group B Streptococcus is a causative agent of urinary tract infections (UTI). The spectrum of GBS UTI includes asymptomatic bacteriuria (ABU), cystitis, pyelonephritis, urethritis and urosepsis (Edwards and Baker, 2005; Mckenna *et al*., 2003).Clinically, UTI caused by GBS in many times may be indistinguishable from those caused by other organisms. However many studies suggest that microbiological and clinical features may be different depending upon the causative agent (Edwards *et al*., 2005; Bliss *et al*., 2002). Various rate of GBS UTI rate have been reported in pregnant and non-pregnant adults (Nolla *et al*., 2003). GBS UTI rate have been different from 1% to up to one third and even more in these studies (Falagas *et al*., 2006; Toumi *et al*., 2006).

Group B Streptococcus colonization of the urinary tract in women most likely occurs by an ascending route from the vagina, where GBS can persist asymptomatically. The overall prevalence of GBS UTI in the adult population remains unclear (Ulett *et al*., 2009).

Penicillin G and Ampicillin are the antibiotics of choice for intrapartum prophylaxis and treatment of invasive infections, while Clindamycin is the recommended agent for patients who are allergic to beta-lactams (Matteson *et al*., 2008). However, although *S. agalactiae* remains almost always susceptible to Penicillins, there is a significant and rising resistance to macrolides and lincosamides (Gygax *et al*., 2006).

Two major resistance mechanisms to macrolides and lincosamides have been reported: alteration of the antibiotic target site (encoded by erm genes) and active drug efflux pump (encoded b mef gene) (Domelier *et al*., 2008). Macrolides is usually mediated by the erm and mef genes, erm-mediated resistance includes macrolid-lincomycin-streptogramin b-inducible (iMLSB) and constitutive (cMLSb) phenotypes. Both MLSb phenotypes are E-resistant and clindamycin resistant (CC^R) but iMLSb requires induction to show CC^R, mef (M phenotype) are E^R and CC-susceptible (CC^S) (Gosnell *et al*., 2005).

There are only few and limited studies to determine the role of GBS in UTI and vaginal infections in non-pregnant adults. The present study was conducted to investigate the role of GBS in both infections in non-pregnant adults from outpatients Healthcare Center and their antimicrobial susceptibility profiles and to assess the Macrolide-inducible resistance to Clindamycin.

Materials and Methods

This study was conducted on female patients visited a primary healthcare out-patient

clinic in UAE. The first non-pregnant group (n= 28) complaining of at least one symptom of urinary tract infection that included dysuria, increased urinary frequency and/or urgency, fever of > 38^0C, flank and/or lumbar tenderness. The other non-pregnant group (n= 19) included patients complaining of vaginal infection in the form of discharge in the period from January 2015 to December 2015.

Morning mid-stream urine samples were collected in sterilized, leak-proofcontainers before the start of antibiotic. The samples were cultured on blood, MacConkey agar by calibrated loop(1ul) and incubated 24 hs in 37^0C. Urine was examined microscopically and chemically by iQ200 2nd generation Automated Urine Microscopy Analyzer-HVL (High volume laboratories) which can process 60 samples/h (Iris Diagnostic. Chatsworth, CA).

Iris iQ200 2nd generation

It is an in-vitro diagnostic use device composed of the iQ200 Automated Urine Microscopy Analyzer, connected physically and electronically to the AUTION MAX TM AX- 4280 Automated Urine Chemistry Analyzer and a workstation. It is a walk-away system that uses flow imaging analysis technology and Auto-Particle Recognition (APR, Iris Diagnostic) software to classify particles based on multiple parameters. Images are stored and can be viewed on the workstation screen, thereby eliminating the need for manual microscopy.

High vaginal swab (HVS) samples were cultured on blood, MacConkey, Chocolate agar in (5-10% CO_2) for 24 hs and Sabouraud' s agar for 48 hrs in 37^o C. Wet smears were examined microscopically for *Trichomonas*, WBCs, RBCs, epithelial cells or yeast. Direct film stained with Gram stain was examined for Clue cell, Gram Negative diplococci or yeast.

Identification of GBS was based on the recognition of beta-hemolytic colonies on sheep blood agar, Gram-positive cocci on Gram stain, absence of catalase production and latex agglutination with group-specific antiserum (BioMerieux).

