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THE ENDOCRINE PSYCHO-SYNDROME IN DYSTHYROIDISM

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ABSTRACT. The study aims to prove an obvious link between the increased TSH level and the normal or low FT4 levels and/ or the ATPO (anti-peroxidase antibodies) presence and the pathology of the bipolar or unipolar affective disorders in the context of the initial presence of crude hypothyroidism or clinically expressed and the occurrence of the depression. The gathered data show us the possibility of developing a depression without obvious clinic manifestations (proven only by increased dosages of TSH and normal values of FT4 – which proves in the laboratory of the crude hypothyroidism or of those with certain hypothyroidism – that is increased TSH values and low values of FT4). The achieved results prove without doubt a relation between the thyroid dysfunction and the depression which support the necessity of a double therapeutic approach, both psychiatrical and endocrine.
KEYWORDS: Crude hypothyroidism, bipolar affective disorder, SSRI inhibitors for re-capturing the serotonin, NaSSA mirtazapine, anti-depressives, TSH, FT4, ATPO

INTRODUCTION

It is that there are characteristic neuroendocrine failures in the disorders. Stressing them in the affective disorders may have a thyroid pathogen component. The positive diagnosis is put based on symptoms such as anorexia, asthenia, adynamia, sleep disorder, psycho-motor agitation, and delirious ideas of micro-manic in depression type or of expansive type in mania associated with a series of vegetative changes: bradycardia or tachycardia, peripheral vasoplasticity, changes in the digestive transit. The digestive disorders occur in over 40% of the patients with hypothyroidism, and on the other hand, the hypothyroidism represents a risk factor for the depressive disorders, and it is signalised in 56% of the cases.

The group of the affective disorders, particularly the depressive ones, is heterogeneous. Actually, for the known clinical sub-types, there is weak descriptive validation and feeble inter-ratter fidelity which do not allow for a high degree of concordance between the clinicians on the diagnosis for the same patient.

We underline that there is a diversity of taxonomic systems situated between the phenomenological customs and the clinical-biological criteria of depressive type of disorders.

Several authors described the serotoninergic depression also called cortisol inducted as associated to anxiety and aggressiveness. Another subtype of depression is represented by the noradrenergic one, expressing itself by cognitive disorders, apathy, bradypshychia, besides the depressive mood.

The clinical studies stress a reduction in the number of β1 adrenergic receptors at the level of the central nervous system affected by hypothyroidism, with the decrease of the adenylate cyclase and of the AMPc at the cerebellum Purkinje cells level. T3 administration causes a comeback to normal levels for the adenylate cyclase and of the AMPc .

It is known that, in the depressions, a reduction occurs in the serotonin concentration and activity. The presence of hypothyroidism was signalised in the persons treated for depressive disorders. Before getting to anti-depressive medication, it is recommended to perform an investigation of the thyroid activity.

MATERIALS AND METHODS

The clinical studies were performed on 3 directions, on the following groups: depressive patients with euthyroidism (20 pacients), group 2 with crude hypothyroidism and signs of depression (30 pacients), the paraclinic qualifying criterion: the level of TSH >=4 and <= 10 mU/ml. The third lot with 30 patients presenting expressed hypothyroidism and signs of depression. The depression has been established using a modified option of the "Edinburgh postpartum" depression scale, comprising a 10 points scale initially elaborated for the study of the postnatal depression which was also validated for use in women without postpartum debut in another nomenclature – "Edinburgh Depression Scale".

For each studied patient, a clinic observation chart was issued, including the main personal data and a table with the 10 suggestive symptoms for depression. The presence of each symptom received 1 point, and the addition of the points summed up to 10 points scoring depending on the frequency and presence of the symptoms (asthenia, paraesthesia, dry teguments, oedemas of the eyelids, bradychardia, bradypsychia, bradylalia, hypoacusis.

TSH, FT4 and ATPO dosages. These involved the Delfia method for TSH and FT4 and the Elisa method for ATPO.

