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Original Research Article

Evaluation of vitamin D levels in allergic and non-allergic asthma

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ABSTRACT

Background and objective: Some researches show that low vitamin D may play a role in asthma pathogenesis. The aim of this study was to evaluate the serum vitamin D level in asthmatics with different phenotypes and to determine its associations with lung function, IgE, eosinophil count and body mass index (BMI).

Materials and methods: The study population comprised 85 patients with asthma and 73 healthy persons. Patients with asthma were divided into groups according to phenotypes. Allergy was assessed using a skin prick test and measuring eosinophil count in peripheral blood and total IgE in serum. Lung function was evaluated by spirometry. Concentration of vitamin D (25(OH)D3) was measured using a commercial ELISA kit. Smoking history was assessed and BMI was calculated for all individuals.

Results: The vitamin D level was lower in asthmatics than in the control group (14.36 ± 0.57 vs. 22.13 ± 0.84 ng/mL, $P < 0.01$). There were no significant differences in the vitamin D level between the groups with allergic and non-allergic asthma (14.36 ± 0.77 vs. 14.35 ± 0.74 ng/mL). The low vitamin D level increased the risk of asthma 1.2 times (OR, 1.194; 95% CI, 1.109–1.286, $P < 0.01$). The vitamin D level did not correlate with lung function and markers of allergy in asthmatic patients. The vitamin D level correlated with FEV1/FVC ($rs = 0.72$, $P < 0.05$) in smoking patients with asthma. Correlation between the vitamin D level and BMI was found in all studied subjects ($rs = -0.18$, $P < 0.05$).

Conclusions: The vitamin D level was lower in asthmatic patients than in healthy individuals despite their hypersensitivity and increase risk of asthma. There was no relation between the vitamin D level and lung function, eosinophil count and total IgE level, whereas the lower vitamin D level was associated with higher BMI.

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1. Introduction

Asthma is a chronic inflammatory airway disease, which pathogenesis has not been fully investigated yet. Despite the progress of medical science in asthma field, morbidity of this disease remains high worldwide [1]. A more comprehensive understanding of asthma mechanisms may lead to discover more specific diagnostic methods and ways of treatment and prevention.

It is known that many cells are involved in the development of asthma: mast cells, eosinophils, neutrophils, T lymphocytes, macrophages and epithelial cells [2]. T helper 2 cells (Th2) activates interleukin (IL) 5 and granulocyte macrophage colony-stimulating factor (GM-SCF), which induce angiogenesis, differentiation and chemotaxis of eosinophils, and IL 13, which increases airway remodeling and inflammation [3,4]. However, scientists still discover new cytokines, mediators, proteins and other substances that may be involved in asthma pathogenesis.

Vitamin D is a fat-soluble nutrient, which is the best known as a key factor in bone mineralization [5,6]. Vitamin D3 is converted to 25(OH)D in liver and later 25(OH)D is converted into the active form 1,25(OH)2D in kidneys [6]. Some studies showed that role of vitamin D was wider [7–9]. This nutrient may participate in pathogenesis of oncological, endocrine, cardiovascular, psychiatric, autoimmune and allergic diseases including asthma [10–14]. The role of vitamin D in asthma may be explained by its impact on T cell [3,4,12,15]. It was shown that Th1/Th2 ratio and elevated inflammatory mediators were significantly correlated to 25(OH)D levels [12]. Vitamin D induces a higher level of IL-10, which is known as anti-inflammatory cytokine [15]. Receptors of vitamin D are localized in number of tissues including respiratory epithelial cells and bronchial smooth muscle [16]. Pulmonary vitamin D receptors are important for the conversion of 25(OH)D into 1,25 (OH)D in respiratory epithelial cells, moreover, vitamin D receptors and hydroxylase that metabolizes 1,25(OH)D synthesis are increased in bronchial smooth muscle cells [17,18]. 1,25(OH)D has been shown to have anti-inflammatory effect in many tissues including lung tissue [19]. Moreover, a number of genes related to vitamin D may be involved in asthma pathogenesis: some of genes are associated with asthma and atopy; other genes, only with asthma [20].

We aimed to assess serum vitamin D level in asthmatics with different phenotypes according to their allergic and smoking status, and to evaluate its possible relation to lung function, total IgE, eosinophil count and body mass index (BMI).

2. Materials and methods

2.1. Study population

A total of 158 individuals aged more than 18 years were enrolled into the study. Of them, 85 patients had stable asthma, which was controlled with low and medium doses of inhaled glucocorticoids (diagnosed according to the Global Initiative for Asthma [GINA] recommendations) and 73 were healthy subjects [1]. Subjects with asthma were divided into two groups according to their allergic status: with allergic asthma ($n = 56$) and non-allergic asthma ($n = 29$). Patients with allergic asthma were additionally subdivided into 3 subgroups according to the results of a skin prick test: hypersensitivity to one inhaled allergen, to more than one inhaled allergen, and to mixed allergens (inhaled and food). None of the subjects showed signs of acute respiratory infection at least one month before the study. The exclusion criteria were as follows: any acute or chronic respiratory diseases (except asthma), pregnancy, autoimmune and oncologic diseases.

Subjects were divided according to their smoking history into asthmatic smokers ($n = 11$) who were current smokers and asthmatic never-smokers ($n = 74$). Smoking was assessed in pack-years expressed as the product of tobacco use (in years) and the average number of cigarettes smoked per day/20.

BMI was calculated according to the formula BMI = weight (kg)/height (m^2) for all individuals [21].

The study was approved by the Regional Bioethics Committee at the Lithuanian University of Health Sciences (No. BE-2-31). Subjects gave their informed consent.

2.2. Lung function testing

Lung function was evaluated using a CustovitM pneumotachometric spirometer (Custo Med, Germany). All subjects were asked to avoid the use of short-acting β2-agonists for at least 8 h before the testing. Patients were investigated in a sitting position, and a nose clip was used. Forced expiratory volume in one second (FEV_1) and forced vital capacity (FVC) were measured. FEV1/FVC ratio was calculated. The best value of the three measurements was selected. Normal values were defined according to Quanjer et al. [22].

2.3. Evaluation of allergic sensitization

Allergic sensitization was assessed using the skin prick method of Pepys [23] with standard (glycerin-preserved) allergens (Stallergens, France). Skin prick testing was performed on the forearm with common aeroallergens and food allergens as well as histamine (positive control) and glycerin (negative control). The reaction was measured after 20 min. The reaction was considered as positive if the diameter of the wheal was 3 mm or greater.

2.4. Peripheral blood collection and processing

Blood eosinophil count and serum total IgE were measured in peripheral blood. Peripheral blood was collected by peripheral venipuncture according to the standard procedure. Blood samples were drawn into BA vacutainer K3 EDTA tubes for further enumeration of eosinophils with the ADVIA 120 automated hematology analyzer (Germany) and into serum tubes, stored at room temperature for 30–60 min and centrifuged for 15 min at 4000 rpm. Serum samples were immediately frozen at 70 °C for further analysis.

2.5. Measurements of vitamin D level and IgE

Concentration of vitamin D (25(OH)D3) in serum was measured by the enzyme-linked immunosorbent assay ELISA using DIAsource 25OH vitamin D Total ELISA kit (Louvain-la Neuve,