Original article

Evaluation of hepatic metabolic activity in non-alcoholic fatty livers on 18FDG PET/CT

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A B S T R A C T

Objective: The liver has been used as a reference organ for the assessment of abnormal 18F-fluoro-2-deoxy-D-glucose (18FDG) in the body. Some researchers found that hepatosteatosis significantly changed the hepatic 18FDG uptake, while others did not. This study investigated whether the liver’s 18FDG uptake was affected by the diffuse fatty infiltration of the liver.

Material and methods: A total of 156 cases were included in this study. Different patient groups were defined according to the liver Hounsfield unit (HU). The HU was calculated from an unenhanced CT, for all patients, and we calculated the mean standardized uptake value (SUVMedio) and maximum standardized uptake value (SUVmax) on an 18FDG PET scan. For this purpose, we placed regions of interest (ROIs) on the liver image. We statistically compared the SUVMedios and SUVmaxes measured in the fatty liver patients and the control group.

Results: The average SUVMedio and SUVmax values were calculated as 2.58 ± 0.66 and 3.94 ± 1, respectively, in the patient group and 2.54 ± 0.57 and 3.7 ± 0.88, respectively, in the control group. We found the average SUVMedio and SUVmax values in the fatty liver group and its subsets were not significantly different from the values in the control group (p > 0.05). We also did not find any statistically significant correlation between average liver density (HU) and the average SUV values (p > 0.05).

Conclusion: Fatty infiltration may not have a significant effect on the liver’s 18FDG uptake. Thus, the liver may be used as a reference or comparator on 18FDG PET scans in patients with fatty liver disease.

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Palabras clave:
Hígado graso
18FDG
PET/TAC
SUV

Evalúación mediante PET/TAC con 18FDG de la actividad metabólica hepática en pacientes con hígado graso de origen no alcohólico

R E S U M E N

Objetivo: El hígado se ha usado como órgano de referencia para evaluar la captación anormal de la 18F-fluoro-2-deoxi-D-glucosa (18FDG). Algunos trabajos publicados muestran que la estesostatosis hepática modifica la captación hepática de 18FDG, mientras que otros no encuentran modificaciones. Este trabajo ha estudiado si la captación hepática de 18FDG se ve afectada por la infiltración grasa difusa del hígado.

Material y métodos: En este estudio se incluyeron 156 casos. Se definieron diferentes grupos de pacientes de acuerdo a las unidades Hounsfield (HU) del hígado. En todos los pacientes, las HU se calcularon a partir de la TAC sin contraste. Además se calculó el SUVMedio y el SUVmax en el estudio PET con 18FDG. Para ello se dibujaron regiones de interés (ROI) sobre el área hepática. Se realizó una comparación estadística del SUVMedio y SUVmaxes en los pacientes con hígado graso y en un grupo control.

Resultados: En el grupo de pacientes, los promedios del SUVMedio y SUVmax fueron 2.58 ± 0.66 y 3.94 ± 1, respectivamente; en el grupo control fueron 2.54 ± 0.57 y 3.7 ± 0.88, respectivamente. Encontramos que los promedios del SUVMedio y SUVmax en los pacientes con hígado graso no fueron significativamente diferentes a los valores del grupo control (p > 0.05). Asimismo, no detectamos correlación estadísticamente significativa entre los valores de la densidad media del hígado, medida en HU, y el promedio de los SUV (p > 0.05).

Conclusión: La infiltración grasa no parece tener un efecto significativo sobre la captación hepática de 18FDG. Por ello, el hígado se puede usar como órgano de referencia en los estudios PET con 18FDG realizados en pacientes con enfermedad grasa hepática.

