Frequency of Hypothyroidism in Patients with Fibromyalgia Syndrome.

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Summary:

Background: Fibromyalgia (FMS) is characterized by chronic widespread pain and allodynia (pain from stimuli which are not normally painful with pain that may occur other than in the area stimulated) of more than 3 months duration. The current hypothesis of the etiology of FMS includes inflammatory and neuroendocrine disorders. Many features of fibromyalgia and hypothyroidism are virtually the same, and thyroid hormone treatment trials have reduced or eliminated fibromyalgia symptoms. These findings led the authors to test the hypothesis that fibromyalgia patients are hypometabolic compared to matched control.

Patients and Methods: The samples were taken from the patients attending the outpatient clinic in Medical city- Baghdad Teaching Hospital – Rheumatology and Rehabilitation Consultation Unit, where the anthropometric tests were performed, from the period Nov. 2010 till June 2011. The samples were taken from 57 fibromyalgia patients and 34 apparently healthy controls. Blood samples were collected and T3, T4, and TSH were estimated in serum of patients and control.

Results: The present results show that only 6.6% of the FMS patients had elevated serum TSH level, and the T3, T4, and TSH results were non-significant.

Conclusion: There is no conclusive evidence of specific thyroid dysfunction in FMS, although, it remains an important diagnosis to exclude when making a primary diagnosis.

Key words: Fibromyalgia, hypothyroidism, stress.

Introduction:

Fibromyalgia Syndrome (FMS) is a chronic disorder of diffuse pain in the muscle or joints, accompanied by tenderness on examination at specific, predictable anatomic sites known as tender points [1,2]. It also can be defined as an idiopathic, non-articular pain defined by a widespread musculoskeletal pain and generalized tender points [3]. FMS signs are: Chronic, widespread pain, and heightened pain, tender points, depressive disorder, fatigue and sleep problems, musculoskeletal pain, stress, localized pain, cognitive disturbances, and hypersensitivity [4,5,6]. The chronic widespread pain of muscle origin is reflected in the criteria for diagnosis published by the Multicenter Criteria Committee of the American College of Rheumatology (ACR). Fibromyalgia has been estimated to effect up to 2-4% of the general population of industrialized countries [7]. Although, a pathophysiologic mechanisms remain unknown, no structural, inflammatory, metabolic or endocrine abnormality has been identified [2]. Histologic and histochemical studies, suggested a possible metabolic myopathy [2]. The thyroid gland controls how quickly the body uses energy, makes proteins, and controls the body’s sensitivity to other hormones. It participates in these processes by producing thyroid hormones. The principal ones being triiodothyronine (T3) and thyroxine (T4) [8]. Hormonal output from the thyroid is regulated by thyroid-stimulating hormone (TSH) produced by the anterior pituitary, which itself is regulated by thyrotropin-releasing hormone (TRH) produced by the hypothalamus [8]. Hypothyroidism is a disorder that occurs when the thyroid gland does not make enough thyroid hormone to meet the body’s needs to regulate metabolism - the way the body uses energy – and affects nearly every organ in the body [9,10]. Hypothyroidism can be associated with the following symptoms: [11,12] Fatigue, weight gain, puffy face, cold intolerance, joint and muscle pain, constipation, dry skin, decreased sweating, heavy or irregular menstrual periods and impaired fertility, depression, and slowed heart rate. Although there are some uncommon symptoms, include: Impaired memory, impaired cognitive function (brain fog). There is currently no consensus on the underlying mechanism of FMS. However, a line of evidence collectively indicates that inadequate thyroid hormone regulation, due to hypothyroidism or the peripheral type of cellular resistance to thyroid hormone (PRTH), is the main.

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Compared to a 1–5% incidence of primary hypothyroidism in the general population, the reported incidence among FMS patients is 10–24% [14, 15]. In a study, TSH level positively correlated with patients’ pain distribution, and the FT3 level inversely correlated with pressure-pain threshold. In view of previous studies showing a high incidence of primary hypothyroidism in FMS, the positive correlation between TSH level and pain distribution raises the possibility that pain distribution in FMS is associated with primary hypothyroidism [13]. Evidence exists that most cases of FMS are associated with difficulties in thyroid production or utilization, while others have documented the similar appearance of FMS and hypothyroidism [16]. On the other hand, it was found that autoimmune thyroiditis is present in a high percentage of FMS, and it has been associated with the presence of typical symptoms of the disease [17]. One cross-sectional study found that the prevalence of thyroid microsomal antibodies was significantly higher in people suffering from long-term musculoskeletal complains throughout the body, and evidence exists of an association between thyroid autoimmunity and FMS [18].

