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Multiresistant *Shigella* Species Isolated from Childhood Diarrhea Cases in Kolkata, India

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Antimicrobial therapy for shigellosis reduces the duration and severity of the disease and can also prevent potentially lethal complications (1). However, over the past few decades *Shigella* spp. have become progressively resistant to most of the first-line drugs used, and the prevalence of multidrug-resistant strains is an important concern of treatment (2,3). In this report we analyze the occurrence rate and multidrug-resistance pattern of *Shigella* isolates obtained from childhood diarrhea cases.

From January 2001 to June 2002, a total of 1,295 stool samples was collected from children suffering from acute diarrhea who attended the Diarrhoea Treatment Unit at Dr. B.C. Roy Memorial Hospital for Children, Kolkata, and were cultured for *Shigella* spp. All isolates were confirmed serologically by slide agglutination using commercially purchased antisera (Denka Seiken, Tokyo). Antimicrobial susceptibility tests were performed using an agar diffusion technique method in accord with the National Committee for Clinical Laboratory Standards guidelines (4). The sixty-eight strains of *Shigella* spp. isolated were distributed as follows: 37 (54.4%) were *S. flexneri*, 19 (28%) were *S. sonnei*, 9 (13.2%) were *S. boydii*, and 3 (4.4%) were *S. dysenteriae*. Antimicrobial susceptibility testing of *Shigella* spp. showed a high degree of resistance to the commonly used antimicrobials, including ampicillin (41%), co-trimoxazole (95%), tetracycline (87%), and nalidixic acid (59%), and low level resistance against norfloxacin (9%) and ciprofloxacin (6%). We observed a different antimicrobial resistance pattern among four *Shigella* spp., necessitating the identification of serogroups along with resistance patterns for purposes of treatment of shigellosis. Overall, 90% of *Shigella* isolates were found resistant to two or more antimicrobial agents. Multidrug resistance also varied significantly among species (Table 1). Antimicrobial resistance has been occurring among *Shigella* spp. since the 1940s, when sulfonamide resistance among *Shigella* organisms was first recognized in Japan (5). By 1970, widespread resistance of this disease to sulfonamides limited their therapeutic usefulness (6). In 1984, an epidemic of shigellosis, caused primarily by multidrug-resistant *S. dysenteriae* 1, broke out in the eastern part of India and caused many deaths in and around Kolkata (7). During this period, the
efficacy of nalidixic acid in the treatment of multi-resistant
* Shigella dysenteriae* 1 infection was reported from Kolkata (8). However, within a short period, the widespread use of this
drug resulted in the emergence of nalidixic acid-resistant
* Shigella dysenteriae* type 1 (9). The development of resistance of
* Shigella* spp. to common drugs in Kolkata had been reported
earlier (10). Shigella isolates resistant to multiple drugs have
been reported from several parts of the world (11,12). Our
results revealed that multi-resistant strains of
* Shigella* are present in Kolkata, and emphasize the importance of main-
taining surveillance of these strains in order to assess local
susceptibility patterns and empiric therapy. Multidrug
resistance is an emerging problem in the clinical management
of shigellosis, particularly in children in third world countries
where diarrheal diseases are a major cause of childhood
morbidity and mortality.

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### Table 1. Multidrug-resistance pattern of 68 *Shigella* isolates

<table>
<thead>
<tr>
<th><em>Shigella</em> spp.</th>
<th>Resistance pattern*</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>S. dysenteriae</em> (n = 3)</td>
<td>A, CO, T, Na, Nx, Cf</td>
<td>3</td>
</tr>
<tr>
<td><em>S. flexneri</em> (n = 37)</td>
<td>None</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>CO, T</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>CO, Na</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>A, CO</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>A, CO, T</td>
<td>7</td>
</tr>
<tr>
<td></td>
<td>A, CO, Na</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>CO, T, Na</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>A, CO, T, Nx</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>A, CO, T, Na</td>
<td>11</td>
</tr>
<tr>
<td></td>
<td>A, CO, T, Na, Nx</td>
<td>1</td>
</tr>
<tr>
<td><em>S. boydii</em> (n = 9)</td>
<td>None</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>CO, T</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>A, T</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>A, CO, T</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>CO, T, Na</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>A, CO, T, Na</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>A, CO, T, Na, Nx, Cf</td>
<td>1</td>
</tr>
<tr>
<td><em>S. sonnei</em> (n = 19)</td>
<td>None</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>CO, T, Na</td>
<td>15</td>
</tr>
<tr>
<td></td>
<td>CO, T</td>
<td>3</td>
</tr>
</tbody>
</table>

*: A, ampicillin; CO, co-trimoxazole; Na, nalidixic acid; Nx, norfloxacin; Cf, ciprofloxacin; T, tetracycline.

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