Postural hypotension and electrolyte disturbances in a 61-year-old man

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A 61-year-old man, in apparent previous good health, presented to hospital with persistent dizziness and two attacks of postural syncope, all with a duration of less than a month. His blood pressure was 100/70 and 80/55 mmHg in the recumbent and upright postures, respectively. His pulse was 72 beats/min, regular, and all accessible peripheral pulses were palpable; the rest of the cardiovascular system examination was normal. He denied any drug therapy or gastrointestinal fluid losses. His serum electrolytes are shown in table 1.

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Table 1 Serum and urinary electrolytes

	<i>Serum (ref range)</i>	<i>Urine</i>
Sodium (mmol/l)	120 (133–149)	<10
Potassium (mmol/l)	2.5 (3.6–5.2)	21
Chloride (mmol/l)	72 (96–108)	—
Bicarbonate (mmol/l)	33 (24–30)	—
Urea (mmol/l)	26.3 (2.3–6.3)	—
Creatinine (μmol/l)	162 (50–130)	12 800
Osmolality (mOsm/kg)	266 (285–295)	401

Questions

- 1 What diagnoses would the serum and urine electrolytes suggest?
- 2 What treatment should this man receive?

Answers

QUESTION 1

The combination of hyponatraemia, hypokalaemia, raised bicarbonate (indicative of probable metabolic alkalosis but blood gases not performed) and uraemia is indicative of sodium, potassium and fluid loss. The uraemia in our patient is most likely to be pre-renal since urine sodium is undetectable, fractional sodium excretion is $<0.1\%$, urine to serum (U:S) creatinine ratio is 79, and U:S osmolality ratio is 1.51 (pre-renal uraemia is generally characterised by urine sodium <10 mmol/l, a fractional sodium excretion of $<1\%$, a U:S creatinine ratio of >25 and a U:S osmolality ratio of >1.5).

The diagnosis of Addisonian crisis is suggested by the hypotension, uraemia and hyponatraemia; however, the absence of clinical features suggestive of Addison's disease, as well as the absence of hyperkalaemia and increased renal sodium loss, makes this diagnosis extremely unlikely. The urinary results do not support a primary renal loss of fluid and electrolytes as the cause of hypotension in this man. In health, hyponatraemia (and plasma hypotonicity) leads to suppression of antidiuretic hormone secretion; this appropriate homeostatic response leads to the production of dilute urine, increased free water excretion and restoration of plasma sodium to normal. However, when intravascular volume depletion co-exists with hyponatraemia, the hypovolaemia produces a marked increase in antidiuretic hormone secretion over-riding the suppressive effect of hyponatraemia (as exemplified by our patient); this leads to high urine osmolality in the face of hypotonic serum, highlighting the body's attempts to maintain blood volume and tissue perfusion in preference to maintaining electrolyte balance.

In our patient, in the absence of obvious gastrointestinal losses, unadmitted diuretic or laxative abuse must be considered. The urine

measurements do not exclude the possibility of recent diuretic abuse.

QUESTION 2

This man should be given isotonic saline with potassium supplements to restore euhydration and electrolyte balance. A search for the cause of his electrolyte and fluid losses must continue.

Further clinical course

Soon after the initiation of fluid and salt repletion as an in-patient, a mucous liquid (approximately 800 ml per day) rectal discharge was noted. He admitted that he had noticed mild mucoid rectal discharge averaging 3–4 times per day for the previous 6 months. The composition of the faecal fluid is shown in table 2.

Table 2 Composition of serum and faecal fluid

	Serum	Faecal
Sodium (mmol/l)	124 (133–149)	159
Potassium (mmol/l)	3.3 (3.6–5.2)	34
Chloride (mmol/l)	74 (96–108)	157
Bicarbonate (mmol/l)	37 (24–30)	—
Urea (mmol/l)	27.4 (2.3–6.3)	14.5
Creatinine (μ mol/l)	179 (50–130)	19
Osmolality (mOsm/kg)	272 (285–295)	408

The main source of the fluid and electrolyte losses in this man is probably the lower gastrointestinal tract. The lack of a positive faecal osmolal gap probably excludes osmotic diarrhoea. The secondary hyperaldosteronism, following rectal loss of fluid and electrolytes, leads to further renal potassium loss, compounding the faecal potassium loss. Sigmoidoscopy revealed a villous adenoma whose extent was delineated by a barium enema (figure). Tumour fragments obtained by biopsy following sigmoidoscopic examination did not reveal a malignant change. A fasting gut hormone profile for vasoactive intestinal polypeptide, pancreatic polypeptide, gastrin, glucagon, somatostatin and neurotensin was negative. This man is currently awaiting surgery for excision of the tumour.

