Pseudohypoaldosteronism following Resection of Ileum and Colon

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Dear Sir,

Pseudohypoaldosteronism is a disorder of mineralo-corticoid resistance manifested by hyponatremia and/or hyperkalemia with normal or increased plasma aldosterone [1–7]. Characteristically, raising plasma mineralocorticoid level by administration of exogenous mineralocorticoid does not correct serum electrolyte imbalance in pseudohypoaldosteronism. We report a patient with a colon cancer who, after subtotal colectomy and ileal resection, developed the picture of pseudohypoaldosteronism. In contrast to the established cases of pseudohypoaldosteronism [1–7], this case was not caused by an aldosterone receptor defect [1–3] or an abnormality of the kidney in handling sodium or potassium [4, 5]. The primary abnormality was the resection of the ileum and colon, key sites for sodium and water absorption. A marked deficit of body sodium and water caused the picture of aldosterone resistance.

Case Report

An 82-year-old man was admitted to the hospital because of acute onset of abdominal pain with the clinical picture of intestinal obstruction. An exploratory laparotomy revealed adenocarcinoma in the descending colon and infarction of a portion of his ileum. He underwent subtotal colectomy, resection of 40% of the small bowel including the ileum, and jejunostomy. Total parenteral nutrition was provided initially, and later he was given 0.9% sodium chloride intravenously. As oral intake increased, his intravenous fluids were gradually tapered and discontinued. Seven days later he developed hyponatremia and hyperkalemia without clinical evidence of Addison's disease. Serum electrolytes showed Na of 125 mEq/l, K of 6.1 mEq/l, Cl of 90 mEq/l, and CO₂ of 25.5 mEq/l. BUN and serum creatinine were 51 mg/dl and 2.2 mg/dl, respectively. Plasma cortisol level obtained at 2:00 PM was 25 µg/dl. Plasma renin activity was 28.9 ng/ml/h (upper limit: 27 under dehydrated condition) and serum aldosterone level was 120 ng/ml (normal: 3–31). He responded to intravenous administration of 5% glucose/0.9% sodium chloride with normalization of serum electrolytes, BUN, creatinine, plasma renin activity, and serum aldosterone levels. Hyponatremia and hyperkalemia were reproducible 6–7 days after discontinuation of intravenous fluid therapy. A balance study was carried out with oral ingestion of 5–10 g salt and 40 mmol potassium daily without intravenous fluid. Before the study, he received 9 g sodium chloride daily by intravenous infusion and oral salt ad libitum. Figure 1 shows the results of the balance study. Fecal volume and fecal sodium excretion exceeded his oral intake of water and sodium, indicating loss of water

and sodium in the gastrointestinal tract. Urinary excretion of sodium and potassium progressively decreased even with 10 g salt intake. Because of oliguria, the balance study was terminated at the end of the 4th day; during this period serum sodium and potassium did not change significantly.

Comments

Our patient presented the classic electrolyte disturbance seen in aldosterone deficiency but with a paradoxically high serum aldosterone level. In addition, a high level of serum aldosterone was ineffective in correcting serum electrolyte imbalance. Therefore, this case fits the picture of pseudohypoaldosteronism. Two types of familial pseudohypoaldosteronism have been described [1–5]. Type 1 is renal sodium wasting in an infant due to an aldosterone receptor defect [1–3]. Type 2 is characterized by hyperkalemia and hyperchloremic acidosis with normal renal function; the basic abnormality is an increased reabsorptive avidity of the distal nephron for chloride, which impairs aldosterone-mediated potassium and hydrogen ion excretion [4, 5]. An acquired pseudohypoaldosteronism has been reported following renal transplantation [6] and in a patient with methicillin-induced nephritis [7]. The primary abnormality of serum electrolyte in our case was the resection of the colon and the ileum, key sites for sodium and water absorption [8, 9].

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1

3

i	1	1	i	1
12	3	4		

DAYS

Fig. 1. The patient received intravenous normal saline infusion until 8:00 a.m. of day 1; thereafter only oral feeding was given. Urine and fecal samples were collected from 8:00 a.m. to 8.00 a.m.

Depletion of body sodium and water activated the renin-aldosterone system. The kidney responded appropriately to sodium and water depletion by decreasing urinary sodium excretion (fig. 1), and there was no evidence of renal sodium wasting. Although we did not continue the balance study until the development of hyponatremia and hyperkalemia, it is conceivable that aldosterone could no longer facilitate potassium excretion in the presence of a profound decrease of renal tubular sodium.

ORAL INTAKE

NaCl/day

4000 -

2000 0

VOLUME

(ml/24h) URINE

1000 VOLUME (ml/24h) 50° 0

50

Na (mmol/24h) 25

0

50

K (mmol/24h) 25

0
FECES
4000
VOLUME (ml/24h)
2000
0
400
Na (mmol/24h) 200
0
50
K (mmol/24h) 25
0
10g

This could cause hyperkalemia. Elevation of plasma aldosterone is common in patients whose terminal ileum and a half colon are resected [9]. It is conceivable that serum electrolytes are maintained at the marginal level by the elevated aldosterone level in these patients. When sodium and water absorption becomes critically low, aldosterone can no longer maintain normal serum electrolyte ; this causes the picture of pseudohypoaldosteronism. Our case is such an example. Since this disorder has not been described as pseudohypoaldosteronism, we present the case here.

References

- Cheek, B.; Perry, W.: A Salt wasting syndrome in infancy. *Archs Dis. Childh.* 55:252–256 (1958).
- Oberfield, E.; Levine, S.; Carey, M.; Bejar, R.; New, I.: Pseudohypoaldosteronism: multiple target organ unresponsiveness to mineralocorticoid hormones. *J. clin. Endocr. Metab.* 48: 228–234 (1979).
- Armanini, D.; Kuhnle, U.; Strasser, T; Dorr, H.; Butenandt, I.; Weber, C; Stockigt, R.; Pearce, P.; Funder, W.: Aldosterone receptor defect. *New Engl. J. Med.* 575:1178–1181 (1985).
- Schambelan, M.; Sebastian, A.; Rector, C, Jr.: Mineralocorticoid-resistant renal hyperkalemia without salt wasting (type II pseudohypoaldosteronism): role of increased renal chloride reabsorption. *Kidney int.* 19:716–727 (1981).
- Licht, J.H.; Amundson, D.; Hseuh, W.A.; Lombardo, J.V.: Familial hyperkalemic acidosis. *Q. Jl Med.* 213:161–176 (1985).
- Uribarri, J.; Oh, S.; Butt, H.; Carrol, J.: Pseudohypoaldosteronism following kidney transplantation. *Nephron* 31: 368–370 (1982).
- Cogan, M.; Arieff, I.: Sodium wasting, acidosis and hyperkalemia induced by methicillin interstitial nephritis. *Am. J. Med.* (54:500–503(1978).
- Hill, L.; Mair, J.; Goligher, C: Impairment of ileostomy adaptation in patients after ileal resection. *Gut* 75:982–987 (1974).