IMPACT OF BODY MASS INDEX ON LIVER ENZYMES IN POSTMENOPAUSAL WOMEN

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ABSTRACT

Objectives: To assess the correlation of BMI with liver enzymes in postmenopausal women.

Methodology: The study was conducted on 100 postmenopausal women in the age group of 45-55 years. Subjects were divided into 3 subgroups: Normal, Overweight and Obese on the basis of BMI. Body weight and height was taken and BMI was calculated using Quetlet index. Early morning Blood samples were taken after a minimum of 8 hours of fasting for assessment of liver enzymes AST and ALT. All parameters were reported as mean and standard deviation. The statistical differences in mean values were tested using Levene’s test for equality of variances followed by t-test for equality of means. ANOVA was used to do comparison within the subgroups. Pearson correlation was used to calculate the correlation between variables. A p-value <0.05 was considered statistically significant and p-value >0.05 was considered non-significant.

Conclusion: A highly significant correlation was seen between liver enzyme AST and BMI in normal, overweight and obese postmenopausal females. There is significant rise of AST levels in obese postmenopausal women.

INTRODUCTION

Menopause, a normal biological event marked for most women by the end of menstrual periods, signifies the depletion of functional ovarian follicles that are responsible for estradiol production[1]. Estrogen deficiency, due to natural menopause or surgical menopause, has been suggested to have an effect on insulin resistance[2,3,4].

Now a days, due to urbanization & industrialization, there is a dramatic change in lifestyle, consisting of physical inactivity, diet rich in fat, sugar and salt, coupled with a high level of mental stress, weight gain and lifestyle diseases associated with it, which can be prevented due to lifestyle modifications such as diet and exercise[5]. So, the current interest is focussed on the abrupt endocrine changes during menopausal transition which have important impact on the physiology of female body, which exacerbate risk for many diseases and disabilities during postmenopausal life[6]. This study elucidates the effect of BMI on liver enzymes as a positive relationship of BMI, exists with major coronary risk factors, hypertension and metabolic syndrome[7].

There is deterioration of metabolic profile with increase in body mass index[8]. Early postmenopausal status is associated with a preferential increase in intra-abdominal fat that is independent of age and total body fat mass[9].

Menopause results in an increase in fat mass and redistribution of body fat, with a relative increase in the proportion of android fat[10]. These results may be influenced by a modified energy balance (lower energy expenditure because of less physical activity) but also a decrease in the basal metabolic rate in postmenopausal women[11].

In the present study, liver enzymes AST and ALT were measured in postmenopausal women. These were measured to elucidate the correlation of BMI with liver enzyme. Obesity is one of the risk factors most frequently associated with increased liver enzymes[12].

MATERIAL AND METHODS

Selection Procedure of the Subjects

Subjects were selected from different outpatient departments of Government Medical College, Jammu. Subjects included healthy postmenopausal women in the age group of 45 to 55 years. All those women with H/O diabetes mellitus, hypertension, neurological disorders, any other illness known...
to affect fasting blood glucose, were excluded from the study, as were smokers and alcoholics. For body mass index, weight was measured in kilograms; on a weighing scale. Height in meter was also measured. Body mass index was calculated using Quetelet’s Index\[13\].

According to the currently recommended cut-offs of BMI recommended by World Health Organization subjects with a BMI of 18.5-24.9 kg/m\(^2\) were classified as normal, while those with a BMI of 25.0-29.9 kg/m\(^2\) were classified as overweight and those with a BMI of \(\geq\) 30 kg/m\(^2\) were classified as obese. So, subjects were divided into subgroups namely normal, overweight and obese.

**Specimen collection and handling:** Venous blood samples were obtained from the cubital vein after 8-12 hours overnight fasting. Separated plasma or serum specimens are stable for 8 hours at room temperature, 2 days at 2-8\(^\circ\) Celsius\[14\].

**Estimation of liver enzymes**

1. **AST (Aspartate aminotransferase):** The AST method based on the dimension clinical chemistry system is an in vitro diagnostic test intended for the quantitative determination of AST activity in human serum or plasma.

   The aspartate aminotransferase method is an adaptation of the methodology recommended by the International Federation of Clinical chemistry \[15\]. The method uses the coenzyme pyridoxal 5-phosphate to activate the apoenzyme and lactic acid dehydrogenase (LDH) to eliminate pyruvate interference.

2. **ALT (Alanine aminotransferase):** The ALT method is based on the dimension clinical chemistry system is an in vitro diagnostic test intended for the quantitative determination of ALT in human serum or plasma.

   The alanine aminotransferase method is an adaptation of the recommended procedure of the International Federation of Clinical Chemistry \[16\]. The procedure is based on the principles outlined by \[17\] but is modified to contain pyridoxal 5-phosphate (PSP) as an activator and to replace phosphate buffer with tris (hydroxymethyl) aminomethane.

**Statistical Analysis**

The statistical differences in mean values were tested using Levene’s test for equality of variances followed by t-test for equality of means. ANOVA was used to do comparison with in the subgroups. Pearson correlation was used to calculate correlation between various variables.

