

Pseudo Bartter Syndrome from Surreptitious Purging Behaviour in Anorexia Nervosa

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Abstract

Pseudo Bartter syndrome is a rare disorder characterized by metabolic alkalosis, hypokalaemia, hyperaldosteronism, hyperreninism, normal blood pressure and hyperplasia of the juxtaglomerular apparatus.

The most dangerous complication of Pseudo Bartter syndrome is hypokalemia.

Hypokalemia caused by vomiting, diarrhea, prolonged fasting, abuse of potassium-depleting drugs, may be present in patients with binge /purging form of anorexia or bulimia nervosa.

We report a case of a 19-year-old girl with anorexia nervosa (BMI 16.15 kg/m²) and severe prolonged hypokalemia (1.9 mEq/l), metabolic alkalosis and severe protracted secondary hyperaldosteronism (i.e. Pseudo Bartter's syndrome) from surreptitious purging behaviour (vomit and laxative abuse).

An intensive multidisciplinary day-hospital treatment including long-term potassium supplementation, a potassium-sparing diuretic was necessary to resolve the case and to allow the young girl to admit her previous purging behaviour and after three months to get at a normal kalemia without any potassium supplementation and BMI at a normal value (20 kg/m²).

Given the dangers to the heart electrical and mechanical functions set by severe potassium deficiency, it is mandatory to find out the true cause so that a proper treatment can be started.

Keywords: Secondary hyperaldosteronism; Hypokalemia; Alkalosis metabolic; PseudoBartter syndrome; Anorexia nervosa; Surreptitious purging

Introduction

Pseudo Bartter syndrome is a rare disorder characterized by metabolic alkalosis, hypokalaemia, hyperaldosteronism, hyperreninism, normal blood pressure and hyperplasia of the juxtaglomerular apparatus [1].

Surreptitious vomiting, diuretic or laxative abuse for purpose of "dieting" are known to cause a primary loss of: water, sodium, potassium, hydrogen and secondary loss of mainly potassium and hydrogen through renal aldosterone-regulated adaptive mechanisms. The whole picture is not so easy to be described for two main reasons. First the whole balance integrates several organs (stomach, small intestine, colon, kidneys); second, the disrupting factors (vomiting, laxative, diuretic drugs) when acting simultaneously or successively can make up a very complex picture ("metabolic madness") in which the core of the PseudoBartter's syndrome e.g. hyperaldosteronism, hypokalemia, metabolic alkalosis may be shaped in different ways [2,3].

The most dangerous complication of PseudoBartter's syndrome is hypokalemia.

Among the many functions of potassium in the body we may mention: regulation of heart function, reduction of blood pressure, fluid and acid/alkali balance regulation and normal nerve and muscle function.

Normal people absorb about 90% of the daily intake (80-90 mEq) and excrete an equivalent amount of potassium in urine (72-80 mEq) and feces (8-10 mEq).

Hypokalemia usually ensues if such an equilibrium is altered.

In western countries hypokalemia is most commonly caused by the use of diuretics, then by diarrhea (spontaneous or due to laxative abuse) or vomiting.

Hypokalemia due to vomiting is only partly due to direct loss of potassium, because vomiting has a further effect of provoking an increased potassium loss in the urine to compensate for blood alkalosis [4].

Hypokalemia caused by vomiting, diarrhea, prolonged fasting, abuse of potassium-depleting drugs, may be present in patients with binge /purging form of anorexia or bulimia nervosa [5].

Anorexia Nervosa is a serious, potentially life-threatening illness characterized by severe malnutrition without physical obstacles to eating, abnormally low body weight (BMI < 17.5 kg/m² or body weight at least 15% below the expected value); nearly always there is also an intense fear of weight gain and an undue emphasis on weight and shape in self-evaluation. Amenorrhea (i.e. loss of three consecutive menstrual cycles) is currently required for the diagnosis, but often is hidden by contraceptive drugs. Anorexia nervosa may also be accompanied by purging behaviours as vomiting, abuse of laxative and/or diuretics in about 30% of cases.

Anorexia nervosa can as well cause significant clinical complications in every organ system.

Undernutrition especially if associated to purging nearly always leads to marked changes in body spaces (e.g. intra-extracellular water)

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in body masses (e.g. potassium, phosphate, magnesium overall and compartmental stores).

Moreover, we also must consider that anorexia nervosa patients display the highest death rate of all mental illnesses and a relevant part of mortality has to be attributed to undernutrition.

Here we report a case of a girl affected by an eating disorder and severe hypokalemia, metabolic alkalosis and protracted, severe, secondary hyperaldosteronism, i.e. a Pseudo Bartter's syndrome.

Case presentation

The patient, a 19-year-old caucasian girl, was referred to our Eating Disorders Unit by the Medical Department of our Hospital.

She was affected by severe long-term hypokalemia requiring daily potassium supplementations.

In the previous six months she was admitted seven times to various general medicine, cardiology or gastrointestinal units with a pattern of hypokalemia, hypochloremia and metabolic alkalosis (extreme values of 1.9, 70 and 38.6 mEq/l).

She was treated with i.v. KCl, oral K-salts supplementation and the amount of added potassium ranged from 40 to 80 mEq/day.

The only sign exhibited was parotid gland enlargement. She denied her underweight condition and any eliminatory behavior, specifically vomiting, use of diuretic, emetic or laxative substances; but her past medical history was significant for anorexia nervosa, for which she was not treated nor seeking treatment.

