Central Obesity and Normochromic, Normocytic Anemia

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Introduction

According to the World Health Organization (WHO) reports, more than 1 billion adults are overweight and at least 300 million of them are obese (1). It was reported that Obesity is associated with anemia in adults in some countries (2-10). Inflammation associated with obesity which is a mechanism that links anemia with obesity (11). Adipose tissue is currently recognized as an endocrine organ. It can contribute to the development of an inflammatory process by secreting pro-inflammatory cytokines and adipokines. Adipose tissue produces many cytokines and adipokines such as interleukin-6 (IL-6), interleukin-1β (IL-1β), interleukin-8 (IL-8), tumor necrosis factor alpha (TNF-
alpha), leptin, adiponectin, resistin, lipocalin-2, C-reactive protein (CRP), monocyte chemoattractant protein 1 (MCP-1), complement components, plasminogen activator inhibitor-1 (PAI-1). (12) Cepeda-Lopez et al has shown that in obese Mexican women and children, CRP concentrations were 4 times more than normal-weight counterparts. (13) Also studies have shown that the CRP concentration decreases significantly after weight loss. (14) Anemia of inflammation (anemia of chronic disease) is characterized by impaired mobilization of iron stores, blunted response to erythropoietin and decreased erythrocyte life span. It is a hypo proliferative anemia accompanied by mildly elevated serum ferritin, hypoferremia, and low transferrin saturation despite adequate reticuloendothelial iron stores (15). Karlee J. Ausk et al has showed that in overweight and obese persons were not more likely to be anemic compared with normal-weight persons but they saw that increasing BMI is associated with higher serum ferritin levels and lower serum levels of iron. Ferritin, although reduces during iron-deficiency anemia but it is an acute phase reactant so in obesity will increase. (2) Hepcidin is a negative regulator of iron metabolism. It inhibits the absorption of iron in the small intestine and the release of recycled iron from macrophages. The synthesis of hepcidin in the liver is increased by inflammation. Hepcidin is an important hormone regulating iron metabolism during anemia of chronic disease (16) it can explain why anemia induced by chronic disease can get hypochromic and microcytic (17) while it is usually Normochromic and normocytic (18) The main mechanism that explained obesity-induced anemia is inflammation. We know that anemia of inflammation is Normochromic, normocytic anemia. The aim of our study was to clarify how much of Normochromic, normocytic anemia is accompanied by central obesity.

Materials and Methods

In this cross sectional study we collected data from 406 persons (18-65 years old) that referred to endocrine clinic in Yazd province of Iran from January 2012 to January 2013. These patients were selected from 2120 patients. The patients referred to the clinic with a complaint other than anemia or obesity. They were excluded from the study if they had history of malignancy, hypo or hyperthyroidism, diabetes, growth hormone disorder, rheumatologic disorder, corticosteroid consumption.

For the patients, we request a complete blood count (CBC) and also their weight, height, waist circumference and hip circumference were measured. The normal range for these criteria has been shown in Table 1. Patients with Normochromic, normocytic anemia were diagnosed based on hemoglobin concentration, mean cell volume and mean cell hemoglobin. Central obesity was determined according to waist and hip circumference. In this study, central obesity was assessed by waisttohip ratio. We compare the central obesity frequency in patients with Normochromic, normocytic anemia and control group (the others). The data were evaluated by SPSS17 software and T-test was done.

Results

In this study, 406 people who were referred to endocrine clinic in Yazd province of Iran were studied. Totally 67 patient had anemia, that

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<tr>
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among them 26 patient suffering from Normochromic, normocytic anemia (NNA). From these patients, 11 patients (%42.3) had central obesity according to waist to hip ratio. At last, 57% of patients had central obesity and 10% were anemic but among patients with central obesity 12% were anemic.

As it has been shown in Table2, there were no significant difference between central obesity criteria (W/H ratio) in patients with Normochromic normocytic anemia and the others. (P value >0.05)

The obese patients who had central obesity and also Normochromic, normocytic anemia, were analyzed for their iron parameters. Iron/TIBC was abnormal only in 3 of them. As it is shown in Table2, the NNA is more frequent in men than women but hypo chromic microcytic anemia was seen in women more than men (%68 Vs. %32) but it wasn’t significant. (P value > 0.05)

In the next step we compare the type of anemia in patients with central obesity that were anemic. Overall, 49 patients had anemia and central obesity together. About 53% of them had Normochromic normocytic anemia and the other hypo chromic microcytic anemia.

**Conclusion**

In this study, from 406 patients who was screened for anemia, we obtain 26 patients with Normochromic, normocytic anemia and %42.3 among of these patients had central obesity. In control group, 58.4% had central obesity. As it was mentioned, central obesity introduced as an inflammatory state because adipose tissue can secrete inflammatory agents. (12)

One study showed that diabetic patients had higher ferritin levels than non-diabetic patients (22). Also non-diabetic patients with metabolic syndrome showed higher ferritin levels than non-diabetic patients without metabolic syndrome (23). It can reject the hypoferremia as the reason of anemia in diabetic and obese patients because during iron deficiency anemia, ferritin is lower than normal range. In spite of all that, some studies conclude that iron deficiency due to hepcidine secretion is the mechanism of anemia in obese patients (2). The most patients with anemia and central obesity had Normochromic normocytic anemia. This fact supports this hypothesis that inflammation is the main mechanism of anemia in central obesity.

In this study, we did not find any correlation between Normochromic, normocytic anemia and central obesity. we excluded the patients with inflammatory path physiology in this study but it is not practical that exclude all of them. Nevertheless, we had 11 persons who had anemia of chronic disease but we don’t find any source of inflammation except central obesity. Thus it can introduce central obesity as a differential diagnosis for anemia of chronic disease.

Because central obesity is a common and prevalent problem in world due to low physical activity and high caloric foods, it is necessary to evaluate central obesity in large sample size and also check inflammatory cytokines such as interleukins for this purpose. Also we recommend that in future studies, comparison of ferritin level in persons with central obesity and control group would be done.
References