ABSTRACT

Objectives: Despite newly developed treatment modalities, colorectal cancer is still the leading cause of cancer deaths. In recent years, studies have been carried out to suggest that lipid metabolism may play a role in cancer development and metastasis.

Methods: Lipid metabolism both in conventional chemotherapy. It has also been found that it plays a central role in resistance to targeted therapies. In our study, we planned to compare the atherogenic plasma index levels of patients diagnosed with colon cancer before chemotherapy and in the month of chemotherapy.

Results and Conclusions: Evaluating the effect on the atherogenic plasma index after chemotherapy, we thought that lipid-regulating treatments would contribute to both cancer development and disease control.

Keywords: Atherogenic plasma index; lipoprotein; cancer; colon.

1. INTRODUCTION

Despite the newly developed treatment modalities, colorectal cancer is still the 3rd in cancer deaths [1,2]. Studies have been carried out in recent years that lipid metabolism may play a role in cancer development and metastasis. A relationship has been found between lipids and disease formation in cancers such as breast prostate lung [3-7]. The molecular
mechanisms of drug resistance include alteration of the drug-specific binding site, decreased drug permeability that can enzymatically deactivate the drug, and/or gene mutations that can increase the pumping out of drugs across the plasma membrane. Many of these processes are associated with altered lipid metabolism. Lipid metabolism has been found to play a central role in both conventional chemotherapy and resistance to targeted therapies. Atherosclerosis and cancer have been associated with chronic inflammation. Uncontrolled cell proliferation and oxidative stress are common factors in the formation of both diseases. Inflammation plays a central role in the formation of atherosclerosis. Changes in blood lipid values after chemotherapy cause negative effects on chronic inflammation and may contribute to recurrence and metastasis in later periods. Monitoring lipid changes after treatment may contribute to disease control. The atherogenic plasma index (AIP) is an index that consists of triglycerides and high-density lipoprotein cholesterol. It has been used to measure blood lipid levels and is widely used as an optimal indicator of cardiovascular diseases associated with dyslipidemia [8]. In our study, we planned to compare the atherogenic plasma index levels of patients diagnosed with colon cancer before chemotherapy and at the 6th month of chemotherapy. The effect of colon cancer chemotherapy treatment on lipids is unclear. We evaluated the effect on the atherogenic plasma index after chemotherapy and thought that lipid-regulating treatments would contribute to both cancer development and disease control.

2. MATERIALS AND METHODS

The blood lipid profile and hemogram parameters, which were routinely checked in cases over the age of 18 with a histopathological diagnosis of colon cancer, were scanned and recorded until 01.06.2017 -30.06.2021. The lipid parameters, which are routinely checked in the patients who received chemotherapy for the treatment of colon cancer, were recorded before the treatment and at the 6th month of the treatment. The atherogenic plasma index (log (triglyceride/HDL-C)) was calculated before and after the chemotherapy and compared. The study was approved by Afyon University of Health Sciences clinical research ethics committee with the number 519- 2011-KAEK-2.

3. RESULTS

As seen in Table 1, 67.6% of the 37 individuals participating in the research were men and 32.4% were women.

As seen in Table 2, the average weight of the individuals participating in the research was 69.59±11.47, and the average age of the individuals was 61.40±11.16.

Table 1. Descriptive statistics for participants

<table>
<thead>
<tr>
<th>Variable</th>
<th>Groups</th>
<th>F</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td>Male</td>
<td>25</td>
<td>67.6</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>12</td>
<td>32.4</td>
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<tr>
<td></td>
<td>Total</td>
<td>37</td>
<td>100.0</td>
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</tbody>
</table>

Table 2. Descriptive statistics for the participants

<table>
<thead>
<tr>
<th>Individuals</th>
<th>N</th>
<th>M&lt;sub&gt;age&lt;/sub&gt;</th>
<th>sd</th>
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</thead>
<tbody>
<tr>
<td>Weight</td>
<td>37</td>
<td>69.59</td>
<td>11.47</td>
</tr>
<tr>
<td>Age</td>
<td>37</td>
<td>61.40</td>
<td>11.16</td>
</tr>
</tbody>
</table>

