PREVALENCE OF SALMONELLA TYPHI CARRIER STATE IN
PATIENTS WITH GLUCOSE – 6 – PHOSPHATE
DEHYDROGENASE DEFICIENCY

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ABSTRACT

A case control study was conducted in Basrah at the period from March to
September 2002. The population included in this study was divided into two groups. The
first group was included patients with G6PD deficiency and the second group was included
normal individuals as a control group.

Stool samples were collected from both groups for bacteriological examination.

This study revealed that; there was an association between G6PD deficiency and
Salmonella typhi carrier state and this association was statistically significant in urban
rather than rural area.

INTRODUCTION

A carrier is a person that shed the microorganism without clear clinical symptoms
and serves as a potential source of infection (Benenson 1995, Muhsen & Bakr 2001).

More than 5% of patients with typhoid fever become chronic carrier after recovery
( Geeds et al 1995); and the most common site of microorganism was gall bladder

The carrier state may follow acute or mild or subclinical infection (Benenson
1995); but as many as one third of chronic carriers give no history of typhoid fever (Foot
and Hock 1979). Underlying biliary and urinary tract disease especially stone formation
increase the probability of chronic enteric and urinary carrier state in patients with typhoid

This study was done to evaluate the susceptibility of patients with glucose – 6 –
phosphate dehydrogenase deficiency to infection with typhoid fever since these patients
have defective phagocytosis activity (Muhsen 2002).

MATERIALS AND METHODS

1. Patients and control group

A-Patients group: This group comprised (110) patients with glucose – 6 – phosphate
dehydrogenase deficiency. Stool samples for bacteriological examination were collected
from this group. All patients reported to have no typhoid fever for at least three months.
B-Control group: This group comprised (210) individuals known to have no family history of Glucose - 6 - phosphate dehydrogenase deficiency. Stool samples for bacteriological study were collected from nurseries and students.

2. Laboratory examination

A-culture

Stool samples were cultured immediately in tetrahydrofuran broth and incubated at 37°C for 24 hours; then subcultured on MacConkey's agar; SS agar; Brilliant green agar and Brinntph sulfite agar (Jawetes et al. 1998).

B-Biochemical test

The following biochemical test were done according to procedures described by Fingold and Baron 1986 to identify Salmonella typhi:
1. Triple sugar iron agar test.
2. Lysine decarboxylase test.
3. Uras test
4. Citrate utilization test
5. Methyle Red – Voges Proskauer test
6. Catalase test
7. Oxidase test
8. Indol test

C-Serology

Slide agglutination test using Salmonella polyvalent agglutinating sera and then Salmonella typhi monovalent agglutinating sera (Welcome) as a final identification method of cultured Salmonella typhi (Pennevom 1995).

RESULTS

1. Characters of patients and control group

The patients with glucose - 6 - phosphate dehydrogenase deficiency were with an age range from 4-31 years and the mean age was 22.42±8.75; while age range of control group was from 6-41 years and mean age was 18.2±5.22. There was no statistical difference in the mean of age between patients and control group (SNID= 0.4 P=0.05).

2. Association between glucose -6-Phosphate dehydrogenase deficiency and salmonella typhi carrier state (table 1).

Salmonella typhi was isolated from 14 out of 110 (12.7%) stool sample collected from patients with glucose -6-phosphate dehydrogenase deficiency and 9 out of 210 (4.2%) stool sample collected from control group. There was an association between Salmonella typhi carrier state and glucose -6-phosphate dehydrogenase deficiency (OR=3.25) and this association was statistically significant ($X^2=7.7$ P<0.05).

3. Association between Salmonella typhi carrier state and glucose -6-phosphate dehydrogenase deficiency in relation to residency.

a. In urban area: -

Salmonella typhi was isolated from 9 out of 55 (16.3%) stool samples collected from patients' group and from 4 out of 105 (3.8%) stool samples collected from control group. There was an association between S typhi carrier state and glucose-6-phosphate dehydrogenase deficiency (OR 4.94) and this association was statistically significant ($X^2=7.6$ P<0.05). Table (2).
(Table 1) Association between G6PD deficiency & *S. typhi* carrier state.

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<th>Total</th>
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<tbody>
<tr>
<td>G6PD DEFICIENCY PATIENT</td>
<td>14</td>
<td>96</td>
<td>110</td>
</tr>
<tr>
<td>Normal Person</td>
<td>9</td>
<td>201</td>
<td>210</td>
</tr>
<tr>
<td>Total</td>
<td>23</td>
<td>297</td>
<td>320</td>
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</tbody>
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OR = 3.25 → $X^2 = 7.7$ → *P* < 0.05

(Table 2) Association between G6PD deficiency & *S. typhi* carrier state in urban area.

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<tbody>
<tr>
<td>G6PD DEFICIENCY PATIENT</td>
<td>9</td>
<td>46</td>
<td>55</td>
</tr>
<tr>
<td>Normal Person</td>
<td>4</td>
<td>101</td>
<td>105</td>
</tr>
<tr>
<td>Total</td>
<td>13</td>
<td>147</td>
<td>160</td>
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$X^2 = 7.6$ → OR = 4.94 → *P* < 0.05

(Table 3) Association between G6PD deficiency & *S. typhi* carrier state in rural area.

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</table>

OR = 0.5 → $X^2 = 1.16$ → *P* > 0.05

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DISCUSSION

1. There was significant association between Salmonella typhi carrier state and glucose – 6 phosphate dehydrogenase deficiency. Since the carrier state gives ideas about infection; from this result we can conclude that patients with glucose – 6 phosphate dehydrogenase deficiency are more susceptible to infection with typhoid fever than normal individuals and G6PD deficiency can be considered as an important risk factor for infection with typhoid fever. Deficiency of phagocytosis activity in patients with glucose – 6 phosphate dehydrogenase deficiency (Muhsen 2002) can explain this result.

2. The association between Salmonella typhi carrier state and glucose – 6 phosphate dehydrogenase deficiency was significant in urban rather than rural area. This result can be explained by that; In heavily contaminated area, both immunocompetent and immunocompromized individuals affected equally and risk factor play no role for infection in rural area; while in urban area where hygiene and sanitation were better than rural area; immunocompromized patients are more susceptible to infection than normal population and risk factor play an important role for infection in urban area (Muhsen 1998).

REFERENCES