CASE REPORT

Eating disorders should be considered in the differential diagnosis of patients presenting with acute kidney injury and electrolyte derangement

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SUMMARY

We present a case of a 40-year-old woman with a history of ongoing anorexia nervosa and bulimia nervosa who has required multiple admissions to hospital for management of acute kidney injury (AKI) and electrolyte derangement. This case is of interest as recent studies have highlighted the significant prevalence of disordered eating and the major public health implications this may have. We discuss the unusual finding of hypercalcaemia in this case and address the investigation and management of AKI and electrolyte disturbance in a patient with anorexia and bulimia.

BACKGROUND

Although the lifetime prevalence of eating disorders is relatively low, the full spectrum of disordered eating is likely to be considerably higher than previously estimated,[1] proving an increasing public health problem with clinical manifestations including hypothermia, hypotension, electrolyte imbalance, endocrine disorders and kidney failure.[2] This case reinforces the importance of considering disordered eating as a potential cause for electrolyte abnormalities and kidney failure.

CASE PRESENTATION

A 40-year-old woman presented to the renal outpatient clinic having had recurrent episodes of acute kidney injury (AKI) with associated hypercalcaemia. She had a longstanding history of eating disorder having suffered with bulimia for many years requiring multiple admissions for correction of electrolyte abnormalities and for treatment of AKI. Her primary electrolyte abnormalities have been hypercalcaemia, alkalosis, hypokalaemia, hypoalbuminaemia and low zinc levels leading to daily headaches, nausea, palpitations, abdominal cramps and diarrhoea. She denied any recent laxative or diuretic abuse. There is no other significant medical history.

INVESTIGATIONS

On examination, blood pressure was 102/72, weight 42 kg and body mass index 15.4. Jugular venous pressure at 1 cm, auscultation of the chest was clear. On examination of the abdomen, no loin or suprapubic tenderness was found, and with no palpable bladder.

Her blood tests and quantity of proteinuria have been variable; her most extreme results include: creatinine 54–362 umol/L depending on hydration status, bicarbonate 47 mmol/L, potassium 2.4 mmol/L, albumin 24 g/L and adjusted calcium 3.37 mmol/L.

On admission, her estimated glomerular filtration rate was 84 μmol/L, giving an epidermal growth factor receptor of approximately 52 mol/L (using Cockcroft-Gault equation).

Urine protein:creatinine ratio was 42.4 mg/mol.

Chest X-ray revealed normal-looking hilar areas and no obvious interstitial changes.

Renal ultrasound (US) showed normal-sized kidneys with increased cortical echogenicity and increased reflectivity of the pyramids with punctuate calcification of the medulla, and with no pelvicalyceal dilation.

Transvaginal US showed retroverted uterus, normal ovaries and no adnexal masses or free fluid. During her multiple admissions, she had normal thyroid-stimulating hormone (0.88 μ/L), normal cortisol (419 nmol/L), normal ACE (45 IU/L), normal parathyroid hormone (2.33 pmol/L), low 25-hydroxy vitamin D (49 nmol/L) and normal creatine kinase (69 U/L). Serum protein electrophoresis revealed no paraprotein band. Antinuclear antibody was negative.

DIFFERENTIAL DIAGNOSIS

- Myeloma
- Sarcoidosis
- Paraneoplastic disease
- Hyperthyroidism
- Hyperparathyroidism
- Eating disorder

TREATMENT

During her multiple admissions, the patient was managed in accordance with local guidelines with assessment of volume status and fluid resuscitation where appropriate. Intercurrent infection was excluded. Electrolyte supplementation was administered. When seen in the renal clinic, she was started on an ACE inhibitor (ACEi) and long-term oral potassium supplementation. She has been referred for eating disorder counselling.

OUTCOME AND FOLLOW-UP

The patient still struggles with eating disorder and has had two further admissions to hospital with dehydration since initiating long-term treatment in the renal clinic. Following the introduction of an ACEi and potassium supplementation, her potassium has been better controlled (2.9–5.2 mmol/L)

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and she has had fewer episodes of hypercalcaemia. She is followed up regularly in the renal clinic.

DISCUSSION

It is well documented that the prevalence of eating disorders is increasing in men and women[3] with considerable medical, social and functional burdens.[4] There are three postulated mechanisms of renal involvement in anorexia nervosa:

1. Chronic dehydration and hypokalaemia;
2. Nephrocalcinosis;
3. Chronic rhabdomyolysis.[2]

Chronic hypokalaemia results in renal tubular cell hyperplasia in association with tubular atrophy, interstitial macrophage infiltration and interstitial fibrosis. This hypokalaemic nephropathy occurs secondary to local ischaemia and vasoconstriction, intrarenal complement activation, local expression of angiotensin II and endothelin[5] and impaired angiogenesis.[6]

Nephrocalcinosis and renal impairment in patients suffering from anorexia nervosa/eating disorder are well described and have been seen in the context of hypocalcaemia and hypercalcaemia.[2 7–9] Proposed mechanisms for this include nephrocalcinosis as a result of chronic volume depletion[2] and progressive hypercalciuria and negative calcium balance as a result of phosphate depletion and bone resorption.[10] It has also been shown that aldosterone directly affects urinary calcium excretion,[11] which may represent a further predisposition for increased urinary calcium and nephrocalcinosis in these patients.

Rhabdomyolysis in association with eating disorder has been detailed in case reports[12] and is thought to be a consequence of hypokalaemia and deranged phosphate metabolism.

