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• 临床研究 •

孕晚期孕妇B族链球菌感染情况及不良妊娠结局分析

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【摘要】 目的 研究孕晚期孕妇B族链球菌(GBS)病原学特点和对妊娠结局的影响。方法 收集400例于本院就诊并携带GBS孕晚期孕妇资料作为研究对象,并收集400例健康孕晚期孕妇对照组。收集标本并采用触媒试验和微生物鉴定仪进行菌株鉴定。采用K-B纸片法测试GBS耐药情况。回顾性分析孕晚期孕妇GBS易感因素和不良妊娠结局。

结果 GBS对红霉素、克林霉素、四环素、阿奇霉素、诺氟沙星和环丙沙星耐药率分别为84.95%、78.76%、71.24%、82.53%、58.06%和51.88%。GBS感染组年龄 27.12 ± 3.61 岁,对照组 27.37 ± 3.93 岁。GBS感染组BMI $27.67 \pm 2.51 \text{ kg/m}^2$,对照组 $26.91 \pm 2.18 \text{ kg/m}^2$ 。GBS感染组患有贫血、高血压和糖尿病,以及职业不稳定人员和有流产史构成比分别为17.75%、4.50%、15.50%、49.25%和9.00%;对照组分别为12.50%、4.00%、10.75%、42.25%和2.50%。GBS感染组阴道pH值<4.4,患有滴虫、真菌、线索细胞感染和阴道与宫颈炎症构成比分别为24.00%、2.75%、14.50%、12.75%和11.50%;对照组分别为54.25%、0.75%、8.75%、8.00%和3.00%。GBS感染组和对照组年龄和是否患有高血压差异无统计学意义($P > 0.05$),而两组患者BMI、患有贫血、糖尿病、滴虫、真菌、线索细胞感染和阴道与宫颈炎症,以及职业不稳定人员和有流产史的构成比差异有统计学意义($P < 0.05$)。GBS感染组的胎膜早破、羊膜炎、胎儿窘迫、产后出血和产后感染发生率分别为20.75%、9.25%、10.50%、8.75%和16.00%,对照组分别为2.75%、1.25%、4.00%、2.25%和0.75%。两组患者胎膜早破、羊膜炎、胎儿窘迫、产后出血和产后感染发生率据差异有统计学意义($P < 0.05$)。GBS感染组的新生儿肺炎、病理性黄疸、新生儿脓血症和新生儿窒息的发生率分别为20.75%、9.25%、10.50%、8.75%和16.00%,对照组分别为3.75%、0.00%、0.00%和0.00%。两组患者新生儿肺炎、病理性黄疸和新生儿脓血症发生率差异有统计学意义($P < 0.05$),而新生儿窒息发生率差异无统计学意义($P > 0.05$)。结论 GBS对大环内酯类、喹诺酮类抗生素产生了一定的耐受性,对青霉素等抗生素仍敏感。肥胖、糖尿病、贫血、有流产史、工作不稳定和阴道微生态差的患者易患GBS感染,发生GBS感染的孕妇易发生不良妊娠结局。

【关键词】 孕妇;B族链球菌;耐药;妊娠结局

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Analysis of Group B Streptococcus infection and adverse pregnancy outcomes in late pregnant women

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【Abstract】 **Objective** To study the pathogenic characteristics of Group B *Streptococcus* (GBS) in late pregnancy and its impact on pregnancy outcomes. **Methods** The data were collected from 400 pregnant women who visited our hospital and carried GBS for late pregnancy as research subjects, and collect 400 healthy late pregnancy pregnant women as a control group. The specimens were collected and strains were identified by catalyst tests and microbial identification instruments. The resistance of GBS were tested by K-B paper method. The GBS susceptibility factors and adverse pregnancy outcomes in late pregnancy women were analyzed. **Results** The resistance rates of GBS to erythromycin, clindamycin, tetracycline, azithromycin, norfloxacin, and ciprofloxacin were 84.95%, 78.76%, 71.24%, 82.53%, 58.06%, and 51.88%, respectively. The age of GBS infection group was 27.12 ± 3.61 years old, while the control group was 27.37 ± 3.93 years old. The BMI of the GBS infection group was $27.67 \pm 2.51 \text{ kg/m}^2$, while the control group was $26.91 \pm 2.18 \text{ kg/m}^2$. The proportions of people with anemia, hypertension, diabetes, occupational instability and abortion history in the GBS infection group were 17.75%, 4.50%, 15.50%, 49.25% and 9.00%, respectively; The control groups were 12.50%, 4.00%, 10.75%, 42.25%, and 2.50%, respectively. The proportions of the vaginal pH value less than 4.4, trichomonads, fungi, clue cells, and vaginal and cervical inflammation in the GBS infection group were 24.00%, 2.75%, 14.50%, 12.75%, and 11.50%, respectively. The control groups were 54.25%, 0.75%, 8.75%, 8.00%, and 3.00%, respectively. The age and hypertension of GBS infection group and control group were not statistically significant ($P > 0.05$).