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Introduction

Fatty liver disease is the excess accumulation of lipids, in the form of triglycerides and cholesterol, within the hepatocytes in the liver.\(^1\) Fat infiltration decreases the value of liver attenuation (Fig. 1). Fatty liver disease has an estimated prevalence of 25–35% in some populations.\(^2,3\) This condition is generally a reversible process, but it can progress to cirrhosis.\(^4\) Fatty liver disease may be in one of three main stages; hepatic steatosis, non-alcoholic steatohepatitis, and cirrhosis.\(^4,5\) Non-alcoholic fatty liver disease is the most common type of the hepatic steatosis. Obesity, type 2 DM, hyperlipidemia, chronic hepatic viral infection, some liver storage disorders, and some drugs and toxins may cause non-alcoholic hepatosteatosis.\(^1,5\)

A histopathologic examination of the liver sample is the standard method for diagnosing hepatosteatosis.\(^6\) Non-invasive imaging techniques, such as ultrasonography (US), computed tomography (CT), magnetic resonance (MR) imaging, and proton (hydrogen-1) MR spectroscopy, have been used to detect hepatic steatosis.\(^5,7–9\) Several criteria have been used for the evaluation of non-contrast CT in hepatic steatosis.\(^10–18\)

18FDG positron emission tomography (PET) scanning is a non-invasive imaging technique that displays the glucose uptake of the cells throughout the body. Therefore, local metabolic activity can be revealed easily using this technique.\(^1\) This metabolic activity can be calculated semi-quantitatively as the standardized uptake value (SUV) in which some factors, such as the injected activity of 18FDG, patient weight, and the time after injection, are taken into account. The average SUV (SUV\(_{\text{mean}}\)) value of a region or maximum SUV (SUV\(_{\text{max}}\)) point in a lesion can be easily calculated using an 18FDG PET scan to compare the 18FDG uptake of pathologic lesions. The liver’s 18FDG uptake has been presumed normal by some authors and is used as a reference in their studies.\(^1,19–25\) If the 18FDG uptake of a lesion is greater than that of the liver in a certain degree, that 18FDG accumulation is considered abnormal.\(^20,27\)

In some studies, no association was found between liver attenuation and the liver’s 18FDG uptake,\(^28\) while others found that hepatosteatosis resulted in a statistically significant change in hepatic 18FDG uptake.\(^1,29,30\) In this study, we investigated whether the liver’s 18FDG uptake, measured on PET/CT scans as SUV\(_{\text{max}}\) and SUV\(_{\text{mean}}\), were affected by the diffuse fatty infiltration of the liver.

Material and methods

Subjects

We performed this study retrospectively (from 21.11.2011 to 21.01.2012) on patient’s medical charts from Konya University Meram Medical Faculty Hospital that were dated May 2008 to January 2012. Demographic data were collected from chart records. All patients had been diagnosed with cancer. Both the patient and control groups had been referred to the Nuclear Medicine Department for 18FDG PET/CT scanning during their diagnosis, staging, or cancer follow-ups. Seventy-nine patients with reported cases of hepatosteatosis on previously contrast-enhanced CTs and 77 control cases that did not have a hepatosteatosis diagnosis on contrast-enhanced CT scans were included in this study. Patients with evidence of hepatic metastasis or any disorder that affected the liver were excluded from the study. On the contrast-enhanced CT and PET scan, normal findings, other than the hepatosteatosis, were reported in the patient group, and normal PET/CT liver findings were reported in the control cases. The age, gender, history of DM and chemotherapy, weight and height of the subjects, serum ALT and AST levels, blood glucose level prior to the PET scan and the elapsed time between the 18FDG injection and onset of PET scan were noted in all cases (Table 1).

