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Plasma levels of omentin-1 and visfatin in senile patients with coronary heart disease and heart failure

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ABSTRACT

Objective: To investigate the alteration of plasma levels of omentin-1 and visfatin in elderly patients with coronary heart disease (CHD) and heart failure. **Methods:** Plasma omentin-1 and visfatin levels were measured in 90 subjects (29 stable angina pectoris (SAP) cases, 30 unstable angina pectoris (UAP) cases and 31 age- and sex-matched healthy controls (age $\geqslant$ 60 years) by enzyme-linked immunosorbent assay methods. According to the New York Heart Association classification, 59 CHDs were divided into three groups: functional I class, 11 cases; functional Ⅱ/Ⅲ class, 36 cases; and functional IV class, 12 cases. **Results:** The plasma level of omentin-1 in CHD patients was significantly lower than that of the control group. Omentin-1in SAP group and UAP group were significantly lower compared to the control group (there was no statistical significance between UAP group and SAP group; $P>0.05$). The plasma level of visfatin in CHD patients was significantly higher than that of the control group. Similarly, visfatin in SAP group and UAP group were all significantly higher compared to the control group, while there was no statistical significance between UAP group, and SAP group. The plasma omentin-1 level was negatively correlated with SBP ($r=-0.264$, $P<0.05$), positively correlated with HDL-c level ($r=0.271$, $P<0.05$); the plasma visfatin level was positively correlated with TC ($r=0.292$, $P<0.05$), negatively correlated with HDL-c level ($r=-0.266$, $P<0.05$). There was a negative correlation between plasma omentin-1 and visfatin levels ($r=-0.280$, $P<0.05$). Moreover, multiple linear stepwise regression analysis showed that omentin-1 and visfatin levels might be affected by HDL-c level. Logistic regression analysis showed that visfatin could be an independent risk factor of CHD. **Conclusions:** Decreased levels of omentin-1 and increased levels of visfatin may be involved in the occurrence and development of CHD. Omentin-1 and visfatin, independently, may be protective and pro-inflammatory cytokines. Additionally, both omentin-1 and visfatin may be related to lipid metabolism. Visfatin may be an independent risk factor of CHD.

1. Introduction

Coronary heart disease (CHD) is one of the primary diseases leading to death worldwide. CHD is due to coronary atherosclerosis (AS) stenosis or occlusion that leads to myocardial ischemia hypoxia. Coronary artery disease (CAD) not only includes lipid accumulation within the artery wall; it also involves inflammatory reactions and other immune processes in the occurrence and development of AS. Recent studies have found that in addition to regulating glucose and lipid metabolism, some adipocytokines, such as omentin and visfatin, also play an important role in regulating immune response and inflammation[1].

In 2003, Yang *et al*[2] suggested omentin as a new adipocytokine secreted from the omental adipose tissue. Omentin was demonstrated to enhance insulin-mediated

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glucose-uptake in adipocytes, activate proteinkinase Akt/PKB, and improve the insulin sensitivity of fat cells. Yamawaki *et al*[3] demonstrated for the first time that omentin plays an anti-inflammatory role by preventing the TNF- α -induced COX-2 expression in vascular endothelial cells. Kazama *et al*[4] found that omentin inhibits TNF- α -induced inflammation of invascular smooth muscle cells (SMCs), and its anti-inflammatory role is attributed, at least in part, to the inhibition of superoxide production. Omentin has two isoforms: omentin-1 and omentin-2; omentin-1 was shown to be the major circulating isoform in human plasma[3]. In addition, omentin-1 was also regarded as a protective cytokine in the metabolic imbalance condition of the body[5]. Previous research found that the plasma levels of omentin were decreased in patients with CHD, which indicated that omentin-1 may also be involved in the occurrence of coronary AS. However, research in the involvement of omentin in the development of heart failure patients with CHD is limited.

In 2005, Fukuhara *et al*[6] discovered visfatin-another adipocytokine mainly secreted from visceral adipose tissue that has+ insulin-mimetic effects and roles in glucose and lipid metabolism regulation. The inflammatory response, immune regulation, and other biological activity are closely associated with obesity, type 2 diabetes and insulin resistance. Sonoli *et al*[7] found that visfatin had antiapoptotic activity and had a regulatory role in inflammation. Plasma levels of visfatin increased with the increase of C-reactive protein (CRP) levels, further suggesting visfatin's inflammatory effects[8]. Relevant research indicated that these inflammatory effects increased the risk of cardiovascular events in patients with obesity. Malyszko *et al*[9] reported that visfatin is also related to hs-CRP. Moreover, several studies have found that visfatin can up-regulate the expression of inflammatory cytokines (such as IL-6, IL-8, MCP-1), and consequently, these inflammatory cytokines can increase the expression of visfatin, thus accelerating the development of cardiovascular disease. Taken together, these research results suggest that there may be a close link between inflammation and adipokines. Other studies, however, found that visfatin had no correlation with hs-CRP; thus, visfatin may be unrelated to the pro-inflammatory effect. Although there are arguments about whether visfatin is a pro-inflammatory cytokine or an anti-inflammatory factor, recent studies lean towards the claim that visfatin is a novel inflammatory regulator with pro-inflammatory effects. Lastly, research suggests that visfatin also plays an important role in the process of unstable atherosclerotic plaque[10]; however, the mechanism of visfatin in cardiovascular diseases is still not clear. Therefore, our study's aim is to observe the changes of plasma omentin-1 and visfatin levels, and discuss their effects in the occurrence of CHD within a group of elderly CHD patients.

2. Materials and methods

2.1. Research objects

All patients were selected at the Second Xiangya Hospital of Central South University from July 2012 to December 2012 by coronary angiography or coronary CT angiography. Fifty-nine elderly patients diagnosed with CHD were chosen and 31 healthy subjects served as a control group. All subjects were aged over 60 years. Based on clinical symptoms, the CHD group was divided into two groups: a stable angina pectoris (SAP) group and an unstable angina pectoris (UAP) group. With regards to the CHD grading of New York Heart Association (NYHA) classification method, the CHD group was divided into three groups based on the conscious activity found within patients, namely functional Ⅰ class, 11 cases; functional Ⅱ/Ⅲ, class 36 cases; and functional Ⅳ, class 12 cases. All subjects signed informed consent.

2.2. Diagnostic criteria

Stable angina pectoris is due to typical angina pectoris attack caused by myocardial ischemia and hypoxia. Its clinical manifestation is relatively stable within 1 to 3 months, with same pain-onset time daily and weekly, with equal emotional and labor pain evoked, and with no changes in nature/ range/ duration in each attack. Relief time of pain is also similar with nitroglycerin.

Unstable angina pectoris is a formerly stable angina pectoris. Within one month, the pain increased seizure frequency, severity, and duration. Factors, such as nitrates, with alleviating effect are reduced. Moreover, new angina pectoris occur within one month and pain occurs due to lighter load; it can occur while at rest or can be induced by slight activity.

Heart function classification: according to the NYHA classification method proposed in 1928, the degree of activity induced heart failure symptoms and heart function was divided into four levels.

Diagnostic criteria of the elderly: in accordance with the elderly branch of standard Chinese Medical Association in 1990, subjects should be 60 years of age or older.

2.3. Exclusion criteria

Subjects with the following were excluded: severe liver/ kidney dysfunction; severe systemic disease (such as the diseases of respiratory system/ digestive system/ nervous system etc.); malignant tumor; acute/ chronic infectious

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