



Visual Vignette

Central Hypothyroidism due to Pituitary Iron Overload

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Case Presentation

A 90-year-old man was seen by the inpatient endocrine service for hypothyroidism. He had been admitted for exertional dyspnea and chest pain 2 days before. He had an extensive medical history, including coronary artery disease, end-stage renal disease on peritoneal dialysis, pulmonary embolism, diabetes, and chronic obstructive pulmonary disease. He did not have a history of head trauma or visual changes. He had been found to have pure red cell aplasia 5 months before and required weekly transfusion of 1 to 2 units of red blood cells in

the previous 5 months. At admission, myocardial infarction was ruled out. The patient was about to be discharged; thyroid function was tested due to lethargy and weakness in the previous few weeks. The thyroid-stimulating hormone level was 0.06 $\mu\text{IU/mL}$ (normal, 0.3–4.7), free thyroxine level was 0.2 ng/dL (normal, 0.8–1.7), and free triiodothyronine level was 95 pg/dL (normal, 222–383). Other pituitary tests showed testosterone level of <6 ng/dL (normal, 200–1000), luteinizing hormone level of 9.8 mIU/mL (normal, 2–12), and normal insulin-like growth factor 1 and cortrosyn stimulation results. The thyroid-stimulating hormone level had been 1.5 and 0.77 $\mu\text{IU/mL}$ 5 and 3 months before this admission, respectively, without concurrent free thyroxine or triiodothyronine measurement. Liver functions were normal. Pituitary magnetic resonance imaging without gadolinium showed no lesions in the hypothalamus or pituitary stalk, normal-appearing pituitary on T1 imaging (Fig. 1 A, arrow), and very low pituitary signal on T2 imaging (Fig. 1 B, arrow).

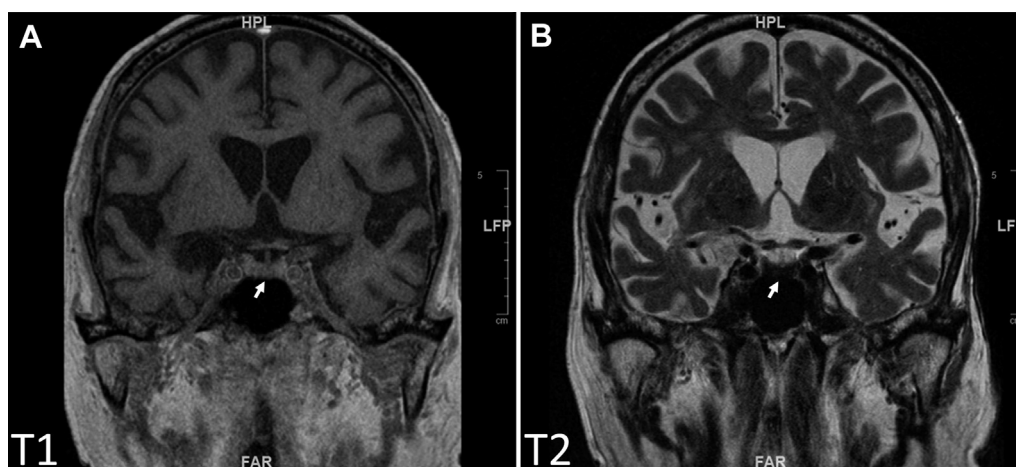


Fig. 1.

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What is the diagnosis?

Answer

Central hypothyroidism due to pituitary iron overload. His iron and ferritin levels had been 116 µg/dL (normal, 41–179) and 552 ng/mL (normal, 8–350), respectively, 14 months before presentation, 205 µg/dL and 1219 ng/mL, respectively, 5 months before presentation, and 230 µg/dL and 3450 ng/mL, respectively, when hypothyroidism was found. He had, thus, already had iron overload 5 months before presentation, likely secondary to pure red cell aplasia; the ensuing frequent transfusions caused more iron overload.¹ In iron overload, iron tends to accumulate in endocrine organs, perhaps due to expression of L-type Ca²⁺ channels in those organs, especially in the thyrotrophs of the pituitary.^{1,2} There have been no studies, however, demonstrating a quantitative correlation between the severity of iron overload and the incidence or severity of central hypothyroidism. Imaging evidence of iron overload in the pituitary classically includes a low signal on magnetic resonance T2 imaging,³ which was clearly present in this patient. Pituitary iron overload can cause central hypothyroidism, in addition to

hypogonadism, due to the toxic effects of iron in the thyrotrophs and gonadotrophs.^{1,2} This patient's central hypothyroidism was consistent with pituitary iron overload. He was treated with levothyroxine. Unfortunately, he developed aspiration pneumonia and sepsis and was discharged to palliative care after an extended hospital stay.

Disclosure

The authors have no multiplicity of interest to disclose.

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