Effect of Toxoplasmosis on Lipid Profile and Thyroid Hormones in Aborted Women

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Abstract

This study addressed the effect of toxoplasmosis on levels of thyroid hormones (Triiodothyronine [T3], Thyroxine [T4] and Thyroid Stimulating Hormone [TSH], and lipid profile such as (Cholesterol, Triglycerides, High-Density Lipoprotein [HDL], Low-Density Lipoprotein [LDL] and very Low -Density Lipoprotein [vLDL] in 100 samples which included 80 aborted women, who had positive test for toxoplasmosis and 20 samples act as control group during the period from August 2015 to January 2016 which were taken from Al-Yarmouk Teaching Hospital in Baghdad City. Infected and control women had similar age that ranged between (17-47) years old. Serum samples were collected and tested by ELISA technique, lipid profile tests were performed by using commercial kits, in addition to measured thyroid hormones by using the Mini Vidus apparatus. The results revealed chronic infection [IgG] recorded the highest percentage (68.75%) and the acute infection [IgM] showed the lowest percentage (31.25%). There were significant decrease in the total cholesterol, Triglycerides, LDL and vLDL (110.96±1.55, 30.93±0.56, 41.38 ±1.55 & 6.40 ±0.18 mg/dl respectively) compared with control group (200.05 ±5.83, 56.05 ±1.98, 142.85±2.05 & 36.90±1.24 mg/dl respectively). On the other hand, a significant increase in HDL, T3 and TSH was recorded (64.51±1.27 mg/dl, 12.74 ±0.53 and 11.64±0.58 mlU/L respectively) compared with control group (53.45±1.57 mg/dl, 1.49 ±0.16 & 2.29 ±0.25 mlU/L respectively). A significant decline in T4 was estimated (2.81±0.12 mlU/L) compared with control group (8.35±0.80 mlU/L). So, we concluded that the Toxoplasma gondii had important role in changes of lipid profile levels & thyroid hormones in infected women.

Keywords: Toxoplasmosis, lipid profile, thyroid gland hormones, Toxoplasma gondii.

Introduction

Toxoplasma gondii is the most widespread parasitic protozoa and an liable for some of the most destructive common diseases of humans [1], classified in the phylum Apicomplexa and coccidian class [2]. This parasite accesses the host through the digestive system due to consuming contaminated food or drink with oocysts or tissue cysts [3]. Toxoplasma gondii replicates inside a host cell in a specialized vacuole known as the parasitophorous vacuole (PV) [4], so the replication of this parasite inside the (PV) need substantial amount of the specific lipids for membrane biogenesis [5]. It is autonomously synthesizes phospholipids, but can too easily search lipid predecessor from host cells [6]. Thyroid gland produce two necessary thyroid hormones into blood stream, the first one called thyroxine (T4) and the other called triiodothyronine (T3) [7]. T3 & T4 are synthesized from iodine and tyrosine, they elevate optimal growth, development, function and conservation of all body tissues. The synthesis and secretion of these hormones effect by a hormone released via the pituitary gland called Thyroid – Stimulating Hormone (TSH), also this gland produce calcitonin which plays a role in calcium homeostasis [8], the abnormal thyroid function has multiple effect for public health, because it construct and stores hormones which are help regulate the heart rate, body temperature, blood pressure, body weight and cholesterol [9]. In addition to the thyroid hormones are necessary for the function of every cell in the body [10]. The purpose of this study is to confirm the relationship between lipid profile, thyroid hormones levels and toxoplasmosis aborted women in Baghdad City.

Materials and Methods

Subjects

The study included 80 blood samples collected from aborted women infected with toxoplasmosis, and 20 samples as control group. All these samples had similar age ranged...
between 17-47 years old, and collected between August 2015 to January 2016. They were taken from Al-Yarmouk Teaching Hospital in Baghdad, then centrifuged and sera were kept frozen at -20°C till they were analyzed.

