Original Article


L’hypersécrétion colique de potassium : un nouveau mécanisme de diarrhée associé aux pseudo-obstructions ? À propos de cinq cas

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Summary
Objective. — To report the mechanism of diarrhoea in patients with subacute colonic pseudo-obstruction, profuse secretory diarrhoea and hypokalemia.
Patients. — Five consecutive patients who developed colonic pseudo-obstruction, profuse watery diarrhoea and severe hypokalemia. Investigations excluded mechanical intestinal obstruction. Usual cause of diarrhoea were ruled out. Abdominal distension and diarrhoea improved simultaneously in all cases after colonoscopic decompression or intravenous neostigmine.
Results. — Faecal ionograms showed a low osmotic gap and high faecal potassium concentration explaining the hypokalemia: 100 to 180 mEq/kg (usually inferior than 50 mEq/l in case of secretory diarrhoea) and low faecal sodium concentrations. Potassium salts were the only factor identified as the driving osmotic force for the diarrhoea.
Conclusion. — Secretory diarrhoea is classically due to chloride active secretion with passive sodium secretion or to inhibition of sodium absorption. In five cases of Ogilvie’s syndrome we evidenced an original mechanism of secretory diarrhoea due to active potassium secretion responsible of a profound hypokalemia. This novel type of diarrhoea may be a hallmark of colonic pseudo-obstruction due to colonic distension.

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K+ was low (18 mmol/l). Potassium concentration in the lower limit of the normal range. Urinary excretion of K+ by the colon has been described[3].

Colonic pseudo-obstruction may be associated with Ogilvie’s syndrome and secretory diarrhoea evident with diarrhoea of unclear mechanism. Recently, a patient developed secretory diarrhoea with large amounts of mucus in the stools and high faecal potassium concentrations responsible of profound hypokalemia. These cases confirmed the original mechanism of diarrhoea associated with colonic pseudo-obstruction that may be frequent in intestinal pseudo-obstruction, although not currently recognized.

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Acute or subacute colonic pseudo-obstruction (so-called ‘‘Ogilvie’s syndrome’’) occurs mainly in hospitalized elderly patients with severe illness or after surgery [1,2]. The precise pathogenesis is incompletely understood: it is thought to result from an imbalance in the activity of the autonomic innervation of the gut with relative excess of sympathetic stimulation or of parasympathetic suppression [1]. Several factors are known to contribute to the colonic dilatation, including drugs and electrolytic disorders such as hypokalemia. Colonic pseudo-obstruction may be associated with diarrhoea of unclear mechanism. Recently, a patient with Ogilvie’s syndrome and secretory diarrhoea evidencing a novel mechanism mediated by excess of potassium secretion by the colon has been described [3].

We report five consecutive patients hospitalized in our institution with acute colonic pseudo-obstruction who developed secretory diarrhoea with large amounts of mucus in the stools and high faecal potassium concentrations responsible of profound hypokalemia. These cases confirm the original mechanism of diarrhoea associated with colonic pseudo-obstruction that may be frequent in intestinal pseudo-obstruction, although not currently recognized.

Cases report

Case 1

A 79-year-old man was admitted in intensive care unit for severe bilateral pneumonia and bleeding duodenal ulcer. Under total parenteral nutrition, he developed a marked abdominal distension and watery profuse diarrhoea, with large amounts of mucus in the stools and was transferred in gastrointestinal unit. CT-scan showed a diffusely dilated colon up to 9 cm without small bowel distension. Severe hypokalemia developed requiring continuous intravenous KCl 12 g/day (160 mEq/day of K+) to maintain kalemia at the lower limit of the normal range. Urinary excretion of K+ was low (18 mmol/l). Potassium concentration in the faecal fluid was high (137 mEq/l) and sodium concentration low (17 mEq/l); chloride concentration was 117 mEq/l and bicarbonates concentration 55 mEq/l. Search for bacteria, Clostridium difficile toxins, parasites and virus in the stools was negative. Total colonoscopy excluded colonic obstruction and showed a marked dilatation of the sigmoid colon “crying tears of mucus”: biopsies showed a hypercrinic mucosa. Intravenous neostigmine was ineffective and colonic decompressions with rectal tube insertions were performed twice over one month: diarrhoea and colonic distension gradually improved but general condition declined and the patient died of sepsis.

