SERUM LEVELS OF ACETYLCHOLINESTERASE IN METABOLIC SYNDROME

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It is known today that low grade inflammatory mechanisms play a pivotal role in the pathogenesis of cardiometabolic diseases including the metabolic syndrome (1-4). In the same vein, in addition to its role in cholinergic neurotransmission, acetylcholine exerts a potential anti-inflammatory effect mediated by “cholinergic anti-inflammatory reflex” (5). Accordingly, a positive correlation of the blood serum level of acetylcholinesterase, butyrylcholinesterase and C-reactive protein (CRP) was reported in obese and diabetic patients (6-10).

In the present study we measured serum levels of acetylcholinesterase, an enzyme hydrolyzing acetylcholine, and CRP, a known inflammatory marker, in patients with mild stage (n=14) and with advanced stage (n=14) of metabolic syndrome, according to the criteria of the United States National Cholesterol Education Program’s Adult Treatment Panel III (ATP III). The control group comprised of age- and sex-matched healthy subjects (n=7).

In patients with advanced metabolic syndrome both acetylcholinesterase and CRP serum level was significantly elevated as compared to that in mild metabolic syndrome patients and in controls.

In mild metabolic syndrome acetylcholinesterase level was in the range of 8 972 U/L, a similar to that in controls. Whereas in advanced metabolic syndrome the level of acetylcholinesterase was significantly increased: 12 000 U/L $\pm$ [F(1.26)=31.96, $p<0.01$] (Fig. 1); this correlated with the elevated CRP level (data not shown).

Figure 1. Serum levels (in U/L) of acetylcholinesterase in patients with mild and advanced metabolic syndrome as compared to controls.

Since the increased acetylcholinesterase serum level may serve as a biomarker for the progression of inflammation in metabolic syndrome, the inhibitors of acetylcholinesterase as well as agonists of nicotinic acetylcholine receptors (11) may open new field of research on the pharmacotherapy of metabolic syndrome.

We have previously reported that the circulating levels of both nerve growth factor (NGF) и brain-derived neurotrophic factor (BDNF) were significantly reduced in patients with advanced metabolic syndrome (12, 13). Since cholinergic anti-inflammatory control is compromised in diabetes and metabolic syndrome (6-10 and the present data) and in Alzheimer’s disease

(14, 15) and since NGF and BDNF are also implicated in the pathogenesis of cardiometabolic and neurodegenerative diseases (16, 17), further studies are required on the possible role of NGF/BDNF and acetylcholinesterase/CRP in the development of these diseases.

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