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

Rahman Kadhum Muhsen

Department of Microbiology ;College of Veterinary Medicine, University of Basrah, Basrah,Iraq.

(Received 7 September 2004,Accepted 7 January 2005)

Keywords;G6pD,Salmonella,Infection.

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 patents 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 crrier :- is a Person that shed the microorganism without clerar clinical symptoms and serves as a potent 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 (Jawetez et al 1998 , Muhsen & Bakr 2001).

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 billiary and urinary tract disease especially stone formation increase the probability of chronic enteric and urinary carrier state in patients with typhoid fever (Christie 1980 , Muhsen & Bakr 2001).

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 tetrathionate broth and incubated at 37°Cfor 24 hours ; then subcultured on Macconkeys agar ; SS agar , Brilliant green agar and Bismuth 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 tast .
2. Lysine decarboxylation test
3. Urase test
4. Citrate utilization test
5. Methyle Red – vogus 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 indentification method of cultured *Salmonella typhi* (Benenson 1995).

RESULTS

1.Characters of patients and control group .

The patients with glucose – 6 – phosphate dehyrogenase 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 (SND= 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 associated 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 4 out of 105 (3.8 %) stool samples collected from control group . There was an association between *S.typhi* carrier state and glucos-6-phosphate dehydrogenase deficiency (OR 4.94) and this association was statistically significant(X^2=7.6 P<0.05). Table (2).

a- In rural area :-

Salmonella typhi was isolated from 9 out of 55(16.3%) stool samples collected from patients group and 4 out of 105(3.8%) stool samples collected from control group ;these was an association between *Salmonella typhi* carrier state and glucose – 6 – phosphate dehydrogenase deficiency (OR = 0.5) but this association was statistically not significant ($x^2 = 1.16$ $P > 0.05$) table (3)

(table 1) Association between G6PD deficiency & *S.typhi* carrier state .

	+ve	-ve	Total
G6PD DEFICIENCY PATIENT	14	96	110
Normal Person	9	201	210
Total	23	297	320

OR = 3.25 ⟶ $X^2 = 7.7$ ⟶ $P < 0.05$

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

	+ve	-ve	Total
G6PD DEFICIENCY PATIENT	9	46	55
Normal Person	4	101	105
Total	13	147	160

$X^2 = 7.6$ ⟶ OR= 4.94 ⟶ $P < 0.05$

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

	+ve	-ve	Total
G6PD DEFICIENCY PATIENT	9	46	55
Normal Person	4	101	105
Total	13	147	160

OR = 0.5 ⟶ $X^2 = 1.16$ ⟶ $P > 0.05$

DISCUSSION

1. There was significant association between *Salmonella typhi* carrier state and glucose – 6 – phosphate delydrogenase deficiency . Since the carrier state give idea 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 phogocytosis activity in patients with glucose – 6 – phosphate dehydrogonase 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)

انتشار صفة الحامل للجرثومة المسببة للحمى التايفونيدية في الأشخاص المصابين بنقص أنزيم محلل الكلوكوز .

رحمن كاظم محسن

فرع الأحياء المجهرية ، كلية الطب البيطري ،جامعة البصرة ،البصرة، العراق

الخلاصة

أجريت هذه الدراسة الضبطية في مدينة البصرة للفترة من آذار إلى أيلول ٢٠٠٢ وشملت هـذه الدراسـة ٣٢٠ شخصاً قسموا إلى مجموعتين ضمت المجموعة الأولى الأشخاص المصابين بنقص محلل الكلوكوز وضـمت المجموعة الثانية أشخاص أصحاء كمجموعة سيطرة وجمعت نماذج البراز من كلا المجموعتين وخضعت للفحـص الجرثومي وأظهرت نتائج الدراسة إن هناك علاقة إحصائية بين نقص أنزيم محلل الكلوكوز وانتشار صفة الحامـل للجرثومة المسببة للحمى التايفوئيديه وكانت هذه العلاقة واضحة أحصائيا فى المناطق الحضرية وغير واضحة فى المناطق الريفية .

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