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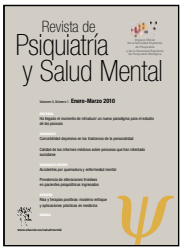

CARTA CIENTÍFICA

Myxoedema psychosis as the first manifestation of hypothyroidism

Locura mixedematosa. Hipotiroidismo que debuta como psicosis

Hypothyroidism is the most common hormonal disorder in day-to-day clinical practice, with a prevalence ranging between 0.5%and 18%, depending on the study population.[1] With a generally insidious onset,[2] it is most common in middle-aged women and is responsible for a wide variety of somatic and neuropsychiatric disorders.[2]

Besides the classic clinical signs and symptoms the patient may have symptoms secondary to the hormonal disorder[3] such as memory and attention disorders, psychomotor slowness, and visual perceptual or executive disorders. For these reasons it can be a difficult clinical entity to diagnose and sometimes goes unnoticed.[4]

Psychiatric symptoms are a frequent manifestation of this thyroid disorder. Around 40% of hypothyroid patients present symptoms and signs of depression, and there is more and more evidence relating treatment-resistant depression with thyroid disorders, even subclinical or underlying ones.[2] A relationship between hypothyroidism and bipolar disorder has also been reported in the literature, but it is more common for maniform symptoms to be related to hyperthyroid states.[5] Other associated psychiatric disorders are psychotic depressions or pure psychoses without cognitive impairment or disorientation.[5]

Myxoedema psychosis combines the presence of psychotic symptoms with the existence of myxoedema, which is characterised by non-pitting oedema, general slowness, and lethargy caused by hypothyroidism.

We present 2 case studies in which hypothyroidism went undiagnosed because it was presented with psychotic symptoms. We will also try to explain the relationship between both phenomena.

Case study 1

Female, 47 years old, referred by the accident and emergency department with behavioural disorders and hetero-aggression in her family environment. She had no history of somatic disease or known drug habits.

Since her youth she has had non-specific behavioural disorders. A possible brief psychotic outburst many years ago characterised by delusions of harm and control. Treated with olanzapine. She abandoned treatment and follow-up after a few months.

In the final months she suffered irritability and frequent fits of rage with agitation, treated with olanzapine (7.5mg/day); She abandoned treatment after a few weeks.

Of note in the first interview was her swollen appearance, and lack of expressiveness. With regards her mental state, she was lucid, cold and distant, and oriented times 3. Hypoprosexic. No data of amnestic disorders, except amnesia for episodes of agitation. Bradypsychia. Speech was slow, lacked fluency, circumstantial, and at times tangential. Empty, vague and with a tendency to concretise. Digressions. Changes in the course and content of thought characterised by non-egodystonic ruminations about strange ideas that she was unable to express. Distorted perception. Affective indifference. Irritability. No anxiety or somatisation. Social withdrawal. No loss of sleep or appetite.

The diagnosis tends toward a psychotic disorder, probably a schizotypal personality disorder (F21, ICD-10) and she was prescribed quetiapine at 400mg/day. Slight improvement with the antipsychotic treatment, but behavioural disorganisation persisted. After the first month of treatment she had side effects in the form of galactorrhoea, so a complete analysis was performed revealing hyperprolactinaemia (prolactin 123.4ng/ml), TSH 49.970mU/l, free T4 0.7ng/dl and anti-TPO 352.2UI/ml. Diagnosed with primary hypothyroidism due to chronic thyroiditis and hyperprolactinaemia secondary to hypothyroidism; began substitute treatment with levothyroxine sodium up to 50mg/day. After 2 months of treatment hormone levels were partially normalised (prolactin 99.6ng/ml, TSH 10.63mU/l, T4 1.1ng/dl and anti-TPO 390.7UI/ml) and she showed significant improvements in content and coherence of thought. No behavioural alterations. She commented on previous alterations. Cognitive and affective levels restored to normal.

Case study 2

54 year-old woman. No history of somatic disease or drug habits of interest. No personal or family history of psychiatric disorders.

Emergency hospitalisation due to behavioural disorders and psychomotor agitation at home. After the death of her husband, she stopped going out and made delusional interpretations of the circumstances surrounding her husband's illness and death, convinced that someone put pills in his food and people burgled the house at night. She had indurated oedemas on her legs, face and hands, which had worsened in the previous months.

When examined on admission she was conscious, orientated, suspicious but collaborative, verbose and demanding, giving detailed accounts of all the events and beliefs upholding her paranoid construct. Anxious and irritable. Delusions of harm and hypochondriacal; delirious interpretations and auditory hallucinations. No awareness of psychiatric disorder.

Of note in the physical examination was her straw-coloured skin, swollen eyelids, alopecia at the tail of her eyebrows, and generalised indurated, non-pitting oedemas.

Imaging and blood tests were performed, highlighting thyroid hormone disorder with TSH 127.10mUI/l, T4 0.23ng/dl, and T3 0.72pg/ml, cardiomegaly in the chest x-ray, and severe pericardial effusion without signs of cardiac tamponade on the echocardiogram.

Treated with a saline drip to avoid the collapse of the right cardiac cavities, and levothyroxine up to 100mcg/day, improvement is observed in the oedemas and the pericardial effusion. Risperidone treatment was continued at 6mg/day. Diagnosed with severe hypothyroidism (myxoedema) and organic delusional [schizophrenic-like] disorder (F06.2, ICD-10). No behavioural alterations on discharge but delusional memories persist with a lack of affective resonance.

Discussion

The combination of myxoedema and psychotic symptoms was first described in 1883 by the Committee on Myxoedema of the Clinical Society of London,[6] based on the descriptions of changes in the mental state of 109 patients with myxoedema. Later, these clinical symptoms appeared in Cronin's novel, *The Citadel,* in 1937 in which the main character examines a patient suffering from psychotic symptoms within the context of hypothyroidism.

In 1949, Asher[7] created the concept of myxoedema psychosis, and suggests myxoedema is one of the most common causes of organic psychosis after discovering 14 patients with the physical characteristics of myxoedema combined with psychotic symptoms.

Psychotic symptoms usually appear after long-term thyroid disease, lasting months or years,[8] and they are associated with clinical or subclinical hypothyroidism as there seems to be no correlation between the analytical data and the presence of psychotic symptoms.[9]

Its aetiopathological mechanism is unknown. Some authors cite dopaminergic alterations[10] secondary to the hypothyroidism as the possible cause, or even brain blood-flow alterations.[11]

The treatment of choice for recovering normal thyroid functioning is hormone substitute therapy, with the psychosis and myxoedema usually disappearing after 1 week of this treatment.[12] However, they may remain even with the correct treatment and when analytical data have returned to normal, above all if the thyroid treatment is begun late.[2] The initial doses must be low and increase slowly to avoid episodes of confusion and exacerbations of the psychotic symptoms.[13] Psychotic symptoms may reappear if the hormone treatment is withdrawn,[14] and some patients can even suffer symptoms again after an initial improvement.[14]

Psychopharmacological treatment with atypical antipsychotics seems to be well tolerated[14] and hastens the improvement in psychotic symptoms compared with the exclusive use of hormone treatment.

Thyroid pathologies must be taken into consideration as the cause of psychotic disorders, in particular when they have these atypical characteristics.

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