Study of Fatty Liver Syndrome Frequency in Dairy Cattle by Assessment of TG, APO-A and APO-B Serum Values in Tabriz

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Abstract: Fatty liver syndrome (Hepatic lipidosis) or fat cow syndrome is a major metabolic disorder in many dairy cattle’s in early period of lactation. The aim of this study was to evaluating fatty liver syndrome in dairy cattle in Tabriz by measurement of TG, APO-A and APO-B serum values. The results showed that TG has a negative correlation with APO-A and APO-B. Thus with elevating of TG serum values, APO-A and APO-B contrary diminished.

Key words: Fatty liver, dairy cattle, TG, APO-A, APO-B, Iran

INTRODUCTION

Fatty liver syndrome (Hepatic lipidosis) or fat cow syndrome is a major metabolic disorder in many dairy cattle’s in early period of lactation (Bruss, 1993; Eddy, 1992) and it is combined with decrease in health and reproduction rate of livestock (Goff and Horst, 1997; Jorritsma et al., 2001).

Fatty liver syndrome was documented in forties (decade, 1940) but there were few researches about it until mid-seventies. In early 70 and 80 decades, this syndrome was reported around parturition widely and it was recorded in many countries (Bruss, 1993; Eddy, 1992). When this disorder is severe, milk production and appetite of cow both are decreased.

So, effective prevention of fatty liver can save millions of dollars every year and prevent from decrease in milk production (Drackley, 1999). Incidence of fatty liver in dairy cattle is mainly in 1st 4 weeks after parturition (Grummer, 1993) when >50% of cows show different degrees of Triacylglycerol (TAG) accumulation in their livers (Jorritsma et al., 2001).

One of the reasons is that daily nutrition of cow is not sufficient and it can not meet increasing need of energy in cattle that is producing milk. In this condition, None Esterified Fatty Acid (NEFA) is released from adipose tissue often more than it is needed and extra amount is transferred to liver, especially in fat cows (Mcnamara, 2000). Fatty liver occurs when liver harvesting of lipids is more than their Oxidation and secretion by the liver and it is with high plasma concentrations of NEFA that is resulted from high adipose tissue (Drackley, 1999; Grummer, 1993). Extra fat is stored in liver as TAG and results in decrease of metabolic function of liver (Drackley, 1999). Liver is classified to three types, according to fat level; normal liver, liver with average fat and liver with very high fat (Drackley, 1999; Grummer, 1993). The latter type is categorized to non-encephalopathic fatty liver (Bobe et al., 2004) and hepatic encephalopathy (Drackley, 1999; Grummer, 1993). Unbalanced or insufficient nutrition, overweight and high concentration of estrogen are involved in etiology of fatty liver (Goff and Horst, 1997).

The disorder can be accompanied with high rate of dystocia infectious and inflammatory disease, long interval between parturitions and reduction of milk and longevity average (Goff and Horst, 1997).

Forasmuch as even slight fatty liver is dual with decrease in health and reproduction status of cow, prevention of its occurrence with supplying enough food and creating an isolated place at preparation period for parturition can reduce decline rate of producing milk and it would be the most efficient therapeutic procedure among the other methods (Wensing et al., 1997).

However, this prevention is not enough for fat cows or the ones that are not feed well, the cows that have problem during parturition or had twins, the cows that have metabolic or infectious disease and the ones that
have developed severe energy imbalance because of producing high amount of milk immediately after parturition (Wensing et al., 1997). Assuming existence of about 9 million dairy cattle all over the America, annual charges of fatty liver in this country is estimated >60 million dollars (Bohe et al., 2004). If there are more studies about molecular changes and relationship between the disease and immunity function, better remedies and more efficient ways to prevent fatty liver can be presented (Zerbe et al., 2000).

In the country because of industrial methods that speed for nurture and maintenance of dairy cattle and because of producing more milk, more nutrition is considered, occurrence of this syndrome is most likely. According to these conditions, providing exact diagnose of this syndrome and estimate it is incidence rate and finally how to prevent it in our country is a necessity and this case made us do the first study about this disease in Tabriz. It’s possible that origin of many diseases happening around parturition could be fatty liver incidence in this region’s dairy cattle.

RESULTS AND DISCUSSION

TG serum value average: Based on Table 2 and ANOVA test and t-test results revealed that there is a significant difference between 2 groups and this variation in group -1 was more significant than +1 (p<0.001).

APB-A serum value average: Based on Table 3 and ANOVA test and t-test results revealed that there is a significant difference between two groups and this variation in group +1 was more significant than -1 (p<0.001).

APB-B serum value average: Based on Table 4 and ANOVA test and t-test results revealed that there is a significant difference between two groups and this variation in group -1 was more significant (p<0.001).

Relationship between TG and APB-A: Based on Table 5 and Pearson’s correlation index revealed that there is a significant and direct correlation between TG and APB-A serum values so that correlation index was \( r = 0.807 \). This index indicates a negative effect of TG on APB-A serum values. Thus with elevating of TG serum values, the APB-A values was decreased.