The serological tests
were carried out by using the onsite Toxo IgG / IgM (Rapid Test Kit, USA), which was a lateral flow chromatographic immunoassay for the simultaneous detection and differentiation of IgG and IgM anti-toxoplasma gondii in human sera or plasma, in addition to serum level of IgG and IgM for Toxoplasma gondii was estimated by ELISA technique (BioTek, USA).

Lipid Profile Tests
Level of lipid profile included Total Cholesterol[C], Triglycerides [TGS] and High-Density Lipoprotein [HDL] which were estimated by enzymatic colorimetric (CHOD-PAP, N.S. BIO-TEC, U.K.). While Low – Density Lipoprotein [vLDL] was calculated according to Friedewald formula [11]:

\[
\text{LDL}\text{mg/dl} = C - \text{HDL} - \text{TGS}/5 \\
\text{vLDL}\text{mg/dl} = \text{TGS}/5 \\
C= \text{Cholesterol} \\
\text{TGS}= \text{Triglycerides}
\]

Measurement of thyroid hormones
Thyroid hormones (Triiodothyronine [T3], Thyroxine [T4] and Thyroid Stimulating Hormone [TSH] were measured by Mini-Vidas apparatus which is a Compact Automated Immunoassay Analyzer, product from BioMerieux company – France. The principle of it based on the Enzyme Linked Fluorescent Assay (ELFA). The Mini- Vidas is a compact model fitted with a reinforced – in computer, so the results were taken automatically by internal printer. Thyroid hormone levels were taken into account as normal T3 hormone level (0.7-1.9 mIU/L), T4 (4.5-12 mIU/L) and TSH (0.25-5.0 mgU/L).

Statistical Analysis
The Statistical Analysis System (SAS) program was used to outcome of different groups in study parameters. Slightest significant difference – LSD test was used to significant compare between means in this study [12].

Results and Discussion
Results shown in Table (1) show that higher prevalence of anti-Toxoplasma antibodies IgG (68.75%) in women and (31.25%) of IgM antibodies with highly significant differences (P≤0.01). Specific IgM antibodies appear during the first 2 weeks of acute illness, and peak within 4 to 8 weeks, finally become undetectable, but they may be present for as long as 18 months after acute infection, while IgG antibodies arise further slowly and peak in 1 to 20 months and may remain high in addition to stable for months to years, assays for Toxoplasma IgM lack specificity [13].

<table>
<thead>
<tr>
<th>Antibody</th>
<th>No.</th>
<th>(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>IgG</td>
<td>55</td>
<td>68.75</td>
</tr>
<tr>
<td>IgM</td>
<td>25</td>
<td>31.25</td>
</tr>
<tr>
<td>Total</td>
<td>80</td>
<td>100</td>
</tr>
</tbody>
</table>

\**(P≤0.01)**

As shown in Table (2), the mean of cholesterol, Triglycerides, LDL and vLDL revealed a significant decrease levels (110.96±1.55, 30.93±0.56, 41.38±1.55 and 6.40±0.18 mg/dl respectively), compared with control group (200.05±5.83, 56.05±1.98 & 36.90±1.24 mg/dl respectively). On the other hand, mean of HDL appeared increased level (64.51±1.27 mg/dl) compared with control group (53.45±1.57 mg/dl), (P≤0.05).

Many workers have recorded that medium containing lipoproteins supported the growth of the parasites in vitro, it is feasible that all these parasites use LDL-like receptors to endocytosis different lipoprotein particles [14]. The parasites make significant variation in lipid parameters, as has been appeared in vitro study, where exchange of serum by lipid/cholesterol in medium (in vivo), thus changes in lipid profile take place in patients having active infections with most of the parasites [15]. Cholesterol is a major

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component of eukaryotic membranes, in addition it plays a main role in cellular membrane organization, dynamics, function and categorization [16]. While parasites membranes contain cholesterol, *Toxoplasma* needs cholesterol biosynthetic enzymes and must scavenge it from its host [17], for this reason *Toxoplasma gondii* exploits host low density lipoprotein receptor-mediated endocytosis for cholesterol obtaining by the LDL pathway by proliferating parasites triggers mobilization of cholesterol from the liver to the periphery, a process that is specifically increased in infected cells [14]. This result is confirmed by [18], who revealed that *Toxoplasma gondii* plays an important role in changes of lipid profile values in infected women with appeared in significant decrease cholesterol, triglyceride, and LDL levels, with a significant increase in HDL level.

