

Prevalence of Glucose-6-Phosphate Dehydrogenase
Deficiency in Typhoid Fever Patients at Children's Hospital

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OBJECTIVE: To determine the prevalence of glucose-6-phosphate dehydrogenase (G-6-PD) deficiency among hospitalized typhoid fever patients at Children's Hospital.

BACKGROUND: The clinical staff of Bangkok Children's Hospital, responsible for the management of typhoid fever patients, has noted a high prevalence of G-6-PD deficiency among hospitalized typhoid fever patients. Approximately 35% of 50 typhoid fever patients at Children's Hospital were G-6-PD deficient using the method of Brewer (1). This method may not detect heterozygotes and may be normal if the red cell population is young.

In the present study, the more sensitive methemoglobin elution technique of Gall (2) was used.

Hemolysis as a clinical expression of G-6-PD deficiency following the administration of various drugs, or in association with specific illnesses, is well known. Hemolysis in patients with typhoid fever and G-6-PD deficiency is well recognized.

Thirty-six male African patients hospitalized with typhoid fever had G-6-PD determinations performed. Fourteen were deficient. This incidence (39%) was more than twice the general incidence for G-6-PD deficiency in Accra, Ghana (3).

The relationship between sickle cell anemia and salmonella osteomyelitis is well recognized. Several studies have shown that bactericidal mechanisms which operate to eliminate salmonella are severely impaired in animals with hemolysis.

PROGRESS: Blood was drawn for G-6-PD determination from 34 patients admitted to Children's Hospital with typhoid fever, confirmed by culture and Widal test. Fourteen of 34 (41%) were either homozygous or heterozygous for G-6-PD deficiency (Table 1).

Fifteen patients with suspected typhoid fever but with negative serial cultures for *Salmonella typhi* and serial Widals of 1:40 or less have had G-6-PD determinations performed. One of 15 was a homozygote (7%) (Table 2).

DISCUSSION: This high prevalence of G-6-PD deficiency among hospitalized typhoid fever patients is remarkable.

The prevalence of G-6-PD deficiency among hospitalized patients with fever, but having no evidence to support the diagnosis of typhoid fever, is approximately the same as the prevalence of G-6-PD deficiency in the general population (10%).

This high prevalence of G-6-PD deficiency among typhoid fever patients suggests that individuals with heterozygous or homozygous deficiency are more susceptible to the *Salmonella typhi* organism. This might

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Table 1. G-6-PD Determinations in 34 Typhoid Patients

Sex	Normal	G-6-PD Deficient	
		Homozygotes	Heterozygotes
Male	12	6	0
Female	8	2	6
Subtotal	20	8	6
Total	20	14	

Table 2. G-6-PD Determinations in 15 Non-Typhoid Patients

Sex	Normal	G-6-PD Deficient	
		Homozygotes	Heterozygotes
Male	7	1	0
Female	7	0	0
Subtotal	14	1	0
Total	14	1	

be a direct result of the G-6-PD metabolic defect, or it might be possible that in G-6-PD deficient patients the reticuloendothelial system is already preoccupied with the handling of the products of red cell destruction.

It may be that G-6-PD deficient individuals who acquire typhoid fever are more likely to be hospitalized than G-6-PD normal individuals who acquire typhoid. For this reason a study of typhoid fever outpatients and controls is planned.

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