INTRODUCTION

Thyroid hormones are required for normal growth, development and function of nearly all tissues with major effects on oxygen consumption and metabolic rate (Yen, 2001). Disorders of thyroid gland results primarily from autoimmune processes that either stimulate the overproduction of thyroid hormones or cause glandular destruction and hormone deficiency (Larry Jameson and Anthony Weetman, 2005). Thyroid disease constitutes the most common endocrine abnormality in recent years, diagnosed either in clinical or sub-clinical form (Hollowell, Staehling and Flanders et al., 1988-1994). Liver is known to synthesize a number of plasma proteins that bind the lipophilic thyroid hormones (Youssef and Mullen, 2002). Also, liver is the major site for cholesterol and triglyceride metabolism and thyroid hormones play an integral part in hepatic lipid homeostasis (Ness et al.,1998). In addition to the central role in deiodination to activate and deactivate thyroid hormones, the liver performs specific functions relating to thyroid hormone transport and metabolism (Murray, 1974). Clinical features of hyperthyroidism are diverse, involving nearly every system in the body and can be conveniently divided into hepatic or cholestatic disease. The mechanism of hepatic injury appears to be relative hypoaemia in the perivenricular regions, due to an increase in hepatic oxygen demand without an appropriate increase in hepatic blood flow. In mild cases, liver histology shows non-specific changes, consisting of a mild lobular inflammatory infiltrate consisting of polymorphic neutrophils, eosinophils and lymphocytes, associated with nuclear changes and kupffer cell hyperplasia(Huang and Liaw, 1995). In patients with cholestatic injury, histological features are similar to non-specific changes seen in hepatic injury. However,in addition ,there appears to be centri-lobular intrahepatocytic cholestasis (Sola et al.,1991).Increase in AST and ALT may be induced by changes in thyroid function(Kubota et al., 2008). So, the present study is planned to access the effects of hyperthyroidism in perturbing liver functions.

MATERIALS AND METHODS

80 subjects were taken from outpatient department of Government Medical College, Jammu in the age group of 20-65 years. Subjects were divided into 2 groups of 40 euthyroid and 40 hyperthyroid patients. All subjects with pre-existing diseases like diabetes mellitus, renal disorders or any other chronic inflammatory medical conditions were excluded from the study. Physical measurements like weight in kgs and height in (meters) were taken. Thyroid function tests (T3, T4 and TSH) were estimated in human serum and plasma by chemiluscent microparticle immunoassay (Patel et al.,1972; Sterling and Lazarus,1977). In estimation of T3,T4 and TSH, ARCHITECT Total T3 Reagent Kit (7K64) ,ARCHITECT Total T4 Reagent Kit (7K66) and ARCHITECT Total TSH Reagent Kit (7K62) were used respectively. Liver function tests (albumin estimation, alanine aminotransferase, aspartate aminotransferase, serum bilirubin estimation) were estimated in plasma by using chromatic endpoint technique. Albumin estimation is an adaptation of the bromocresol purple dye-binding method (Carter,1970; Louderback et al.,1968).

STUDY OF LIVER FUNCTION TESTS IN HYPERTHYROID PATIENTS

Navneet Kour, Meenakshi Sharma and Sunil Sachdev

Department of Physiology, Government Medical College, Jammu

A R T I C L E   I N F O

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A B S T R A C T

Aim: To study liver function tests in hyperthyroidism
Materials and Methods: 80 subjects in the age group of 20-65 years were taken from the outpatient department of Government Medical College, Jammu and liver function tests including serum alanine aminotransferase, serum aspartate aminotransferase, serum bilirubin and serum albumin were evaluated in these subjects. Thyroid function tests were also determined.
Results: There is significant difference in mean AST (aspartate aminotransferase) and mean ALT (alanine aminotransferase) between euthyroid and hyperthyroid subjects whereas there is no significant difference in mean total bilirubin and mean total serum albumin between euthyroid and hyperthyroid subjects.
Conclusion: Thyrotoxicosis has a significant effect on liver that is reflected in increased level of liver specific enzymes i.e. AST and ALT.
Standard procedures for specimen collection and storing were used. The liver function tests were assessed according to Clinical Diagnosis and Management by Laboratory Methods (Henry, 2003).

Statistical Analysis

Data was subjected to one way analyses of variance (ANOVA, Kryplot version 2). In all the cases, means are used as units of analyses and are represented as Mean ± SD. A p-value of less than 0.05 was considered statistically significant.

RESULTS

Table 1 Comparison of mean total bilirubin of euthyroid and hyperthyroid subjects.