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0.05), while the data of BMI, anemia, diabetes, trichomonas, fungi, clue cell infection, vaginitis and cervicitis, occupational instability and abortion history of the two groups were statistically significant ($P < 0.05$). The incidences of premature rupture of membranes, amniotic, fetal distress, postpartum hemorrhage, and postpartum infection in the GBS infection group were 20.75%, 9.25%, 10.50%, 8.75%, and 16.00%, respectively, while those in the control group were 2.75%, 1.25%, 4.00%, 2.25%, and 0.75%, respectively. There were statistically significant differences in the incidences of premature rupture of membranes, amniotic, fetal distress, postpartum hemorrhage, and postpartum infection between the two groups of patients ($P < 0.05$). The incidences of neonatal pneumonia, pathological jaundice, neonatal sepsis, and neonatal asphyxia in the GBS infection group were 20.75%, 9.25%, 10.50%, 8.75%, and 16.00%, respectively, while those in the control group were 3.75%, 0.00%, 0.00%, and 0.00%, respectively. There were statistically significant differences in the incidence of neonatal pneumonia, pathological jaundice, and neonatal sepsis between the two groups of patients ($P < 0.05$), while the incidence of neonatal asphyxia was not statistically significant ($P > 0.05$). **Conclusion** GBS had a certain degree of tolerance to macrolides and quinolones, but remains sensitive to penicillins and other antibiotics. Patients with obesity, diabetes, anemia, history of abortion, unstable work and poor vaginal microflora were prone to GBS infection. Pregnant women with GBS infection were prone to adverse pregnancy outcomes.

【Key words】 Pregnant women; Group B *Streptococcus* (GBS); drug resistance; pregnancy outcomes

B族链球菌(Group B *Streptococcus*, GBS)又可称为无乳链球菌,属 β 溶血革兰阳性菌。它成对或链状排列,是条件致病菌,可定植于人类生殖道和消化道等部位^[1]。由于女性生殖道生理结构特点易发生感染,GBS是寄生在女性生殖道及肛门周围的常见条件致病菌,严重威胁着孕妇和新生儿健康^[2]。Russell一份关于综合世界范围内85个国家的报道显示孕妇GBS定植率约为18.0%^[3]。我国学者对2000-2018年国内的相关报告显示孕妇GBS定植率约为11.3%^[4]。GBS对绒毛膜具有较强的吸附能力和穿透能力,常引起胎膜早破、早产和羊水污染等不良妊娠结局^[5]。孕妇感染GBS是新生儿感染GBS的高风险因素,有研究显示,孕妇GBS感染后,若未进行干预治疗,约有50%会引发新生儿感染,可引发新生儿脑膜炎、肺炎和脓毒症等疾病^[6-8]。GBS已成为公认的威胁新生儿生命的致病菌之一。因而,早期诊断和干预治疗能够有效的减少不良妊娠结局发生。美国于1996年发布了《围产期GBS感染筛查和预防指南》^[9]和2019年发布了《预防新生儿早发型GBS病委会共识》^[10],欧洲国家也于2014年制定了《产时B族链球菌筛查和预防欧洲共识》^[11],我国则在2011年和2021年分别发布了《孕前和孕期保健指南》和《预防围产期B族链球菌病(中国)专家共识》。其中《预防围产期B族链球菌病(中国)专家共识》建议将GBS检查列入了孕妇常规筛查项目,最佳检测时间在孕35-37周。由于不同地区GBS定植率存在一定差异,本次研究分析本院就诊孕晚期孕妇GBS感染情况和危险因素及不良结局。

材料与方法

1 材料

1.1 一般资料 选取2021年1月至2022年12月本

院就诊并于孕35~37周进行GBS筛查并进行分娩的400例发生B组链球菌感染孕妇为研究对象。选取同期400例健康产妇作为对照组。纳入标准:1)自然受孕,单胎妊娠;2)筛查前两周无性交史;3)筛查前两周未进行抗生素治疗和阴道栓剂。排除标准:1)有吸烟、喝酒等不良嗜好;2)存在其他严重疾病;3)档案资料不齐全。

