Glucose 6 phosphate dehydrogenase deficiency diagnosed with hemolytic anemia triggered by type 1 diabetes mellitus

B. Orman[1], S. Çetinkaya[1], N. Oner[2], M. Akçaboy[3], A. Fettah[2], N. G. Lafcı[4], S. S. Erdeve[1],

University of Health Sciences, Dr. Sami Ulus Obstetrics and Gynecology, Children's Health and Diseases Training and Research Hospital, Ankara, Turkey

[1]Department of Pediatric Endocrinology,

[2]Department of Pediatric Hematology,

[3]Department of Pediatrics

[4]Department of Genetics

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INTRODUCTION

Glucose 6 phosphate dehydrogenase (G6PD) is expressed in all tissues and is necessary for the oxidant stress capacity of cells. G6PD deficiency is the most common enzymopathy in humans; it is among the important causes of hemolytic anemia.[1,2,3] It has been reported that severe hemolytic anemia due to G6PD deficiency may develop in newly diagnosed diabetes mellitus, especially during the correction of hyperglycemia. To date, 9 cases have been described in the literature.[4]

AIM

We present a case of hemolytic anemia due to G6PD deficiency secondary to newly diagnosed diabetes mellitus.

CASE REPORT

A 4-year-old male patient presented with the complaint of the polyuria-polydipsia.

Blood glucose was 750 mg/dl.

In examination;

He was weak, tachypneic and dehydrated.

Height (SDS) : 105 cm (-0,18)

Body Weight (SDS) : 13 kg (-2,29)

No hepatosplenomegaly

No goiter

No acantosis nigricans

Testicular volüme 2/2 ml

Laboratory examination in the admission are given in Table 1.

The patient was diagnosed with diabetic ketoacidosis and given intravenous insulin and hydration therapy.

Hb (g/dl)	13.1
WBC (mm^3)	6310
Platelet (mEq/L)	341.000
Glukoz (mg/dl)	750
BUN (mg/dl)	15
Creatinin (mg/dl)	0.76
Na (mEq/L)	126
K (mEq/L)	4.55
pH	7.29
HCO3(mmol/L)	12.4
BE	-12.3
Urine ketone	+
HbA1c (%)	15.3
Insulin (mIU/ml)	2.2
c-peptide (ng/ml)	0.3
Islet cell antibody	+
Anti-GAD	+
Anti- Insulin antibody	-

Table 1: Laboratory examination

On the 10th day of his hospitalization, while receiving subcutaneous insulin therapy, his hypoglycemia (45mg/dl) was accompanied by tachycardia (160/min). After his hypoglycemia resolved, his tachycardia continued. The patient, whose tachycardia continued after the hypoglycemia resolved, was examined. There was no jaundice and organomegaly on the physical examination.The patient was examined. **Hgb 8.3 g/dl** , thrombocyte 639,000/mm3, leukocyte 13.170/mm3. Anisocytosis and normoblasts were seen in the peripheral smear. Reticulocytosis (10%) was found. Hemolytic anemia was considered with these examinations. G6PD level was found to be low at 0.56 U / gr Hgb. Control Hgb was 7 g/dl, erythrocyte suspension was given, hemoglobin level was found to be 12.2 g/dl. Genetic examination of our patient was performed, and C.653C>T(p.S218F)(p.Ser218Phe) hemizygous mutation was detected in the G6PD gene.

CONCLUSIONS

Our case was diagnosed with type 1 diabetes mellitus with ketoacidosis. While the tachycardia, which developed secondary to hypoglycemia after blood glucose regulation, continued after the hypoglycemia was recovered, hemolytic anemia due to G6PD deficiency was detected. It should be kept in mind that G6PD deficiency may be present in diabetic individuals in the presence of severe anemia.

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CONTACT INFORMATION

burceayvazoglu@gmail.com