A SMALL OUTBREAK OF DIARRHOEA ASSOCIATED WITH THE PARACOLON BACILLUS

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The term ‘paracolon bacillus’ is generally used in bacteriological literature to describe a certain variety of coliform bacillus not infrequently isolated from human faeces, especially in the tropics, and the following definition of Abdoosh (1934) may usefully be accepted: ‘Members of genus *Bacterium* that fail to ferment lactose in 24 hours and fail to ferment the same sugar even after 3 weeks, that ferment glucose and mannite, acid and gas, produce indole and fail to ferment saccharose.’

Other important characteristics of the paracolon bacillus not mentioned in this definition are as follows (Stewart, 1926; Wilson, 1929; Abdoosh, 1934; Sandiford, 1935; Mackie and McCartney, 1938): motility, variable, but usually negative; methyl red test, positive; Voges-Proskauer reaction, negative; H₂S, very seldom produced; gelatin, not liquefied; fermentation of inosite, negative, and of dulcite, variable; reaction in litmus milk, variable.

The paracolon bacillus is thus seen to have fairly distinctive properties which enable it to be recognized without great difficulty by simple biochemical and cultural tests. It is probable, however, that more than one species may be included in the paracolon group.

With regard to pathogenicity, it has been claimed that paracolon strains can, under certain circumstances, produce diarrhoea. For example, Hassmann & Herzmann (1934) found that such organisms were associated with diarrhoeal states after measles and other infectious diseases (see also Hassmann, 1935).

Other workers have, however, expressed different opinions. Thus, Stewart (1926), after examining a number of strains, considered that their presence was symptomatic rather than aetiological, and Sandiford (1935), who studied forty-one strains isolated from stools sent in to the Central Laboratories at Cairo for examination for dysentery, and six strains from normal stools, concluded that the presence of the paracolon bacillus in a stool had no pathological significance. He was partly influenced in arriving at this conclusion by the finding of negative blood cultures in persons excreting the organism in their stools, and by the fact that serum agglutinins against the homologous bacillus were not developed. As further evidence of their non-pathogenic role, coliform organisms of the paracolon type, known as Intermediate Type II, have been found in water and are thought to be of soil origin (see *Ministry of Health Report*, 1940).
Diarrhoea associated with the paracolon bacillus

Although there thus seems little evidence in favour of the primary causal role of the paracolon bacillus in intestinal disease, it is the object of this paper to record a small outbreak of diarrhoea in a mental hospital which was associated with and perhaps produced by an organism of this type.

CLINICAL FEATURES

The outbreak of diarrhoea occurred in two wards of a mental hospital in which five female patients were affected. The first patient (D. Price) had several loose motions containing blood and mucus on 16 October 1941; her temperature was 102°F. Except for a transient recurrence of diarrhoea on 22 October her condition rapidly improved. Two other patients in the same ward (M. Gorman and A. Owens) suffered from similar symptoms on 20 and 21 October respectively. These patients also recovered in a few days. On 22 and 23 October two patients (A. Jarman and E. M. Barnett) in another ward suffered from similar symptoms, and also recovered rapidly.

SOURCE OF INFECTION

To trace the origin of 'asylum dysentery' is notoriously difficult, and careful inquiry in this outbreak failed to detect any obvious source of infection. It is probable, however, that the patient Price infected the other two cases in her ward by more or less direct contact. No real suspicion rested on food handlers, and the stools of three scullery workers in the first ward to be affected were examined bacteriologically with negative results.

ORIGINAL BACTERIOLOGICAL FINDINGS

Stools were plated on MacConkey's medium, shortly after being passed, and brilliant green enrichment was also carried out. In some cases 'Difco' S-S agar was also used. Non-lactose-fermenting colonies were readily obtained by all these procedures and subcultures were usually prepared direct from the primary MacConkey plate. As all 'pale' colonies appeared to be of one type, only one colony was subcultured in each case. The following tests were then performed:

(a) Fermentation and other tests

The results of fermentation and other tests follow in Table 1.

From Table 1 it will be seen that strains Barnett, Owens, and Gorman had identical properties and that strain Jarman only differed in that dulcite was fermented. These reactions place the organisms in the paracolon group.

(b) Agglutination tests

To exclude the possibility of these organisms being concomitants or secondary invaders, sera were obtained from all five patients on 1 November, i.e. 10–14 days after the onset of diarrhoea, in order to test for the presence of agglutinins for the paracolon bacillus. From four strains (Owens, Barnett,
Gorman, Jarman) O antigens were prepared according to the method recommended by Mackie & McCartney (1938). The agglutination tests were incubated at 55° C. for 4 hr. and then at 37° C. overnight. The results are given in Table 2.

From Table 2 it will be seen that agglutinins were well developed for the homologous paracolon antigens in the case of patients Owens, Gorman, and Jarman, while the agglutinin response was not so marked in the case of Barnett.

