

## **THYROID MYOPATHY - A CASE STUDY**

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### **REZUMAT**

Miopatia tiroidiană apare de obicei la 1 - 3 luni după instalarea tirotoxicozei, din punct de vedere clinic pacienții prezentând scăderea forței musculare, dureri, crampe și atrofii musculare de grade diferite. Prezentăm cazul unui bărbat de 45 de ani, care s-a prezentat pentru un consult neurologic cu următoarele simptome: tulburări ale mersului, scăderea forței musculare, tremor, fatigabilitate și scădere în greutate, cu evoluție progresivă. De asemenea, el a prezentat dispnee severă, anxietate, iritabilitate, labilitate emoțională și insomnie.

**Cuvinte cheie:** miopatie, tirotoxicoză, boală Graves

### **ABSTRACT**

The thyroid myopathy usually appears 1 - 3 months after the onset of thyrotoxicosis, clinically the patients presenting muscle weakness, pain, cramps, and muscular atrophy in various degrees. We present the case of a 45 years old male with an atypical form of Graves' disease which was referred for neurological evaluation for the following complains: gait disturbance with muscle weakness, tremor, fatigue and weight loss, with progressive evolution. He also presented severe shortness of breath, anxiety, irritability, emotional lability and insomnia.

**Key Words:** myopathy, thyrotoxicosis, Graves' disease

### **BACKGROUND**

The thyroid gland has an important role in tissue metabolism, secreting thyroxine (3,5,3',5' tetraiodothyronine) – T4 and small amounts of 3,5,3' triiodothyronine – T3, both having systemic effects.

The symptoms due to hyperthyroidism were first described in the 19<sup>th</sup> century. Robert Graves, an Irish physician, in 1835, and Carl Adolf von Basedow, a German practitioner, in 1840, were the first who described muscle weakness and atrophy during thyrotoxicosis.<sup>1,2</sup>

Between hyperthyroidism and thyrotoxicosis a distinction can be made, hyperthyroidism denoting a sustained increased production and release of thyroid hormones, whereas thyrotoxicosis describing the clinical syndrome that results from excess of thyroid hormones.

Hyperthyroidian induced myopathy is assumed to be secondary to the disturbance in the function of the muscle fibers due to increased mitochondrial respiration, accelerated protein degradation and lipid oxidation and increased beta-adrenergic sensitivity caused by excessive concentration of thyroid hormones.

### **CASE REPORT**

We present the case of a 45 years old male referred for a neurological evaluation for the following complains: gait disturbance with muscle weakness, tremor, fatigue and weight loss with progressive onset. He also presented severe shortness of breath, anxiety, irritability, emotional lability and insomnia.

His history reveals that his health problems apparently started four months ago, when he had fever, diarrhea and dehydration. He was treated for acute enterocolitis with some improvement of the symptoms, but soon he developed motor deficit with fatigue induced even at small efforts (especially when climbing stairs), muscular cramps at the level of the calves and predominantly at night, insomnia, irritability, heat intolerance. He also had a significant weight loss (14 kg in one month), despite an exaggerate appetite. Because the usual laboratory exams showed a mild anemia, he was treated with iron compounds.

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Nevertheless, his symptoms progressed, the muscular cramps accentuated, he continued to lose weight, developed hyperdefecation (increased frequency of normally formed bowel movements) and tachycardia. Because of his digestive complaints and weight loss neoplasia was suspected but ruled out eventually.

When we have examined the patient he had the following status:

- At the general examination: the general aspect of a profoundly ill person, significant and severe weight loss (25 kg in 4 months) (despite the increased appetite), fine and thinning hair, transpirations and heat intolerance, sinus tachycardia (with 125 beats/minute), severe dyspnea and hyperdefecation.

- At the neurological examination: a fine rhythmic tremor in hands, proximal muscle weakness, affecting predominantly the pelvic girdle, with a waddling myopathic gait, difficulties in rising from a squat and climbing stairs. Also, there was symmetrical muscle atrophy with proximal onset. Tendon reflexes were slightly brisk. Otherwise his neurological examination was normal.

The laboratory exams showed a mild microcytic anemia (with 13 g/dl of hemoglobin, mean corpuscular volume of 77.6 fl, mean corpuscular hemoglobin 25.6 pg), normal leucocytes level but increased lymphocytes (55.3%), increased erythrocyte sedimentation rate (23 mm/h). Blood levels of serum thyroid-stimulating hormone (TSH) were decreased (0.005  $\mu$ U/ml), the free triiodothyronine (T3) was > 50 pg/ml and thyroxine (T4) > 7.77 ng/dl.

Thyroid ultrasound showed a gland of 68.9 ml of volume, with diffuse, not homogeneous hypoechogenicity.

The patient was sent for an endocrinology consultation, where the diagnosis of Graves' disease with hyperthyroidism was established and a treatment with Thyrozol, 10 mg four times a day and beta blockers (Metoprolol 50 mg qd) was recommended.