本次研究均向患者说明并签署知情书。

1.2 主要仪器与试剂 VITEK® 2COMPACT 30/60全自动微生物鉴定仪,法国梅里埃(bioMerieux);恒温培养箱,德国Eppendorf公司;YA0180麦氏比浊管,北京索莱宝科技有限公司;低温冰箱,日本三洋。培养基,英国OXOID公司。

2 方法

2.1 标本采集 对孕35~37周就诊患者进行采用,采样前对外阴进行擦拭和消毒,采用棉拭子在阴道1/3处轻轻旋转取样。然后再用棉拭子于肛门括约肌上2~3cm处沿肠壁旋转取样。将采集到的标本接种于5%血琼脂培养基上,于35℃,浓度5%CO₂培养箱中培养20~24h。检出菌落做进一步鉴定。

2.2 细菌鉴定 选取直径约1mm呈半透明和产生 β -溶血菌落进行革兰染色。选取革兰染色初步鉴定为链球菌的菌株进行触媒试验,结果阴性为链球菌。采用微生物鉴定仪进行菌株鉴定。

2.3 药敏试验 采用K-B纸片法测定GBS对青霉素、红霉素、氨苄西林、克林霉素、头孢唑啉钠、头孢曲松、庆大霉素、阿米卡星、阿奇霉素、诺氟沙星、环丙沙星、万古霉素和利奈唑胺耐药情况。

2.4 资料分析 回顾性分析孕妇年龄、孕前BMI、孕次、产次、学历、是否作息规律、妊娠期性生活、糖尿病、高血压、贫血、阴道滴虫和念球菌感染等因素对GBS

感染的影响。分析胎膜早破、羊水污染和产后感染等不良因素以及新生儿败血症、新生儿肺炎等疾病与GBS感染之间的关系。

3 统计分析

本次研究数据采用SPSS 25进行统计分析,学历、是否作息规律、糖尿病、高血压、贫血、阴道滴虫、念球菌感染、胎膜早破、羊水污染和产后感染等计数资料采用例(%)表示,组间对比采用 χ^2 检验,对年龄、孕前BMI、孕次和产次等计量资料采用“ $\bar{x} \pm s$ ”表示,组间对比采用t检验,以 $P < 0.05$ 为差异有统计学意义。

结 果

1 GBS 耐药情况

本次研究共分离400株GBS菌株,对红霉素、克林霉素、四环素、阿奇霉素、诺氟沙星、环丙沙星产生了耐药,耐药株数分别为316、293、265、307、216和193株,耐药率分别为84.95%、78.76%、71.24%、82.53%、58.06%和51.88%。GBS未对青霉素、氨苄西林、头孢唑啉钠、头孢曲松、庆大霉素、阿米卡星、万古霉素和利奈唑胺产生耐受性(表1)。

表1 GBS对部分常用抗生素耐药情况

Table 1 Resistance of GBS to some commonly used antibiotics

抗生素 Drugs	耐药 Resistance		中介 Intermediary		敏感 Sensitive	
	株数 No.	率(%) Rates	株数 No.	率(%) Rates	株数 No.	率(%) Rates
青霉素	0	0.00	0	0.00	372	100.00
红霉素	316	84.95	11	2.96	45	12.10
氨苄西林	0	0.00	0	0.00	372	100.00
克林霉素	293	78.76	8	2.15	71	19.09
四环素	265	71.24	5	1.34	102	27.42
头孢唑啉钠	0	0.00	0	0.00	372	100.00
头孢曲松	0	0.00	0	0.00	372	100.00
庆大霉素	0	0.00	0	0.00	372	100.00
阿米卡星	0	0.00	0	0.00	372	100.00
阿奇霉素	307	82.53	6	1.61	59	15.86
诺氟沙星	216	58.06	9	2.42	147	39.52
环丙沙星	193	51.88	8	2.15	171	45.97
万古霉素	0	0.00	0	0.00	372	100.00
利奈唑胺	0	0.00	0	0.00	372	100.00