A total of 156 cases (mean age, 55 ± 2.2 years; age range, 20–83 years) were included in this study, including 83 (53.2%) men (mean age, 54.8 ± 13.1 years; age range, 20–83 years) and 73 (46.8%) women (mean age, 55.2 ± 11.2 years; age range, 27–83 years). There were 79 subjects (mean age, 53.4 ± 11.4 years; age range, 20–73 years) in patient group and 77 subjects (mean age, 56.7 ± 12.9 years; age range, 26–83 years) in control group. The PET/CT indication included colon cancer in 28 patients (17.9%), lung cancer in 43 patients (27.6%), stomach cancer in 16 patients (10.3%), lymphoma in 8 patients (5.1%), breast cancer in 30 patients (19.2%), malign melanoma in 5 patients (3.2%), genitourinary cancer in 10 patients (6.4%), pancreatic cancer in 4 patients (2.6%), head and neck cancer in 10 patients (6.4%) and endometrial cancer in 2 patients (1.3%). The control group had 77 patients with a mean liver HU (HU\(_L\)) value greater than their mean spleen HU (HU\(_S\)) value. The patient group included 79 patients in whom the mean HU\(_L\) value was lower than or equal to the mean HU\(_S\) value. To achieve a more detailed analysis, the fatty liver disease group was divided into subsets: the patient subgroup with a HU\(_L\)–HU\(_S\) < 0 (−10) was called “Group HU\(_L\) < HU\(_S\) = 0 (−10)” which had 42 patients, the patient group with a HU\(_L\)–HU\(_S\) = 0 (−10) was called “Group HU\(_L\) = HU\(_S\) = 0 (−10)” which had 18 patients, the patient group with a HU\(_L\)–HU\(_S\) < −20 was called “Group HU\(_L\) = HU\(_S\) = −20” which had 19 patients, the patient group with a HU\(_L\) < 40 was called “Group HU\(_L\) < 40” which had 42 patients, and the patient group with a HU\(_L\) < 40 was called “Group HU\(_L\) < 1.1” which had 51 patients.

18FDG PET/CT scan

All the patients fasted for at least 6 h before they received a 370 Megabecquerel (10 mCi) injection of 18FDG. The patients’ blood glucose levels were measured before scanning. PET/CT scans were obtained using an integrated scanner (Siemens, Biograph True Point 6 PET/CT, Germany) at an average 62 min (range, 50–72 min) after the injection. A whole-body CT scan was performed using 130 kV, 50 mAs, a 1.5 pitch, 5 mm section thickness, and a 70 cm field of view, without intravenous contrast administration. A PET scan was performed immediately after an unenhanced CT scan, and it acquired images from the base of the skull to the upper thigh using a three-dimensional acquisition mode with a 3-min acquisition per bed position. The PET attenuation correction was based on the unenhanced CT data.

SUV measurements

In all cases, a total of four circular regions of interest (ROI) about 3 cm in diameter were drawn over segments II, IV, V, and VI of the liver, and three circular ROI about 2 cm in diameter were drawn on or more different sites of the spleen’s mid section. During the drawing process, we avoided biliary, vascular, and peritoneal structures. Using the same ROIs for each case, the average HU for liver and spleen and the average SUV\(_{\text{mean}}\) and SUV\(_{\text{max}}\) were measured using software on the PET/CT system’s workstation (TrueD, Siemens Medical Systems). SUV calculations were based on the following formula: SUV = [radioactivity concentration (Bq/ml)/injected dose (Bq)/body weight (g)]. ROIs were reviewed by a nuclear medicine physician with 10 years of experience.

Statistical analysis

Statistical analysis was performed using SPSS 15.0 software (SPSS Inc., Chicago, Illinois, USA). Data were stated as the...
mean ± the standard deviation (SD). To compare age, BMI, 18FDG dose, elapsed time after injection, glucose level, and the PET/CT’s quantitative parameters of patients with fatty livers and control subjects, an independent samples t-test, or Mann–Whitney U-test, was used. Categorical data of gender, DM, chemotherapy status, ALT, and AST elevation were analyzed using Chi-square testing. The Pearson’s correlation coefficient was used to discover the relationships between the liver HU, SUVmean, and SUVmax measurements. A p-value of less than 0.05 was considered statistically significant.