Table 1. Reactions of five strains of paracolon bacillus

<table>
<thead>
<tr>
<th>Strains</th>
<th>Price</th>
<th>Owens</th>
<th>Gorman</th>
<th>Barnett</th>
<th>Jarman</th>
</tr>
</thead>
<tbody>
<tr>
<td>Motility after 24 hr. in peptone water</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Glucose, 24 hr.</td>
<td>AG</td>
<td>AG</td>
<td>AG</td>
<td>AG</td>
<td>AG</td>
</tr>
<tr>
<td>Lactose, 14 days</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Dulcite, 14 days</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Saccharose, 14 days</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Mannite, 24 hr.</td>
<td>AG</td>
<td>AG</td>
<td>AG</td>
<td>AG</td>
<td>AG</td>
</tr>
<tr>
<td>Inosite, 14 days</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Indole, 24 hr.</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Voges-Proskauer</td>
<td>---</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Methyl red</td>
<td>---</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Koser's citrate</td>
<td>---</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Liquefaction of gelatin, 14 days</td>
<td>---</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Reaction in litmus milk, 14 days</td>
<td>---</td>
<td>a</td>
<td>a</td>
<td>a</td>
<td>a</td>
</tr>
<tr>
<td>Blackening of lead acetate</td>
<td>---</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Spreading growth on agar at 37° C.</td>
<td>---</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

0 = negative. — = not carried out. a = slight acidity. AG = acid and gas.

Table 2. Agglutination reactions between patient's sera and paracolon bacillus strains

<table>
<thead>
<tr>
<th>Serum from</th>
<th>Paracolon antigen from</th>
<th>Agglutination</th>
</tr>
</thead>
<tbody>
<tr>
<td>Owens</td>
<td>Owens</td>
<td>1/240</td>
</tr>
<tr>
<td>Barnett</td>
<td>Barnett</td>
<td>1/60</td>
</tr>
<tr>
<td>Gorman</td>
<td>Gorman</td>
<td>1/240</td>
</tr>
<tr>
<td>Jarman</td>
<td>Jarman</td>
<td>1/480</td>
</tr>
</tbody>
</table>

Sera and homologous antigens

<table>
<thead>
<tr>
<th>Serum from</th>
<th>Paracolon antigen from</th>
<th>Agglutination</th>
</tr>
</thead>
<tbody>
<tr>
<td>Price</td>
<td>Owens</td>
<td>1/240</td>
</tr>
<tr>
<td>Barnett</td>
<td>Owens</td>
<td>1/60</td>
</tr>
<tr>
<td>Gorman</td>
<td>Owens</td>
<td>1/240</td>
</tr>
<tr>
<td>Jarman</td>
<td>Owens</td>
<td>1/60</td>
</tr>
</tbody>
</table>

Sera and heterologous antigens

No antigen was prepared from strain Price, but it was found that this patient's serum agglutinated antigen from the patient Owens to a titre of 1/240. Agglutinins against strain Owens were also developed in the sera of the other three patients.

These results definitely suggest that only one strain of paracolon bacillus was present in all the patients affected.

As controls, sera from five mental patients, who were in perfect physical health at the time, were tested against the 'O' antigen prepared from strain Gorman. No agglutination occurred, even at a dilution of 1/30, showing that
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The positive findings recorded in Table 2 were not due to the presence of natural agglutinins.

(c) Examination of stools of other inmates

The organism associated with this outbreak was evidently not widely distributed in the institution, as the stools of seven persons in contact with the original patients showed no paracolon bacilli.

Re-examination of stools

Faeces from all cases were examined for the presence of the paracolon bacillus on various occasions after the initial attack of diarrhoea with the following results:

Table 3. Showing the results of examinations of the stools of the patients after recovery

<table>
<thead>
<tr>
<th></th>
<th>6. xi. 41</th>
<th>10. xi. 41</th>
<th>14. xi. 41</th>
<th>17. xi. 41</th>
</tr>
</thead>
<tbody>
<tr>
<td>Price</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Owens</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Barnett</td>
<td>0</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Jarman</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Gorman</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

It is thus seen that in two cases paracolon bacilli were still being passed more than three weeks after the initial attack of diarrhoea.

Summary

A small outbreak of diarrhoea is reported which involved five patients in a mental hospital. An organism showing the characteristics of the paracolon bacillus was isolated from all these cases. Agglutinins to this organism were present to significant titre in the sera of all the patients, suggesting that this race of the paracolon bacillus was the aetiological agent in this outbreak.

I have to express my thanks to Dr W. Stanley Hughes and Dr R. O. Smyth, of the Salop Mental Hospital, who have kindly described the clinical features of this outbreak and afforded me every facility for investigation.

References


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