Introduction

Obesity is carelessly associated with the premature development of atherosclerosis, increased risk of stroke, and development of congestive heart failure (Douketis & Sharma, 2005). Studies have indicated that the adiposity secretes a variety of adipokines involved in energy metabolism, inflammation, and cardiovascular functions such as leptin, adiponectin and TNF (Mohamed et al., 1998; Trayhurn and Wood, 2004). Leptin is an adipocyte-derived hormone that has attracted much attention because β-cell may be adversely affected by chronic hyperglycemia and hyperinsulinemia (Hukshorn et al., 2004). It has been reported that low serum levels of adiponectin are associated with the risk of metabolic syndrome and coronary heart disease (Nakashima et al., 2010; Kajikawa et al., 2011). Tumor necrosis factor alpha (TNF-α) is a polypeptide cytokine produced primarily by mononuclear phagocytes and play a key role in the initiation of the inflammatory response but has relevant effects in many tissues that can regulate many cellular and biological processes such as immune function, cell differentiation, proliferation, apoptosis and energy metabolism (Bazzone & Beutler, 1996). In adipose tissue TNF-α is produced by macrophages in response to inflammation (Tzanavari et al., 1998) and obesity in humans (Clement et al., 1998), because β-cell may be adversely affected by chronic increased leptin levels (Hukshorn et al., 2004).

From the most prominent adipokines produced by adipocyte is adiponectin. Adiponectin has been shown anti-inflammatory, anti-diabetic and anti-atherogenic effects through its ability to increase insulin sensitivity by regulating fatty acid β oxidation and glucose metabolism (Yamauchi et al., 2002 and Minokoshi et al., 2002). LEPR, is a member of the class I cytokine receptor family that is expressed in the hypothalamus (Considine et al., 1996), plays a critical role in the regulation of appetite and metabolism (Zhang et al., 1994) and influence on lipid metabolism. Mutations resulting in a deficient leptin receptor cause obesity and diabetes in animals (Chen et al., 1996) and obesity in humans (Clement et al., 1998), because β-cell may be adversely affected by chronic increased leptin levels (Hukshorn et al., 2004).
Obese diabetic patients had fasting blood glucose level 179.3 mg/dl. These data indicate that fasting blood glucose level in obese have significant differences than normal individuals. Also, obese diabetic patients have highly significant elevation in blood glucose than both obese and normal subjects.

**Table 1:** Clinical and biochemical parameters in the studied groups regarding obesity index, hypertension and hyperglycemia

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Control</th>
<th>Non-diabetic Obese</th>
<th>Diabetic Obese</th>
</tr>
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<tbody>
<tr>
<td>Number of subject</td>
<td>34</td>
<td>49</td>
<td>8</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>21.83±0.2428</td>
<td>35.61±0.6045***</td>
<td>34.13±1.137</td>
</tr>
<tr>
<td>Waist circumference (cm)</td>
<td>84.26±1.105</td>
<td>120.0±1.434***</td>
<td>121.0±1.732</td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>125.1±3.221</td>
<td>134.3±2.253***</td>
<td>160.0±7.498***</td>
</tr>
<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>85.18±2.541</td>
<td>91.15±1.781***</td>
<td>109.8±2.498***</td>
</tr>
<tr>
<td>Fasting blood glucose level</td>
<td>84.32±1.438</td>
<td>92.04±1.394***</td>
<td>179.3±12.47***</td>
</tr>
<tr>
<td>Postprandial 2 hours blood glucose level</td>
<td>108.8±3.252</td>
<td>118.0±2.811</td>
<td>257.3±11.78***</td>
</tr>
<tr>
<td>HbA1c</td>
<td>5.11±0.0917</td>
<td>5.54±0.088***</td>
<td>8.025±0.387***</td>
</tr>
<tr>
<td>Leptin/adiponectin ratio</td>
<td>0.66±0.156</td>
<td>4.98±0.403***</td>
<td>4.40±0.509</td>
</tr>
</tbody>
</table>

Data are expressed as mean ± SE, letter * and ** referred to the significant difference between control and non-diabetic obese and between non-diabetic obese and diabetic obese, respectively, where the number of asterisk (*) indicates the levels of significant at p<0.05, 0.01, 0.0001.

As in Figure 1 leptin level in non-diabetic obese and diabetic obese showed a significant increase in comparison with normal subjects. Plasma adiponectin level in Figure 2 was significantly decreased in both non-diabetic obese and diabetic obese than normal subjects. However, plasma level of TNF-α and lymphocytes count showed a significant increase in non-diabetic obese and diabetic obese in comparison with normal subjects as in Figure 3 and Figure 4 respectively.

**Figure 1:** Leptin level in plasma of the studied groups. Letter * referred to the significant difference between control and non-diabetic obese at p < 0.0001.
Obese diabetic patients have high blood pressure, and adipocytokines have clinical implications as useful markers and potential therapeutic targets of glucose, lipid and cardiovascular abnormalities in all age groups. Blood leptin, adiponectin and TNF-α levels may accurately predict the presence of hyperlipidemia and/or diabetes mellitus (Margoni et al., 2011). The present results showed a significant increase in plasma level of leptin and a significant decrease in adiponectin in both non-diabetic obese and diabetic obese in comparison with control subjects. These results were consistent with the previous results obtained by Arita et al. (1999), Hamed et al. (2011), Nayak et al. (2010) who found that blood leptin concentration was significantly increased and adiponectin level was significantly reduced among obese subjects in comparison with lean control subjects. However, Coimbra et al. (2014) found that adiponectin and leptin levels in elderly patients with type 2 diabetes seem to be closely linked to obesity and to the length of the disease. Moreover, adiponectin and leptin levels in type 2 diabetes patients are more associated with obesity and less with diabetes (Neuparth et al., 2013).

The increase in leptin level was positively correlates with BMI (Hardie et al., 1997; Haitao et al., 2014). However, serum adiponectin was negatively correlates with BMI (Yoshida et al., 2005). Hence, Kowalska et al. (2006) suggested that adiponectin could play a role in the pathogenesis of insulin resistance in lean offsprings of type 2 diabetic subjects. Moreover, there is significant relationship between serum TNF-α and insulin resistance between diabetic and non-diabetic subjects, but there is no significant relation in obese type 2 diabetes alone (Al-Dhar and Jiffri, 2010). In the present study individuals diagnosed with obesity and type 2 diabetes were characterized by high level of TNF-α and lymphocytes count. These indicate that obese diabetic patients have high inflammation risk than obese patients; also obese subjects have risk of inflammation than normal individual (Lago et al., 2007; Mavridis et al., 2008; Gauthier and Ruderman, 2010). This overproduction of TNF-α by adipose tissue may be explaining the hyper insulinemia and/or insulin resistances as major determinant of the hypoadiponectinemia in obesity and type diabetes (Assal et al., 2007).

### Conclusion

The present results showed in obesity with or without type 2 diabetes plasma levels of leptin and TNF-α and lymphocytes count were increased, however, adiponectin level was decreased. Moreover, the high level of TNF-α reflect risk factor for vascular diseases like hypertension.

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### References