Results

The mean and SD of the clinical factors and comparison data are stated in Table 1. There were statistically significant differences between the BMI, ALT elevation, AST elevation (p < 0.0001), DM, and glucose levels (p = 0.003) of the patients with fatty livers and those from the control group. All the subjects in the control group had a liver HU greater than 40. There were no statistically significant difference between the fatty liver disease group and control group for the mean and maximum liver SUVs (p > 0.05). Also, the mean and maximum liver SUVs for the subsets of the patients with fatty liver disease were not significantly different from those in the control group (p > 0.05) (Table 2). We did not find any statistically significant correlation between average liver density (HU) and average SUVs (p > 0.05) (Fig. 2).

Discussion

In this study we assessed whether liver 18FDG uptake was affected by hepatosteatosis. Through statistical analysis, we found that the SUVmean and SUVmax values in the fatty liver group and its subsets were not significantly different from the values in the control group (p > 0.05) (Table 2). We did not find any statistically significant correlation between average liver density (HU) and average SUVs (p > 0.05).

We used different semi-quantitative, unenhanced CT methods from the literature to identify fatty livers. Thus, we developed several patients groups: (a) HU_{S,L} ≤ 0, (b) HU_{S,L} = − (10–20), (c) HU_{S,L} = − 20– (40), (d) HU_{S,L} < 40, and (f) HU_{S,L} / HU > 1.1. In this study, we included cases who had a normal contrast-enhanced CT findings other than the hepatosteatosis for the liver. To avoid potential errors due to lesions, such as haemangioma, small cysts, vascular anomalies, or isoattenuating isometabolic metastases that may not be identifiable in CT images, we placed four ROIs on different regions of the liver at segments II, IV, V, and VI and calculated the average HU.

Table 1
Clinical characteristics of patients with fatty liver disease, control subjects, and comparison data.

<table>
<thead>
<tr>
<th>Clinical parameters</th>
<th>Patients with fatty livers (n = 79)</th>
<th>Control subjects (n = 77)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (year)</td>
<td>53.4 ± 11.4 (20–73)</td>
<td>56.7 ± 12.9 (26–83)</td>
<td>0.091</td>
</tr>
<tr>
<td>Gender (male/female)</td>
<td>36/43</td>
<td>47/30</td>
<td>0.053</td>
</tr>
<tr>
<td>DM</td>
<td>20 (25.3%)</td>
<td>6 (7.8%)</td>
<td>0.003</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>29 ± 5.6</td>
<td>24.1 ± 5.4</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>ALT elevation</td>
<td>25 (31.6%)</td>
<td>0</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>AST elevation</td>
<td>26 (32.9%)</td>
<td>3 (3.8%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Elapsed time after injection (min)</td>
<td>62 ± 14</td>
<td>60 ± 10.7</td>
<td>0.395</td>
</tr>
<tr>
<td>Glucose level (mmol/L)</td>
<td>120 ± 30.9</td>
<td>104.8 ± 18.8</td>
<td>0.003</td>
</tr>
</tbody>
</table>

DM, diabetes mellitus; BMI, body mass index; ALT, alanine aminotransferase; AST, aspartate aminotransferase.
Table 2

| (a–f) Demonstrate mean and standard deviation of the data and statistical comparison results in patient groups and control group. |