Slidex Strepto plus

It is a latex agglutination test done for Streptococci bacteria of lancefield group A, B, C, D, F and G. It is latex particles conjugated to group specific antisera will bind to the corresponding antigen to result in visible clumping of the latex particles. Most beta hemolytic Streptococci possess group specific antigens that can be extracted and identified with group specific antisera.

After growth, the isolated colonies of *Streptococci* are removed and placed in a tube containing the extraction enzyme. The group specific antigen is enzymatically extracted from the streptococcal cell wall. Antigen in the extract is identified using latex particles sensitized with group specific anti-streptococcal antibody. Visible aggregates form in the specific latex particle suspension which reacts with the extracted antigen. The latex will remain in suspension if the antigen is not present in the extract.

Methods

For the preparation of the extract: transfer 0.4 ml of the extraction enzyme into test tube, then pick 3-5 typical colonies according to their size and emulsify them in the extraction enzyme.

Mix using a vortex-type mixer and incubate for ten min at 37^0C, then dispense 1 drop of each latex reagent into the corresponding

reaction fields (A,B,C,D,F and G). Then dispense 15 ml of the extract beside each together and spread over the whole surface of the field. Rotate the card gently for a maximum of 2 min and read under normal lighting.

A positive result is indicated by the development of an agglutination pattern showing clearly visible clumping of the latex particles in less than 2 min.

A negative result is indicated when agglutination doesn't occur. Homogenous suspension or very fine granular appearance.

The result is uninterruptible if agglutination is observed in several of the latex suspension. This may indicate a polyagglutinating strain or a mixture of strains. In this case, reperform the isolation and test.

After serotyping, we did Automated identification and susceptibility on Vitek 2 Compact (BioMerieux, France), machine used for fast (5-8 hs) and accurate microbial identification.

VITEK 2 compact machine

It includes an expanded identification database, andreads every 15 min for greater speed in identification.

It uses Advanced Colorimetry ™

We use Gram +ve identification and AST ST01 Streptococcus Susceptibility card; the panel includes: Ampicillin (AM), Benzylpenicillin (P), Cefotaxime (CTX), Ceftriaxone (CRO), Clindamycin (CM), Erythromycin (E), Levofloxacin (LEV), Linezolid (LNZ), Tetracycline (TE), Timethoprim/ Sulfamethoxazole (SXT) and Vancomycin (VA).

VITEK 2 compact test for inducible Clindamycin Resistance (ICR): for Strept. Pyogen and Strept. Aglactiae

Clindamycin (CM) 0.5 ug/ml, Clindamycin/Erythromycin 0.25/0.5 ug/ml.

This test is quantitative growth based detection algorithm using predetermined growth thresholds.

Statistical analysis

Data were analyzed using SPSS (Statistical Package for Social Science) version 19. Qualitative data was presented as number and percentage. Quantitative data was presented as mean and standard deviation. The Chi-square was used to compare between variables of qualitative data. The P value of< 0.05 indicates a significant difference while P value of < 0.001 indicates a highly significant difference.

Result and Discussion

Of the 745 urine samples collected from non-pregnant females complaining of UTI; 549 (73.7%) displayed no growth while 196 (26.3%) were giving growth; from which 28 (14.3%) were GBS, yielding a prevalence of 3.8% in total urine samples from non-pregnant females.

Of the 199 HVS samples from non-pregnant females complaining of vaginal infection; 122 (61.3%) were giving no isolated pathogenic organism and 77 (38.7%) showed pathogenic growth; from which 19 (24.7%) were GBS, yielding a prevalence of 9.5% in total HVS from non-pregnant females. There were four patients with the same vaginal and urinary infection with GBS infection.

Demographic data and microscopic examination of the urinary and vaginal samples were shown in table (1). A total number of 47 (28 urinary and 19 HVS) isolates of GBS were recovered out of 944 (4.98%) samples collected from female patients complaining of manifestations of infection. There was insignificant difference between both groups of UTI and HVS samples as regard age, WBCs, RBCs and pus (≥ 10 WBCs/HPF) counts.

Frequency of GBS in different months were shown in table (2). Most of UTI positive samples were detected in October and November (25%, 21.4%), while HVS positive samples were highest in June (21%).

Seasonal variation in each group was shown in table (3). Both UTI and vaginal infections were common in autumn (50%, 36.8%) but without statistically significant difference.

