

La anatomía patológica descartó malignidad y no se observaron bacilos tipo *Helicobacter*. A pesar de que el número de casos fue pequeño, se trataba de una complicación grave.

La úlcera por decúbito con este tipo de sonda ya había sido descrita previamente: inicialmente, en casos aislados, y ya en 2002 y 2012 en series más amplias en las que figuraba un riesgo mayor de úlcera gástrica. Después de la publicación en la que se demuestra un aumento del riesgo de úlcera por presión de 2,27 veces, se hizo una valoración de nuestra casuística y se iniciaron los procedimientos administrativos oportunos para el cambio.

Podemos concluir que la HDA secundaria a una úlcera por decúbito de la sonda de gastrostomía con extremo distal fuera del globo es una complicación poco frecuente pero grave. Velar por la seguridad del paciente es un requisito fundamental a la hora de elegir entre los distintos tipos de sondas de gastrostomía para la nutrición enteral.

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Analytical interference in the corticotropin immunoassay in patients with adrenal adenomas



Interferencia analítica en el inmunoensayo de corticotropina en pacientes con adenomas suprarrenales

Analytical interference in the corticotropin (ACTH) assay is an uncommon event (<1%).¹ However, when it occurs, it can interfere with the diagnostic orientation and management of adrenal disease leading to inappropriate clinical decisions, especially when patients show alterations in imaging tests.

Case #1

A 79-year-old woman was referred for a right adrenal mass (23 × 16 mm) incidentally discovered in a thoracic-abdominal CT scan performed after trauma. The patient had always had cats. She did not report tachycardia, palpitations, headaches or hyperhidrosis, and did not show

any clinical sign of hypercortisolism. Hyperpigmentation was absent.

Hormone analysis showed marked hypercorticotropinemia (ACTH 242 pg/ml; normal range, *N*: 5–46) with serum cortisol (16.1 mcg/dl; *N*: 3.7–19.4), nighttime (23:00) salivary cortisol (0.1 mcg/dl; *N* < 0.28), and 24-h urinary free cortisol (UFC, 40 mcg/24 h; *N*: < 140) within the normal range. Mineralocorticoid function [aldosterone 4.4 ng/dl (*N*: 3–35.5), plasma renin activity, PRA 1.78 ng/ml/h (*N*: 0.3–7.0)] and medullary adrenal function [24-h urinary metanephrines 168 mcg/24 h (*N*: 50–825)] were also normal. A second plasma ACTH determination confirmed hypercorticotropinemia (ACTH 311 pg/ml).

The presence of hypercorticotropinemia with normal values of serum, urinary and night salivary cortisol in the absence of clinical adrenal dysfunction forced us to rule out ACTH dependent Cushing syndrome (ACTH-dependent CS) and Addison's disease. We performed a 1-mg dexamethasone (23:00 h) suppression test (serum cortisol 1.4 mcg/dl; *N* < 1.8) and a short ACTH (250 mcg iv) stimulation test (serum cortisol at 0, 30, and 60 min: 12.8, 21.3, and 23.3 mcg/dl). Antiadrenal antibodies were also negative. A normal pituitary MRI and a negative ^{99m}Tc-EDDA/HYNIC-TOC