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SCIENTIFIC LETTERS

Hyperthyroidism induced by topical iodine☆

Hipertiroidismo inducido por yodo tópico

Exposure of the body to large amounts of iodine sometimes causes thyroid function changes, and may result in both hyperthyroidism and hypothyroidism. Potential iodine sources include dietary supplements, drugs, iodine-rich antiseptic solutions, or iodinated contrast agents used for imaging tests.[1] Povidone iodine is a non-toxic, low cost chemical agent with high germicidal power which is widely used in hospital practice as a disinfectant and sterilizing agent. The case of a patient who experienced subclinical hyperthyroidism after repeated topical administration of iodine is reported below.

This was a 39-year-old male with Cacchi–Ricci disease. This condition, also known as medullary sponge kidney, is an uncommon congenital disease consisting of the cystic malformation of distal collecting tubules and usually causes recurrent urinary tract infection and renal stones. The patient was admitted for elective ureterorenoscopy with left lithotripsy, performed uneventfully. After surgery, however, he experienced septic and hemorrhagic shock that required admission to the ICU. Fluid therapy was started, followed by an infusion of norepinephrine 0.58 μg/kg/min due to persistent hypotension. Because of the critical condition of the patient despite the actions taken, urgent left nephrectomy was required, as well as arteriography with urgent embolization for stump bleeding after nephrectomy.

High-dose catecholamines were maintained for two weeks, and the dose was tapered after patient stabilization. As the result of the administration of high doses of vasoactive drugs over a long time period, ischemic lesions developed in both feet, requiring the daily application of povidone iodine for periods ranging from 60 to 90 min. The patient stayed at the ICU for 26 days and was later transferred to the hospitalization ward. Evaluation by the nutritional department was requested. The patient had lost 20% of his usual weight (75 kg) and had an estimated weight of approximately 60 kg (a body mass index [BMI] of 19.6 kg/m^2) and anthropometric parameters (mid-upper arm circumference [MUAC], tricipital fold [TF], and arm muscle circumference [AMC]) below the 10th percentile. A physical examination revealed marked muscle atrophy and cachexia, with no edema. Blood tests showed decreased visceral protein levels (albumin, 2.2 g/dL; total protein, 8.2 g/dL; transferrin, 82 mg/dL) and malnutrition parameters (total cholesterol, 130 mg/dL; lymphocyte count, 1.6×10^3/μL) with no thyroid function impairment (TSH, 3.280 mU/L [0.350–4.940 mU/L]; free T4, 1.23 ng/dL [0.70–1.48 ng/dL]). Acute, severe mixed malnutrition was diagnosed.

Subsequently, the patient was regularly monitored at the clinic for approximately one year, during which he experienced a gradual improvement in his nutritional status and recovered his usual weight. However, routine tests performed 6 months after hospital discharge showed the following levels: TSH, 0.003 mU/L (0.350–4.940 mU/L); free T4, 1.42 ng/dL (0.70–1.48 ng/dL), and free T3, 3.38 pg/mL (1.70–3.70 pg/mL). There was no associated clinical evidence of low thyroid function. This finding, consistent with subclinical hyperthyroidism, persisted over time. The patient had no family or personal history of thyroid disease, thyroid gland palpation revealed no goiter or nodules, thyroglobulin antibodies were slightly positive (11.89 U/mL, 0.05–6 U/mL), and peroxidase antibodies were negative. A thyroid ultrasound examination was normal. A thyroid scan with technetium 99m (Fig. 1) showed low thyroid uptake, which was attributed to repeated application of povidone iodine to the ischemic lesions of the feet. The use of povidone iodine was discontinued, and chlorhexidine was used instead. The patient showed a favorable course, with a normalization of TSH levels two weeks after iodine exposure was stopped (TSH, 0.784 mU/L; free T4, 0.95 ng/dL; and free T3, 2.27 pg/mL).