2 GBS 感染危险因素分析

GBS感染组年龄 27.12 ± 3.61 岁,对照组 27.37 ± 3.93 岁,两组患者年龄差异无统计学意义($t = 0.956, P > 0.05$)。GBS感染组BMI 27.67 ± 2.51 kg/m²,对照组 26.91 ± 2.18 kg/m²,两组患者BMI差异有统计学意义($t = -4.537, P < 0.05$)。GBS感染组贫血患者71例,占17.75%;对照组贫血患者50例,占12.50%。GBS感染组高血压患者18例,占4.50%;对照组高血压患者16例,占4.00%。GBS感染组糖尿病患者62例,占15.50%;对照组糖尿病患

者43例,占10.75%。GBS感染组职业不稳定患者197例,占49.25%;对照组职业不稳定患者169例,占42.25%。GBS感染组有流产史患者36例,占9.00%;对照组贫血患者10例,占2.50%。两组患者一般资料对比显示GBS感染组贫血、糖尿病、有流产史和职业不稳定的患者的比例显著高于对照组($P < 0.05$)。两组患者患高血压的比例差异无统计学意义($P > 0.05$)(表2和3)。

表2 两组孕妇一般资料比较
Table 2 Comparison of general information between two groups of pregnant women

因素 Factors	GBS感染组 (n=400)		对照组 (n=400)		χ^2	P		
	GBS infection group		Control group					
	例数 No.	率(%) Ratio	例数 No.	率(%) Ratio				
贫血	71	17.75	50	12.50	4.2941	0.0382		
高血压	18	4.50	16	4.00	0.1229	0.7259		
糖尿病	62	15.50	43	10.75	3.9575	0.0467		
职业不稳定	197	49.25	169	42.25	3.9485	0.0469		
流产史	36	9.00	10	2.50	15.5922	0.0001		

表3 两组孕妇阴道微生物比较
Table 3 Comparison of vaginal microbiota between two groups of pregnant women

因素 Factors	GBS感染组 (n=400)		对照组 (n=400)		χ^2	P		
	GBS infection group		Control group					
	例数 No.	率(%) Ratio	例数 No.	率(%) Ratio				
pH值<4.4	96	24.00	217	54.25	76.84001	0.0000		
滴虫	11	2.75	3	0.75	4.652854	0.0310		
真菌	58	14.50	35	8.75	6.436404	0.0112		
线索细胞	51	12.75	32	8.00	4.852884	0.0276		
阴道与宫颈炎症	46	11.50	12	3.00	21.48899	0.0000		

3 两组患者不同妊娠结局比较

GBS感染组胎膜早破患者83例,占20.75%;对照组胎膜早破患者11例,占2.75%。GBS感染组羊膜炎患者37例,占9.25%;对照组羊膜炎患者5例,占1.25%。GBS感染组胎儿窘迫患者42例,占10.50%;对照组胎儿窘迫患者16例,占4.00%。GBS感染组产后出血患者35例,占8.75%;对照组产后出血患者64例,占16.00%。GBS感染组产后感染患者64例,占16%;对照组产后感染患者3例,占0.75%。两组患者妊娠结局对比显示胎膜早破、羊膜炎、胎儿窘迫、产后出血和产后感染的发生率差异有统计学意义($P < 0.05$)(表4)。

4 新生儿并发症比较

GBS感染组新生儿肺炎29例,发生率7.25%;对照组新生儿肺炎15例,发生率3.75%。GBS感染组病理性黄疸17例,发生率4.25%;对照组病理性黄疸0例。GBS感染组新生儿脓血症4例,发生率

1.00%；对照组新生儿脓血症0例。GBS感染组新生儿窒息3例，发生率0.75%；对照组新生儿脓血症0例。两组患者新生儿并发症比较结果显示，新生儿肺炎、病理性黄疸和新生儿脓血症的发生率差异有统计学意义($P<0.05$)，而新生儿窒息的发生率差异无统计学意义($P>0.05$)（表5）。

表4 两组孕妇妊娠结局比较

Table 4 Comparison of pregnancy outcomes between two groups of pregnant women

因素 Factors	GBS感染组 (n=400)		对照组 (n=400)		χ^2	P		
	GBS infection group		Control group					
	例数 No.	率(%) Ratio	例数 No.	率(%) Ratio				
胎膜早破	83	20.75	11	2.75	62.4917	0.0000		
羊膜炎	37	9.25	5	1.25	25.7319	0.0310		
胎儿窘迫	42	10.50	16	4.00	12.5662	0.0112		
产后出血	35	8.75	9	2.25	16.2578	0.0276		
产后感染	64	16.00	3	0.75	60.6137	0.0000		

