Effect of Red Blood Cell Glucose-6-Phosphate Dehydrogenase Deficiency on Patients with Dengue Hemorrhagic Fever

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Abstract

Eighty nine males aged 1-13 years diagnosed with dengue haemorrhagic fever (DHF) and admitted to the Department of Pediatrics Siriraj Hospital from March 1998 to April 2000 were included in this study. 17 cases (19.1%) had red blood cell glucose-6-phosphate dehydrogenase (G-6-PD) deficiency and 72 cases (80.9%) had normal G-6-PD enzyme activities. Most of the patients were classified as DHF grade II in severity. 3 of 17 G-6-PD deficient cases had serious complications and all of them had acute intravascular hemolysis requiring blood transfusions. One of these also had hematemesis, one had azothemia and the other one had renal failure and severe liver failure with hepatic encephalopathy.

In the cases without obvious hemolytic or hepatic complications, G-6-PD deficient cases had mildly but significantly higher total biririubin and indirect bilirubin, as well as a lower hematocrit than those who had normal G-6-PD. Reticulocyte count was low during the acute phase, however, during recovery, the levels were significantly increased in both groups. In the non G-6-PD deficient group, G-6-PD enzyme levels were significantly decreased during the acute phase compared to the normal controls but rose significantly to normal levels during the recovery phase. There were no statistically significant differences in other laboratory data. All patients recovered fully from DHF.

The prevalence of G-6-PD deficiency in male patients who had DHF in this study was 19.1 per cent which was higher than the prevalence in a previous study of 12 per cent in Bangkok. This may imply that G-6-PD deficient males suffer more from DHF compared to normal G-6-PD subjects.

Key word : G-6-PD Deficiency, Dengue Hemorrhagic Fever, Liver Failure, Acute Hemolysis

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Dengue haemorrhagic fever (DHF) is a disease caused by the dengue virus which is highly endemic in Southeast Asian countries including Thailand[1]. The morbidity and mortality of this disease is high especially in cases who have complications or unusual manifestations. These include shock, severe bleeding, hepatic failure, encephalopathy and secondary infection[1-3].

Red blood cell glucose-6-phosphate dehydrogenase (G-6-PD) deficiency is highly prevalent in Thai males and ranges from 3-18 per cent depending upon the geographic region and the method used. In Bangkok the prevalence assessed by enzyme assay in cord blood revealed 12.08 per cent and 11.1 per cent in 2 studies[4,5]. Although almost all G-6-PD deficient persons are symptomless, acute hemolysis may occur during oxidative stress[6]. There have been reports of severe hyperbilirubinemia and massive hemolysis in patient with viral infection especially viral hepatitis[7-10]. Some DHF patients were observed to have severe complications including anemia with dark-colored urine and G-6-PD deficiency was suspicious but not proven due to blood transfusion and/or their mortality. It was the purpose of the authors to prospectively study the effect of red blood cell G-6-PD deficiency on male patients with DHF.

SUBJECTS AND METHOD

Male children who were clinically diagnosed to have DHF and admitted to the Department of Pediatrics, Siriraj Hospital from March 1998 to April 2000 were prospectively studied. Paired serum samples were obtained from the patients for serological testing by hemagglutination inhibition (HI) and enzyme-linked immunosorbent assay (ELISA) for IgM[1]. Acute samples were inoculated into mosquito larvae for virus isolation[1]. The acute dengue titer of over 1 : 1,280 or 4-fold rising of antibody titer or positive results of dengue virus isolation after mosquito innoculation were diagnostic[1, 11]. The diagnosis was based on the clinical, hematological and/or viral study. The severity of DHF was classified in 4 grades as previously described[11, 12]. Serial measurements of complete blood count were done, G-6-PD assay was performed in all cases and G-6-PD levels of less than 5 IU/dl red blood cell on admission or on follow-up were considered G-6-PD deficiency[4]. The clinical findings, days of hospitalization, blood chemistry including liver function test, renal function test and other complications were recorded. The statistical analysis was performed using the Student *t*-test.