Discussion

The short duration of mucoid diarrhoea and occult faecal fluid and electrolyte losses in this man with villous adenoma is similar to that described in previous studies.¹ Most modern standard textbooks do not highlight the profound hyponatraemia that can occur in patients with villous adenomata, although hypokalaemia secondary to these tumours continues to be emphasized. The entire clinical picture in this man is attributable to the sudden development of sodium, potassium and fluid losses, reversed entirely by isotonic saline and potassium. We found no evidence of a humoral-mediation of faecal losses of fluid and

Figure Barium enema studies of colonic villous tumour showing (A) coating of the tumour surface by residual barium following evacuation, and (B) barium-filled rectosigmoid colon showing the outline of the tumour mass. The arrows indicate the site of the tumour

electrolytes such as those reported in tumours secreting excessive vasoactive intestinal polypeptide secretion. Our patient had no symptoms attributable directly to a local effect of the tumour, either as tenesmus or rectal bleeding, which may have contributed to the late clinical presentation.

Villous adenomata arise mainly in the rectum and the rectosigmoid, with equal sex distribution, usually in persons over the age of 50 years.² Malignancy is a common complication in these tumours and multiple histological sections may be required to exclude this possibility in any given patient. Diarrhoea of varying duration (3 months to 15 years) and volume (0.3–3.4 l/24 h) is commonly seen with these tumours; an average of 120, 44 and 123 mmol/l of sodium (range 40–120), potassium (range 15–107) and chloride (80–163), respectively, has been described in the faecal fluid.³ Vague abdominal pain, anorexia, weight loss, excessive thirst, nausea and vomiting may have been present for some time prior to total collapse.¹ Not all of these tumours produce fluid and electrolyte depletion⁴ but those that do may cause marked dehydration, lethargy, weakness, oliguria, metabolic acidosis, mental confusion, and hypotension. Ability to compensate for loss of fluid and electrolytes may extend over many years until the tumour enlarges sufficiently, intake is decreased or fluid loss increased to the point that compensatory mechanisms become ineffectual; inability to compensate may develop rapidly and dramatically, the previously asymptomatic patient presenting as an emergency.¹ Symptoms such as rectal bleeding, change in bowel habit, tenesmus, prolapse of the tumour, and elimination of tumour tissue have also been described.¹ Lesions can be over-

looked on rectal palpation because of their softness or a more proximal location. These tumours are usually single and located within reach of a sigmoidoscope, as was the case in our patient, although multiple and more proximal lesions have been described.⁵ Typically the tumour arises in the rectum, is sessile, reddish-grey, bulky and may involve the entire circumference of the bowel. Barium enema studies are helpful, as seen in our patient, in outlining the extent of the lesions as well as establishing the presence or absence of multiple lesions. Definitive diagnosis must be confirmed by histological examination.^{5–6} Lack of awareness has been credited with fatal outcome, up to 20% in some series^{1–3}; such fatal outcome is invariably secondary to the incomplete correction of fluid and electrolyte imbalance produced by these tumours. Complete excision of the lesion with a margin of normal tissue is curative and the treatment of choice in benign lesions. If the tumour is shown to be malignant, the type of surgery should be the same as for any other carcinoma of colon or rectum.

We were unable to find recent publications describing the electrolyte and fluid imbalance that may occur with villous adenoma. This case is a reminder that severe electrolyte and fluid disturbances may occur with these tumours and energetic replacement is required in the management of these patients.

Final diagnosis

Villous adenoma causing severe electrolyte and fluid disturbances.

Keywords: villous adenoma; hyponatraemia; hypokalaemia; electrolyte imbalance

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