Table (4) showed the frequency of colony count of positive cultures in UTI with WBCs/HPF. We found 21/28 (75%) with high colony count and with low WBCs count (<10/HPF). However, we found only 7/28 (25%) with high colony count and high WBCs count (≥ 10/HPF) with statistically insignificant difference (P= 0.2).

We detected thirteen urine samples showing Erythromycin resistance(13/15) (86.7%) and inducible Clindamycin positive; and clindamycin become resistant with highly statistically difference (P= 0.000). However, ten HVS samples were Erythromycin resistant (10/13) (76.9%) and inducible clindamycin positive; clindamycin become resistant with highly statistically significant difference (P= 0.001).

Clindamycin resistant was detected in (3/16) (18.8%) urine samples and (2/12) (16.7%) HVS with negative inducible clindamycin with highly statistically significant difference (P= 0.000) (Table 5).

GBS was highly susceptible to Ampicillin, Benzyl Penicillin, Cefotaxime, Ceftriaxone, Linezolid, TMP/SMZ and Vancomycin. Levofloxacin showed good susceptibility for UTI (82.1%) and for vaginal infection (78.9%). However urinary and vaginal infections showed high resistance to Erythromycin (53.6%, 68.4%) and for Clindamycin (57.1%, 63.2%)respectively. The highest resistance was shown for Tetracycline (96.4%, 94.7%) respectively (Table 6).

*Streptococcus agalactiae*is generally known to cause invasive infection in pregnant women and neonates since it commonly colonizes the vaginal and gastrointestinal tracts of healthy women (Farley *et al*., 1993). However, infection in non-pregnant adults has been increasingly reported worldwide (Falagas *et al*., 2006; Huang *et al*., 2006; Matsubara and Yamamoto, 2009; Phares *et al*., 2008). This study showed that positive urine culture for GBS was found in 3.8% of all non-pregnant females subjects and 14.3% in females with positive culture.Shayanfar *et al*.,2012; Munoz *et al*., 1992;Ulett *et al*., 2009 reported lower prevalence of GBS (1.8%, 2%, 1.1%) respectively. Rahbar *et al*., 2012 was in agreement with our results who detected the frequency rate of GBS was 4.22% among female patients engaging UTI.

GBS was detected in 9.5% of the total HVS from non-pregnant females, Lambiase *et al*., 2012 detected higher prevalence 879/2156 (40.8%) from vaginal and recto-vaginal swabs that were obtained from pregnant and non-pregnant women, a 4-year study from 2005-2008.

The highest rates of cultures positive for GBS were seen in October and November

(25%, 21.4%) respectively for UTI and June (21%) for vaginal infection. Both types of infections were common in autumn. Shayanfar *et al*., 2012 found that the highest rates of culture positive for GBS were seen in December and January (11.4% for each). Chaiwarith *et al*., 2011 observed that the number of *Strept. aglactiae* was higher in September (autumn) and they did not know the explanation for this finding but there is controversy about the seasonal variation due to geographical distribution and variation in seasons.

The mean WBCs in urine samples in our study was 85.9±315 (ranging from 1-1600), most of the study subjects (75%) showed WBCs count <10/HPF and (25%) cases > 10/HPF. The mean WBCs reported by Shayanfar *et al*., 2012 was very low 6.6±10.1 (ranging from 0 to 50) but they were in agreement with our study that the majority of the studied subjects (83.7%) showed WBCs <10/HPF and only 16.3% with WBCs>10/HPF.

Our study showed that 18/28 (64.3%)of the patients had a colony count of > 10^5 and 10/28 (35.7%) had a colony count 10^4-10^5.This was in agreement with a study by Munoz *et al*., 1992, the GBS colony count > 10^5 in urine culture of 63% of the subjects, however Shayanfar *et al*.,2012 showed that only 10% of patients had a colony count of > 10^5. The cause of this difference could be attributed to the study population.

We detected 13/15 (86.7%) urine samples and 10/13 (76.9%) HVS which were Erythromycin resistant and inducible clindamycin resistance positive with highly statistically difference (P=0.00). Gosnell *et al*., 2005 found lower percentage than our results of Erythromycin resistance and inducible clindamycin resistance positive 37/64 (57.8%). Also, Browling *et al*., 2010 founded Erythromycin resistance and positive inducible clindamycin in 16/32 (50%) of GBS and in 46/100 (46%) of total Beta-hemolytic streptococcus isolates.