RESULTS

One hundred males who were initially diagnosed to have DHF were included in this study. However, laboratory confirmation of the diagnosis was achieved in 89 cases. Definitive diagnosis of DHF was confirmed by serology result and/or viral isolation in 73 cases. These comprised of 59 cases whose diagnosis was based on serology study, 26 cases had acute antibody titer of >1 : 1,280 while 33 cases had a 4-fold rise of antibody titer. Among 29 cases who had positive results on viral isolation, it revealed dengue virus type 1, 2 and 3 in 8, 12 and 9 cases respectively. The other 16 cases in whom no positive data on viral study were available, the diagnosis was based on the clinical and hematological parameters i.e. hemoconcentration, presence of transformed lymphocyte and low platelet. Of 89 confirmed cases, their age ranged from 1-13 years. There were 17 cases (19.1%) who had red blood cell G-6-PD deficiency and all of them had G-6-PD activities at O IU/dl Rbc. Most of the patients (69.7%) were classified as DHF grade II in severity, patients in grade I, III and IV were 9, 18 and 3.4 per cent respectively.

Among 17 G-6-PD deficient cases, 3 had serious complications. The first case was a 9-year-old boy with grade II DHF and acute intravascular hemolysis with frank hemoglobinuria. His hematocrit dropped to 18 per cent and he had transient azothemia. He was hospitalized for 10 days and finally recovered after packed red cell transfusion and other supportive treatment. The second case was a 5-year-old boy with grade IV DHF who had both hemoglobinuria and hematemesis without renal complication. He recovered well after packed red cell transfusion and other supportive measures. The third case was a 9-year-old boy with grade IV DHF who had hematemesis, frank hemoglobinuria and shock. The clinical signs and laboratory results were compatible with acute renal shut down and hepatic failure with encephalopathy. The treatments included blood transfusions and 2 exchange blood transfusions and other supportive measures. He finally recovered and was sent home after 40 days of hospitalization. The detail of these cases will be presented elsewhere.

Comparison of the laboratory data was made between the other 14 cases who had G-6-PD deficiency but there was no obvious hemolytic complication (Group I) to the non G-6-PD deficient group (Group II). It was shown that patients in Group I had slightly but significantly lower hematocrit, in addition both total and indirect bilirubin were higher than those of normal G-6-PD group. (Table 1, 2).

There were no statistically significant differences in other laboratory data i.e. renal function, liver function, days of hospitalization and the outcome between the 2 groups.

During acute illness, the reticulocyte count in both groups were significantly lower than the levels observed 2 weeks later, during recovery. This was in agreement with the increased G-6-PD enzyme activity in the recovery period in Group II (Table 3). Table 4 shows the prevalence of abnormal initial laboratory data in patients of both groups. It was shown that the patients in Group I tended to have lower hematocrit, higher creatinine, total and indirect bilirubin as well as transaminase enzymes compared to Group II.

Table 1. Laboratory data of non complicated cases of dengue hemorrhagic fever who had G-6-PD deficiency (Group I) and non deficiency (Group II).

Parameter	Group I ($\bar{X} \pm SE$)	Group II ($\bar{X} \pm SE$)
Hospitalized days	3.79 ± 0.26	4.51 ± 0.27
Reticulocyte (%)	1.43 ± 0.59	0.83 ± 0.001
Hematocrit (%)	37.79 ± 1.15	40.99 ± 0.71*
White blood cells (x $10^3/\mu l$)	3.56 ± 0.33	4.80 ± 0.33
Platelet count (x $10^3/\mu l$)	94.71 ± 17.91	82.93 ± 8.5
Blood urea nitrogen (mg/dl)	12.67 ± 1.65	14.5 ± 0.77
Creatinine (mg/dl)	0.74 ± 0.001	0.74 ± 0.001

* Statistically significant difference between the 2 groups P = 0.026

Table 2. Liver function test and biochemical data in non complicated patients with dengue hemorrhagic fever who had G-6-PD deficiency (Group I) and non deficiency (Group II).

Parameter	Group I ($\bar{X} \pm SE$)	Group II ($\bar{X} \pm SE$)
Total bilirubin (mg/dl)	0.88 ± 0.001	0.38 ± 0.001*
Direct bilirubin (mg/dl)	0.24 ± 0.001	0.12 ± 0.001
Indirect bilirubin (mg/dl)	0.63 ± 0.001	0.17 ± 0.001*
Aspartate amino transferase (μ/L)	223.147 ± 65.03	255.87 ± 58.61
Alanine amino transferase (μ/L)	101.07 ± 29.2	108.38 ± 20.59
Alkaline phosphatase (μ/L)	170.36 ± 16.36	162.92 ± 7.27
Creatine phosphokinase (μ/L)	136.64 ± 37.42	341.40 ± 130.96
Lactate dehydrogenase (μ/L)	1,068 ± 156.31	1,100.2 ± 89.56

* Statistical difference between the 2 groups P = 0.001

Table 3. Reticulocyte count and G-6-PD enzyme activities in patients with DHF.

