

Don't ignore the tanned woman

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Abstract

This case report highlights one of the various ways people with adrenal insufficiency can present and how to recognise them. In this case report hyper pigmentation associated with hyponatremia and hyperkalaemia alerted us to the diagnosis. Timely diagnosis and clinical management of this condition are critical, and physicians in all areas of medicine should be aware of the signs and symptoms that herald adrenal insufficiency and its proper management.

Case report

A 66 years old Caucasian woman presented with a 4 day history of vomiting and diarrhoea along with confusion. She had a background of hypothyroidism and osteoporosis and was on L-thyroxin 50 micrograms daily along with weekly bisphosphonate and calcium/vitamin D supplementation. Physical examination showed marked hyper pigmentation postural hypotension and non-specific weakness with an abbreviated mental test score of 3/10. Initial investigation revealed a urea: 7.1 mmol/L (2.5-6.7 mmole/l), creatinine: 45 mmole (70-150 mmole/l), sodium: 103 mole/l (135-145 mmole/l), K: 5.5 mmole/l (3.5-5 mmole/l).

A short ACTH stimulation test was performed and patient commenced on intravenous hydrocortisone and intravenous fluids. There was marked clinical improvement with gradual normalization of electrolytes. An absent cortisol response to ACTH stimulation confirmed the diagnosis of adrenal insufficiency. Her thyroid function results came back as normal.

The patient was commenced on oral hydrocortisone and fludrocortisone and was doing well with normal Urea and Electrolytes when seen in the clinic a month later. By that time her antibody screen was back and it showed positive antiadrenal cortex antibodies. CT adrenal demonstrated normal appearance.

Discussion

Adrenal insufficiency has protean manifestations. The cardinal clinical symptoms of adrenocortical insufficiency, as first described by Thomas Addison [1] in 1855, include weakness, fatigue, anorexia, and abdominal pain, with orthostatic hypotension, salt craving, and characteristic hyper pigmentation of the skin occurring with primary adrenal failure. It is most conspicuous in areas exposed to light and chronic friction. The acute syndrome constitutes a medical emergency since it may result in a severe hypotensive crisis and clouded sensorium, together with pain in the muscles, joints, or abdomen and fever [2,3].

Hyponatremia is the most common electrolyte disorder, with a marked increase among hospitalized and nursing home patients. A 1985 prospective study of inpatients in a US acute care hospital found an overall incidence of approximately 1% and a prevalence of approximately 2.5%. Hyponatremia has been observed in approximately 30% of patients treated in the intensive care unit [4]. A

2009 study of 98,411 hospitalized patients found that even mild degrees of hyponatremia were associated with increased in-hospital, 1-year and 5-year mortality rates [5]. A 2009 study in Copenhagen concluded that hyponatremia in the range of 130-137 mEq/L is also associated with increased mortality rates in the general population [6]. Adrenal insufficiency — the clinical manifestation of deficient production or action of glucocorticoids — is a life-threatening disorder that may result from either primary adrenal failure or secondary adrenal disease due to impairment of the hypothalamic-pituitary axis [2,3].

Primary adrenal insufficiency, which can be acute or chronic, may be caused by the anatomic destruction of the gland. This destruction can have various causes, including fungal infection, other diseases infiltrating the adrenal glands, and haemorrhage. However, the most frequent cause is idiopathic atrophy, which is probably autoimmune in origin. Primary adrenocortical insufficiency is rare and occurs at any age. The male-to-female ratio is 1:1.

Secondary adrenal insufficiency may be caused by hypopituitarism due to hypothalamic-pituitary disease or may result from suppression of the hypothalamic-pituitary axis by exogenous steroids or endogenous steroids (*i.e.*, tumor). Secondary adrenocortical insufficiency is relatively common. Extensive therapeutic use of steroids has greatly contributed to increased incidence.

In the diagnostic workup for the disorder, the capacity of the adrenal cortex to respond to ACTH is tested with the use of the standard short ACTH test, which measures the serum cortisol level before and 30 or 60 minutes after an intravenous or intramuscular injection of 250 microgram of ACTH [7]. In emergency situations, it is important not to delay treatment of presumed adrenal insufficiency during diagnostic testing. Treatment with dexamethasone allows ACTH stimulation testing without affecting or interfering with the measurement of serum cortisol levels.

References

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