Thyrotoxic psychosis: a case report

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Psicosis tirotóxica: a propósito de un caso

Summary

The first step in the assessment of a patient who presents psychiatric symptoms is to discard somatic illness. We present a case of a patient whose symptoms began with confusion, behavior alterations and agitation, which were followed by psychomotor inhibition with visual hallucinations, with underliyng thyrotoxicosis. In the discussion, we analyze the aspects to consider in order to detect similar cases and their treatment, since, although it is a rare form of presentation of hyperthyroidism, it should be taken into account due to the seriousness of the picture.

Key words: *Psychosis. Thyrotoxicosis, Endocrinopathy. Organic.*

Resumen

El primer paso en la valoración de un paciente con síntomas psiquiátricos es descartar la patología somática. Presentamos el caso de una paciente cuya clínica se inició con síntomas confusionales, desestructuración conductual y agitación, seguida de inhibición psicomotriz y alucinaciones visuales, con una tirotoxicosis subyacente. En la discusión se analizan los aspectos a tener en cuenta para la detección de casos similares y su tratamiento, ya que, aunque sea una forma poco común de presentación del hipertiroidismo, debe tenerse en cuenta por la gravedad del cuadro.

Palabras clave: *Psicosis. Tirotoxicosis. Endocrinopatía. Orgánico.*

We present the case of a 36 year old woman, foreigner, allergic to penicillin, with no known toxic habits, with two episodes of post-partum depression recorded in the anamnesis and with no other background of interest. Two days before her admission, she presented a confusional picture that resulted in psychomotor agitation with auto and heteroaggressiveness. She was administered 10 mg of diazepam and 25 mg of levomepromacin, both intramuscularly, as an out-patient and she was sent for hospital admission. The patient was examined with the following symptoms: fever of 38.4º C, heart rate 120 beats per minute, blood pressure 110/60 mmHg and respiratory rate 20 per minute was observed. It stands out that her skin is warm and sweaty, there is mucous dryness, significant thinness and fine tremor of the hands. Significant restlessness with incoherent speech, destructured behaviors and temporal-spatial disorientation. Cardiac auscultation is rhythmic without murmurs or rubbing. The respiratory auscultation presented conserved vesicular murmur with added upper respiratory tract sounds. In the abdominal examination, an enlarged bladder is palpated without masses or megalies with increased peristaltism. No goiter or abnormal lymph nodes are palpated. In the neurological examination, there are no abnormalities of the cranial pairs, the patient moves her four limbs and there are no apparent focalities. A waiting attitude is taken with serum therapy, vital signs control and bladder probe with control of diuresis. Blood analysis is performed with the following results: complete blood count and leukocyte formula in normal limits, prothrombin time of 62%, APTT normal; glucose, 7.3 mmol/l; K, 4.3 mEq/l, and Na, 147 mEq/l. The simple chest X-ray shows a normal cardio-thoracic index and no condensations are observed. Normal urine sediment. During admission, she begins to present fluctuations in consciousness level, psychomotor inhibition, crying, visual hallucinations, perspiration, low grade fever, increase in distal tremor of limbs, oligomenorrhea and dysphagia that requires nasogastric tube. The result of the thyroid tests is: TSH, 0.01 mU/l; T_3, 1.07 ng/ml; T_4, 12.2 microg/dl, and free T_4, 1.43 ng/dl; positive antithyroglobulin antibodies, 361; positive antimicrosomal antibodies, 312; the reference values being positive after 250 and 125 respectively. With all this, the diagnosis of thyrotoxicosis is suspected and she is sent to the general hospital to complete the study.

The term thyrotoxicosis refers to clinical, physiological and biochemical manifestations that occur after exposure and response of the tissues to the excessive supply of thyroid hormone[1]. Thyrotoxicosis is generally produced by hyperthyroidism, but may sometimes be due to an excessive intake of drug preparations of the

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thyroid hormone or, in rare cases, to an extrathyroideal origin such as struma ovarrii. Causes of hyperthyroidism are Graves' disease, single hyperfunctioning adenoma, multinodular goiter, some forms of subacute or chronic thyroiditis and, rarely, a TSH producing primary hypophyseal lesion[2]. The most common cause is Graves' disease, which is more frequent in women and in the third and fourth decade of life. The common finding of serum antibodies against thyroidal microsomes and thyroglobulin shows the humoral autoimmunity[3].

The most frequent clinical manifestations of hyperthyroidism are: irritability, emotional lability, restlessness, tiredness, tremor, insomnia and anxiety, that is generally the principal psychiatric symptom[4]. In relationship with these symptoms, it has been proposed that a beta adrenergic dysfunction is produced in the central nervous system[4,5]. Some data that can lead to the diagnosis towards hyperthyroidism are: heat intolerance, perspiration, warm skin, weight loss with increased appetite, palpitations, tachycardia, exophthalmos and stronger osteotendinous reflexes[6,7]. In general, the neurological symptoms predominate in younger patients, while the cardiovascular and myopathic ones do so in the elderly[1]. An atypical form of presentation of hyperthyroidism is the so-called «apathetic hyperthyroidism» that shows characteristics of depression, apathy and psychomotor delay, the differential diagnosis with major depression being difficult[4,8]. It generally occurs in the elderly and does not respond to antidepressive treatment[9,10], but does respond to thyroid correction[4].

