

# EVALUATION OF S-100B, ANTIOXIDANT AND OXIDATIVE CAPACITY BEFORE AND AFTER THE TREATMENT IN CHILDREN WITH DIABETIC KETOACIDOSIS

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## Background:

Diabetic ketoacidosis is a serious condition with high rates of morbidity and mortality in children with type 1 diabetes mellitus. Calcium-binding protein S100B is a cell damage marker glycopeptide that is mainly produced by astrocytes. Oxidative stress might be defined as an imbalance between anti-oxidative defense of the body and free radical production responsible for peroxidation of lipid layer of cell walls.

## Objective and hypotheses:

In patients with diabetic ketoacidosis, high blood glucose concentration may increase oxidative stress and consequent changes in neurotransmitter by which may increase the level of S100B. In this study we aimed to investigate S100B protein levels, oxidant and antioxidant capacity in children with diabetic ketoacidosis.

## Methods:

49 healthy children and 49 children with diabetic ketoacidosis included into this study. Peripheral venous bloods were taken for tests during diabetic ketoacidosis and after recover diabetic ketoacidosis. Measurement of serum S100B was performed by ELISA kits. TAS (Total antioxidant status), TOS (Total oxidant status) and OSI (oxidative stress index) were studied at peripheral venous blood by Erel method.

## Results:

Comparing pretreatment and post-treatment of TAS, TOS, S100B levels were significantly higher in patients with diabetic ketoacidosis than healthy control group ( $p < 0.05$ ) (Table 1, Table 2). When the pre-treatment values compared with after treatment values in the patients; TAS and TOS levels were significantly higher in the pre-treatment group ( $p < 0.05$ ) but no significant difference in the levels of S100B and OSI ( $p > 0.05$ ). In our study there was also a positive correlation between S100B with the TOS and OSI values. (Respectively  $r: 0.235$ ,  $r: 0.244$ ;  $p: 0.006$ ,  $p: 0.005$ ).

Table 1.

	Before Treatment (n:49)	Controls (n:49)	p
TOS ( $\mu\text{mol H}_2\text{O}_2$ Eqv./L)	29.38 $\pm$ 8.06	16.85 $\pm$ 3.31	<0.000
TAS (mmol Trolox Eqv./L)	1.17 $\pm$ 0.21	0.78 $\pm$ 0.16	<0.000
OSI (Arbitrary Unite)	2.64 $\pm$ 1.23	2.22 $\pm$ 0.56	0.019
S100B ( $\mu\text{g/mL}$ )	130.11 $\pm$ 35.16	106.35 $\pm$ 30.14	<0.000

Table 2.

	After Treatment (n:49)	Controls (n:49)	p
TOS ( $\mu\text{mol H}_2\text{O}_2$ Eqv./L)	23.79 $\pm$ 4.60	16.85 $\pm$ 3.31	<0.000
TAS (mmol Trolox Eqv./L)	0.91 $\pm$ 0.27	0.78 $\pm$ 0.16	0.007
OSI (Arbitrary Unite)	2.87 $\pm$ 1.23	2.22 $\pm$ 0.56	0.001
S100B ( $\mu\text{g/mL}$ )	136.4 $\pm$ 42.01	106.35 $\pm$ 30.14	<0.000

## Conclusions:

In diabetic ketoacidosis, increase of oxidative stress and S100B may be depending on high glucose level. Increasing concentrations of these factors in blood may be therefore indicative for either neuronal damage, impaired blood brain barrier function, or both.