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Acute renal injury as a manifestation of Addison syndrome in a 66-year-old female patient

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Mr. Editor:

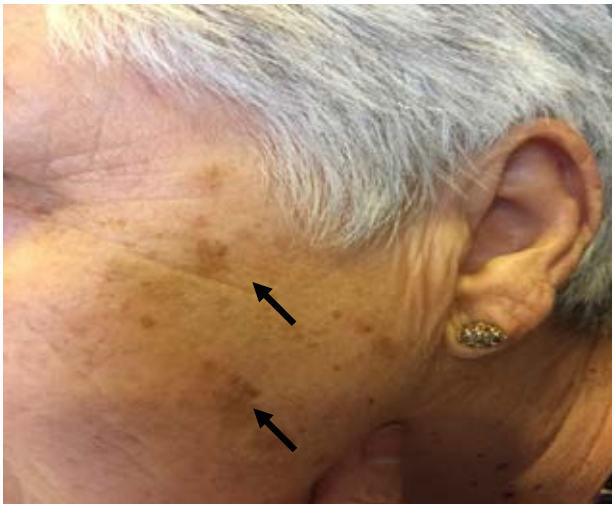
Addison syndrome is a hormonal deficiency caused by damage to the adrenal gland which leads to hypofunction or primary adrenal insufficiency. It is a very rare disease, with an incidence of less than 1 in 100,000 inhabitants, with a prevalence of 4–6 per 100,000 people[1]. The causes are multiple and include infections (tuberculous, mycotic or granulomatous, viral). Among the autoimmune causes are polyglandular autoimmune syndrome type II and I[2]. Clinical presentation includes nausea and vomiting, low blood pressure, hyperpigmentation of the skin and mucous membranes or weight loss. Renal function involvement as the start of Addison disease is rare and tends to lead to misdiagnosis.

A 66-year-old woman came to the emergency room in October 2017 presenting generalized weakness, vomiting and deterioration of renal function. Her medical history included diabetes mellitus type II, multinodular goiter and hypothyroidism diagnosed 12 years ago. In June 2017, prior to the current admission, the patient presented similar renal deterioration requiring hospital admission, and was later discharged, with normal renal function.

She was re-admitted in October 2017. Physical examination revealed some brownish macules on both cheeks (Figure 1). At admission, she presented blood pressure 90/60 mmHg, afebrile, dry skin and mucous membranes. Analytically the patient presented creatinine 1.7 mg/dl, glomerular filtration rate 38 ml/min/m2, sodium 120 mEq/L, potassium 4.9mEq/l, bicarbonate 17 mEq/l, hemoglobin 13 mg/dl. The remaining parameters and abdominal echography were normal.

Figure 1

Skin colour characteristic of Addison syndrome.



With undiagnosed Addison syndrome suspected, ACTH, cortisol, aldosterone and anti-alpha 21 hydroxylase levels were requested. All was normal, except for ACTH levels of 1312 pg/ml and cortisol 0.22 ug/dl. Aldosterone levels were <21 pg/ml. Computerized tomography showed marked glandular atrophy .

Hydric repletion was performed and treatment with hydroaltesone started at 20 mg/day in a decreasing dose. An endocrinology evaluation was made, confirming the diagnosis. The patient was discharged with normal renal function.

Addison syndrome is a hormone deficiency caused by damage to the adrenal gland.

With the loss of more than 50% of the gland, low levels of cortisone stimulate ACTH-CRH, increasing levels of ACTH causing hyperpigmentation, while cortisol deficit decreases gluconeogenesis, producing hypoglycaemia, asthenia and muscular fatigability[3,4].

As the glomerular zone is destroyed, renin and angiotensin II levels increase to maintain a normal aldosterone secretion, leading to a deficit in mineralocorticoid secretion, at which point loss of sodium and water due to the prevailing hyperaldosteronism creates a situation of hypovolemia characterized by arterial hypotension[5].

In a patient with the above clinical picture, acute prerenal renal failure might be suspected; however, sustained hypoglycaemia, arterial hypotension and skin coloration should raise other suspicion. Although Addison syndrome does not tend to debut with acute renal failure, it is necessary to remember this could be the case to make a correct diagnosis and provide the patient with proper treatment.

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