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RESEARCH ARTICLE

HYPERPROLACTINEMIA AND HASCHIMOTO'S THYROIDITIS: WHEN ONE CAUSES AND POTENTIATES THE OTHER (CASE REPORT).

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ABSTRACT

Hyperprolactinemia corresponds to an increase in the level of prolactin, the symptoms vary according to the sex and the length of time the disease has been present. It can be secondary to several situations: functional or organic. Hypothyroidism is considered to be one of the classic functional causes of hyperprolactinemia. On the other hand, prolactin is known for its stimulating effects on the immune system. Therefore, hyperprolactinemia is incriminated in the occurrence of autoimmune diseases, especially hypothyroidism. We report an observation of hyperprolactinaemia revealing the rare association of peripheral hypothyroidism and a macroprolactinoma.

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INTRODUCTION

Hyperprolactinaemia is defined as prolactin levels above 20 ng/ml in men and 25 ng/ml in women(1). A common endocrine disorder, it occurs in 1 to 1.5% of the general population and in 5 to 14% of patients with secondary amenorrhoea (2). The most common cause after drug-induced hyperpolactinemia is prolactinoma. Other clinical conditions may be associated with moderate to mild functional hyperprolactinaemia : chronic renal failure, hepatic insufficiency and peripheral hypothyroidism (a classic but very rare cause of hyperprolactinaemia) (1). Objective of the work: Highlighting 2 points: Do not ignore an organic cause of hyperprolactinemia in the presence of a functional cause, and the possible causal link between hyperprolactinemia and the occurrence of autoimmune diseases.

OBSERVATION: A 36-year-old female patient with a family history of Hashimoto's thyroiditis consult fot spontaneous bilateral multipore galactorrhoea.

The interrogation revealed marked asthenia, psychomotor retardation and secondary amenorrhoea. The clinical examination revealed signs of hypometabolism and a diffuse homogeneous stage 1 goitre. The hormonal test revealed: hyperprolactinemia at 85 ng/ml (confirmed by a second assay), profound peripheral hypothyroidism with a TSH at 58 mIU/l (0.5- 5) and positive anti-TPO antibodies at 256 U/ml (< 34). Cervical ultrasound showed thyroiditis, hepatic and kidney function tests were normal.

The patient was put on L-Thyroxine substitution at progressive dose. The clinical and biological evaluation at 6 months showed persistent galactorrhea and hyperprolactinemia despite normalization of the thyroid check-up. Magnetic resonance imaging (MRI) showed a left lateralized pituitary macroadenoma measuring 13mm/8mm (Figure 1, 2,3), justifying the prescription of dopaminergic agonists (Cabergoline at 2 ug/week). The evolution was marked by the disappearance of galactorrhoea, amenorrhoea, normalisation of prolactin levels and disappearance of the adenoma on MRI.