<table>
<thead>
<tr>
<th>Classification of subjects</th>
<th>T. Bilirubin (mg%)</th>
<th>Mean ± SD</th>
<th>F-value</th>
<th>P-value</th>
<th>Significant difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Euthyroid</td>
<td>0.54±0.24</td>
<td>F(1,78)=1.2687</td>
<td>P= 0.2635</td>
<td>Significant as P &gt; 0.05</td>
<td></td>
</tr>
<tr>
<td>Hyperthyroid</td>
<td>0.49±0.16</td>
<td>F(1,78)=1.0673</td>
<td>P= 0.3048</td>
<td>Significant as P &gt; 0.05</td>
<td></td>
</tr>
</tbody>
</table>

Table 2 Comparison of serum albumin of euthyroid and hyperthyroid subjects.

<table>
<thead>
<tr>
<th>Classification of subjects</th>
<th>S. Albumin (mg%)</th>
<th>Mean ± SD</th>
<th>F-value</th>
<th>P-value</th>
<th>Significant difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Euthyroid</td>
<td>3.87±0.36</td>
<td>F(1,78)=1.0673</td>
<td>P= 0.3048</td>
<td>Significant as P &gt; 0.05</td>
<td></td>
</tr>
<tr>
<td>Hyperthyroid</td>
<td>3.95±0.39</td>
<td>F(1,78)=1.0673</td>
<td>P= 0.3048</td>
<td>Significant as P &gt; 0.05</td>
<td></td>
</tr>
</tbody>
</table>

Table 3 Comparison of mean ALT levels of euthyroid and hyperthyroid subjects.

<table>
<thead>
<tr>
<th>Classification of subjects</th>
<th>AST(IU/L)</th>
<th>Mean ± SD</th>
<th>F-value</th>
<th>P-value</th>
<th>Significant difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Euthyroid</td>
<td>25.18±7.68</td>
<td>F(1,78)=18.4279</td>
<td>P= 0.0001</td>
<td>Significant as P &lt; 0.05</td>
<td></td>
</tr>
<tr>
<td>Hyperthyroid</td>
<td>34.48±11.37</td>
<td>F(1,78)=18.4279</td>
<td>P= 0.0001</td>
<td>Significant as P &lt; 0.05</td>
<td></td>
</tr>
</tbody>
</table>

Table 4 Comparison of mean ALT levels of euthyroid and hyperthyroid subjects.

<table>
<thead>
<tr>
<th>Classification of subjects</th>
<th>ALT (IU/L)</th>
<th>Mean ± SD</th>
<th>F-value</th>
<th>P-value</th>
<th>Significant difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Euthyroid</td>
<td>35.7±10.54</td>
<td>F(1,78)=16.6364</td>
<td>P= 0.0001</td>
<td>Significant as P &lt; 0.05</td>
<td></td>
</tr>
<tr>
<td>Hyperthyroid</td>
<td>46.45±12.91</td>
<td>F(1,78)=16.6364</td>
<td>P= 0.0001</td>
<td>Significant as P &lt; 0.05</td>
<td></td>
</tr>
</tbody>
</table>

DISCUSSION

Thyroid hormones influence the function of all body organs and cells. The data presented here clearly indicates how biochemical markers of liver may be affected by alteration in the level of thyroid hormones in the body. Our findings are in consensus with the study by Ohshima et al., (1990) that liver has an important role in metabolism of thyroid hormone and autopsies have shown hepatic inflammation, fibrosis and centrilobular necrosis in patients with hyperthyroidism. Our study is also in accordance with the theory that thyrotoxicosis may cause a defect in bilirubin metabolism by decreasing bilirubin UDP-glucouronyl transferase (Greenberger et al., 1964). The physiological effects of hyperthyroidism may cause an increased hepatopexy oxygen consumption without an equal increase in blood flow, causing focal hypoxiaemia and hepatic dysfunction (Venditti et al., 2006). It has also been hypothesized that these abnormalities are in part related to congestive heart failure and venous congestion caused by hyperthyroidism, although features of congestive hepatopathy were not evident in liver biopsy (Dooner et al., 1967). Thyroid hormones regulate the basal metabolic rate of all cells including hepatocytes and hence modulate hepatic function (Malik and Hodgson, 2002). Lim et al., 1993 in a study found that hyperthyroidism in a well documented cause of abnormal liver enzymes. The cases in the present study were mainly the referral cases from different outpatient departments of the hospital. A significant difference was observed in TSH, T3 and T4 levels of euthyroid and hyperthyroid patients.

CONCLUSION

We conclude that thyrotoxicosis has a significant effect on liver that is reflected in increased levels of AST and ALT levels.

References

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