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Pituitary

CENTRAL INVOLVEMENT OF MULTISYSTEM TUBERCULOSIS AS A CAUSE OF HYPOPITUITARISM THAT HAS NOT BEEN DISCOVERED FOR A LONG TIME

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INTRODUCTION: Tuberculous meningitis (TBM) can occur in isolation or along with a pulmonary focus. The clinical spectrum of TBM is broad and may be non-specific making early diagnosis difficult. Hypopituitarism has been reported due to pituitary or hypothalamic abnormalities in TBM, and it may become evident years after recovery, apparently due to progressive scarring of either the hypothalamus itself or adjacent tissues. Here, we present a case of hypopituitarism thought to have developed after tuberculous menengitis during a multisystemic tuberculosis infection approximately 40 years ago.

CASE PRESENTATION: A 60-year-old male was sent to our endocrinology clinic because of low FT4 and TSH levels. Physical examination revealed pale skin and hypogonadal facial appearance. Axillary and pubic hairs were very scarce. The blood pressure was 90/60 mmHg. He had a scar on his left cervical region. He was diagnosed with colon carcinoma and received radiotherapy and chemotherapy after surgery in 2015 and was in remission thereafter. He indicated that he had decreased sexual function for more than 25 years. He gave the information of a long lasting treatment when he was 20 years old for left cervical masses without neck pain and/or stiffness. But there was discharge from these masses and hearing loss developed after an injection therapy, which was a part of the treatment. Based on past medical history, we thought that the patient was most probably treated for tuberculous lymphadenitis. His pituitary function tests were as follows: FT3: 2.8 pmol/L (3.1-6.8), FT4: 9.4 pmol/L (12-22), TSH: 2.6 mIU/L (0.27-4.2), IGF-1: 27.5 ng/mL(43-220), LH: 0.1 mIU/mL (1.7-8.6), FSH: 0.25 mIU/mL (1.5-12.4), Total Testosteron: 0.025 ng/mL (1.32-8.92), PRL: 16 ng/mL (4-15), basal cortisol: 4.77 ug/dL and 1 mcg ACTH stimulation test showed insufficient adrenal response. The patient denied to have polyuria or polydypsia, and his blood Na+ level was 141 mmol/L. These results showed central hypogonadism, central hypothyroidism and secondary adrenal insufficiency. Pituitary MRI revealed disseminated calcification compatible with menengitis sequelea of tuberculosis and both cranial MRI and CT showed suprasellar calcification adjacent to pituitary stalk and hypothalamus (Figure1). He had no visual field defect. The chest X-ray and thorax CT revealed apical lesions compatible with sequelea of pulmonary tuberculosis. During current evaluation, active tuberculosis was excluded. Based on this data, early diagnosis and treatment of multisystem tuberculosis probably caused the symptoms of TBM to be mild. The patient begun to receive replacement therapies with glucocorticoid and thyroid hormone. He was warned about stress conditions and recommended to receive steroid protection.

CONCLUSION: Although hypopituitarism associated with meningitis is rarely reported, these cases may be more recognizable by careful physical examination and questioning of the patient's medical history.

KEYWORDS: Tuberculous meningitis, hypopituitarism, central hypothyroidism

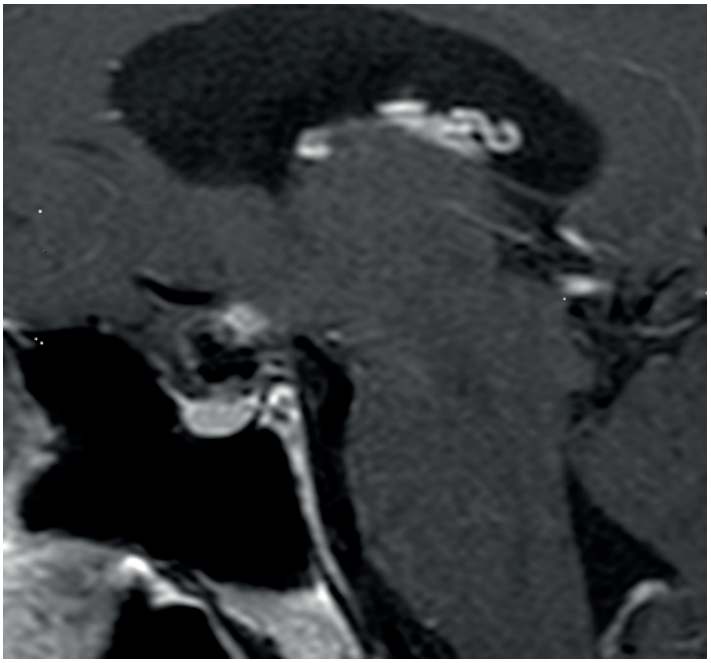


Figure 1. Sagittal section of pituitary MRI (T1) showing suprasellar calcification