	Number	Mean ± SE
Normal control		
G-6-PD (I.U./dl Rbc)	20	198.60 ± 5.24
Reticulocyte (%)	33	1.46 ± 0.94
Group I		
G-6-PD^{+} (IU/dl Rbc)	17	0*
Reticulocyte^{+} (%)	10	1.47 ± 0.65
Reticulocyte$^{\#}$ (%)	10	3.67 ± 0.39*,**
Group II		
G-6-PD^{+} (IU/dl Rbc)	40	125.30 ± 7.00*
G-6-PD$^{\#}$ (IU/dl Rbc)	40	183.60 ± 4.60**
Reticulocyte^{+} (%)	32	0.82 ± 1.20*
Reticulocyte$^{\#}$ (%)	32	1.82 0 ± 13*,**

$^{+}$: Initial data
$^{\#}$: Data at recovery
* : Significant difference from controls ($p<0.001$)
** : Significant difference from $^{+}$ ($p<0.001$)

DISCUSSION

Persons who have G-6-PD deficiency have a risk of hemolysis during oxidative stress such as infection, drugs, metabolic derangement whenever it outweighs the bodies' defensive mechanism[6]. Viral hepatitis, for instance, is one example of infections in which severe hyperbilirubinemia and massive hemolysis occurred in G-6-PD deficient persons[7-10]. Since the prevalence of G-6-PD deficiency is high in Thailand, the prevalence of males with G-6-PD deficiency in Bangkok is around 12 per cent thus, male DHF patients who have G-6-PD deficiency are also expected to be 12 per cent. Besides, recently there have been reports of DHF cases who developed severe liver failure[2,3,13-15], whether or not G-6-PD deficiency has an effect on the clinical course such as liver failure in patients with DHF is our question.

Among 100 male children clinically suspicious of having DHF, only 89 cases were confirmed for the diagnosis. Definitive diagnosis by viral study was achieved in 73 cases (82%)[1]. Of the remaining 16 cases, the diagnosis was made by clinical and hematological parameters which is also considered high in predictive value[1,16].

From the present study, there were 17 cases (19.1%) who had G-6-PD deficiency. This figure is higher than the prevalence of G-6-PD deficiency of

Table 4. Abnormal initial laboratory tests in G-6-PD deficient (Group I) and non deficient G-6-PD patients (Group II).

Parameters	Group I			Group II		
	Number	%	Total	Number	%	Total
Hematocrit >40%	3	17.6	17	46	63.9	72
White blood cells <4.0 x $10^6/\mu l$	9	52.9	17	40	55.6	72
Blood urea nitrogen >20 mg/dl	2	13.3	15	10	14.9	67
Creatinine >1 mg/dl	1	6.7	15	2	2.9	67
Total bilirubin >1 mg/dl	8	50	16	1	1.6	63
Direct bilirubin >0.2 mg/dl	10	62.5	16	6	9.4	64
Indirect bilirubin >0.8 mg/dl	8	50	16	1	1.6	63
Aspartate amino transferase >100 μ/L	14	82.3	17	41	59.4	69
Alanine amino transferase >100 μ/L	7	41.2	17	18	26.0	69
Alkaline phosphatase >500 μ/L	0	0	17	0	0	64
Lactate dehydrogenase >500 μ/L	14	87.5	16	52	88.1	59
Creatine phosphokinase >150 μ/L	5	35.7	14	25	43.1	58

12 per cent in the population previously studied in Bangkok[4,5]. This implies that patients who have G-6-PD deficiency suffer from DHF more often than non deficient cases.

Among 72 non G-6-PD deficient cases, there were 4 cases (5.6%) who had gastrointestinal (GI) bleeding but no one had acute intravascular hemolysis. In the G-6-PD deficient group, the patients succumbed to more complications i.e. one case had GI bleeding but no evidence of hemolysis, three cases (17.61%) had acute intravascular hemolysis, one of which also had GI bleeding, the other one had azothemia and the last one had bleeding, shock, renal and liver failure.

