INFLUENCE OF HBA1C ON NITRIC OXIDE LEVEL IN PATIENTS WITH TYPE 2 DIABETES MELLITUS

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Key words: type 2 diabetes, glycated gemoglobin, Nitric Oxide.

Introduction: In 21-st century, type 2 diabetes (T2D) has become a global health and social problem in the whole world. The number of persons, suffering from T2D is huge: more than 382 million people have diabetes, predominantly T2D, and by 2035 this number will have risen to 592 million. The goal of treatment for patients with T2D is to prevent complications of diabetes - macrovascular diseases (heart disease, stroke and peripheral vascular disease) and microvascular diseases (retinopathy, neuropathy and nephropathy). Obtaining HbA1c below 7% is the main condition to achieve this goal. Nitric oxide (NO) plays an important role in maintaining vascular homeostasis. Loss of NO function is one of the earliest indicators of disease and its progression especially in patients with T2D (activation of NO synthase (NOS) is under insulin control through the Akt pathway).

Aim: to compare NO level between patients with well and bad controlled glycemia in T2D.

Methods: The study included 32 patients with T2D. The diagnosis of T2D was confirmed due to International Diabetes Federation (IDF) criteria 2015. Patients were divided into two groups: with well controlled glycaemia (HbA1c < 7%) and bad controlled glycaemia (HbA1c > 7%). The control group consists of 15 healthy subjects.

Results: NO level in patients with T2D is significantly higher (27,2 ±3,1 μmol), compared to controls (18,86±0,9 μmol; p < 0,001). A significant difference in NO level was found between patients with bad controlled glycaemia (25,9±2,2 μmol) and well controlled glycaemia (28,7 ± 3,0 μmol; p<0,01). The study showed a moderate negative correlation between NO level and HbA1c (-0,399; p< 0,05).

Conclusions: Production of NO is impaired in patients with T2D, especially with bad controlled glycaemia. With increase in HbAc serum NO decreases. This can be the main target for prevention vascular complication in T2D.

DRUG-INDUCED ESOPHAGITIS: NEW LOOK ON OLD PROBLEM

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Key words: esophageal mucosa, drug side effects, H_2S.

Drug-induced esophagitis is being recognized increasingly in the past few years. Since 1970 more than 30 or more medications have been reported caused drug-indused esophagitis (old name pill-induced esophagitis).

Clinical data have shown that drug-induced esophageal injury tends to occur at the anatomical site of narrowing, with the middle third behind the left atrium predominating (75.6%) patients on nonsteroidal anti-inflammatory agents whose injury is aggravated by gastroesophageal reflux (21.8%) (reflux aggravated).

Severe esophageal injury has been reported in some women taking biphosphonates as treatment for postmenopausal osteoporosis. Endoscopic findings in such patients with esophageal injury generally suggested a chemical esophagitis, with erosions or ulcerations and

exudative inflammation accompanied by thickening of the esophageal wall. Thus, the most important aspect of therapy is to make the correct diagnosis and then to avoid re-injury with the drug.

Last data have shown that H_2S contributes significantly to mucosal defence in the esophagus, as in other parts of the GI tract. Modification of endogenous H_2S synthesis with enrichment provides a novel approach that can be useful in NSAID-related drug-induced esophagitis.

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