The minimum recommended iodine intake is 150 μg/day.[2] Exposure to iodine overload causes thyroid self-regulation, consisting of transient decreases in iodine organification and thyroid hormone synthesis (the Wolff–Chaikoff effect). A escape phenomenon occurring at 2–4 weeks normalized hormonal production. Iodine excess may, however, also cause hyperthyroidism (the Jod–Basedow effect) due to thyroid disease hyperproduction and sudden release in response to the administration of large amounts of iodine. This most commonly (but not only) affects people with prior thyroid

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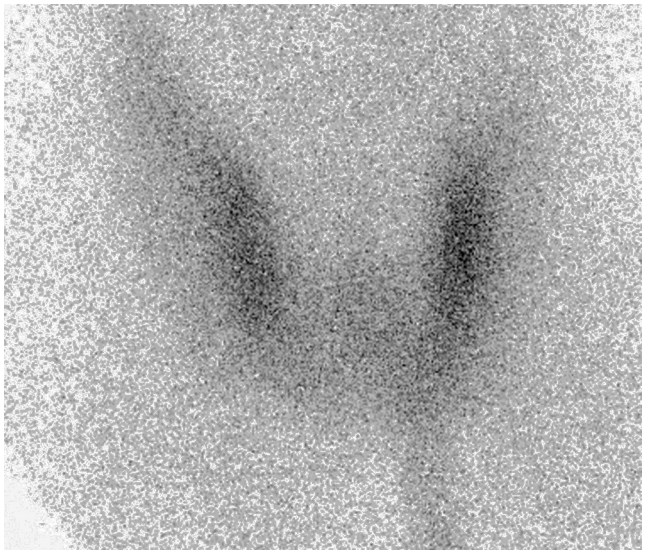

Figure 1 Thyroid scan with ^{99m}Tc: low quality examination due to inadequate thyroid uptake of the tracer. The condition may be attributed to the repeated application of povidone iodine to foot lesions.

disease such as endemic goiter, toxic multinodular goiter, or iodine deficiency.[3]

However, iodine overload is an uncommon cause of hyperthyroidism. A detailed clinical history is therefore essential to detect the condition, and should include data on the use of drugs or dietary supplements[4] with high iodine contents, the use of antiseptic solutions such as povidone iodine, or imaging tests with contrast agents. Most cases of iodine-induced hyperthyroidism are self-limited and resolve when exposure ceases.[5]

Tu sum up, a case of subclinical hyperthyroidism induced by excess topical iodine is reported in a patient with no prior thyroid disease. The condition resolved after povidone iodine was replaced by a iodine-free antiseptic solution. The use of iodinated antiseptics is a widespread practice in hospitals, especially for patients undergoing surgery or with ulcers of both arterial and venous origin.[6] In cases with long-term exposure, potential thyroid dysfunction should be considered because, as in the reported cases, it may not be exceptional even in the absence of a history of thyroid disease.

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Black adrenal adenoma causing Cushing's syndrome: 40 years ago and today

Adenoma suprarrenal Negro causar el síndrome de Cushing: Hace 40 años y hoy

Dear Editor,

Black adrenal adenomas are adrenal cortical tumors that are black or dark brown on cut sections. The first case of black adrenal adenoma was reported in 1938.[1] Autopsy studies published in the early 1970s suggest that the pigments in black adrenal adenomas are made of lipofuscin, a lysosomal material, and that these tumors are common autopsy findings (10% on random adrenal sections and 37% on fine sections) but do not secrete hormones.[2] In 1973, two of us (G.D.B. and R.R.E.) cared for and studied a patient with a black adrenal adenoma that caused ACTH-independent Cushing's syndrome. We here describe the case and discuss it in historical background and in light of the literature on this topic in the last 40 years.

A 42-year-old Caucasian female had been well until 1966 when she developed hypertension, edema, and hyperglycemia during her third pregnancy. In 1969, she developed right femoral head aseptic necrosis. She also noted a 40-pound weight gain, rounding of face, the development of a dorsal fat pad, ruddy complexion, facial hair, weakness, easy fatigability, emotional liability, irregular menses, and easy bruising. In January 1973, she was seen at Harbor General Hospital (now Harbor-UCLA Medical Center). She denied skin darkening, exogenous steroid ingestion, or family history of endocrine diseases. Physical examination revealed a hypertensive, Cushingoid female. Endocrine evaluation revealed absence of suppression of plasma or urinary