The thyrotoxic episode is manifested by the immediate increase of the signs and symptoms of thyrotoxicosis. This syndrome is characterized by extraordinary irritability, delirium or coma, fever of 41º C or more, tachycardia, restlessness, hypotension, vomiting and diarrhea.

The key to the diagnosis is the blood determination of thyroid hormones and TSH. The isolated test that has the greatest value is the TSH measurement[2]. In general, the patients with thyrotoxicosis show undetectable levels (less than 0.1 mU/l) and the normal subjects show values between 0.3 and 3 mU/l (1). In cases of serious disease, serum concentration of T_3, T_4 and free T_4 may be within the normal limits[2].

DISCUSSION

Patients with thyroid disorders, both due to excess as well as defect, may present psychiatric symptoms, even acute psychosis[4,6,11,12]. Psychosis or cognitive disorder are rare in our setting as symptoms of thyroid disorder but may be the presentation of an untreated hyperthyroidism[4, 13-15]. The first and most important step in the assessment of a patient with psychiatric symptoms is to eliminate the somatic causes[16] (see table 1). In a retrospective New Zealand study of a 20 year period[17], 18 patients who required admission to a psychiatric unit due to «thyrotoxic psychosis» were recorded. None of them presented a history of previous thyrotoxicosis, however four had undergone psychiatric admissions some years before. A total of seven had depression, seven mania, one schizophreniform disorder, one paranoid symptoms and one delirium. Some studies have mentioned that principally the illusions and other sensoperception disorders may accompany a thyrotoxic picture with maniform characteristics and precede a thyroid storm[18]. Hyperthyroidism may be the precipitating factor or may exacerbate a chronic psychosis, especially the affective ones, and may go unnoticed when treating psychotic symptoms[7,17]. Furthermore, this type of patients may report the suggestive symptoms poorly[7,13]. On the other hand, transitory elevation of the thyroid hormone level has been described in the psychiatric population, especially during exacerbations of the disease and in hospitalization periods[7]. Thus, there is the possibility of false positives and the laboratory tests should be repeated at 2 weeks[7]. In fact, some individuals present elevations of thyroid hormone that disappear during the periods of minimum stress and reappear during episodes of stress, producing a clinical hyperthyroidism[4]. However, the presence of life stressors can be incidental in relationship with the presence of any disease[7]. In the past, hyperthyroidism was considered as a prototype of psychosomatic disease (7,7) and was related with an emotional trauma[4]. A comparative study found a greater number of negative life events, with statistically significant difference, in the group of 208 patients with Graves' Disease than in the control group of 372 persons, during the 12 months prior to the diagnosis of hyperthyroidism[19].

In most of the cases, the psychiatric symptoms remitted with treatment of the underlying thyrotoxicosis[4], however it must be taken into account that some patients have presented psychotic symptoms after antithyroid treatment[4,20]. Caution must be taken with some psy-

TABLE 1. Aspects that suggest an organic origin of the behavior disorders [6]

Some atypical characteristics for a specific psychiatric diagnosis
Behavior symptom that is more spectacular than that expected according to the isolated psychiatric syndrome
Late onset age in a new behavioral symptom
Absence of personal or familial history of psychiatric disease
Sudden onset of psychosis or dementia
Poisoning or withdrawal of some substance, or the prescription of substances with psychoactive properties (i.e. digoxin)
Obvious systemic disease
Evidence of increase of intracranial pressure
Non-auditory hallucinations (visuals, distortions and illusions are the most suggestive)
Time relationship between physical state and onset, exacerbation and end of psychiatric symptoms
Appearance of autonomic dysfunction without there being pre-morbid dysfunction
Resistance to psychiatric treatment
Alterations of consciousness level, orientation or memory

chopharmaceuticals: the tricyclic antidepressants are associated with a greater risk of toxicity and are generally avoided[4]. Lithium can exacerbate the exophthalmos of Graves' disease, however its antithyroid properties should be considered for the treatment[4]. Antipsychotic agents may be necessary to control the psychotic symptoms, considering that haloperidol has been involved in neurotoxicity in some cases[4].

Endocrinopathies should be considered in the differential diagnosis of a large spectrum of psychiatric symptoms. Early treatment of the hormone or metabolic alteration can minimize the morbidity of a secondary psychopathology. In addition, in most of the patients, it is enough to correct the psychiatric symptoms, although, in a subgroup of them, the resolution of some pictures, such as depression, may not occur after the treatment of the endocrine or metabolic dysfunction and may require specific psychiatric treatment[4].

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