Group B streptococci — gastrointestinal organisms?

CSF EASMON, A TANNA, P MUNDAY, * S DAWSON†

From the Departments of Bacteriology and †Genito-urinary Medicine, St Mary's Hospital and Medical School, London W2 1PG and *Division of Communicable Diseases, Clinical Research Centre, Northwick Park Hospital, Watford Road, Harrow, Middx HA1 3UJ

SUMMARY Matched perianal swabs, rectal swabs, and faecal samples from a group of male homosexual patients attending a clinic for sexually transmitted disease were examined for the presence of group B streptococci (GBS). GBS recovery rates were as follows: perianal skin 31/115 (27%), rectal mucosa 18/72 (25%) and faeces 7/115 (6%). The recovery of GBS from faeces was similar to that obtained from faecal samples sent to the laboratory for routine investigation (5%). Although there was no difference in GBS recovery rates from rectal and perianal swabs, the latter did show heavier colonisation. These results suggest that gastrointestinal GBS carriage is mainly limited to the rectum and anal canal and that this may represent contamination from perianal skin.

Group B streptococci (GBS), together with Escherichia coli are the most important bacterial pathogens in the neonatal period. In “early onset” GBS disease the organism is usually acquired from the mother’s genital tract at birth.1 Both Badri et al.2 and Franciosi et al.3 found that rectal GBS carriage rates were consistently higher than the corresponding vaginal carriage rates. The two groups, however, differed in their interpretation of these findings. Franciosi et al.3 suggested that the isolation of GBS from rectal cultures was the result of perianal skin contamination from the primary site of carriage in the vagina, while Badri et al.2 thought that this represented gastrointestinal carriage. More recently, Islam and Thomas4 found a much lower GBS carriage rate in faecal samples than in rectal samples, although the numbers of the latter were small. They suggested that the perianal skin rather than the gastro-intestinal tract was the primary site of GBS carriage.

In our study we have not only examined routine faecal samples for GBS carriage but also have compared GBS carriage in matched faecal samples, rectal mucosal swabs, and perianal swabs from a group of homosexual patients attending a clinic for sexually transmitted diseases.

Material and methods

Faecal specimens submitted to the laboratory for routine examination were included in this study. The remaining samples were taken from male homosexual patients admitting to passive sexual intercourse who attended the Praed Street Clinic for sexually transmitted diseases. Some of these patients had symptoms of proctitis. Swabs were taken from the skin around the anal margin and from the rectal mucosa using a proctoscope. Swabs from the rectal mucosa were not taken from the first 50 patients in the survey. Where possible faecal samples were taken directly through the proctoscope; otherwise they were obtained as soon as possible after the rectal and perianal swabs had been taken. Ethical Committee consent was obtained for the study of asymptomatic clinic patients.

The swabs were plated directly on to Islam’s starch serum agar plates5 containing nalidixic acid (15 mg/l) and gentamicin (4 mg/l) which were then incubated overnight anaerobically at 37°C using the Gaspak system (BBL). GBS produced an orange yellow pigment when grown anaerobically on this medium.6 Swabs and faecal samples were cultured in Todd-Hewitt broth (BBL) supplemented with 5% defibrinated sheep blood and with gentamicin and nalidixic acid as above6 and after overnight incubation at 37°C, subcultured on to Islam’s agar without antibiotics. Pigmneted colonies were serogrouped by coagglutination with the Phadebact system (Pharmacia).

Results

The Table shows that there was no difference between the rate of recovery of GBS from the routine faecal samples and those from the clinic. The GBS colonisation rates from patients who provided samples from all three sites were as follows: rectal mucosa at 24% and perianal skin at 25% were similar while both
Group B streptococcus isolation rates from faeces, perianal, and rectal swabs

<table>
<thead>
<tr>
<th>Specimen</th>
<th>No positive/No examined (%)</th>
<th>Routine samples</th>
<th>Clinic samples from</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Those giving faecal samples</td>
<td>All patients</td>
</tr>
<tr>
<td>Faece</td>
<td>13/245 (5)</td>
<td>7/115 (6)</td>
<td>7/115 (6)</td>
</tr>
<tr>
<td>Rectal swab</td>
<td>Not done</td>
<td>18/72 (25)</td>
<td>24/105 (23)</td>
</tr>
<tr>
<td>Perianal swab</td>
<td>Not done</td>
<td>31/115 (27)</td>
<td>41/157 (26)</td>
</tr>
</tbody>
</table>

were very much higher than the 6% found in faeces. When the figures for all patients who provided rectal or perianal samples or both were included, there was little change in rectal (23%) or perianal (26%) recovery rates.

