To Study the Levels of Serum Fetuin - A in Patients with Non-Alcoholic Fatty Liver Disease

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Abstract: The aim of study was to measure serum level of Fetuin A in NAFLD and compare with healthy control subjects. In this study 90 NAFLD and 90 healthy subjects (age and gender matched) were enrolled. BMI, Glucose, HOMA IR, Insulin and Fetuin A were assessed. Serum Fetuin A levels were higher in NAFLD subjects compared with healthy controls (324.0 ± 98.23 pmol/l vs 225.0±75.0 pmol/l, respectively, P<0.0001). Increased serum levels of Fetuin A in NAFLD other than healthy subject.

Keywords: Fetuin A, NAFLD

1. Introduction

NAFLD is defined as the accumulation of excessive fat in the liver of patients without history of alcohol abuse or other causes of hepatic steatosis. NAFLD comprises a wide spectrum of diseases ranging from simple steatosis (SS) (i.e., fat accumulation in the liver) to Nonalcoholic steatohepatitis (NASH), which leads to variable grades of fibrosis and ultimately cirrhosis with its complications including Hepatocellular carcinoma (HCC) (Buzzetti E et al. 2016). NAFLD is characterized by mild to moderate increase in aspartate transaminase (AST), alanine transaminase (ALT), or both. Increased γ-glutamyl transferase (GGT) and serum alkaline phosphatase (ALP) can also be observed (Angulo P. 2002).

Fetuin-A is a carrier plasma glycoprotein synthesized by the liver and has many functions (Schafer C et al.2003). Fetuin-A binds the calcium and phosphate in the medium to form calciprotein particle (CPP). CPP thus removes the calcium from the medium. It is internalized mainly by the Kupffer cells of the liver and macrophages in the splenic marginal zone and carries out the calcium clearance (Heiss A et al.2003, Herrmann M et al.2012). Various studies have demonstrated an association between reduced plasma fetuin-A levels and increased vascular calcification and cardiovascular mortality in dialysis patients, increased risk of peripheral arterial disease, and coronary artery disease in patients with type 2 diabetes (Ketteler M et al.2003, Bilgir O et al.2010, Eraso LH et al.2010). Studies have defined reduced plasma fetuin-A levels as a new cardiovascular risk factor and demonstrated that severity of atherosclerosis increases with low plasma fetuin-A levels (Mundelain A et al.2012, Szeberin Z et al 2011). In addition, in humans, fetuin-A has been suggested to provide an important link between obesity and insulin resistance (Mori K et al.2006, Ix JH et al.2006).

Although there are many evidences linking obesity, serum Fetuin A and NAFLD. Data about serum Fetuin A concentration in NAFLD is limited. Therefore, present study was undertaken to evaluate serum Fetuin A levels in patients with NAFLD and to compare it with healthy controls.

2. Materials & Methods

The present study was conducted on USG proven Non-alcoholic fatty liver disease (NAFLD) patients admitted or attending Department of General Medicine, J.L.N. Medical College, Ajmer. Diagnosis of NAFLD was confirmed by an experienced physician. Anthropometric parameters and other variables i.e. Age, Weight, Height, Body mass index (BMI), systolic and diastolic blood pressure were measured. Smoking status and alcohol consumption were also noted in the present study. Venous blood sample was obtained by aseptic technique in plain tubes. Serum was separated by centrifugation at 2500 RPM for 10 minutes and stored in labeled tubes. Consent from all the subjects was obtained for the study.

3. Results and Observation

In this study, 90 cases of NAFLD were compared with 90 healthy controls.

Table 1: Anthropometric parameters ofNAFLD subjects & Healthy controls

<table>
<thead>
<tr>
<th>Parameters</th>
<th>NAFLD Cases (Mean ± SD )</th>
<th>Healthy Controls (Mean ± SD )</th>
<th>P-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>AGE (yrs)</td>
<td>49.7 ± 7.35</td>
<td>50.42 ± 8.05</td>
<td>0.644 (NS)</td>
</tr>
<tr>
<td>WEIGHT (kg)</td>
<td>51.0± 5.9</td>
<td>73.34 ± 4.5</td>
<td>-</td>
</tr>
<tr>
<td>HEIGHT (cm)</td>
<td>155.72 ± 5.1</td>
<td>157.52 ± 1.2</td>
<td>-</td>
</tr>
<tr>
<td>BMI (kg/m2)</td>
<td>21.20 ± 5.13</td>
<td>29.4 ± 3.86</td>
<td>&lt;0.001 (HS)</td>
</tr>
</tbody>
</table>

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We have no competing interests.

Table 2: Biochemical parameters of NAFLD subjects & Healthy subjects

<table>
<thead>
<tr>
<th>Parameters</th>
<th>NAFLD Cases (Mean ± SD)</th>
<th>Healthy Controls (Mean ± SD)</th>
<th>P-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>S. Fetuin-A</td>
<td>324.0 ±98.23</td>
<td>225.0±75.0</td>
<td>&lt;0.0001 (HS)</td>
</tr>
<tr>
<td>Alanine Aminotransferase (U/L)</td>
<td>71.32 ± 6.27</td>
<td>30 ± 5.30</td>
<td>&lt;0.0001 (HS)</td>
</tr>
<tr>
<td>Aspartate Aminotransferase (U/L)</td>
<td>45.28 ± 7.14</td>
<td>27.18±7.07</td>
<td>&lt;0.0001 (HS)</td>
</tr>
<tr>
<td>Gamma-Glutamyl Transferase (U/L)</td>
<td>40.53 ±15.22</td>
<td>28.21 ±7.52</td>
<td>&lt;0.0001 (HS)</td>
</tr>
</tbody>
</table>

P value <0.0001 is considered highly significant while p<0.01 is considered significant.

Basic anthropometric parameters of NAFLD subjects and healthy subjects are summarized in table-1. There was no significant difference between NAFLD subjects and healthy subjects regarding mean age (49.7 ± 7.35 vs. 50.42 ± 8.05 yrs.). BMI mean ± SD in kg/m² in NAFLD and healthy subjects was (21.20 ± 5.13 vs. 29.4 ± 3.86) and it was highly significant. Biochemical parameters of NAFLD subjects and healthy subjects are presented in table-2. NAFLD subjects had higher Fetuin A levels compared to healthy subjects (324.0 ±98.23 vs 225.0±75.0, P<0.0001).

4. Discussion

In the present study, NAFLD subjects have significantly higher levels of Fetuin A as compared to healthy control subjects. A number of articles have reported increased levels of serum Fetuin A in NAFLD and obese patients, but NAFLD subjects have not been studied extensively to know whether the increase in the circulating Fetuin A levels begin before the onset of NAFLD. Our result was inconsistent with Dariusz M. Lebensztejn et al (2014) who observed that the results demonstrated a significantly higher Fetuin A serum concentration in the obese diagnosed with NAFLD compared to the control group. However, their study group included only 12 children with NAFLD and 14 in the control group. Similar results were obtained in the adult population with the biopsy-proven NAFLD. Results of this study suggest that plasma levels of Fetuin A are increased in patients with NAFLD.

5. Limitations of Study

Our sample size was relatively small.

6. Acknowledgements

NIL

7. Conflicts of Interest

We have no competing interests.

8. Funding

NIL

9. Conclusion

From the present study it is concluded that serum Fetuin A levels gets increased prior to onset of NAFLD. Moreover the relation between the Fetuin-A and the liver may act as a major player in the link between the metabolic syndrome and the NAFLD. It could be considered among therapeutic agents used in the prevention of NAFLD and in the prevention or reduction of its critical complications.

References