**RESULTS**

**Table 1 Mean AST of subjects**

| Subjects       | Mean ALT ± SD (Range) (in IU/ml) |
|----------------|----------------------------------|
| Postmenopausal | 40.93 ± 13.31 (18–25)            |

**Table 2 Correlation of BMI with AST in postmenopausal women**

| BMI (Kg/m\(^2\)) | Classification | Postmenopausal ALT (Group II (no.)) | Mean ALT ± SD (Range) (in IU/ml) | Pearson correlation with p-value |
|------------------|----------------|-------------------------------------|---------------------------------|---------------------------------|
| 18.5             | Normal weight  | 37                                  | 42.51 ± 9.89  (30–75)           | 0.723                           |
| – 24.99          |                |                                     |                                 |                                 |
| 25               | Overweight     | 34                                  | 57.94 ± 17.59  (25–85)          | 0.963                           |
| – 29.99          |                |                                     |                                 |                                 |
| ≥ 30.00          | Obese          | 29                                  | 76.24 ± 16.08  (32–110)         | 0.876                           |
|                  |                |                                     |                                 |                                 |

**Table 3 Mean ALT of subjects**

| Subjects       | Mean ALT ± SD (Range) (in IU/ml) |
|----------------|----------------------------------|
| Postmenopausal | 57.54 ± 19.97  (25 – 110)        |

**Table 4 Correlation of BMI with ALT in Postmenopausal women**

| BMI (Kg/m\(^2\)) | Classification | Postmenopausal ALT (Group II (no.)) | Mean ALT ± SD (Range) (in IU/ml) | Pearson correlation with p-value |
|------------------|----------------|-------------------------------------|---------------------------------|---------------------------------|
| 18.5             | Normal weight  | 37                                  | 42.51 ± 9.89  (30–75)           | 0.723                           |
| – 24.99          |                |                                     |                                 |                                 |
| 25               | Overweight     | 34                                  | 57.94 ± 17.59  (25–85)          | 0.963                           |
| – 29.99          |                |                                     |                                 |                                 |
| ≥ 30.00          | Obese          | 29                                  | 76.24 ± 16.08  (32–110)         | 0.876                           |
|                  |                |                                     |                                 |                                 |

**DISCUSSION**

Findings in the study were supported by the fact that liver is profoundly affected by obesity where it may be associated with hepatomegaly, increased liver biochemistry values and alterations in liver histology like macrovesicular steatosis, steatohepatitis, fibrosis and cirrhosis\[18\].

Also, correlation of BMI with ALT levels in the three subgroups (normal, overweight and obese), in perimenopausal women, is highly significant. This is analogous with the study that the mean ALT in persons with increased BMI is higher than those with normal BMI\[19\].

Assessed body weight composition in postmenopausal women and determined correlations with metabolic and hormonal parameters\[20\]. The authors found that body mass index values positively correlated with age, time since menopause, parity and glucose, thereby displaying significant correlations with hormonal and metabolic parameters. This is in disagreement with our study as we did not observe significant difference between perimenopausal and postmenopausal women in terms of blood sugar and AST but ALT levels are significantly higher in obese postmenopausal women as compared to perimenopausal women.

Liver plays a major role in carbohydrate homeostasis. Hepatocellular glycogen accumulation leads to hepatic abnormalities in poorly controlled diabetes patients. In hyperglycaemic states, there is accumulation of glycogen in the hepatocytes due to increased glycogen synthesis, causing findings of mild to moderately elevated aminotransferases\[21\].

Non-alcoholic fatty liver disease is the main cause of chronic liver disease associated with obesity\[22\]. Diabetes, steatosis, and age are all significant indicators of cirrhosis.

Liver accumulation of fat in patients with diabetes mellitus or with the insulin resistance syndrome is mainly related to increased lipolysis of adipose tissue, with increased flux of free fatty acids to the liver that exceeds the liver’s capacity to export VLDL\[23\].

Relationship is present between abdominal obesity and serum alanine transferase (ALT) levels and between body mass index and alkaline phosphatase (ALP) levels. These findings suggest that serum ALP, particularly liver ALP, is derived from adipose and hepatic tissue\[24\].
Serum alanine transferase (ALT) is commonly used as a diagnostic marker and predictor of many liver diseases. The mean ALT in persons with increased BMI is higher in than those with normal BMI. Liver enzyme tests are commonly used to screen for liver diseases such as fatty liver and hepatitis[25]. Increased levels primarily of alanine aminotransferase (ALT) and triglycerides, and secondarily of gamma-glutamyltransferase (GGT), appear to be the most sensitive biochemical indicators of the presence of hepatic steatosis[26,27]. Even a minor elevation of ALT is a good predictor of mortality from liver disease[28].

CONCLUSION

We conclude that highly significant correlation was seen between liver enzyme AST and BMI in normal, overweight and obese postmenopausal females. There is significant rise of AST levels in obese postmenopausal women.

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**How to cite this article:**

Meenakshi Sharma et al (2017) 'Impact Of Body Mass Index On Liver Enzymes In Postmenopausal Women', *International Journal of Current Advanced Research*, 06(03), pp. 2436-2439.