At the first contact her physical examination showed: weight 44.5 kg, height 1.66 m, BMI 16.15 kg/m², blood pressure 95/50 mmHg, heart rate 46 min. Her heart sounds were normal without any murmurs, rubs or gallops, but the ECG showed a prolonged QT interval of 0.52 seconds.

Some pathology values measured before starting the treatment in our Eating Disorders Unit are displayed in the first column of Table 1.

We diagnosed severe undernutrition likely from anorexia and hypothesized secondary hyperaldosteronism due to persistent hypokalemia triggered by vomiting and/or purging, which likely was still "practiced" by the patient, in spite of her denial. As a matter of

fact, aldosterone serum levels turned out to be > 100 µg/dl (149 µg/dl "in standing" and 152 µg/dl "in supine position").

An intensive multidisciplinary day-hospital treatment in our Eating Disorders Unit was started with nutritional rehabilitation, behavioural therapy psychotherapy and family psycho-education program. Long-term potassium supplementations was planned and the electrolyte profile was daily checked. We started by an intravenous infusion of potassium chloride (40- mEq/day) plus oral supplementation of potassium chloride (20-40 mEq/day), a potassium-sparing diuretic was added i.e. canrenone, a renal competitive aldosterone antagonist (100 mg/day).

After 2 months it was possible to stop canrenone and then progressively lower the potassium salts. Lastly, after 3 months of multidisciplinary treatments her kalemia was kept at normal levels without any potassium supplementation; her body weight increased to 55 kg and BMI raised to normal value (20 kg/m²) and the serum aldosterone dropped to 2/3 of initial value (48 µg/l) (Table 1, second column).

At last, the cooperative effort of the whole team allowed the young girl to admit her previous purging vomiting (many times a day) and diarrhea by using laxative.

Discussion

In the seven previous hospital admissions the focus was on hypokalemia. Disfunctions of the intestinal absorption were variously hypothesized. The young patient was investigated with gastroduodenoscopy and colonoscopy with only minimal findings of gastric inflammation and possibly irritable colon; repeated searches of any germ in stool material turned out unsuccessful. There were no findings at all of celiac disease, whether from biopsy or immunochemistry. Also renal diseases as chronic tubulo interstitial nephritis, Bartter syndrome, Gilteman syndrome and diuretics in the urine were explicitly but unsuccessfully investigated.

As a matter of fact the clues for us were the physical findings of anorexia (BMI) and the suspicion of purging behavior which could, among other things, explain the abnormally low chloride output and the unexpectedly severe, protracted hypokalemia. The purging behavior accompanies the anorexia more often than usually expected and quite often is strenuously denied by the patient; one more reason for searching for it.

As a matter of fact, chronic vomiting and diarrhea without diuretics can cause a Pseudo Bartter's syndrome with the full pattern of Bartter's syndrome, but with low chloride urinary output.

Indeed, the urine chloride content can help the clinician: in all the previous conditions, except in diuretic abuse, the urine chloride level is high, while in Pseudo Bartter's syndrome this level is low, because chloride is lost via the oral-intestinal route. Thus, the urine chloride level distinguishes this condition from all other forms of hypokalemic metabolic alkalosis.

The volume depletion from self-induced vomiting and laxative abuse activates the rennin - angiotensin - aldosterone axis. In fact, our patient presented hyperreninism added to hyperaldosteronism. Increased aldosterone secretion acting on the distal renal tubule further exacerbates the hypokalemia and metabolic alkalosis, as in a self-perpetuating vicious cycle [6,7].

To sum up the case, our young patient showed that chronic hypokalemia was in fact produced by a state of systemic chloride

Labs (range)	Admission *	Discharge **
Body weight (Kg)	44.5	55
Height (m)	1.66	1.66
Body mass index (kg/m ²)	16.15	20
Sodium (132 – 143 mmol/l)	133	140
Potassium (3.4 – 5.2 mmol/l)	1.9	4
Chloride (96 – 107 mmol/l)	70	102
Bicarbonate (22 – 30 mmol/l)	39	20
Aldosterone (2-15 µg/dl)	149	48
Renine (2.8 – 39.9 µU/ml)	240	58.6
Urine/ 24 h		
Sodium (mmol/24 h))	24	32
Potassium (mmol/24 h))	179	46
Chloride (mmol/24 h))	23	21
Creatinine (mg)	1206	1503

*during potassium infusion of 80 mEq/24h

**none potassium supplementation

Table 1: Anthropometric, serum and urine values at admission and discharge.

depletion induced by renal-independent factors, such as surreptitious cyclic vomiting and laxative abuse and not by renal dependent factors as happens in Bartter's syndrome [8].

Given the dangers to the heart electrical and mechanical functions set by severe potassium deficiency, it is mandatory to find out the true cause so that a proper treatment can be started.

We should also consider that a too late diagnosis may lead to irreversible complications as end-stage renal disease [9] or central pontine myelinolysis [10], as literature reported in two anorexia nervosa patients.

Finally, I will emphasize the diagnosis of anorexia nervosa can be elusive, because in contrast to most patients with loss of weight for other medical conditions (i.e. hyperthyroidism malignancy etc.) who express concern over their weight loss or alterations of their biochemical values, patients with anorexia nervosa are actively pursuing an abnormally body weight, usually they hide the illness and actively deny their symptoms, so hampering the collection of their true history.

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