RESULTS

The patients with depression and euthyroidism expressed loss of vital impetus, anxiety, asthenia,

sweating, insomnia, uselessness. The basal TSH values were between 1.3-4 mU/ml.

The patients with crude hypothyroidism and with signs of depression expressed loss of vital impetus, anxiety, asthenia, sweating, insomnia, uselessness; the difference being that the basal TSH was between 4-10 mU/ml.

The patients with expressed hypothyroidism and signs of depression presents an emphasis of the above mentions manifestations, which were assessed according to a 1 to 10 scale, obtaining an average of 9.1 and TSH values between 4.8 and 5 (mU/ml). This proved an obvious correlation between the hypothyroid pathology and the depressive disorders.

Administering the selective inhibitors of the serotonin re-capturing such as fluoxetine or of some drugs with dual action NaSSA-mirtazapine in association with dosages of 25 µg of levothyroxine led to substantial reduction of the symptomatology in the group with signs of depression and crude hypothyroidism, especially in the group with expressed hypothyroidism and signs of depression.

DEBATES

A lot of data on the correlation in mental disorders between depression and thyroid hormones have been published. Such studies proved mostly that, changes in the hypothalamus- pituitary – thyroid axis may occur in patients with depression, which would plead for more and less expressed hypothyroidism.

In the same time, there were studies on the potential positive effects of the thyroid hormones in improving the depression. The first issue occurring during the studies was that of more rigorously definition of a lot of female patients with manifestations of depression. Things seem even more complex if we consider the expression of the affective disorders (uni and bi-polar ones). The study on x TSH-thyroid status comprised only the patient with depression of neurotic intensity (reactive disorder).

TSH values in patients with depression and without clinical signs of hyperthyroidism may be considered a useful tip in verifying the existence of a crude hypothyroidism. The results certify higher values in the patients with depression which supports the argumentation from the literature that there is a chronic stimulation of the pituitary TSH due to the TRH hypersection, in depression disorder. Nevertheless, the numerous clinical signs and symptoms found in the thyroid dysfunction are likely to those of depression which means that the depression might be caused by dysthyroidism. The achieved data support for a convergence between the triggering of hyperthyroidism and of the depression.

Starting from these elements, the study that we performed on the patients with high TSH and with more or less severe elements of depression suggests also the need monitor from psychical point of view, the triggering and the evolution of such a frequent disorder such as the depression.

The researches were performed by following up the colouring of the TSH level and the clinical score of pain, and we met a positive correlation between them. The intensity of the depression disorders met in the studied group draws the attention on the previous mental framework with which the patient complies in the hypothyroidism pathology, the depression being certainly linked to hypothyroidism.

The mental pain scale (depression) in patient with expressed hypothyroidism was higher than in those with crude hypothyroidism.

It is also to be noticed that insomnia and other types of manifestations for the depression re-establish the normal circadian rhythm of TSH and the FT4 circulating levels after the therapy with thyroid hormones.

My own data that followed up the link between the TSH level and the antibodies titre stressed that establishing high levels of TSH must be performed in parallel with the APTO calculation.

CONCLUSIONS

The researches on the link between the high levels of TSH and the ATPO and depressive expressions presence was a positive one in over 50% of the cases. The levothyroxine therapy associated with anti-depressives in patients with high TSH and normal or low FT4 and the ATPO presence or absence proved that the thyroid hormones contribute in quick stabilisation of the condition.

The analyse of the symptoms before and after introducing the levothyroxine therapy reduced considerably the pain scale which was present during the anti-depressives therapy.

The disorders of the thyroid function are also relatively frequent in depressions resulting from bipolar affective disorders, particularly in the forms with quick cyclicality. For the therapy-resistant depression, we may appeal to the increase of the anti-depressives dosages or we may opt for the association with thyroid hormones.

The thyroid insufficiency in depressions and the depression in hypothyroidism are the 2 poles to follow up in order to keep a neuro-psychical balance in these two categories of patients.

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