Relationship between TG and APB-B: Based on Table 6 and Pearson’s correlation index revealed that there is a significant and direct correlation between TG and APB-B

Table 2: Mean serum TG in Holstein dairy cows based on pregnancy status

<table>
<thead>
<tr>
<th>Groups</th>
<th>No.</th>
<th>Mean (mg ml⁻¹)</th>
<th>F</th>
<th>p-value</th>
<th>t-test</th>
<th>df</th>
</tr>
</thead>
<tbody>
<tr>
<td>-1</td>
<td>67</td>
<td>7.0±1.35</td>
<td></td>
<td>0.000</td>
<td>15.21</td>
<td>109.42</td>
</tr>
<tr>
<td>+1</td>
<td>83</td>
<td>4.09±0.88</td>
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<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

Table 3: Mean serum APB-A in Holstein dairy cows based on pregnancy status

<table>
<thead>
<tr>
<th>Groups</th>
<th>No.</th>
<th>Mean (mg ml⁻¹)</th>
<th>F</th>
<th>p-value</th>
<th>t-test</th>
<th>df</th>
</tr>
</thead>
<tbody>
<tr>
<td>-1</td>
<td>67</td>
<td>1.14±0.28</td>
<td></td>
<td>0.58</td>
<td>11.74</td>
<td>148</td>
</tr>
<tr>
<td>+1</td>
<td>83</td>
<td>1.64±0.23</td>
<td></td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

Table 4: Mean serum APB-B in Holstein dairy cows based on pregnancy status

<table>
<thead>
<tr>
<th>Groups</th>
<th>No.</th>
<th>Mean (mg ml⁻¹)</th>
<th>F</th>
<th>p-value</th>
<th>t-test</th>
<th>df</th>
</tr>
</thead>
<tbody>
<tr>
<td>-1</td>
<td>67</td>
<td>0.83±0.28</td>
<td></td>
<td>0.301</td>
<td>11.74</td>
<td>148</td>
</tr>
<tr>
<td>+1</td>
<td>83</td>
<td>1.33±0.23</td>
<td></td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

Table 5: Correlation index between TG and APB-A

<table>
<thead>
<tr>
<th>Variables</th>
<th>APB-A (mg ml⁻¹)</th>
</tr>
</thead>
<tbody>
<tr>
<td>TG</td>
<td>( R = 0.807 )</td>
</tr>
<tr>
<td></td>
<td>( p = 0.000 )</td>
</tr>
<tr>
<td></td>
<td>( N = 150 )</td>
</tr>
</tbody>
</table>

Table 6: Correlation index between NEFA and total Bilirubin serum values

<table>
<thead>
<tr>
<th>Variables</th>
<th>APB-B (mg ml⁻¹)</th>
</tr>
</thead>
<tbody>
<tr>
<td>TG</td>
<td>( R = 0.807 )</td>
</tr>
<tr>
<td></td>
<td>( p = 0.000 )</td>
</tr>
<tr>
<td></td>
<td>( N = 150 )</td>
</tr>
</tbody>
</table>
serum values so that correlation index was $r = 0.807$. This index indicates a negative effect of TG on APO-B serum values. Thus with elevating of TG serum values, the APO-B values was decreased.

For awareness of fatty liver syndrome, blood biochemical parameters can be used or we can measure TAG and total fat of hepatic cell. Some researchers inspect fatty liver based on TAB or hepatic fat percent (Wensing et al., 1997). Reid (1980) divided livers in 4 levels depending on severity of fat accumulation in it; normal, slight, average and severe. Now-a-days, general opinion is that a high percent of mature cows show signs of slight or severe fatty liver around parturition (Drackley, 1999, Grummer, 1995). Almost near parturition NEFA increases in blood and moves to liver and can cause ketosis, abomasums displacement, metritis and fatty liver after parturition (Drackley, 1999, 2000; Geelen and Wensing, 2006). In a normal situation and positive energy balance, NEFA value is about 200 meq L$^{-1}$ in blood. This value increases since 3 weeks is parturition and reaches to 300 meq L$^{-1}$ in the last week. In the last days before parturition, it reaches to 800-200 meq L$^{-1}$. After parturition these acids should wane immediately and if it remains $>700$ meq L$^{-1}$ after 7 days, represents negative energy balance and probability of fatty liver incidence.

About 3 weeks after parturition, the amount of these acids should return to normal level (200 meq L$^{-1}$) (Drackley, 2000). Also, the results of this study have conformity with Grummer (1993) results that showed three is lipid aggregation in liver in 1st 4 weeks after parturition.

There was a research in Netherlands about 71 dairy cattle before parturition that showed 5% of liver is occupied with TAG (Johanssen, 1993). Also in a slaughterhouse research in Tehran, aggregation of TAG $>10\%$ in liver in last month of pregnancy was reported. These researchers had not measured NEFA values. In this study, TAG aggregation in liver in last month of pregnancy had occupied $>5\%$ of liver cells and amount of NEFA was $>900$ meq L$^{-1}$ being nonspecific and some other reasons.

CONCLUSION

This study shows that slight and mild forms of fatty liver can destroy hepatocytes and disturb liver function without making any changes in activity of hepatic specific enzymes found in serum.

Measurement of liver enzymes in serum is useful for evaluating fatty liver disease but with certain restrictions such as is being nonspecific. Mild and moderate forms of fatty liver with damaged hepatocytes can cause liver and no specific changes in liver enzymes in serum, liver dysfunction to establish (Bogin et al., 1988; Ruikkwansuk et al., 1999).

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REFERENCES