Erythromycin and Clindamycin in our study showed moderate resistance for urinary and vaginal infections (53.6%, 68.4%) for Erythromycin and (57.1%, 63.2%) for Clindamycin respectively and almost all Clindamycin resistant strains were also resistant to Erythromycin except one case. There are diversity of results, some studies showed lower resistance. Woods *et al*., 2015 found that 32% of GBS isolates were resistant to Erythromycin and 15% were resistant to Clindamycin and 99% of Clindamycin resistant strains were also resistant to Erythromycin. Also, Ulett *et al*., 2009 found resistance to Erythromycin (39.5%) and 26.4% for Clindamycin.

Florindo *et al*., 2014 studied the increasing rate of resistance from 2006 to 2012, GBS isolates that were resistant to Erythromycin ranged from 14% in 2006 to 23% in 2011, whereas the % of GBS isolates with resistance to Clindamycin ranged from 6% in 2009 to 18% in 2012.Rahbar *et al*., 2012 were in agreement with our results, they reported resistance rate to Erythromycin and Clindamycin (70.5%, 66.1%) respectively to GBS. Other studies showed higher resistance rate, Lambiase *et al*., found that the occurrence of Macrolide and Clindamycin resistance was 16.5% in 2005 increasing up to 69.9% in 2008. Also, Tazi *et al*., 2007 reported 73.6% resistance to Erythromycin.

In our study, Tetracycline showed very high resistance rate (96.4%, 94.7%) respectively for urinary and vaginal infections.Mostly all the studies showed the high resistance rate to Tetracycline (Lambiase *et al*, 2012; Florindo *et al*., 2014; Tazi *et al*., 2007). All

GBS resistant to Erthromycin, also resistant to Tetracycline which could be expected considering a putative horizontal gene transfer event involving the same conjugative transposon carrying both genetic resistance determinants (Gherardi *et al*., 2007).There are two main mechanisms of resistance to Tetracycline in Streptococci; efflux by proton antiporters and ribosome protection (Roberts, 2005).

Table.1 Demographic data and microscopic examination of the studied samples (n=47). Values are number (%) or mean ± SD (range).

	Urine (n=28)	**HVS (n=19)**	**P- value**	**95 % confidence interval**	
				Lower	**Upper**
Age (y)	39.25 ± 11.3 (20-63)	38.9± 9.9 (23-65)	0.9	-6.1	6.8
WBC/HPF	85.96 ± 315 (1-1600)	15.36 ±28.7 (1-100)	0.3	-75.8	217
RBCs/HPF	5.89 ±14.4 (1-77)	1.63 ± 0.76 (1-3)	0.2	-2.4	10.9
PUS : High Low	 7 (25 %) 21 (75 %)	 6(31.6 %) 13 (68.4%)	0.4	- 0.21	0.29

HVS: High vaginal swab RBCs: Red blood cells
HPF: High power field WBCs: White blood cells

Table.2 Frequency of group B Streptococci in different months (n=47).

Month	Urine sample (n=28)		HVS (n=19)	
	N	%	N	%
January	0	0	1	5.3
February	2	7.1	1	5.3
March	1	3.6	2	10.5
April	1	3.6	2	10.5
June	1	3.6	4	21.0
July	3	10.7	0	0
August	2	7.1	1	5.3
September	1	3.6	1	5.3
October	7	25	3	15.8
November	6	21.4	3	15.8
December	4	14.3	1	5.3
Total	28	100	19	100

HVS: High vaginal swab

Table.3 Seasonal variation in each group of UTI and vaginal infections.

	Urine sample (n=28)		HVS (n=19)		P-value
	N	%	N	%	
Winter	6	21.4	3	15.8	0.5
Spring	2	7.0	4	21.1	
Summer	6	21.4	5	26.3	
Autumn	14	50	7	36.8	
Total	28	100	19	100	

HVS: High vaginal swab

Table.4 Frequency of Colony count in UTI with WBCs /HPF (n=28).

