

- 5 Chopra IJ, Williams DE, Orgiazzi J, Solomon DH. Opposite effects of dexamethazone on serum concentrations of 3,3',5'-triiodothyronine (reverse T₃) and 3,3',5'-triiodothyronine (T₃). *J Clin Endocrinol Metab* 1975; 41: 911–20.
- 6 Croxon MS, Hall TD, Nicoloff JT. Combination drug therapy for treatment of hyperthyroid Graves' disease. *J Clin Endocrinol Metab* 1977; 45: 623–30.
- 7 Ozawa Y, Daida H, Shimizu T, Shishiba Y. Rapid improvement of thyroid function by using glucocorticoid indicated for the preoperative preparation of subtotal thyroidectomy in Graves' disease. *Endocrinol Japon* 1983; 30: 93–100.
- 8 Volpe R. Immunoregulation in autoimmune thyroid disease. *N Engl J Med* 1987; 316: 44–6.

Congestive cardiac failure following laxative withdrawal

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Summary

We report the case of a 60-year-old woman who presented with weakness and hypokalaemia due to excessive use of laxatives. When the laxatives were withdrawn, she developed severe congestive cardiac failure requiring treatment with a diuretic and angiotensin-converting enzyme inhibitor. There was no underlying cardiac abnormality, and these drugs were eventually stopped with no recurrence of the cardiac failure. The possible mechanisms of heart failure following laxative withdrawal is discussed.

Keywords: laxative abuse, hyperaldosteronism, cardiac failure

Laxatives account for a significant proportion of over-the-counter (OTC) pharmacy sales (£37m in 1994 out of a total of £1268m OTC pharmacy and grocery sales). The wide variety and easy availability can lead to excessive use, inappropriate dosage, and even abuse of these medicines. Loss of fluid and electrolytes in diarrhoea may result in secondary hyperaldosteronism and hypokalaemia. Oedema may be a feature of the recovery phase after laxative withdrawal.

We describe a patient whose excessive laxative intake led to life-threatening cardiac failure after the laxatives were withdrawn.

Case report

A 60-year-old woman underwent a bilateral salpingo-oophorectomy for endometrial carcinoma in December 1987. She made a good recovery and routine follow-up showed no evidence of recurrence. Following the operation, she was told to avoid straining at stool and was prescribed Fybogel sachets and senna tablets. She felt this laxative regimen did not have the desired effect and some months later, on the advice of a friend, she started to take Fam-Lax (phenolphthalein and rhubarb) which she bought over-the-counter. Starting

with the recommended adult dose of two tablets, she soon increased this to five tablets per day. This produced a liquid, easily passed stool within a few hours.

Whilst on holiday in Spain in 1993 she felt weak and exhausted and was told she had a low blood pressure. By the time of her return home the symptoms had become more severe and eventually resulted in her admission to hospital.

On admission, her blood pressure was 105/60 mmHg. Clinically her heart was normal and there was no evidence of cardiac failure. Urea and electrolytes were within the normal range except for serum potassium which was found to be low at 2.6 mmol/l. Her prescribed medication consisted of medroxyprogesterone, sucralfate, ranitidine and co-dydramol.

In the absence of any other obvious cause for her hypokalaemia, laxative abuse was suspected. She admitted to taking the Fam-Lax – she had simply been following instructions not to strain at stool.

The laxatives were withdrawn and oral potassium supplements started (Sando-K 2 bid). Serum potassium rose rapidly to normal levels over the next four days (figure).

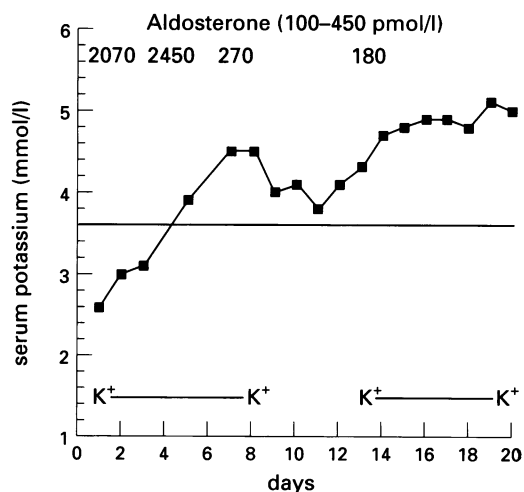


Figure Serum potassium and aldosterone levels

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Hypokalaemia: symptoms and signs

- lethargy
- muscle weakness
- polydipsia and polyuria
- nephropathy
- abnormal ECG
- cardiac arrhythmias
- paralytic ileus

On day 6 some mild generalised oedema was noted, but a chest X-ray was normal and there was no evidence of cardiac failure at this time. Her weight, however, was increasing. On day 8 she began to complain of breathlessness and increased ankle swelling was noted. Her chest remained clear. By day 10, her weight had risen by 15 kg. Her chest X-ray now showed cardiomegaly and bilateral pleural effusions (heart size was normal on admission). She was in severe cardiac failure and treatment with frusemide and captopril was initiated. Potassium supplements were stopped, but the serum potassium began to fall, so they were re-started with daily monitoring (hyperkalaemia can develop with concurrent use of an angiotensin-converting enzyme inhibitor and potassium supplements).

To exclude the possibility of pulmonary embolism as a cause of the cardiac failure, a lung perfusion scan was performed on day 13. This was reported as low probability for pulmonary embolism.

During the next two weeks she made a gradual improvement, the chest X-ray returned to normal, she lost 10 kg in weight and now had no evidence of cardiac failure. An electrocardiogram (ECG) and echocardiogram were normal and the frusemide and captopril were gradually withdrawn. Aldosterone levels are shown in the figure.

In the year since this episode, she has remained well and laxative treatment now consists of docusate and Fybogel. In standard dosage, this combination is unlikely to cause electrolyte imbalance.

Discussion

Laxative abuse is a well known cause of

Learning points

- laxative abuse is a well known cause of hypokalaemia
- withdrawal of laxatives may lead to the formation of oedema due to persisting hyperaldosteronism
- rarely, such fluid retention may be excessive and lead to cardiac failure

hypokalaemia and such patients may present with a wide variety of symptoms and signs.¹ The hypokalaemia is multifactorial.² There is faecal loss of potassium *per se* in the stool, but the primary metabolic deficit is one of sodium and water depletion. This leads to increased renin secretion, and secondary hyperaldosteronism follows. Aldosterone causes increased loss of potassium from the colon and the kidney and renal damage may lead to further potassium loss.

This patient's presenting complaints of weakness and exhaustion are likely to have been due to the hypokalaemia. The weight gain and oedema that followed the withdrawal of laxatives were presumably due to persisting hyperaldosteronism (figure).

Although oedema following laxative withdrawal is not uncommon, it subsides spontaneously over a period of weeks as sodium balance becomes re-established.^{3,4}

In this case, the fluid retention was excessive and led to severe cardiac failure. There was no suggestion of any underlying cardiac disease and frusemide and captopril have been withdrawn without recurrence. We are not aware of any similar case of cardiac failure following laxative withdrawal.

1 Cummings JH. Progress report. Laxative abuse. *Gut* 1974; 15: 758-66.

2 Moriarty KJ, Silk DBA. Laxative abuse. *Dig Dis* 1988; 6: 15-29.

3 Fine KD, Kreis GJ, Fordtran JS. Diarrhoea. In: Sleisenger MH, Fordtran JS, eds. *Gastrointestinal disease* Vol 1. WB Saunders, 1989; pp 306-8.

4 Ullrich I, Lizarralde G. Amenorrhoea and edema. *Am J Med* 1978; 64: 1080-3.