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Secondary Hypoparathyroidism - A Neuropsychological Evaluation Case Study

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Abstract

Hypoparathyroidism is a rare disorder in which the body secretes abnormally low levels of Parathyroid Hormone (PTH). The cognitive function of patients suffering from secondary hypoparathyroidism has not been adequately studied, while during the neuropsychological examination, most patients experience a decrease in their cognitive functions.

Aim: The purpose of the study was to present the case of a 54-year-old female patient with hypoparathyroidism, and the results of her neuropsychological evaluation.

Results: Hypothyroidism has psychological and cognitive implications, usually presenting slowly. It is usually seen in middle-aged women and in many cases is recognized as depression. The literature states that in neglected and severe cases there may be psychotic manifestations such as paranoid ideas and confusion.

Conclusion: The symptoms of this patient were severe cognitive deficits which focused on slowness of perception and attention, disorders of memory and concentration. For this reason, it initially presented a clinical picture of possible onset dementia. The patient's condition was reversible with thyroxine. The antidepressants did not work.

Keywords: Neuropsychological assessment; Hypothyroidism; Hypoparathyroidism

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Introduction

Hypothyroidism is a condition in which the thyroid gland does not produce enough thyroid hormone. The most common cause of hypothyroidism worldwide is often iodine deficiency, but it can be caused by many other factors. It can result from the malfunction of the gland or its partial or total removal, as an autoimmune disease, or from infection and destruction of the gland by radioactive Iodine-131, while it can also be associated with increased stress. The levels of thyroid hormone in the blood have a direct effect on the body's metabolic functions and the function of the sympathetic nervous system, and when they are reduced, a number of dysfunctions occur in various systems of the body. In infants, severe hypothyroidism can lead to cretinism. A 2011 study found that around 8% of women over 50 and men over 65 in the UK suffer from hypothyroidism [1]. Normal is called hyperthyroidism.

Hypothyroidism is usually classified according to the organ responsible for it its appearance [1].

Primary or Thyroid gland

The thyroid gland does not produce enough hormones. The most common forms include Hashimoto's thyroiditis (which is an autoimmune disease) and Iodine-131 thyroid dysfunction or destruction as a result of a nuclear accident or radiotherapy.

Secondary Pituitary

It occurs when the pituitary gland does not produce enough Thyroid-Stimulating Hormone (TSH), the hormone that alerts the thyroid gland to secrete thyroid hormone. Although not all cases have a clear cause, secondary hypothyroidism is usually caused by damage to the pituitary gland, which can be caused by a tumor, radiation, or surgery. Secondary hypothyroidism accounts for less than 5% of all cases [2].

Tertiary Hypothalamus

It occurs when the hypothalamus does not produce enough Thyroaclytine (TRH), the hormone that alerts the pituitary gland to secrete TSH.

Congenital Hypothyroidism

When a newborn has hypothyroidism then we say that he suffers from congenital hypothyroidism. It is due to partial or total insufficiency of thyroid hormones. The incidence of congenital hypothyroidism is approximately 1: 3000.

Symptoms

Early hypothyroidism is often asymptomatic or has very mild symptoms. Subclinical hypothyroidism is a condition in which the levels of thyroid hormone in the blood are normal, and Thyroid-Stimulating Hormone (TSH) levels are slightly elevated. With higher TSH levels and lower free T4 levels, the condition is described as clinical (or overt) hypothyroidism. Hypothyroidism may be associated with the following symptoms:

- Fallen mood and negative feelings
- Depression
- Memory malfunctions
- Slowness, impairment of cognitive functions, inability to focus attention
- Irritability and mood swings

The need for thyroid hormones increases during pregnancy and therefore the risk of a previously unnoticed, subclinical or latent hypothyroidism turning into clinical hypothyroidism. Subclinical hypothyroidism in early pregnancy is estimated to increase the risk of preeclampsia and perinatal mortality. Hypothyroidism, even in its mild or subclinical form, is known to adversely affect fertility [3].

Causal Factors

Iodine deficiency is the most common cause of hypothyroidism worldwide. Iodine is the main component for the synthesis of thyroid hormones and its lack in the diet leads to their insufficient production. In the past, goiter and hypothyroidism were more common in mountain populations living off the sea, precisely because of the lack of iodine in their diet. Nowadays the addition of iodine to table salt ensures minimal iodization of foods regardless of place of residence or consumption of iodine-rich foods. Nevertheless, according to the World Health Organization in 2007 iodine deficiency had about 2 billion people worldwide, a third of whom were school-age children, and only 34 countries had achieved complete iodization of total salt production. Thus, the problem of iodine deficiency remains an important public health issue [4].

In people with iodine deficiency, hypothyroidism is often caused by Hashimoto's thyroiditis, an autoimmune disease in which the body's immune system attacks and destroys the thyroid. Damage to the thyroid gland is also caused by the ingestion of the radioactive Iodine-131.

This radioactive isotope of iodine is released during surface nuclear tests but also after a serious nuclear accident, and can be ingested through contaminated food. It is concentrated in the thyroid gland and its breakdown can cause thyroid cancer and various thyroiditis or morphological and functional abnormalities in the gland, especially when ingested by children. I-131 is also used in nuclear medicine, administered to intentionally destroy the thyroid to treat Graves' disease or hyperthyroidism.

Congenital hypothyroidism is a rare condition, accounting for about 0.2% of cases, and may be due to either thyroid aplasia, a congenital absence or underdevelopment of the gland, or dysfunction of hormone metabolism. This category also includes hypersensitivity to thyroid hormones, although in this case the levels of hormones in the blood are normal or even high [5]. Hypothyroidism can also occur as a consequence of postpartum thyroiditis, which initially appears as hyperthyroidism that can return to normal levels or develop into transient or permanent hypothyroidism. It can also occur as a result of Quervain thyroiditis, an inflammation of the thyroid gland caused by a virus-like infection that can damage the gland.