表5 新生儿并发症比较
Table 5 Comparison of neonatal complications

因素 Factors	GBS感染组 (n=400)		对照组 (n=400)		χ^2	P		
	GBS infection group		Control group					
	例数 No.	率(%) Ratio	例数 No.	率(%) Ratio				
新生儿肺炎	29	7.25	15	3.75	4.7138	0.0299		
病理性黄疸	17	4.25	0	0.00	17.3691	0.0000		
新生儿脓血症	4	1.00	0	0.00	4.0201	0.0450		
新生儿窒息	3	0.75	0	0.00	3.0113	0.0827		

讨 论

GBS为革兰阳性球菌，于1938年首次被发现。链球菌细胞壁有C物质(细胞壁C多糖物质)和S物质(GBS表面荚膜多糖,capsular poly saccharide,cps)。依据C物质不同,链球菌可分为18个族^[12]。GBS细胞壁中的多糖物质属于抗原结构分类中的B族。依据C物质不同,GBS目前已被发现的血清型约有10种^[13]。GBS是定植于女性阴道和直肠的常见病原菌,属于条件致病菌。孕期妇女免疫力较差,阴道微生态失衡,易发生GBS感染。孕期妇女GBS的定植率具有地域性,欧美国家GBS感染率较高,亚洲感染率较低^[14-16]。国内不同报道也存在一定差异性,李芳等^[5]报道中,宁夏地区妊娠晚期孕妇GBS携带率为14.73%。陈敏红等^[16]报道中,温岭市妊娠晚期孕妇GBS检出率约5.97%。

采用抗生素治疗时,GBS对临床常用抗生素耐受性是一个不容忽视的问题。青霉素是治疗GBS的一线药物,它对GBS具有良好的治疗效果。当患者有青霉素过敏史,且病原菌对克林霉素诱导耐药试验成阴性时可以考虑采用克林霉素治疗。近年来有报道显示

GBS有对青霉素升高的趋势,对四环素、红霉素类抗生素具有较高的耐受性^[17-19]。中国细菌耐药监测研究2019-2020年革兰阳性菌监测报告中GBS对青霉素、红霉素、阿奇霉素、克林霉素、环丙沙星和左氧氟沙星耐药率依次为4.8%、82.3%、84.4%、72.8%、65.3%和65.3%^[20]。2021-2022年GBS对青霉素、红霉素、阿奇霉素、克林霉素、莫西沙星和左氧氟沙星耐药率依次为0.0%、82.9%、82.9%、63.8%、47.6%和47.6%^[21]。2021-2022年比2019-2020年,GBS对临床常用抗生素耐药率略有下降。本次研究中GBS未对青霉素等抗生素产生耐药性,而对大环内酯类和喹诺酮类产生了一定的耐药性。GBS对青霉素产生耐受性机制尚不明确,这可能与青霉素结合蛋白(PBPs)中的部分氨基酸突变有关。GBS对大环内酯类抗生素耐药主要有主动外排机制(mef)、核糖体靶位改变(erm)和灭活酶的产生。喹诺酮类抗生素耐药则是作用于细菌DNA促旋酶和拓扑异构酶IV,GBS中喹诺酮类耐药决定区(quinolone resistance determining region,QRDR)的部分氨基酸突变有关。

妊娠期体内微生态环境发生变化,高糖环境则易于病原菌生长,孕期肥胖患者其阴道微生态失衡、贫血患者往往体质虚弱,因而肥胖、并发糖尿病和贫血是GBS的易感因素^[22-23]。阴道微生态环境较差如有滴虫、真菌感染、阴道与宫颈炎症的患者更易感染GBS。GBS上行感染会降低胎膜阻力,它对绒毛膜有较强吸附能力和穿透能力,它通过对炎症细胞吞噬作用和产生的蛋白水解酶破坏胎膜,可引起胎膜早破。胎膜早破后,外界细菌进入,易发宫腔感染、产后感染和新生儿感染。既往研究显示孕妇妊娠期发生GBS感染的新生儿发生GBS感染率显著高于未感染孕妇的新生儿,从而诱发新生儿肺炎、新生儿脓血症和病理性黄疸。本次研究中GBS感染组患者胎膜早破、羊膜炎、胎儿窘迫、产后出血和产后感染均高于对照组,其新生儿肺炎、病理性黄疸和脓血症发病率高于对照组。对妊娠期GBS感染的早期诊断和干预有着重要意义。

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