Table 3. Analysis of the patients’ pre/post treatment values

<table>
<thead>
<tr>
<th>Measurements</th>
<th>Variables</th>
<th>N</th>
<th>M</th>
<th>Std. Deviation</th>
<th>Std.Error</th>
<th>t</th>
<th>df</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pair 1</td>
<td>B.T. monocyte</td>
<td>37</td>
<td>5711</td>
<td>.25136</td>
<td>.04132</td>
<td>.914</td>
<td>3</td>
<td>.36</td>
</tr>
<tr>
<td></td>
<td>A.T. monocyte</td>
<td>37</td>
<td>5378</td>
<td>.26250</td>
<td>.04315</td>
<td>6</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>Pair 2</td>
<td>B.T. HDL-C</td>
<td>37</td>
<td>46,4595</td>
<td>14.69050</td>
<td>2.41510</td>
<td>1.77</td>
<td>.84</td>
<td></td>
</tr>
<tr>
<td></td>
<td>A.T. HDL-C</td>
<td>37</td>
<td>41,1351</td>
<td>12.43463</td>
<td>2.04424</td>
<td>8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pair 3</td>
<td>B.T. LDL-C</td>
<td>37</td>
<td>109,4865</td>
<td>36.74735</td>
<td>6.04123</td>
<td>-</td>
<td>.51</td>
<td></td>
</tr>
<tr>
<td></td>
<td>A.T. LDL-C</td>
<td>37</td>
<td>113,3784</td>
<td>31.73620</td>
<td>5.21740</td>
<td>.660</td>
<td>3</td>
<td></td>
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<tr>
<td>Pair 4</td>
<td>B.T. Total</td>
<td>37</td>
<td>186,0541</td>
<td>39.45106</td>
<td>6.48571</td>
<td>-</td>
<td>.16</td>
<td></td>
</tr>
</tbody>
</table>
As can be seen in Table 3, according to the results of the paired sample t-test analysis performed on the values, between B.T. Monocyte and A.T. Monocyte, between B.T. HDL-C and A.T. HDL-C, between B.T. LDL-C and A.T. LDL-C, between B.T.Total Cholesterol and A.T.

5. DISCUSSION

There was no effect on the atherogenic plasma index measured at the 6th month of our patients who received FOLFOX (5 fluorouracil, folinic acid, oxaliplatin) chemotherapy for the treatment of colon cancer. No significant changes were detected in lipid parameters after chemotherapy. It has been determined in many studies that chemotherapy treatment causes changes in lipid metabolism.

In a study conducted in 18 lymphomas, 18 breast cancers, 14 small cell lung cancers, and 7 urethelial carcinomas, total cholesterol and low-density lipoprotein cholesterol (LDL-C) were increased after chemotherapy in patients other than breast cancer patients (Ref. 9). The tendency to increase in serum triglyceride levels was found to be statistically significant in breast cancer patients [9]. In a study, a decrease in LDL-C and an increase in high-density lipoprotein cholesterol (HDL-C) were found in patients with less metastatic colon cancer who were given combined chemotherapy with antiangiogenic therapy [10]. An increase in triglycerides and a decrease in HDL-C were detected in bexarotene chemotherapy used for the treatment of cutaneous T-cell lymphoma [11].

It is thought that the change in lipids in colorectal cancer affects the prognosis. In a study, an increase in cholesterol levels and a decrease in triglyceride and HDL-C levels were found in patients with colorectal cancer after adjuvant chemotherapy. In addition, HDL-C elevation was found to be prognostic (12). In our study, no change was detected in the serum lipid profile. Radiotherapy and chemotherapy treatment are side effects of heart failure, hypertension, thromboembolism and atherosclerosis [13]. Both radiotherapy and chemotherapy treatment cause inflammation and endothelial cell activation, which leads to the onset of atherosclerosis. It also causes inhibition of thrombolyis. It causes instability in previously formed plaques. Altered expression of thrombolysis-related proteases is involved in atherosclerotic plaque progression and in the process of cancer invasion and metastasis [14].

5. CONCLUSIONS

There are a limited number of studies in the literature examining lipid changes after colorectal cancer chemotherapy. Lipid change and its mechanism after chemotherapy treatment remain unclear. The number of patients in our study is limited. We think that there is a need for more extensive research by increasing the number of patients.

CONSENT

As per international standard or university standard, patients' written consent has been collected and preserved by the author(s).

ETHICAL APPROVAL

The study was accepted by Afyon Karahisar University of Health Sciences clinical research ethics committee with the decision number 519-2011-KAEK-2.

COMPETING INTERESTS

Authors have declared that no competing interests exist.
REFERENCES


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