The aim: Although many studies showed that there was an association between FMS and thyroid autoimmunity, and as the many symptoms of FMS resemble that of hypothyroidism, so the aim of the present study is to exclude hypothyroidism in patients with FMS.

Patients and method:

Patients: The present study includes 57 patients with Fibromyalgia Syndrome (49 females and 8 males), their mean age 43.017 ± 10.945 years. The clinical diagnosis of these patients was confirmed by the consultant rheumatologists of the former hospital according to the ACR 1990 criteria for the diagnosis of FMS. The study was performed during the period from November 2010 till March 2011. The subjects were selected from the people attending the out-patient clinic in Medical city – Baghdad Teaching Hospital – Rheumatology and Rehabilitation Consultation Unit. Control: Thirty-four control individuals apparently healthy (28 females and 6 males) who were age and sex matching with FMS patients, were included in this study. Determination of Thyroid Hormones (Total T3, T4, and TSH): Principle of the assay: The (T3, or T4, or TSH) ELISA is based on the principle of competitive binding between T3 (or T4, TSH) in a test specimen and T3 (or T4, TSH) - peroxidase conjugate for a limited number of binding sites on the anti-T3 (or T4, TSH) sheep coated wells. Thus the amount of T3 (or T4, TSH) - peroxidase conjugate bound to the well in inversely proportional to the concentration of T3 (or T4, TSH) in the specimen. After incubation of specimen and T3 (or T4, TSH) - peroxidase conjugate unbound enzyme conjugate is removed in the equilibrium state by washing. TMB/substrate solution is added, and the blue color develops. The intensity of this color, which changes to yellow after stopping the reaction, is inversely proportional to the amount of T3 (or T4, TSH) in the specimen [19]. Statistical Analysis: The overall values for the results in the studied group were performed according to program of SPSS. Statistics for the data were expressed as mean ± S.D., using independent sample t-test p<0.005, and considered statistically significant.

Results:

Although 5 patients out of the 57 (6.6%) have elevated serum TSH level, but the results of sera thyroid hormones in this study show a non-significant differences between FMS patients and healthy control. The results are illustrated in the table and the figure below.

Table: Thyroid hormones in serum of FMS patients and control.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Specimen</th>
<th>Mean</th>
<th>S.D.</th>
<th>p.value</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>T3 (ng/ml)</td>
<td>Patients</td>
<td>0.843</td>
<td>0.224</td>
<td>0.116</td>
<td>N.S.</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>0.932</td>
<td>0.224</td>
<td></td>
<td></td>
</tr>
<tr>
<td>T4 (µg/dl)</td>
<td>Patients</td>
<td>6.328</td>
<td>1.238</td>
<td>0.128</td>
<td>N.S.</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>5.809</td>
<td>1.177</td>
<td></td>
<td></td>
</tr>
<tr>
<td>TSH (mIU/L)</td>
<td>Patients</td>
<td>2.048</td>
<td>1.063</td>
<td>0.083</td>
<td>N.S.</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>1.576</td>
<td>0.775</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

N.S.: non-significant difference.
Discussion:
The pathogenesis of Fibromyalgia probably reflects an interaction of environmental triggering events on the background of a genetic predisposition. It has been postulated that it can be the consequence of a chronic stress mediated mainly through the hypothalamic-pituitary-adrenal axis (HPA) and the sympathetic nervous system [17]/8.

The secretion of pituitary TSH is suppressed and TSH response to thyrotropin-releasing hormone (TRH) is blunted [20]. The conversion of the relatively inactive thyroxine to the biologically active triiodothyronine in peripheral tissues is decreased during stress [21]. It is known that the incidence of depression and anxiety are increased in subjects with FMS; but, it was found that, there was no association between depression or anxiety and thyroid autoimmunity [16]. As the present study had a limited number of patients and the estimations were limited to T3, T4, and TSH, so for further investigations it will be more fruitful to include more patients and to estimate fT3, and fT4 beside TSH.

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