<table>
<thead>
<tr>
<th>(a) Parameters</th>
<th>HU&lt;sub&gt;L&lt;/sub&gt; ≤ 0 (n = 79)</th>
<th>Control (n = 77)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>HU&lt;sub&gt;L&lt;/sub&gt;</td>
<td>34.6 ± 13.8</td>
<td>56.8 ± 4.6</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>HU&lt;sub&gt;L&lt;/sub&gt; &gt; 0</td>
<td>-12.9 ± 13.4</td>
<td>12.1 ± 6.5</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Liver SUV&lt;sub&gt;mean&lt;/sub&gt;</td>
<td>3.58 ± 0.66</td>
<td>2.54 ± 0.57</td>
<td>0.662</td>
</tr>
<tr>
<td>Liver SUV&lt;sub&gt;max&lt;/sub&gt;</td>
<td>3.94 ± 1</td>
<td>3.7 ± 0.88</td>
<td>0.108</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>(b) Parameters</th>
<th>HU&lt;sub&gt;L&lt;/sub&gt; -0 (–10) (n = 42)</th>
<th>Control (n = 77)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>HU&lt;sub&gt;L&lt;/sub&gt;</td>
<td>43.6 ± 6</td>
<td>56.8 ± 4.6</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>HU&lt;sub&gt;L&lt;/sub&gt; &gt; 0</td>
<td>-3 ± 3.5</td>
<td>12.1 ± 6.5</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Liver SUV&lt;sub&gt;mean&lt;/sub&gt;</td>
<td>2.7 ± 0.59</td>
<td>2.54 ± 0.57</td>
<td>0.145</td>
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<tr>
<td>Liver SUV&lt;sub&gt;max&lt;/sub&gt;</td>
<td>3.99 ± 0.96</td>
<td>3.7 ± 0.88</td>
<td>0.95</td>
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<tr>
<th>(c) Parameters</th>
<th>HU&lt;sub&gt;L&lt;/sub&gt; -10 (–20) (n = 18)</th>
<th>Control (n = 77)</th>
<th>p value</th>
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<tr>
<td>HU&lt;sub&gt;L&lt;/sub&gt;</td>
<td>33.4 ± 6</td>
<td>56.8 ± 4.6</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>HU&lt;sub&gt;L&lt;/sub&gt; &gt; 0</td>
<td>-14.9 ± 2.4</td>
<td>12.1 ± 6.5</td>
<td>&lt;0.0001</td>
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<tr>
<td>Liver SUV&lt;sub&gt;mean&lt;/sub&gt;</td>
<td>2.58 ± 0.67</td>
<td>2.54 ± 0.57</td>
<td>0.936</td>
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<td>Liver SUV&lt;sub&gt;max&lt;/sub&gt;</td>
<td>4.02 ± 0.95</td>
<td>3.7 ± 0.88</td>
<td>0.237</td>
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<th>(d) Parameters</th>
<th>HU&lt;sub&gt;L&lt;/sub&gt; ≤ -20 (n = 19)</th>
<th>Control (n = 77)</th>
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<td>HU&lt;sub&gt;L&lt;/sub&gt;</td>
<td>15.8 ± 12</td>
<td>56.8 ± 4.6</td>
<td>&lt;0.0001</td>
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<tr>
<td>HU&lt;sub&gt;L&lt;/sub&gt; &gt; 0</td>
<td>-32.7 ± 10.2</td>
<td>12.1 ± 6.5</td>
<td>&lt;0.0001</td>
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<tr>
<td>Liver SUV&lt;sub&gt;mean&lt;/sub&gt;</td>
<td>3.23 ± 0.74</td>
<td>2.54 ± 0.57</td>
<td>0.227</td>
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<tr>
<td>Liver SUV&lt;sub&gt;max&lt;/sub&gt;</td>
<td>3.76 ± 1.19</td>
<td>3.7 ± 0.88</td>
<td>0.769</td>
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<th>(e) Parameters</th>
<th>HU&lt;sub&gt;L&lt;/sub&gt; &gt; 40 (n = 42)</th>
<th>Control (n = 77)</th>
<th>p value</th>
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<tr>
<td>HU&lt;sub&gt;L&lt;/sub&gt;</td>
<td>25.3 ± 12</td>
<td>56.8 ± 4.6</td>
<td>&lt;0.0001</td>
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<td>Liver SUV&lt;sub&gt;mean&lt;/sub&gt;</td>
<td>2.46 ± 0.58</td>
<td>2.54 ± 0.57</td>
<td>0.49</td>
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<tr>
<td>Liver SUV&lt;sub&gt;max&lt;/sub&gt;</td>
<td>3.86 ± 1.05</td>
<td>3.7 ± 0.88</td>
<td>0.4</td>
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<th>(f) Parameters</th>
<th>HU&lt;sub&gt;L&lt;/sub&gt;/HU&lt;sub&gt;R&lt;/sub&gt; &gt; 1.1 (n = 51)</th>
<th>Control (n = 77)</th>
<th>p value</th>
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<tr>
<td>HU&lt;sub&gt;L&lt;/sub&gt;/HU&lt;sub&gt;R&lt;/sub&gt;</td>
<td>2.5 ± 3.53</td>
<td>1.29 ± 0.2</td>
<td>&lt;0.0001</td>
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<tr>
<td>Liver SUV&lt;sub&gt;mean&lt;/sub&gt;</td>
<td>2.5 ± 0.7</td>
<td>2.54 ± 0.57</td>
<td>0.695</td>
</tr>
<tr>
<td>Liver SUV&lt;sub&gt;max&lt;/sub&gt;</td>
<td>3.92 ± 1.07</td>
<td>3.7 ± 0.88</td>
<td>0.205</td>
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</table>