Case 2

A 92-year-old man was hospitalized for progressive left hemiplegia related to a large subacute cerebral haematoma. He complained of watery and mucous diarrhoea since 10 days. At the admission, kalemia was only slightly decreased at 3.1 mEq/l. Rectosigmoidoscopy disclosed an important distension of the sigmoid colon. Search for bacteria and C. difficile toxins in the stools was negative. Diarrhoea and abdominal distension persisted and kalemia fell to 2.0 mEq/l. Urinary excretion of K+ was 40 mmol/l. Potassium concentration in a stool sample was high (180 mEq/kg) and sodium concentration low (30 mEq/kg). Without other specific treatment than intravenous KCl (up to 8 g KCl/day) and rectal tube insertion diarrhoea and colonic distension gradually improved over one month. Neostigmine was not used. General condition declined and the patient died of pulmonary inhalation.

Case 3

A 97-year-old patient was hospitalised for severe abdominal distension and watery diarrhoea with mucus discharge for two days. He had a history of hypothyroidism and mild chronic renal insufficiency. At the admission, laboratory results showed acute renal failure and hypokalemia at 2.1 mEq/l. CT-scan disclosed colonic distension up to
8 cm; colonoscopy showed a distended colon without obstruction. After parenteral rehydration renal function improved; 8 g KCl/day were necessary to maintain kalemia in normal range; urinary losses of K⁺ were 23 mEq/day; fecal K⁺ concentration in a stool sample was 110 mEq/kg and Na⁺ concentration was 40 mEq/kg. Colonic distension, watery diarrhea and mucus losses occurred despite three colonoscopic decompressions but improved rapidly after intravenous neostigmine without further recurrence.

Case 4

A 78-year-old man with a history of mild chronic renal insufficiency and ischemic heart disease was hospitalized in intensive care unit for pneumonia complicated by hypovolemic shock, acute renal failure and congestive heart failure. After improvement he developed abdominal distension, watery diarrhea with mucus in the stools and mild hypokalemia at 3,1 mEq/l and was transferred in gastroenterology unit. Four grams per day of KCl were necessary to maintain kalemia at the lower limit of the normal range. Search for bacteria, C. difficile toxins, parasites and virus in the stools was negative. CT-scan and a colonoscopy up to the right flexure excluded colonic obstruction and disclosed a marked dilatation of the sigmoid colon. Biopsies disclosed a hypercrinic mucosa. Faecal K⁺ concentration in a stool sample was 100 mEq/kg and Na⁺ faecal concentration 20 mEq/kg. Urinary excretion of K⁺ was low at 16 mEq/l. After colonic decompression and rectal tube insertion abdominal distension and diarrhea improved over several days and the patient was discharged. Neostigmine was not used.

Case 5

A 93-year-old man with an history of chronic atrial fibrillation was hospitalized for pneumonia. He had a marked abdominal distension and watery profuse diarrhea, with large amounts of mucus in the stools. CT-scan showed a dilated sigmoid colon up to 10 cm. At the admission, laboratory results showed acute renal failure and mild hypokalemia at 3,2 mEq/l but hypokamemia felt at 2,6 mEq/l. Four grams per day of KCl were necessary to maintain kalemia at the lower limit of the normal range. Search for bacteria and parasites in the stools was negative. Rectosigmoidoscopy disclosed an important distension of the sigmoid colon. Biopsies disclosed a hypercrinic mucosa. Faecal K⁺ concentration in a stool sample was 100 mEq/kg and Na⁺ faecal concentration 60 mEq/kg. After colonic decompression and rectal tube insertion abdominal distension and diarrhea improved over several days and the patient was discharged. Neostigmine was not used.

