EDITORIAL

Association between selenium levels and thyroid function in patients with heart disease

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Selenium (Se) is an essential nutritional element, present in nature and in living organisms in organic and inorganic forms. The main organic forms are selenomethionine and selenocysteine, while the most common inorganic forms are selenite and selenate.[1] In human body, Se is largely used to produce the amino acid selenocysteine, which is cotranslationally incorporated into selenoproteins. Selenoproteins are essential components of antioxidant defense, and Se bioavailability is a limiting factor in the synthesis of numerous selenoproteins.[2] As shown by experimental studies, glutathione peroxidase deficiency may be involved in endothelial dysfunction, cardiac abnormalities, development of atherosclerosis, as well as may adversely affect prognosis in patients with coronary artery disease.[2,3] Recent studies have shown that individuals at risk of cardiovascular disease often have low plasma Se concentrations, which are associated with increased levels of acute phase proteins such as C-reactive protein, a phenomenon attributed to the activation of nuclear factor-κB.[2,4]

In a recent study by Frączek-Jucha et al[5] published in this issue of *Kardiologia Polska* (*Kardiol Pol*), the authors investigated Se concentrations in patients with myocardial infarction (MI) or heart failure (HF), as well as their relationship with thyroid hormone levels. A total of 143 participants were divided into 3 groups: MI group (54 patients), HF group (59 patients), and control group (30 healthy volunteers). All groups had blood samples collected for the analysis of Se concentrations, cardiac function, and thyroid activity. Following this, 4 subgroups were distinguished depending on Se concentrations: patients with MI or HF with Se levels below the normal values and patients with MI or HF without reduced Se levels. The results showed a high prevalence of patients with lower Se concentrations (below the recommended level) when compared with controls. However, the authors did not report a strong relationship between the Se concentration and thyroid function. Although they revealed variations in thyrotropin levels during the acute phase of MI, this finding was no longer observed during follow-up. Thus, the authors concluded that patients with both MI and HF had lower Se levels than controls, but there was no significant correlation between reduced Se levels and either thyroid or cardiac function.

Patients with heart disease may show inadequate food intake, proinflammatory state activation, and oxidative stress.[6] All these conditions are associated with reduced nutrient absorption, which may occur due to the presence of abdominal edema as well as increased permeability and chronic inflammation of the mucosa, which in turn may alter the intestinal function and prevent an adequate amount of micro- and macronutrients from being absorbed.[7] Not only the absorption but also the metabolism of some nutrients may be impaired in cardiac patients, who could have oxidative stress that can alter microelements such as Se and zinc as well as systemic inflammatory response that can modify plasma Se levels.[8] Duncan et al[9] reported a negative relationship between the plasma Se concentration and the magnitude of the inflammatory response assessed by C-reactive protein concentrations. Selenium, zinc, iodine, and iron participate directly in the formation of thyroid hormones (triiodothyronine, thyroxine).[10] Both excess and deficiency of thyroid hormones can lead to thyroid dysfunction. The prevalence of thyroid dysfunction in patients with

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HF ranges between 1.3% and 21%, depending on thyrotropin levels, age, and iodine intake.[11] Another factor affecting the prevalence of thyroid dysfunction is the functional class of patients with HF. Iacoviello et al[12] found that more advanced functional classes are associated with a higher risk of thyroid dysfunction. The same was shown by Lima et al[13] and Güder et al,[14] who also found significant differences between functional class and thyrotropin levels, older age, serum urea concentrations, lower body mass index, elevated total cholesterol levels, and systolic blood pressure. In patients with HF, thyroid dysfunction was shown to be related more to the severity of the disease than to nutritional aspects, such as nutrient levels (mainly micronutrients like selenium).

Although Frączek-Jucha et al[5] were able to demonstrate a correlation between reduced Se levels and abnormal thyroid hormone levels in patients with HF and MI, some questions remain unanswered. Research that focused on the importance of Se deficiency in the development of cardiovascular diseases has indicated that the association between low Se intake and cardiovascular disorders might result from increased oxidative stress and its sequelae,[15] highlighting the need to investigate the selenoproteins involved in cardiovascular stress response as well as glutathione peroxidases, thioredoxin reductases, and selenoprotein R.[15] Another issue that should be considered apart from the measurement of circulating Se levels and the activity of Se-dependent enzymes is the presence of a polymorphism that might affect Se distribution and subsequently the development of CVD.[15]

ARTICLE INFORMATION

DISCLAIMER The opinions expressed by the author are not necessarily those of the journal editors, Polish Cardiac Society, or publisher.

CONFLICT OF INTEREST None declared.

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REFERENCES

1 Mehdi Y, Hornick JL, Istasse L, Dufrasne I. Selenium in the environment, metabolism and involvement in body functions. Molecules. 2013; 18: 3292-3311.

2 Lubos E, Sinning CR, Schnabel RB, et al. Serum selenium and prognosis in cardiovascular disease: results from the AtheroGene study. Atherosclerosis. 2010; 209: 271-277.

3 Okuyama H, Langsjoen PH, Hamazaki T, et al. Statins stimulate atherosclerosis and heart failure: pharmacological mechanisms. Expert Rev Clin Pharmacol. 2015; 8: 189-199.

4 Koşar F, Sahin I, Taşkapan C, et al. Trace element status (Se, Zn, Cu) in heart failure. Anadolu Kardiyol Derg. 2006; 6: 216-220.

5 Frączek-Jucha M, Kabat M, Szlósarczyk B, et al. Selenium deficiency and the dynamics of changes of thyroid profile in patients with acute myocardial infarction and chronic heart failure. Kardiol Pol. 2019; 129: 674-682.

6 Sahade V, Montera VSP. Nutritional treatment for heart failure patients [in Portuguese]. Rev Nutr. 2009; 22: 3.

7 Romeiro FG, Okoshi K, Zornoff LAM, Okoshi MP. Gastrointestinal changes associated to heart failure [in Portuguese]. Arq Bras Cardiol. 2012; 98: 273-277.

8 Stefanowicz FA, Talwar D, O'Reilly DS. Erythrocyte selenium concentration as a marker of selenium status. Clin Nutr. 2013; 32: 837-842.

9 Duncan A, Talwar D, McMillan DC, et al. Quantitative data on the magnitude of the systemic inflammatory response and its effect on micronutrient status based on plasma measurements. Am J Clin Nutr. 2012; 95: 64-71.

10 Zimmermann MB, Köhrle J. The impact of iron and selenium deficiencies on iodine and thyroid metabolism: biochemistry and relevance to public health. Thyroid. 2002; 12: 867-878.

11 Pasqualetti G, Tognini S, Polini A, et al. Subclinical hypothyroidism and heart failure risk in older people. Endocr Metab Immune Disord Drug Targets. 2013; 13: 13-21.

12 Iacoviello M, Guida P, Guastamacchia E, et al. Prognostic role of sub-clinical hypothyroidism in chronic heart failure outpatients. Curr Pharm Des. 2008; 14: 2686-2692.

13 Lima LF, Barbosa F Júnior, et al. Heart failure, micronutrient profile, and its connection with thyroid dysfunction and nutritional status. Clin Nutr. 2019; 38: 800-805.

14 Güder G, Gelbrich G, Edelmann F, et al; Competence Network Heart Failure Germany. Reverse epidemiology in different stages of heart failure. Int J Cardiol. 2015; 184: 216-224.

15 Benstoem C, Goetzenich A, Kraemer S, et al. Selenium and its supplementation in cardiovascular disease-what do we know? Nutrients. 2015; 7: 3094-3118.