Assessment Of Visfatin Level In Patients With Coronary Heart Disease

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Abstract. In the current study, seventy of patients were divided into three study groups: acute myocardial infarction (AMI) patients group included 21 subjects, unstable angina (UA) patients group included 23 subjects and stable angina (SA) patients group included 26 subjects, the control group was composed of 20 healthy. The samples were collected from the Coronary Care Unit (CCU) in Al-Sadder Teaching Hospital in Al-Najaf province/ Iraq, during the period from September till December 2018. The ages of patients and control ranged from 30 to 69 years old, Each patients was divided into subgroups according gender, type of disease, and age. The current study indicated a significant increase (P<0.05) in serum VF level of CHD compared with healthy group. The results also revealed a significant increase (p<0.05) in serum VF the level in AMI group as compared with UA group and SA group. The current study indicated no significant differences (p>0.05) in serum level between males and females of CHD patients. The results also indicated when compared with (30-39y), (40-49y), (50-59y) and (60-69y) of CHD, a significant increase (p<0.05) in serum level VF there was a significant increase (p<0.05) among different age. Conclusion: The present study concluded that Visfatin level were good marker for detection and diagnosis of coronary heart disease in male and female patients.

Keywords: Coronary heart disease, Visfatin, Angina, Myocardial infraction

1. Introduction

Coronary heart diseases (CHD) are the most common cause of morbidity and mortality in most countries (Sebregts et al., 2000), also due to the significant impact of coronary heart disease, it is important for identifying the determinants of risk for developing this disease, coronary artery diseases are the generic designation for the three forms of cardiac diseases, i.e., angina pectoris {unstable angina (UA), stable angina (SA)}, sudden cardiac death and acute myocardial infarction (AMI), In most cases, the abnormal outcomes from inadequate blood flow resultant lead to the development of atherosclerosis causing in narrowing of the coronary arteries, thus, CHD is {also often called coronary artery disease (CAD) or ischemic heart disease (IHD)( Rydén et al., 2007; Naveen, 2009).

Visfatin (VF) is a recently discovered adipokine with different functions, Visfatin is mainly found in visceral adipose tissue and mimics insulin in lowering plasma glucose levels (Fukuhara et al., 2005;
Shaker et al., 2011). Visfatin, other than the fat tissue, is expressed by various other cells and tissues such as neutrophils, liver, heart and muscles as well as accepted as a growth factor for the maturation of pro-cells of the B-lymphocytes (Kitani et al., 2003; Gürsoy et al., 2014). The product of Visfatin/PBEF gene was initially identified by Samal and his colleagues in (1994) as a cytokine subsequently named pre-B-cell colony-enhancing factor (PBEF) that is expressed in lymphocytes of peripheral blood and plays a role in lymphocytes maturation and inhibition of neutrophil apoptosis (Jia et al., 2004). There is a direct association between visfatin levels and increased cardiovascular disease (Filippatos et al., 2013) and it had a role in many pathophysiological processes that eventually lead to cardiovascular disease such as hypertension and atherosclerosis (Omer and Mahmood, 2016).

2. Materials and Methods

2.1. Patients and healthy groups:

Seventy of patients were divided into three study groups: acute myocardial infarction (AMI) patients group included 21 subjects, unstable angina (UA) patients group included 23 subjects and stable angina (SA) patients group included 26 subjects, the control group was composed of 20 healthy. The samples were collected from the Coronary Care Unit (CCU) in Al-Sadder Teaching Hospital in Al-Najaf province/ Iraq, during the period from September till December, 2018. The ages of patients and control ranged 30 to 69 years old. Each patients was divided into subgroups according gender, type of disease, and age (AL-Mohammad et al., 2017).

2.2. Blood samples collection:

Five ml of venous blood was acquired by antecubital venipuncture utilizing needle drained from CHD and control subjects between 8:30-10 AM following 12 hour fasting. The blood was permitted to clot in plain test tube at room temperature. The serum was suctioned after centrifugation at 3000rpm for 10 min, divided into aliquots in epindroff tubes and stored at -20°C (Kadhim, 2017).

Determination of serum Visfatin level

Specific kit for measuring human Visfatin concentrations in serum was supplied by Elabscience Biotechnology Co., Ltd. A Catalog No: E-EL-H1763.

Statistical analysis

The data of present study were articulated as (Mean ± Standard Error), the statistical analysis (Descriptive statistics, Correlation coefficients, P-value) were calculated by using Graphpad prism. The comparison between two groups were analyzed by t-test and the comparison among subdivided groups were analyzed by one-way ANOVA. when P-value < 0.05 was statistically a significant.