Discussion

We report five consecutive patients hospitalized in our institution with acute or subacute colonic pseudo-obstruction who had a typical secretory diarrhea persisting during fasting with a low faecal osmotic gap and very high faecal potassium concentrations: 100 mEq/kg, 100 mEq/kg, 110 mEq/kg, 137 mEq/l and 180 mEq/kg of K⁺ respectively in patients 4 and 5, 3, 1 and 2, whereas normal concentrations are approximately 90 mEq/l, and usually inferior than 50 mEq/l in case of secretory diarrhea [3–6]; faecal sodium concentrations were low. These elevated faecal concentrations of potassium in large volume (although not quantified) diarrhea induced important outputs of potassium salts responsible of profound hypokalemia and decreased urinary excretion of K⁺. A potassium compensation as high as 160 mEq/day in patient 1, 105 mEq/day in patients 2 and 3, and 50 mEq/day in patients 4 and 5 was required to maintain a normal kalemia, whereas physiological potassium faecal losses are under 9 mEq/day [5].

The pathophysiology of secretory diarrhea is well known: it is caused by active chloride secretion with passive sodium secretion or inhibition of sodium absorption; abnormalities in potassium transport are not known to cause secretory diarrhea [3–6]. In our patients the potassium salts were the principal ions in the stools and the sole osmotic force explaining the diarrhea; these results evidence an original mechanism of diarrhea due to active potassium secretion involving the colon because large bowel but not small intestine has the physiologic capacity to actively secrete potassium [4,5]. Such a mechanism has been described before only once in a single patient who had also acute colonic pseudo-obstruction [3]. This peculiar mechanism is not found in common etiologies of diarrhea [3,4,6]; actually, we did not evidence any usual cause in our patients despite careful investigations. However, we cannot totally rule out to have missed one because they all had multiple disorders, complex medical histories and had had numerous drug therapies including antibiotics. We assume that the pseudo-obstruction itself explains these excessive faecal losses of potassium and the related hypokalemia; our hypothesis is supported by the fact that diarrhea and potassium requirements persisted during the whole course of the pseudo-obstruction in our five patients, but rapidly resolved after effective colonic decompression. Hypokalemia is common and often refractory in Ogilvie’s syndrome and is classically considered to be responsible of the colonic pseudo-obstruction; on the contrary, we believe that it is more likely to be a consequence of the pseudo-obstruction that can lead to self-aggravation of the colonic distension. Our five patients also had abundant discharge of mucus with large amounts of egg’s white like matter in the watery stools. In patient 1 the distended part of the colon was “‘crying tears of mucus’”; in patients 1 and 4 biopsies disclosed a hypercrinic colonic mucosa. These enhanced mucus secretion are likely to be linked to the secretory diarrhea, and they both may reflect an over stimulated colonic mucosa.

The possible pathogenesis of the increased colonic potassium secretion associated with pseudo-obstruction had been previously exposed comprehensively by van Dinter et al. [3]. They propose two principal hypothesis: first, colonic pseudo-obstruction and secretory diarrhea might be both due to a hyperadrenergic stimulation of the enteric nervous system: a relative excess of sympathetic activity may decrease colonic motility and epinephrine is known to stimulate potassium secretion through beta-adrenergic receptors in rabbit colon [7]; data are lacking in human and the effect of adrenergic stimulation on the colonic mucus secretion is not known [8]. However, adrenergic inhibitors are ineffective for Ogilvie’s syndrome whereas neostigmine,
which increases cholinergic activity, is effective [9,10]. Second, colonic distension may stimulate colonic secretion by itself. Experimental evidences in animal models show that distension of the colon can stimulate VIPergic secretory neurons of the enteric nervous system [11]. VIP can induce mucus and electrolytic secretion in rat colon [8,11]; to our knowledge, experimental data are lacking in humans but in one patient we evidenced that the mucus was secreted exclusively from the dilated part of the colon. The rapid resolution of the diarrhoea after effective colonic decompression in our patients supports this second hypothesis.

We found high faecal concentration of potassium in five consecutive patients with Ogilvie’s syndrome associated with watery and mucous diarrhoea and hypokalemia: these observations suggest that this newly recognized mechanism of colonic secretory diarrhoea associated with colonic distension is probably not rare and could be a not yet recognized hallmark of colonic pseudo-obstruction. However, further studies are necessary to confirm this hypothesis. It is an important concern for clinical practice because it suggests that early decompression of colonic distension by endoscopic or pharmacological (that is neostigmine) treatments may reduce faecal potassium losses, avoid hypokalemia and thereby prevent the constitution of a vicious circle of self-aggravation.

References


