A 54-year-old man with childhood-onset type I diabetes mellitus who had preserved renal function but blindness secondary to his diabetes mellitus presented with a 5-day history of malaise and diarrhea.

On admission, he was dehydrated, hypotensive, and hypo-tensive (96.1°F/35.6°C). Initial laboratory values showed a potassium level of 9.2 (N, 3.6–5.0) mmol/L, and a sodium level of 122 (N, 137–145) mmol/L. The potassium level was confirmed on arterial samples.

The initial ECG showed a regular idioventricular rhythm with bizarre, broad QRS complexes, and a frequency of 32 beats per minute (Figure A). Over a 5-hour period, on correction of the hyperkalemia, he converted to sinus rhythm (Figure B and C) initially with a first-degree atrioventricular block (Figure B). Furthermore, the characteristic peaking of the T waves related to the hyperkalemia normalized (Figure C).

On follow-up, an increased level of adrenocorticotropic hormone, the absence of response to synthetic adrenocorticotropic hormone, and strongly positive adrenal antibodies revealed that the patient had developed autoimmune adrenal insufficiency (Addison disease).

Thomas Addison recognized the potential effects of adrenal insufficiency on the heart, and he described one of the marked features of the condition as “a remarkable feebleness of the hearts action” in his original monograph from 1855.1

To this day, adrenal insufficiency is a condition that cardiologists should be aware of because of the common occurrence of hypoaldosteronism-mediated hyperkalemia, and because it is a differential diagnosis in patients with shock or circulatory collapse.

Conditions predisposing to adrenal insufficiency are tumors, hemorrhage, or infections affecting the pituitary or the adrenal glands; iatrogenous adrenal insufficiency attributable to long-term glucocorticoid therapy; other autoimmune diseases; or, more rarely, hereditary diseases. In some developing countries, tuberculous adrenalitis is still common, whereas an autoimmune diathesis accounts for most cases in the Western world.2,3

Common clinical findings in patients with adrenal insufficiency include malaise, lethargy, abdominal discomfort, hypotension, hypothermia, and sometimes hyperpigmentation or signs of a predisposing state (eg, other autoimmune diseases, carcinomas, or glucocorticoid therapy).

The occurrence of hypoglycemia, metabolic acidosis, affected kidney function, or the classic—but not invariably present—combination of hyponatremia and hyperkalemia may be other clues to the tentative diagnosis of adrenal insufficiency.2–4

Acute adrenal insufficiency or addisonian crisis is a life-threatening condition, and, on suspicion, the administration of intravenous glucocorticoid, rehydration with saline, and treatment directed toward the triggering condition (eg, infections) should be initiated promptly. The glucocorticoid of choice has classically been hydrocortisone in an initial dose of 100 mg intravenously.2–4 If synthetic corticotropin is readily available and if the clinical condition allows for a delay in glucocorticoid administration, a short corticotropin test can easily be performed bedside in 30 minutes.3 The test results should not be awaited. Some authorities recommend intravenous dexamethasone (4 mg) as an alternative to hydrocortisone in patients without a previous diagnosis of adrenal insufficiency, because dexamethasone does not interfere with subsequent evaluation of cortisol levels.

Disclosures
None.

References
Figure. Progressive normalization of ECGs on treatment of severe hyperkalemia.
Hyperkalemia: A Clue to the Diagnosis of Adrenal Insufficiency
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