

Glycolytic Enzymes in Beta-Thalassemia

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Thalassemia is a chronic genetic disorder in which repeated blood transfusions form the mainstay of therapy. The present study was designed to evaluate the glycolytic enzymes in betathalassemia and assess change in their status after transfusion in thalasseemics.

Material and Methods

The study was conducted in the Departments of Pediatrics and Biochemistry, Burdwan Medical College between May, 1987 to August, 1988. The study material comprised 20 cases of thalassemia diagnosed on the basis of clinical, radiological and hematological evaluation, including hemoglobin electrophoresis and 20 healthy age and sex matched controls.

Blood was drawn for estimating the glycolytic enzymes (aldolase, phosphohexose-isomerase and lactic dehydrogenase by

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recommended techniques(1-3). In thalasseemics, blood samples were taken before transfusion and 4 weeks after transfusion.

Results and Discussion

The glycolytic enzymes were significantly higher ($p < 0.001$) in thalasseemics in comparison to controls. Further, there was a significant reduction in the enzymes after transfusion in thalasseemic subjects (*Table I*). The higher activity in thalassemia may be due to increased hemolysis since the glycolytic enzymes are usually located in RBCs.

It was observed that patients with lower levels of phosphohexose-isomerase and lactic dehydrogenase required less frequent transfusions to maintain hemoglobin level.

It is concluded that glycolytic enzymes

TABLE I - Glycolytic Enzymes (mean \pm SD) in Thalassemia

Group	Serum enzymes (units/ml)		
	Aldolase	Phosphohexose isomerase	Lactic dehydrogenase
Controls	9.1 \pm 1.2	9.2 \pm 0.8	180.6 \pm 5.6
Thalassemia*	37.6 \pm 3.1	34.5 \pm 3.6	752.4 \pm 15.2
<i>Thalasseemics</i>			
Pre-transfusion	11.1	21.2	228.5
Post-transfusion ⁺	8.3	15.5	201.5

* Significantly different from controls ($p < 0.001$);

+ Significantly different from pre transfusion ($p < 0.01$).

are higher in thalassemics and that these are reduced after blood transfusion. The possibility of utilizing these levels to predict transfusion requirements should be explored.

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Poisoning with Writing Ink

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Commercially available writing or marking ink contains aniline dyes as a major constituent. Aniline is a product of nitrobenzene. Fatal poisoning by ingestion of this compound, though uncommon, is occasionally reported(1). We report a patient who died of methemoglobinemia following accidental ingestion of writing ink.

Case Report

An 11-year-old girl swallowed about

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30 ml of 'Sulekha' writing ink. One and half hours later, she was found unconscious and complete pale with bluish tint. Her lips were dark purple in colour. Despite gastric lavage, oxygen administration and slow intravenous infusion of 1% methylene-blue solution in the dosage of 1 mg/kg body weight, she died 12 hours later.

Autopsy Examination

The dead body showed marked blueing of the lips, hard palate and finger nail beds. The face was livid and fine froth was present around the nostrils and mouth. Chocolate colored postmortem stains were elicited on the back. Petechial hemorrhages were observed in the cerebral hemispheres while other organs were congested.

Histopathological examination showed evidence of renal tubular necrosis and hepatic centrilobular necrosis. Blood examination by spectrophotometry showed four characteristic absorption bands in the wavelength scale, first at λ 640-628 in the red, second at λ 587-570, third at λ 550-530, and the fourth which was merged in the absorption of the blue rays, at λ 510-490. In other words, the first two absorption bands were seen between D and E, the third was between C and D, while the fourth, rather