Hepatic failure with or without encephalopathy is seen in DHF patients and leads to a high mortality.[2,3,13-15] The contributing factors may be multifactorial i.e. viral replication in the liver, hepatic ischemia, existing hepatitis and drugs. Hepatic ischemia may be the most important etiology because most of the reported cases had shock preceded liver failure. For cases with G-6-PD deficiency in the present study, 3 had acute intravascular hemolysis, 2 of which also had bleeding. Both events led to a drop in hemoglobin which also aggravated ischemia in the patients. One of these 3 cases had both renal and hepatic failure which are serious complications with poor outcome. Surprisingly enough, in most reports of DHF who had liver failure, evaluation of G-6-PD had not been done. One of the reasons may be due to urgent management including blood transfusion or exchange tranfusion. Most cases who had serious bleeding or hemolysis as in 7 cases in the present series, blood transfusions and/or exchange transfusions were needed. The authors recommend evaluating the G-6-PD status before transfusion or if possible at admission in DHF cases in countries where G-6-PD deficiency is prevalent. Also physicians must be aware of all the possible complications. Most of all G-6-PD deficient cases have unmeasurable enzyme activities, so this test can be done either by screening or enzymatic assay [6]. Since about 12 per cent of male donors are also expected to be G-6-PD deficient, red cell G-6-PD should be checked in each bag of blood in order to avoid G-6-PD deficient blood being given to G-6-PD deficient patients because a second episode of hemolysis may occur if oxidative stress is still present in such patients.

When the authors compared the 14 cases with G-6PD deficiency but did not have acute intravascular hemolysis (Group I) to non G-6-PD deficient cases (Group II) it was found that the hematocrit was lower while the total bilirubin and indirect bilirubin were statistically higher in group I (Table 1, 2). Hemoconcentration is one important clinical feature of DHF so that it had been used as one of the criteria for the diagnosis[1,11]. However, the criterion hematocrit value of 40 per cent may not be attained if the patients have bleeding and/or hemolysis. In the present study, an initial hematocrit of over 40 per cent was found in 63.9 per cent in Group II while only 17.6 per cent was seen in Group I. These findings along with higher than normal

levels of both total and indirect bilirubin in the G-6-PD deficient group most likely indicated some extent of extravascular hemolysis in these patients (Table 4). Acute intravascular hemolysis which did not occur indicated the capacity of the body's antioxidative defense against oxidative stress in these cases[6].

In this study, the authors found that the initial reticulocyte count in Group II was significantly lower than the control while in Group I, the reticulocyte were not increased (Table 3). On fol low-up the reticulocyte during recovery rose significantly in both groups but was more marked in Group I. Patients who have hemolysis would be expected to have reticulocytosis. In G-6-PD deficient cases, even without intravascular hemolysis (Group I), the authors expected them to have reticulocytosis in response to postulated extravascular hemolysis but such findings were not found.

During the acute phase of DHF only hematocrit was increased due to hemoconcentration while platelet, leukocyte as well as neutrophil were decreased[16,17]. These findings of cytopenia were also observed in cases of typhoid fever in whom hemophagocytosis had been demonstrated in the bone marrow smear[18,19]. Phagocytized materials included platelet, red cell precursors especially neucleated red blood cell and neutrophil. This phenomenon can be seen in many infectious conditions known as infectious associated hemophagocytic syndrome (IAHS)[20]. This is also observed in patients with DHF[17,20-23]. Hypoplasia of the bone marrow was another observation in DHF patients[17], this, in addition to hemophagocytosis should result in cytopenia in DHF. In DHF patients Group II in the present study, besides reticulocytopenia was observed during the acute phase, G-6-PD enzyme activities were also significantly lower than the normal control. However, during recovery both reticulocytes and G-6-PD enzyme levels increased significantly and caught up the normal control values which is most likely an indicative signs of recovery as has been shown in patients with typhoid fever[18]. This was seen in both groups of patients in the present study and was more obvious in Group I who were more anemic in the acute phase but had more compensation in the convalescent phase[24]. This is well explained by the fact that young red cells have higher enzyme activities and vice versa[6].

It has been previously observed that DHF patients have elevation of liver enzymes especially at a higher grade of severity.[3,11,12] Though the cases in the present study were mostly in grade II, the authors still observed that other than the aforementioned increases of indirect bilirubin in the G-6-PD deficient group, it was also found that transaminase enzymes increased in both groups but there were more cases in the G-6-PD deficient group. Marked elevation of liver enzymes was seen in one of the G-6-PD deficient cases in the presented series who had renal and hepatic failure whose aspartate aminotransferase, alanine transferase and lactate dehydrogenase were as high as 10,200, 5,980 and 27,220 u/L respectively. In both groups of patients in the present study, the highest levels of these enzymes were 3,065, 2,368 and 3,903 u/L respectively. These alterations of laboratory tests may result from hepatic injury seen in DHF[3,11,12]. These abnormal levels will decline to normal levels during recovery.