Of the seven subjects with positive faecal samples, three were negative at both other sites. Of the 106 patients from whom both rectal and perianal swabs were obtained, 35 were positive at one or other site. Of these, 17 were positive in both sites, 11 were perianal-positive and rectal mucosa-negative, while seven were rectal mucosa-positive and perianal-negative. Twenty-two of the 41 (54%) perianal GBS-positive samples were positive on direct plating, compared with only six of the 24 (25%) positives from the rectal mucosa. In three of the 17 patients in whom GBS were recovered from both sites, the perianal swabs were positive on direct culture while the rectal swabs were only positive after broth enrichment.

Discussion

Although the Praed Street Clinic population is highly selected, the faecal GBS recovery rate of 6% did not differ from that in routine clinical samples (5%). The latter, of course, mainly coming from patients with gastrointestinal symptoms cannot necessarily be regarded as “normal.” The Clinic patients, being homosexual, might also be expected to have a higher than average rectal GBS carriage rate, given the reported high incidence of urethral carriage of GBS7 and the possible importance of sexual transmission.

In fact the GBS carriage rate of 25% seen in rectal samples were very similar to the rate (21%) which we have observed, using the same methods, in a concurrent study of over 1000 women attending antenatal clinics.

GBS carriage in the female genital tract is directly relevant to early onset GBS infection in the neonate, but the primary site of carriage of this organism has been a matter of some controversy. The observations that vaginal carriage was not consistent, and that rectal carriage rates tended to be higher than vaginal rates, combined with the fact that in some studies antibiotic treatment had little long term effect on GBS carriage in the vagina, led to the suggestion that the gastrointestinal tract was in fact the primary site of GBS carriage.

Unfortunately there have been very few detailed studies of GBS in the gastrointestinal tract. Unsworth10 used Islam’s medium with neomycin, nalidixic acid and metronidazole to isolate GBS from faeces. The number of specimens examined was small, two out of the 12 being positive. In one of these, however, GBS were present in very low numbers (5 x 10^3/g) and might, therefore, have represented contamination from perianal skin. Hare and Maxted11 found no GBS in a larger number of faecal samples.

The significance of high rectal GBS carriage rates was taken by Franciosi et al.3 to be a reflection of the perianal carriage of GBS from the vagina. Kexel and Beck, however, found that in comparing swabs from the vulva, vagina, and cervix, the rates of GBS colonisation fell steadily from 12.4% (vulva) to 6.7% (cervix). This implied that the perianal skin might be the source of vaginal carriage rather than vice versa.

The use of male subjects in a study of rectal/anal GBS carriage removes any confusion over vaginal carriage. The recovery of GBS from both rectal mucosa and perianal skin was four times higher than the total recovery of GBS from the faeces of the same individuals. There was no significant difference in the overall recovery of GBS from rectal mucosa and perianal skin. A comparison of the relative recovery of organisms from both sites using direct plating and enrichment techniques, however, suggested that perianal carriage might be heavier.

Our results, therefore, agree with those of Islam and Thomas in suggesting that “gastrointestinal” GBS colonisation may be limited largely to the rectal mucosa and perianal skin. Their rectal sample was very much smaller than our own (28 as opposed to 101) and it was not made clear whether the faecal and rectal samples were taken from the same patients, but our overall faecal and rectal carriage rates are very similar. Faecal samples only give an indirect estimate of the bacterial flora higher up in the gastrointestinal tract, but accurate studies of GBS colonisation in such sites will not be easy because of the problem of sampling.

If our impression that perianal GBS carriage is heavier than that associated with rectal mucosa in the same subjects is correct, it provides an interesting parallel with the results of Kexel and Beck. They found that GBS carriage in the female genital tract dropped as samples were taken in turn from the vulva, the vagina, and finally the cervix. The perianal
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skin, rectal mucosa and faeces form a similar sequence.

The Islam starch agar performed well in both selective and non-selective forms. Unlike Islam and Thomas,4 we did not find that the addition of nalidixic acid (15 g/l) affected pigmentation. Not all human strains of GBS produce pigmentation.13 The figures vary but we have tested over 600 GBS strains for pigment production over the past 18 months and found only five non-pigment producers. The chances that any were missed in this study is, therefore, small and would be unlikely to alter our findings significantly.

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References

3 Franciosi RA, Knostman JD, Zimmerman RA. Group B streptococcal neonatal and infant infections. J Pediatr

11 Hare R, Maxted WR. The classification of haemolytic streptococci from the stools of normal pregnant women and of cases of scarlet fever by means of precipitin and biochemical tests. Journal of Pathology and Bacteriology 1935;41:513-20.

Requests for reprints to: Dr CSF Easmon, Department of Bacteriology, St Mary's Hospital and Medical School, London W2 1PG, England.