Temporary hypothyroidism may be the result of the Wolff-Chaikoff effect, in which overdose of iodine, often for emergency reasons, can lead to decreased hormone production due to decreased iodine organization in the gland. The antiarrhythmic agent amiodarone, rich in iodine, can have this effect. Hypothyroidism can also be caused by the use of lithium-based antidepressants, commonly used to treat bipolar disorder. In fact, lithium has been sporadically used to treat hyperthyroidism [6]. Other drugs that can cause hypothyroidism are interferon alfa, interleukin-2, and thalidomide [7].

Risk Factors

Risk factors for hypoparathyroidism may include:

- Recent neck surgery, especially one involving the thyroid
- Family history of hypoparathyroidism
- Certain autoimmune or endocrine disorders (e.g., Addison's disease)

Complications

In adults, there may be heart, kidney and brain calcifications. It can coexist with other diseases if not of surgical etiology, such as cataracts, Addison (lack of corticosteroids due to adrenal insufficiency) and B12 deficiency anemia [8].

The diagnosis is made by finding low calcium, and/or Magnesium with high Phosphorus, and of course low PTH. High calcium in the urine indicates a lack of PTH. In kidney failure, the kidney is no longer able to produce enough vitamin D or remove all the phosphorus produced by the body, leading to low calcium levels. These low levels of calcium stimulate the parathyroid glands to produce more PTH. Over time, this constant stimulation causes the development of parathyroid glands and hyperactivity, and patients can progress to the development of secondary hyperparathyroidism [9].

The cognitive function of patients with secondary hypoparathyroidism has not been adequately studied, while during neuropsychological testing, most show a marked decline in their cognitive functions [10]. This discount can be perceived by the non-specialist as a symptom of dementia because in most cases there is a discount of recent memory, especially in the phases of engraving and retrieval of new information, but also the patient's slowness to respond to the tests. In most cases, during the control, mainly disorders of concentration and attention are found, while there are also disorders of speech and difficulty in finding the appropriate word immediately [11].

Case Study

Ms. TH, 54 years old, was referred with a diagnosis of secondary hypoparathyroidism after a neurological examination for evaluation

and neuropsychological assessment of the level of her cognitive functions. Apart from her laboratory tests, the patient also showed characteristic clinical signs such as bradycardia, edematous face, menstrual disorder, decreased tendon reflexes and intense confusion. Previous antidepressant treatment has shown that it did not work. Her level of education was 9 years. Her neuropsychological assessment array included the tests:

Mini mental state examination (MMSE) for the assessment of the general cognitive level.

WAIS IV: The Wechsler Adult Intelligence Scale: the subtest: Codes that test attention skills. Verbal Fluency Test. A test sensitive to frontal dysfunction and at an early stage of semantic disorder.

Visual-mental tracing test (Trail-making Test, part A, part B): Which assesses attention skills, optomotor speed, visual detection, information processing, mental flexibility, concentration and abstract thinking.

Rey auditory-verbal learning test (RAVLT): This test measures the direct memory field, provides a learning curve, and measures short-term and long-term retention.

Rivermead behavioral memory test (RBMT): The subtest of history (immediate and delayed recall).

Rey ostereith complex figure test (ROCFT): A complex figure designed to examine perceptual organization, visual memory, spatial construction, and design.

Stroop test: Examines the effect of interpolation that an automated process (word reading) has on a process that requires more effort (e.g., naming the colors in which words are written).

Boston naming test (BNT). Examines naming ability.

Neuropsychiatric inventory - NPI.

Neuropsychological examination of the patient focused on memory function (Story memory, reverse number retrieval, verbal flow test), attention (stroop, direct number retrieval), and executive functions (Trail Making Test). Her performance in each sub-test of the assessment array she gave showed skills above the normal average for her age and educational level.

In particular, her score in both immediate and long-term memory tests appeared to be deficient. Regarding the long-term retention, difficulties were observed in the neuropsychological tests that allow the conceptual organization of the elements to be memorized (story memory) while the preservation of the mnemonic traces concerning general knowledge (declarative memory) was observed. As indicated by the slow execution of Part A of the Trail Making Test and the Stroop interpolation condition. It shows reduced accuracy and speed of rotation of the visual attention in a different type of reaction. The difficulty of naming it (disorganization of semantic memory) is an indirect indicator of cognitive decline in dementia. In general, the performance in information processing speed, learning, memory and executive function ranged below the normal limits for its age and level of education.

The neuropsychological evaluation of the patient contributes leads to the picture of severe mental retardation. The reality, however, is not so dramatic for the patient because it is a false pathological assessment of psychological tests, which is due to the under-functioning of the thyroid gland [12-16].

Conclusion

Hypothyroidism, regardless of the etiology, has psychological and cognitive implications, usually presenting slowly. It is usually seen in middle-aged women and in many cases is recognized as depression. The literature states that in neglected and severe cases there may be psychotic manifestations such as paranoid ideas and confusion. Symptoms are severe cognitive deficits which focus on slowness of perception and attention, memory and concentration disorders. For this reason, a possible onset of dementia can often be misdiagnosed. The patient's condition was reversible with thyroxine and antidepressants did not work. In addition to hormone therapy, the administration of psychotropic substances should be done with caution because hypothyroid patients show increased sensitivity with hypotension, sedation and anticholinergic side effects. Long-term administration of lithium reduces thyroid hormone secretion while small doses of benzodiazepines appear to be safer [17].

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