After initiating the treatment, his status started to improve day by day, with evident progress: he started to gain weight, the insomnia disappeared, he was no longer irritable, the motor deficit started to improve and the cardiac rhythm decreased to 80 beats/minute.

## **DISCUSSIONS**

Graves' disease is common, affecting 1 - 5% of the population, with a female to male preponderance of 5 - 10: 1.<sup>3</sup> It is characterized by diffuse goiter, infiltrative ophthalmopathy, thyrotoxicosis and occasionally dermopathy (myxedema), the presenting symptoms

being generalized, neurological signs appearing after some months or years.<sup>4</sup>

Our case is not a typical form of Graves' disease. At the general examination, he had no goiter, no warm and moist skin, and the fine and thinning hair is an easy to miss sign. The characteristic signs of Graves' disease, myxedema and infiltrative ophthalmopathy with periorbital edema, exophthalmia, extraocular muscles dysfunction and chemosis were absent in our patient, making more difficult the diagnosis.<sup>4</sup> He presented tachycardia but not elevated blood pressure.

Dyspnea is very common and it correlates with functional weakness of the diaphragm and patients with no obvious myopathy may complain of respiratory difficulties.<sup>5</sup> Other common complaints are heat intolerance and sweating and, at gastrointestinal level, hyperdefecation, weight loss despite normal or increased appetite, but diarrhea is rare, and the weight loss can be due also to malabsorption. Nervousness, emotional lability, irritability and the tremor of the hands are very often present in hyperthyroidism, but they can be easily mistaken with an anxiety state or a psychoneurosis.

In thyrotoxic myopathy, muscle symptoms appear usually 1 - 3 months after the onset of hyperthyroidism. Common presenting symptoms are: fatigue, poor exercise tolerance and myalgias, patients noting difficulties in climbing stairs, brushing their hair. One half to two thirds have proximal weakness, other one third having as well distal and bulbar weakness, in the most severe forms.<sup>6</sup> In a minority of cases, the clinical picture may resemble myasthenia gravis with dysphagia, dysphonia, respiratory difficulties, there being possible also an association between Graves' disease and myasthenia. Muscular atrophy is of a lesser severity than the weakness.<sup>7</sup> Tendon reflexes can be normal or brisk. The neurological signs that we found in our patient are of a myopathic type.

The first step in confirming the clinical diagnostic of thyrotoxicosis is to investigate the serum levels of thyroid hormones and thyrotropin, normal levels excluding thyrotoxicosis.<sup>4</sup> Finding a decreased TSH and increased level of T3 and T4 in our patient confirmed the diagnosis of thyrotoxic myopathy and the patient was sent for an endocrinology consult for further investigations, the differential diagnosis including Graves' disease, toxic multinodular goiter, thyroid adenoma, iodide - induced hyperthyroidism, excess hormone production by ectopic thyroid tissue, thyroiditis. The diagnosis of Graves' disease was established, the most common cause of thyrotoxicosis.

Other studies, such as electromyography (EMG), were not performed in our patient, because it would have provided limited information, while his diagnosis was clear.<sup>7</sup> EMG might reveal the presence of a myopathy, but a normal finding does not exclude the diagnosis. An abnormal pattern is more likely to be found in the proximal muscles, including reduced duration of mean action potentials and increased mean percentage of polyphasic potentials.<sup>8</sup>

Muscular biopsy is used mainly to exclude other muscle diseases such as myotonia or congenital myopathies. Histological findings in thyrotoxic myopathy can be normal or nonspecific such as: mild atrophy, fatty infiltration, focal myofibrillar degeneration, mitochondrial hypertrophy, focal dilatation of transverse tubular system, increased sarcolemmal nuclei.

The prognosis of thyrotoxic myopathy is good, weakness resolving rapidly during a proper treatment, in Graves' disease being used antithyroid drugs and blocking agents for achieving a euthyroid status.<sup>9,10</sup> Strength recovers more rapidly than the muscle wasting.<sup>11</sup>

## CONCLUSIONS

The thyrotoxicosis may involve every organ or system, as the thyroid hormone receptors are present in every tissue. The severity of symptoms varies with the duration and the degree of hyperthyroid state, the age of the patient, the anterior status of other organ systems and the disease causing thyrotoxicosis.

At the beginning, the symptoms and signs might be unspecific, making the diagnosis more difficult.

Our patient presented an atypical form of Graves' disease, without goiter, ophtalmopathy or myxedema, his major complains being muscle weakness, weight loss and psychological disturbances.

Undiagnosed and untreated, severe and life threatening complications are possible, such as the thyroid storm (with a mortality rate of 20 - 50%), and cardiovascular diseases (myocardial ischemia, atrial fibrillation, congestive heart failure).<sup>12</sup>

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