Differences in hepatic liver function in women with Turner Syndrome and women with POI: A clinical audit

K. Stefanaki[1], A. Kalampalikis[2], G. Ntali[3], S. Ivanidou[2], L. Michala[2]

[1] Department of Therapeutics, "Alexandra" General Hospital, National and Kapodistrian University of Athens. Greece

[2]1st Department of Obstetrics and Gynecology, "Alexandra" General Hospital, National and Kapodistrian University of Athens. Greece

[3]Department of Endocrinology and Diabetes, "Evangelismos" Hospital, Athens, Greece

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Introduction

Both Turner syndrome (TS) and Premature Ovarian Insufficiency (POI) are characterized by estrogen deficiency, which in turn leads to osteoporosis and increased cardiometabolic risk. Different pathophysiologic mechanisms have been examined to interpret the metabolic syndrome which initially presents with liver dysfunction or prediabetes; however, no causing factor has yet been identified. Exogenous estrogen replacement therapy administration plays a protective role for liver function by ameliorating insulin resistance. It has been proposed that an inherent factor predisposes to liver injury in TS, commonly presenting as liver steatosis. However, the impact of the karyotype abnormality of X monosomy is difficult to be estimated.

Objectives

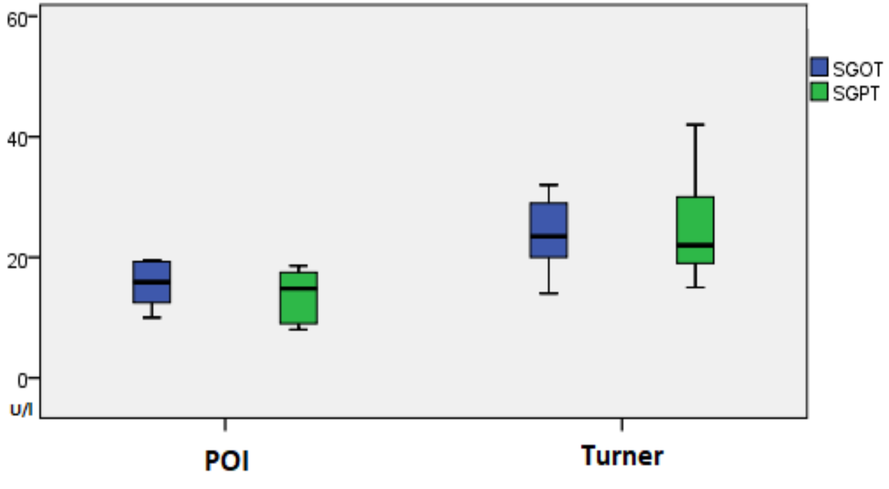
Given the increased risk of women with TS for liver dysfunction, we compared women with TS and POI to identify factors that could lead to this impairment.

Materials & Methods

This was a retrospective review of the notes of all patients with a diagnosis of TS or POI, referred to a tertiary referral center for complex pediatric and adolescent gynecology and reproductive endocrinology conditions, over a five-year period.

Information was collected at the age of the last clinical evaluation for each patient. The karyotype, the age at diagnosis, the type of hormone replacement therapy (HRT), somatometric characteristics, glucose levels, glycosylated hemoglobin, liver function tests (LFTs), lipid profile, and Bone Mineral Density were recorded for each woman.

Quantitative variables were expressed as mean values (SD) or as median values (interquartile range=IQR). Qualitative variables are presented with absolute and relative frequencies. Student's t-tests were computed for the comparison of mean values when the distribution was normal and Mann-Whitney test for the comparison of median values when the distribution was not normal.



Graph 1. Differences observed in the values of SGOT and SGPT in patients with POI and Turner syndrome

Results & Discussion

We identified 21 women with TS and 29 women with POI, all receiving oral preparations of sequential combined HRT.

Their age range was 12-48 and 15—38 years, respectively. The Body Mass Index and lipid profile did not differ between the two groups. Only two women with TS and one with POI had raised LFTs (SGPT 48, 62 and 117U/l respectively). However, liver enzyme concentrations were higher in women with TS (SGOT median/IQR 23.5/9.75, SGPT median/IQR 22/14.75 U/L) when compared to women with POI (SGOT median/IQR 15.9/7.2U/L, SGPT median/IQR 14.8/8.75U/L). The difference was statistically significant (SGOT $p=0.012$ and SGPT $p<0.01$).

Given the fact that all other metabolic parameters between the two groups, were not statistically different, we hypothesize that another, inherent factor may cause raised LFTs in TS.

Liver biopsies performed in women with TS have revealed vascular and steatic abnormalities. Genetic and epigenetic dysregulation caused by the loss of genetic information may cause these abnormalities. Although only a minority of women had overtly raised LFTs, the slightly raised levels may indicate a predisposition for future liver disease.

Conclusions

1. Liver enzyme levels were statistically higher in women with TS.
2. BMI, HDL, LDL, and cholesterol levels did not differ between the two groups.
3. X monosomy, rather than estrogen deficiency is likely to be the cause for this aberration.