Hyporeninemic Hypoaldosteronism Presenting with Hypokalemia

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Sir,

Acquired selective aldosterone deficiency in adult patients is a rare clinical entity, in which adrenal production of aldosterone is reduced without associated reduction in production of cortisol [1]. Since the original case reported by Hudson et al. [2], about 50 additional cases have been described, but there is no case coursing with hypokalemia (instead of hyperkalemia) except that in an Italian journal [3] and our case, who was recently detected in our hospital.

Case Report. A 55-year-old housewife was admitted with dysuria, fever, and vomiting for 3 months. The blood pressure was 150/100 mm Hg; costovertebral angle pain was positive bilaterally. There was no other abnormal finding. Liver and renal function tests were unremarkable. Initially, the blood sugar was 98 mg/dl (5.44 mmol/l), the sodium 135 mEq (135 mmol/l), the potassium 2.9 mEq (2.9 mmol/l), the chloride 105 mEq (105 mmol/l) per liter of serum. The arterial pH was 7.36, the serum HCO₃⁻ 18 mEq/l (18 mmol/l), the PaO₂ 60.1 mm Hg (8.13 kPa), and the PaCO₂ 24.1 mm Hg (3.20 kPa). Table I shows some relevant biochemical values in detail. During hospitalization, she developed an enterobacteria septicemia originating from the urinary system and was given amikacin 15 mg/kg/day. After she had recovered from the septicemia, serum potassium levels were found to be between 2.4 and 3.1 mEq/l (2.4–3.1 mmol/l). She ingested 31 mEq (31 mmol) K⁺ enteric tablets, but hypokalemia did not improve. Having completed hormonal studies at this time, a diagnosis of hyporeninemic hypoaldosteronism with hypokalemia was confirmed (table II). The patient has been monitored at regular intervals, but hypokalemia and low levels of both aldosterone and plasma renin activity have persisted.

The findings in our subject fulfill the criteria for hyporeninemic hypoaldosteronism; the plasma renin activity and aldosterone levels decreased and adrenal and thyroid functions were normal. But our patient had hypokalemia instead of hyperkalemia, which is the expected classical finding. To our knowledge, our subject is only the second case presenting with hypokalemia reported in the literature. A similar case has been described elsewhere [3]. The possible causes of hypokalemia in our patient may be: (a) decreased dietary K⁺ intake (because of vomiting with septicemia); (b) vomiting (this may cause hypokalemia – despite the low levels of aldosterone – by two mechanism: first by external K⁺ loss, and second by increasing urinary K⁺ excretion); (c) hypomagnesemia (due to vomiting) [4].

Table I. Some biochemical values

These values were obtained simultaneously.

240

Sivri/Taşdemir/Turgan/Yasavul/Çaglar

Table II. Some of the hormonal studies

Normal range

Mass units

Plasma renin activity

0.16ng/ml/h 0.42 ng/ml/h 28.5 pg/ml 14.9 µg/dl 890 ng/ml 316.70 ng/l 3.73 mg/dl/day 11.20 µg/dl 1.60 ng/ml

0.51–2.64

0.98–4.18

40–310

5–23

80–1,000

39.73–496.60

3–10

4.5–12

0.8–2

Supine position

Upright position Plasma aldosterone Cortisol (at 7 a.m.) Dehydroepiandrosterone sulfate 17-Hydroxyprogesterone 17-Ketosteroids (urinary) Thyroxine Triiodothyronine

SI units

Normal range

In summary, this syndrome of our case is different from the cases referred so far, in that it presented with hypokalemia and probably was caused by a mild (sub-clinical) nephropathy, involving juxtaglomerular apparatus.

References

- Hirsch, D.; Kashgarian, M.; Boulpaep, E.L.; Hayslett, J.P.: Role of aldosterone in the mechanism of potassium adaptation in the initial collecting tubule. *Kidney int.* 26:798–807 (1984). Hudson, T.B.; Chobanian, A.V.; Reiman, A.S.: Hpoaldosteron-ism a clinical study of a patient with an isolated adrenal mineral -ocorticoid deficiency resulting in hyperkalemia and Stokes-Adams attacks. *New Engl. J. Med.* 257:529–532 (1957). Cotrozzi, G.; Matteni, M.; Relli, P.; et al.: Ipoaldosteronismo iporeninamico con crisi ipokaliemike. *Recent. Prog. Med.* 74: 901–909 (1983).
- Rose, B.D.: *Clinical physiology of acid-base and electrolyte disorders*, pp. 461–465 (McGraw-Hill, London 1977).

We found blood pH between 7.36 and 7.56. This finding probably reflected the sum of the effects of hy-poaldosteronism and vomiting; although one would expect metabolic acidosis in this syndrome.