HU<sub>L</sub>, liver Hounsfield unit; HU<sub>R</sub>, spleen Hounsfield unit; HU<sub>L</sub> &gt; 0, the difference between Hounsfield units of the liver and spleen; SUV<sub>mean</sub>, average standardized uptake value; SUV<sub>max</sub>, maximal standardized uptake value.

Lin et al. evaluated the impact of the liver’s fat infiltration using the SUV<sub>max</sub> of livers from an 18<sup>FDG</sup> PET. They studied a population of 112 persons with fatty livers. The authors excluded oncologic patients from the study, however. The authors diagnosed fatty livers and divided them into three groups based on the severity of the disease, according to a visual assessment of the liver on US. The authors found statistically significant differences in the livers’ SUV<sub>max</sub> on the 18<sup>FDG</sup> PET between control and patient groups. Based on the results, they concluded that the liver could not be used as a comparator for the extra hepatic fat of equivocal increased 18<sup>FDG</sup> activity in patients with fatty liver disease. In contrast, we did not find a statistically significant difference between patient groups and the control group in the livers’ SUV<sub>max</sub>. There are several possible causes for the different results of our study, including the fact that all the subjects in the Lin et al. study were nononcologic, and we studied only on oncologic patients. The different study population may have given different results. Also, the authors’ fatty liver diagnostic tool was US, whereas we used an unenhanced CT for this purpose. Lin et al. evaluated their livers visually and subjectively, so their accuracy depends on the operator and some patient features. Finally, previous studies have shown that US has a sensitivity of 60–94% and a specificity of 66–95% in detecting fatty livers, but we used a semi-quantitative method that is more objective in our study. We also used more strict criteria to diagnose the fatty livers (Table 2). Park et al. have reported that diagnostic performance of unenhanced CT have high performance in qualitative diagnosis of higher (>30%) degrees of hepatic steatosis with 100% specificity and 82% sensitivity using histological analysis as the reference.

Van Werven et al. studied different imaging modalities in their assessment of the hepatic steatosis in patients who were undergoing liver resection. They reported diagnostic accuracy of the different modalities in detecting fatty liver: 71% for US, 74% for CT, 91% for T1-weighted dual-echo MR imaging, and 89% for 1<sup>1</sup>H MR spectroscopy. In our hospital, we did not have a database of fatty liver evaluations by MR spectroscopy, so we could not compare MR spectroscopy to the unenhanced CT and histopathology in fatty liver disease. The literature suggests that MRI and MR spectroscopy are accurate methods for detecting fatty livers. However, MR spectroscopy is expensive and requires special software that may not be available in all MRI units. In our study, we chose Group HU<sub>L</sub> &gt; 0 as one of patient groups with more strict criteria whose HU<sub>L</sub> minus HU<sub>R</sub> was lower than 20 and had a mean HU<sub>L</sub> of 15.8 ± 12. For this group, there was also not a statistically significant difference between patient and control groups with respect to SUVs.