	Colony count		p-value
	$10^4 - 10^5$	$>10^5$	
Low WBCs (< 10/HPF) (n=21)	9 (42.9)	12 (57.1)	0.4
High WBCs (≥10/HPF) (n=7)	1 (14.3)	6 (85.7)	
Total (n=28)	10 (35.7)	18 (64.3)	

Table.5 Relation of inducible clindamycin resistance, Erythromycin and Clindamycin susceptibility among GBS (n=47). Values are number (%).

Inducible Clindamycin	Erythromycin Susceptibility			P	ClindamycinSusceptibility		P
	S	R	I		S	R	
Urine samples							
Negative (15)	11(73.4)	2 (13.3)	2 (13.3)	0.000	12(80)	3(20)	0.000
Positive (13)	0 (0)	13 (100)	0 (0)		0(0)	13(100)	
Total (28)	11 (39.3)	15 (53.6)	2 (7.1)		12 (42.9)	16(57.1)	
HVS							
Negative (9)	6 (66.7)	3 (33.3)	0 (0)	0.003	7 (77.8)	2 (22.2)	0.001
Positive (10)	0 (0)	10 (100)	0 (0)		0.0	10 (100)	
Total (19)	6 (31.6)	13 (68.4)	0 (0)		7 (36.8)	12 (63.2)	

HVS: High vaginal swab

Table.6 Antimicrobial susceptibility pattern of GBS (n=42). Values are numbers (%).

	Urine samples (n=28)			HVS (n=19)		
	S	R	I	S	R	I
Ampicillin	28 (100)	0	0	19 (100)	0	0
Benzyl penicillin	28 (100)	0	0	19 (100)	0	0
Cefotaxime	28 (100	0	0	19 (100)	0	0
Ceftriaxone	28 (100)	0	0	19 (100)	0	0
Clindamycin	12 (42.9)	16 (57.1)	0	17 (36.8)	12 (63.2)	0
Erythromycin	11 (39.3)	15 (53.6)	2 (7.1)	6 (31.6)	13 (68.4)	0
Levofloxacin	23 (82.1)	5 (17.9)	0	15 (78.9)	4(21.1)	0
Linezolid	28 (100)	0	0	19 (100)	0	0
Tetracycline	1 (3.6)	27 (96.4)	0	1(5.3)	18 (94.7)	0
TMP/SMZ	27 (96.4)	1 (3.6)	0	19 (100)	0	0
Vancomycin	28 (100)	0	0	19 (100)	0	0

S=sensitive R=Resistant I=Intermediate
TMP/SMZ:Trimethoprim/Sulfamethoxazole
HVS: High vaginal swab

In present study, GBS was highly susceptible to Ampicillin, Penicillin, Cefotaxime, Ceftriaxone, Linezolid, TMP/SMZ and Vancomycin.Woods, 2015 reported that GBS are uniformly sensitive to Penicillin and Ampicillin. Although resistance to Penicillin or Ampicillin has not be documented, some isolated have shown MIC approaching the upper limits of susceptibility for some of the Beta-Lactam agents (Phares *et al*., 2008).

Many studies reported high rate of susceptibility to Penicillin G (Ko*et al*., 2001; Blancas *et al*., 2004; Huang *et al*., 2006). Shayanfar *et al*., 2012 showed that GBS was sensitive to Cephalothin, Norfloxacin, Ampicillin, Nitrofurantoin, Vancomycin, therefore treatment with Ampicillin or Cephalothin was recommended. Also, Chaiwarith *et al*., 2011, founded that all isolates of GBS were susceptible to Penicillin, Ampicillin and Vancomycin. However, Rahbar *et al*., 2012 showed high resistance to Penicillin, Ampicillin, Ceftriaxone, Cefotaxime (89.4%, 52.5%, 43,8%, 46.6%). Also, in a study performed at Taiwan founded susceptibility to Penicillin 60.7% and 100% for Vancomycin (Fu *et al*., 2004).

As it is obvious from the results of these above reports, antibiotic resistance rates showed different pattern so, it indicates that performance of antibiotic susceptibility test is necessary before any prescriptions (Rahbar *et al*., 2012).

From this study we concluded that we must do culture if the patient complaining of manifestation of UTI even in presence of low WBCs count (absence of pyuria). Physicians can start empirically with Penicillin or Ampicillin for GBS but the

problem in allergic patients to Penicillin so, we must do the antibiotic susceptibility due to the high resistance rate to Clindamycin and Erythromycin nowadays.

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