### Table (2)

**Comparison between *Toxoplasma* and control groups in lipid profile tests.**

<table>
<thead>
<tr>
<th>Group</th>
<th>No.</th>
<th>Mean ± SE</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Cholesterol mg/dl</td>
</tr>
<tr>
<td>Toxoplasma</td>
<td>80</td>
<td>110.96±1.55</td>
</tr>
<tr>
<td>Control</td>
<td>20</td>
<td>200.05±5.83</td>
</tr>
<tr>
<td>LSD</td>
<td>-----</td>
<td>8.425*</td>
</tr>
</tbody>
</table>

*(P≤0.05)*

On the other hand, Table (3) revealed a significant increase in the mean of T3 & TSH hormones (12.74±0.53 & 11.64±0.58 mIU/L respectively), compared with control group (1.49±0.16 & 2.29±0.25 mIU/L respectively). While the mean of T4 hormone showed a significant decrease in level (2.81±0.12 mIU/L) compared with control group (8.35±0.80 mIU/L). Some people with thyroid and autoimmune thyroid conditions have parasitic infections, in addition to parasites can aggravate thyroid problems [19]. Thyroid gland is assist to regulate growth as well as the rate of chemical reactions (metabolism) in the body [20]. There is a high relationship between the thyroid gland damage and *Toxoplasma* parasite, due to direct involvement of thyroid gland by *Toxoplasma gondii*, multiplication & propagation of this parasite in thyroid tissue changes and subsequent alteration of thyroid hormones, also may be due to reactivation of latent toxoplasmosis which may persist after or within 6 months of recovery from first initiation of toxoplasmosis [21]. *Toxoplasma gondii* may cause neurological stimuli for hypothalamus-pituitary thyroid axis for accelerating of proteolysis of the thyroglobulin, which causes release thyroid hormones such as triiodothyronine (T3) into the blood within 30 minutes [22].

Additionaly, when the *Toxoplasma gondii* reaches hypothalamus lead to change its stimulation, resulting to distraction in TSH secretion as well as T3 and T4 abnormal productions [23-24]. In addition to, the elicited T4 responses were distinctly diminished in magnitude, reflecting absence of easily obtainable thyroidal T4 reserves, because the containing synthesis, storage and release of T4 is dependent on the pulsattle of the thyroid by TSH [25]. This result was similar to [26-25], which revealed the effect of toxoplasmosis on thyroid hormones, high rates TSH and T3 were recorded among females in Kirkuk city, and a decline in serum thyroxine T4 (hypothyroxinemia) occurs in female mice infected with *Toxoplasma gondii* might be result of primary thyroid dysfunction.
Table (3)
Comparison between Toxoplasma and control groups in levels thyroid hormones.

<table>
<thead>
<tr>
<th>Group</th>
<th>No.</th>
<th>T3 mIU/L</th>
<th>T4 mIU/L</th>
<th>TSH mIU/L</th>
</tr>
</thead>
<tbody>
<tr>
<td>Toxoplasma</td>
<td>80</td>
<td>12.74±0.53</td>
<td>2.81±0.12</td>
<td>11.64±0.58</td>
</tr>
<tr>
<td>Control</td>
<td>20</td>
<td>1.49±0.16</td>
<td>8.35±0.80</td>
<td>2.29±0.25</td>
</tr>
<tr>
<td>LSD</td>
<td>---</td>
<td>2.147*</td>
<td>0.919*</td>
<td>2.361*</td>
</tr>
</tbody>
</table>

*(P≤0.05)

It was concluded from this study, that *Toxoplasma gondii* has an important role in changes of lipid profile which characterized by a significant decrease in Cholesterol, Triglycerides, LDL & vLDL levels, in addition to a significant increase in HDL level. Also, *Toxoplasma* may impaired thyroid function.

References