A number of electrolyte abnormalities have been reported in association with eating disorder including hypokalaemia, hyponatraemia, hypomagnesaemia, hypophosphataemia, hypocalcaemia and hypercalcaemia.[2 8 13] In our case and the two cases described by Roberts *et al*,[7] the patients were noted to have hypercalcaemia, which is unusual and is likely to be responsible for the nephrocalcinosis in these reports. Despite thorough screening for other causes of hypercalcaemia including hyperparathyroidism, myeloma, sarcoidosis, tuberculosis, thyrotoxicosis, Addison's and vitamin D intoxication, a precipitating cause could not be identified. A transvaginal US was also performed in our case to exclude ovarian malignancy, as rarely benign ovarian tumours have been associated with parathyroid-related peptide production and hypercalcaemia.[14] There have been a number of reports of hypercalcaemia associated with disordered eating as a result of dehydration, thiazide diuretic abuse and the milk-alkali syndrome,[15 16] where patients suffering from bulimia seem to be particularly vulnerable.[17] Our patient denied diuretic abuse, although her urine was not tested. She also denied over indulging in calcium-based foods. She has had several documented episodes of dehydration requiring fluid resuscitation.

Long-term management of these patients poses significant difficulty and a multidisciplinary approach is required to try and resolve the underlying eating disorder. There is emerging evidence that angiotensin-receptor blockade may ameliorate tubulointerstitial injury induced by chronic potassium deficiency, and is therefore a crucial part of management.[18] Given the direct effect of aldosterone on urinary calcium excretion, angiotensinogen blockade may have a further role in the preservation of renal function in this group.

This case highlights the importance of considering eating disorder in patients presenting with AKI and electrolyte derangement and the evidence base for angiotensin inhibition in the management of these patients. The unusual finding of hypercalcaemia in this case was thought to be related to her eating disorder, having excluded the rarer causes of hypercalcaemia.

Learning points

- The full spectrum of morbidity and severity of clinical manifestations associated with disordered eating is likely to be considerably higher than previously estimated, proving an increasing public health problem.
- There are three postulated mechanisms of renal involvement in anorexia nervosa: chronic dehydration and hypokalaemia, nephrocalcinosis and chronic rhabdomyolysis.
- There is evidence that angiotensin-receptor blockade may ameliorate tubulointerstitial injury induced by chronic potassium deficiency and may reduce urinary calcium excretion, and is therefore a crucial part of management.
- Hypercalcaemia is an unusual finding and other rarer causes should be excluded before attributing this to disordered eating.

Contributors Dr SHAL and BEMT have seen this patient in clinic and feel that this case offers an important message and therefore have submitted it as a case report. BEMT performed the literature search and wrote the case report. BEMT will act as guarantor for the finished article. Dr SHAL is responsible for the patient's overall management, as consultant in charge.

Competing interests None.

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REFERENCES

1 Swanson SA, Crow SJ, Le Grange D, *et al*. Prevalence and correlates of eating disorders in adolescents results from the national comorbidity survey. *Arch Gen Psychiatry* 2011;68:714–23.
2 Cavoli G, Rotolo M. Renal involvement in psychological eating disorders. *Nephron Clin Pract* 2011;119:c338–41.
3 Hay P, Mond J, Buttner P, *et al*. Eating disorder behaviors are increasing: findings from two sequential community surveys in South Australia. *PLoS ONE* 2008;3:e1541.
4 Wang L, Nichols L, Austin B. The economic effect of planet health on preventing bulimia nervosa. *JAMA Paediatr* 2011;165:756–62.
5 Suga SI, Phillips MI, Ray PE, *et al*. Hypokalemia induces renal injury and alterations in vasoactive mediators that favour salt sensitivity. *Am J Physiol Renal Physiol* 2001;281:F620–9.
6 Reungjui S, Roncal C, Sato W, *et al*. Hypokalemic nephropathy is associated with impaired angiogenesis. *JASN* 2008;19:125–34.
7 Roberts M, MacGregor D, Paoletti N, *et al*. Severe renal failure and nephrocalcinosis in anorexia nervosa. *Med J Aust* 2005;182:635.
8 McLaughlin A. Anorexia nervosa and senna misuse: nephrocalcinosis, digital clubbing and hypertrophic osteoarthropathy. *Med J Aust* 2008;189:348.
9 Fairburn C, Brownell K. *Eating disorders and obesity: a comprehensive handbook*. 2nd edn. The Guildford Press, 2002.
10 Haglin L. Hypophosphataemia in anorexia nervosa. *Postgrad Med J* 2001;77:305–11.
11 Ferrari P, Bianchetti M, Sansonnens A, *et al*. Modulation of renal calcium handling by 11β-hydroxysteroid dehydrogenase type 2. *JASN* 2002;13:2540–6.
12 Wada S, Nagase T, Koike Y, *et al*. A case of anorexia nervosa with acute renal failure induced by rhabdomyolysis; possible involvement of hypophosphatemia or phosphate depletion. *Intern Med* 1992;31:478–82.
13 Fonesca V, Havard C. Electrolyte disturbances and cardiac failure with hypomagnesaemia in anorexia nervosa. *BMJ* 1985;291:1680–92.
14 Jacobs T, Bilezikian J. Clinical review: rare causes of hypercalcemia. *J Clin Endocrinol Metab* 2005;90:6316.
15 Kallner G, Karlsson H. Recurrent factitious hypercalcemia. *Am J Med* 1987;82/3:0002–9343.
16 Muldowney WP, Mazbar SA. Rolaids-yogurt syndrome: a 1990s version of milk-alkali syndrome. *Am J Kidney Dis* 1996;27:270–2.
17 Medarov B. *Milk-Alkali Syndrome Mayo Clinic Proceedings*. vol. 84, Issue 3, March 2009, pp. 261–7.
18 Suga S, Mazzali M, Ray PE, *et al*. Angiotensin II type 1 receptor blockade ameliorates tubulointerstitial injury induced by chronic potassium deficiency. *Kidney Int* 2002;61:951–8.