3. Result

Serum Visfatin level:

The result in figure (1) exhibit a significant increase (p<0.05) in serum levels of VF CHD group compared with in HT group.
Comparison serum visfatin level according to types of disease in patient with coronary heart disease. The results in figure (2) showed there is a significant increase (p>0.05) in serum level VF in AMI group as compared with UA group and SA group of CHD.

The different letters mean significant differences (P<0.05).

Figure (2): Comparison of serum visfatin level between SA, UA and AMI of CHD patients.

Comparison serum visfatin level according to gender in patient with coronary heart disease. The result in figure (3) indicated no significant differences (p>0.05) in serum level of VF between males and females groups of CHD patients.

(*) Statistically significant differences (p<0.05).
Comparison serum visfatin level according to ages of patient with coronary heart disease. The result indicated a significantly increased (p<0.05) in serum level VF of CHD as compare with HT group among different ages groups as showed in figure (4).

4. Discussion:

In current study indicated a significant increase (p<0.05) in VF level in CHD patients in comparison with healthy group.

Visfatin is highly expressed in adipose tissue, especially stromal cells, (Sethi et al., 2005) Visfatin is differentially expressed in the adipose tissue of different organs, and this may influence its effects on atherogenesis (Spiroglou et al., 2010) Increased visfatin expression has been reported in
diabetes mellitus, obesity, hypertension and cardiovascular disease (Sommer et al., 2008; Zhang et al., 2011). The results of the previous study suggest that increased serum visfatin is an independent risk factor for the development of atherosclerosis in patients (Kong et al., 2014).

Visfatin can promote vascular smooth muscle inflammation, being associated with a potential role in vascular dysfunction and inflammation associated with some metabolic disorders (Romacho et al., 2009). Increased visfatin levels are associated to coronary artery disease (CAD) and acute coronary syndromes even after correction for classic cardiovascular risk factors such as cholesterol, smoking, hypertension, diabetes, and obesity (Liu et al., 2009; Saddi-Rosa et al., 2011).

A cardioprotective effect of visfatin was also suggested in an animal model of non-atherosclerotic ischemia and reperfusion. Visfatin administered at the moment of reperfusion diminished the size of the infarcted area (Lim et al., 2008). Visfatin mRNA expression could be up-regulated in the fat tissue of obesity due to hypoxia (Segawa et al., 2006) which suggest that myocardial ischemia may have up-regulated the expression of visfatin, in participants diagnosed with cardiovascular diseases (Chang et al., 2011). Plasma levels of visfatin in patients with CHD were also found to increase significantly compared to that of the control group, and the plasma levels of visfatin in UA Patients group had an increasing tendency compared to SA patients group (Wang et al., 2014). Other studies (Fu et al., 2009; Mazaherioun et al., 2012) that have shown visfatin could contribute to atherosclerosis, inflammation and plaque destabilization which in turn, leads to myocardial infarction. Plasma levels of visfatin are increased in CAD and in acute ST elevation myocardial infarction (Lu et al., 2012). IL-6 plays an important role in initiating coronary artery disease by recruiting monocytes & macrophages to the vessel wall (Ikeda, 2003). The previous our studies (Al-kraity and Al-Dujaili, 2017a; Al-Dujaili and Al-kraity, 2018) indicated the other inflammatory marker cyclophilin-A and CD147 increase markedly in CHD compare with control, an inflammatory marker in atherogenesis and CHD, the mechanistic role of cyclophilin A in vascular disease progression. The CD147 act as a surface receptor for extracellular CYP-A (Yurchenko et al., 2002)

The incidence of cardiovascular disease and hyperlipidemia increases with age in both gender, but in women the risk increases markedly after menopause (Domenico et al., 1999). These agreement with our previous studies (Al-kraity and Al-Dujaili, 2017b) which indicate CHD increase in women with menopause due to depletion estrogen and gelsolin, the gelsolin’s possible role in cardiovascular diseases; The reduction of gelsolin may perhaps have problems in vascular functions, in atherosclerosis and coronary heart disease (Peddada et al., 2012). And older individuals (65+ years) are more likely to have CHD and die as a result of that, males have a greater risk of CHD compared to females and are also more likely to have coronary events earlier in life (Vliegenthart et al., 2005; AHA, 2010; Jawad et al. 2018). This is due to the fact that the female hormone estrogen provides a consistent protective effect against CHD, through its association with lipid metabolism, once past the menopause however, a woman’s risk becomes similar to a man’s. (Mackay and Mensah, 2004; Jawad et al. 2017). However, the study of Chen et al. (2006) and Sonoli et al. (2011) investigate that there is no significant difference in level of visfatin between males and females in and patients with that agreement with current study.

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