Abikhzer et al. assessed the hepatic metabolic activity in 37 patients with hepatosteatosis on an 18<sup>FDG</sup> PET/CT. The authors calculated the SUV<sub>max</sub> adjusted for body weight and for lean body mass. They did not find a statistically significant decrease in hepatic metabolic activity in terms of SUV<sub>max</sub> adjusted for body weight. Our study findings were consistent with theirs. They also did not reveal any statistically significant difference between liver attenuation values and SUV<sub>max</sub> in their patient group. They diagnosed hepatic steatosis if the HU<sub>L</sub> was greater than 10 HU below the HU<sub>L</sub> on an unenhanced CT. The mean HU<sub>L</sub> was 24.92 ± 9.71. However, in our study, the HU<sub>L</sub> in patient subset HU<sub>L</sub> ≤ -20 was 15.8 ± 12 lower than that in their patient group, so we have more strictly defined the hepatosteatosis than Abikhzer et al. found that...
hepatic steatosis resulted in a small statistically significant decrease in hepatic metabolic activity in terms of SUVmax adjusted for body weight and for lean body mass. In our study, we calculated SUVmax adjusted for body weight but not for lean body mass because we do not routinely use SUVmax adjusted for lean body mass in our clinics.

Abele et al. studied 142 patients to investigate the association between diffuse fatty infiltration of the liver and average liver 18FDG uptake.26 The authors accepted HU_L ≤ -10 as fatty liver disease. They defined fatty liver disease more strictly as HU_L ≤ -10. They found no association between liver attenuation (in HU) and 18FDG uptake measured in terms of SUVmean and SUVmax. We also did not find any relationships between the liver attenuation and SUVmean and SUVmax values in our patient groups including the HU_L ≤ -20 group. Our study findings were consistent with the authors’ results.

In the present study, the BMI of the patient group was 29 ± 5.6, which is within the overweight range, and the control group’s BMI was 24.1 ± 5.4 kg/m², which is within the normal range. We observe a statistically significant difference between the BMI of patients with fatty livers and the control subjects (p < 0.0001). This finding showed that there was a relationship between obesity and hepatosteatosis. To date, this fact has been reported by many researchers.5,28-30

We also found the serum glucose level higher in the patient group than in the control group (p = 0.003). Of the patients with fatty livers, 25.3% had been diagnosed with DM, whereas 7.8% of the control group shared the diagnosis. A significant association between DM and fatty livers has been reported in many studies.5,29

In the patient group, 31.6% of subjects had elevated ALT and 32.9% had elevated AST, whereas in control group, 3.8% had elevated AST and none of the group had elevated ALT. There were a significant difference between the patient and control groups with respect to ALT and AST elevation (p < 0.0001). The elevation of serum ALT and AST may occur in some patients with severe fatty livers due to damaged liver cells. Our findings were also consistent with the literature.5

One limitation of present study was that histopathology was not used to identify patients with fatty liver disease. We identified fatty livers according to HU unit measurements on an unenhanced CT. Instead, we used more strict criteria to define fatty livers in some of patient groups. In these patient groups, we also did not find statistically significant differences in SUVmean and SUVmax from the control group’s values. Also, all the patients enrolled in this study had been diagnosed with a malignant tumour. Most of them had received chemotherapeutics and other drugs. Some of the patients may have had a metabolically affected liver. Finally, there may be invisible metastatic focuses in the patients’ livers.

In conclusion, our study findings demonstrated that there was no significant association between the SUVmean, SUVmax, and fatty livers. Hepatosteatosis may not have had a significant effect on the liver’s 18FDG uptake. Based on these findings, the liver can be used as a reference or comparator on 18FDG PET scan in patients with fatty liver disease.

Conflict of interests

The authors declare not to have any conflict of interests.
References