In conclusion, patients with DHF have a higher prevalence of G-6-PD deficiency than the general population. These patients had high risk of acute intravascular hemolysis, renal and hepatic failure. In suspected cases of DHF in whom hemoconcentration are not correlated with clinical features or alteration of either clinical or biochemical parameters, G-6-PD should be evaluated. Close follow-up and prompt management such as maintenance of blood pressure, fluid balance and proper correction of anemia as well as avoidance of known toxic drugs and other supportive measures should be instituted in DHF cases who had G-6-PD deficiency in order to prevent serious complications and improve the outcome.

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ผลของภาวะพร่องเอ็นซัยม์ กลูโคส-6-ฟอสเฟต ดีฮัยโดรจีเนส ต่อผู้ป่วยไข้เลือดออก

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คณะผู้วิจัยได้ศึกษาผู้ป่วยเด็กชายอายุ 1-13 ปี จำนวน 89 รายที่ป่วยเป็นโรคไข้เลือดออก และเข้ารับการรักษาในภาควิชากุมารเวชศาสตร์ คณะแพทยศาสตร์ศิริราชพยาบาล ตั้งแต่มีนาคม 2541-เมษายน 2543 พบว่า 17 ราย (19.1%) มีภาวะพร่องเอ็นซัยม์ กลูโคส-6-ฟอสเฟต ดีฮัยโดรจีเนส (จี-6-พีดี) ในเม็ดเลือดแดง ผู้ป่วยส่วนใหญ่เป็นโรคไข้เลือดออกเกรด II

มีผู้ป่วย 3 ราย จาก 17 ราย ที่พร่อง จี-6-พีดี และมีภาวะแทรกซ้อนรุนแรงคือ มีภาวะเม็ดเลือดแดงแตกเฉียบพลัน ซีดมากจนต้องให้เลือด โดยมีผู้ป่วย 1 ราย อาเจียนเป็นเลือดด้วย 1 ราย มีหน้าที่ไตผิดปกติชั่วคราว อีก 1 ราย มีไตวาย ตับวาย และมีอาการทางสมองร่วมด้วย

ในผู้ป่วยไข้เลือดออกที่ไม่มีภาวะแทรกซ้อนจากเม็ดเลือดแดงแตกรุนแรง หรือตับวาย กลุ่มที่พร่อง จี-6-พีดี จะมีระดับบิลิรูบินสูงกว่า และระดับฮีมาโตคริตต่ำกว่ากลุ่มที่มี จี-6-พีดีปกติอย่างมีนัยสำคัญทางสถิติ และพบว่าเรติคูโลไซต์ต่ำในระยะต้นของโรค แต่สูงขึ้นอย่างมีนัยสำคัญในระยะพักฟื้นในผู้ป่วยทั้งสองกลุ่ม ในผู้ป่วยกลุ่มที่ไม่พร่อง จี-6-พีดี ระดับเอ็นซัยม์ต่ำกว่าค่าคนปกติอย่างมีนัยสำคัญทางสถิติในระยะไข้ของโรค แต่ระดับเอ็นซัยม์จะสูงขึ้นอย่างมีนัยสำคัญสู่ระดับปกติในระยะพักฟื้น ส่วนผลการตรวจทางห้องปฏิบัติการอื่น ๆ ไม่แตกต่างกัน และผู้ป่วยทุกรายหายเลาและหายจากโรคจนเป็นปกติ

ความชุกของภาวะพร่อง จี-6-พีดี ในเด็กชายที่ป่วยเป็นไข้เลือดออกในการศึกษานี้เท่ากับร้อยละ 19.1 ซึ่งสูงกว่าความชุกร้อยละ 12 ที่พบในผู้ชายในกรุงเทพฯ ดังที่เคยศึกษาไว้ แสดงว่า ผู้ชายที่เป็นไข้เลือดออกร่วมกับมีภาวะพร่อง จี-6-พีดี ในเม็ดเลือดแดง น่าจะมีอาการของโรครุนแรงกว่าผู้ชายที่มีเอ็นซัยม์ จี-6-พีดี ปกติ

คำสำคัญ : ภาวะพร่อง จี-6-พี ดี, ไข้เลือดออก, ตับวาย, เม็ดเลือดแดงแตกเฉียบพลัน

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