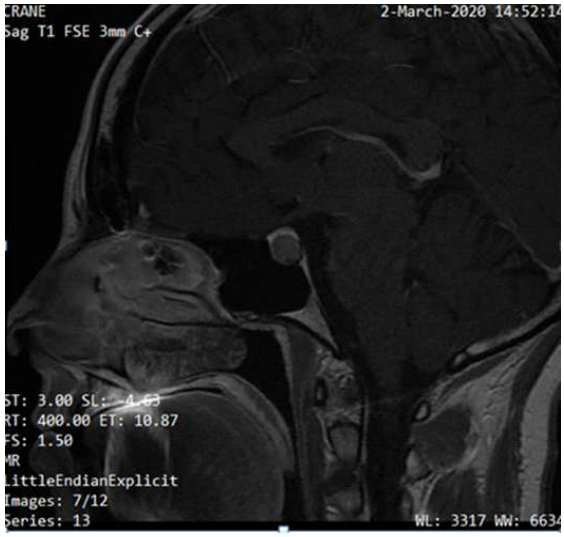

Figure N1. Sagittal injected section showing the adenoma

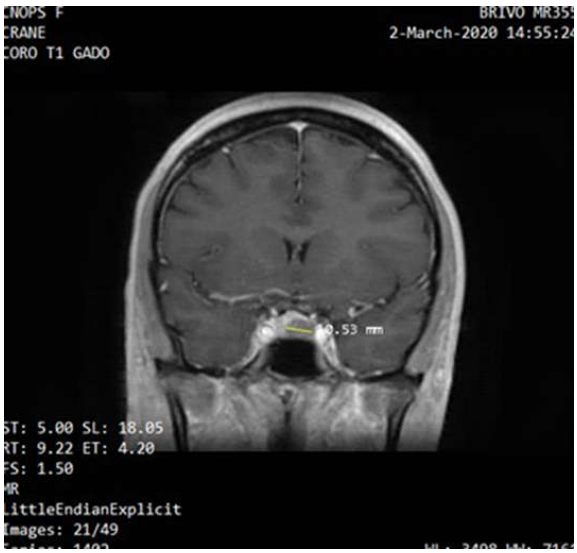

Figure N2. T1 coronal section after Gadolinium injection showing the pituitary adenoma

DISCUSSION

Hyperprolactinaemia is a common condition (1 to 1.5% of adults) (2). The clinical manifestations are more obvious and earlier in women than in men and depend on the sex and the age of the hyperprolactinaemia (1). The discovery of hyperprolactinaemia goes through two steps: The first is to verify the reality of the hyperprolactinaemia by a check-up in a specialised hormonal laboratory. The second is to eliminate functional causes, often responsible for moderate to mild hyperprolactinaemia (1,3): drug-induced origin, renal or hepatic insufficiency and peripheral hypothyroidism (1), in which hyperprolactinaemia is secondary to hyperplasia of lactotropic cells following the rise in hypothalamic TRH (4). In the absence of a functional cause, the possibility of a tumor of the hypothalamic-pituitary region should be considered: prolactinoma, pituitary adenoma of another nature or a sellar tumor responsible of disconnection hyperprolactinemia (1). In prolactinoma, serum prolactin levels is usually correlate to tumor size. However, a prolactin adenoma can be associated with any prolactin level (1). Recent research has shown that prolactin has stimulatory effects on the immune system. Its receptor is widely expressed in various cells of the immune system: monocytes, lymphocytes, macrophages, NK cells, granulocytes, thymic epithelial and dendritic cells(5,6). Binding of prolactin to its receptor activates signalling ways including proliferation, differentiation, secretion, survival of immune cells and antibody production (5,6,7). The PRL gene is located on the short arm of chromosome 6, near the HLA-DRB1 region, which is known to be associated with various immune-mediated disorders (8). This suggests a role for prolactin in the immunomodulation, occurrence and pathogenesis of autoimmune diseases. Data on the association between PRL level and disease activity are inconsistent (6). Hyperprolactinaemia has been described in various autoimmune diseases, in particular in systemic lupus erythematosus, multiple sclerosis, systemic sclerosis, Sjögren's syndrome and rheumatoid arthritis (6,7). In dysthyroidism, hyperprolactinemia has been found in 20% of patients with autoimmune thyroid disease with higher frequency in patients with hypothyroidism. Approximately 90% of patients with Hashimoto's thyroiditis had significantly elevated prolactin levels. The role of dopamine agonists in the treatment of autoimmune thyroiditis remains to be determined (8). However, hyperprolactinemia can be seen in peripheral hypothyroidism and prolactinomas, but it may be responsible for the occurrence of primary autoimmune hypothyroidism because of its action on the immune system. Two clinical situations responsible for the same manifestation, and one of which may support and provoke the occurrence of the other.

CONCLUSION

Our patient's case demonstrates the diagnostic difficulty and rarity of the association of two pathologies that can give the same clinical and biological presentation. Through this observation, we would like to underline 2 main points: the first is the importance of not ignoring an organic cause of hyperprolactinemia in the presence of a functional cause, and the second is probably the causal link between hyperprolactinemia and the occurrence of autoimmune disorders. Conflict of interest statement: The authors do not have any financial, real or perceived conflict of interest in the publication of this manuscript.

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