CASE REPORT

Cardiac arrest: first presentation of anorexia nervosa

Sian-Lee Ewan,[1] Patricia C Moynihan[2]

[1]Department of Psychiatry, Northern Ireland Deanery, Downpatrick, UK
[2]Department of Psychiatry, Downshire Hospital, Downpatrick, UK

Correspondence to
Dr Sian-Lee Ewan, sianlee.ewan@gmail.com

SUMMARY

A 16-year-old girl collapsed in cardiac arrest in a hospital car park. Investigations revealed a potassium level of 1.8. Following a 5-day intensive care unit admission she described behaviours consistent with restrictive-purging type anorexia nervosa, which had been concealed from her parents and health professionals. Long-term management has been difficult due to poor patient engagement. Further, recurrent episodes of hypokalaemia continue to feature. Here we explore the cardiac complications of anorexia nervosa and challenges with long-term management of this condition.

BACKGROUND

Anorexia nervosa is a challenging condition to manage with significant medical complications, having a higher mortality rate than any other psychiatric disorder.[1] Up to 80% of patients with anorexia nervosa suffer from cardiovascular complications, and these account for one-third of related deaths.[2]

Recent studies have shown that cardiovascular complications are present in adolescents early in the disease process.[3] Therefore, it is important for clinicians to recognise the presenting signs, monitoring and management strategies.

CASE PRESENTATION

A cardiac arrest call was made to attend a 16-year-old girl in the car park of a hospital who had collapsed. Following cardiopulmonary resuscitation and two episodes of defibrillation, a cardiac output was restored. She was transferred to the intensive care unit (ICU) and investigations revealed hypokalaemia (K^+ 1.8) as the precipitating cause.

Following a 5-day ICU admission the patient was transferred to a medical ward where further assessment could be completed. She gave a 4-year history of food restriction, self-induced vomiting and excessive exercise. Specifically, she could purge on demand up to 30 times daily, and would perform 200–300 sit-ups, often until she collapsed. She weighed 38 kg, with a BMI of 14.8. A diagnosis of anorexia nervosa was made and the hypokalaemia was explained by frequent vomiting.

There were no obstetric issues with her birth. She grew up in the family home where she was the youngest of four siblings. At primary school she was noted to have a lisp and was less academically able than her siblings. She developed low self-esteem and accompanying perfectionism with difficulty with emotional expression. Her body image became more of a concern around age 11 when she wanted to be the 'skinniest prettiest Barbie'. At this point she began to engage in dietary restriction, self-induced vomiting and excessive exercise. Her parents had noticed that she had began to withdraw, but were unaware of these eating behaviours until her presentation to hospital in cardiac arrest.

INVESTIGATIONS

Investigations from her initial presentation with cardiac arrest:

- Full blood count: Hb 125, WCC 14.1, Plt 279
- Urea and electrolytes: Na 137, K 1.8, Cl 93, CO2 25, Urea 3.6, Cr 60
- Urinary electrolytes: Na 33, K 87, Urea 178. Urine osmolality 486.

Investigations for long-term monitoring:

- ECGs: often normal, but during episodes of hypokalaemia have shown increased QTc (up to 475 ms) which resolves upon electrolyte correction.
- Electrolyte monitoring: continues to experience frequent episodes of hypokalaemia requiring medical admission.
- Dual emission X-ray absorptiometry bone scan showed an increased risk of fractures to left hip and L1–L4 vertebrae.

DIFFERENTIAL DIAGNOSIS

Differential diagnosis for hypokalaemia:

1. Decreased potassium intake
   - Prolonged fasting
2. Gastrointestinal loss:
   - Vomiting (secondary to infection or self-induced)
   - Diarrhoea (secondary to infection or laxative/enema abuse)
   - Pancreatic fistulae
3. Renal loss:
   - Thiazide and loop diuretics
   - Renal tubular acidosis
   - Primary hyperaldosteronism
   - Cushings syndrome
   - Liddle's syndrome
   - Bartter's syndrome
   - Fanconi's syndrome
   - Magnesium deficiency
4. K^+ shift from extracellular fluid
   - Secondary to medications; insulin, β_2-agonists, catecholamines

OUTCOME AND FOLLOW-UP

This patient was discharged with community follow-up from the child and adolescent mental health services. Unfortunately, she continued to lose weight and 5 months later was admitted to an adolescent unit for 11 months. She received

To cite: Ewan S-L, Moynihan PC. *BMJ Case Rep* Published online: [*please include* Day Month Year] doi:10.1136/bcr-2013-200876

individual psychotherapy and family therapy. Decisions regarding pharmacological intervention were difficult as many antipsychotics carry a risk of QTc prolongation. However, as her weight increased, she was started on fluoxetine for low mood and olanzapine for agitation and anxiety.

Ten days after discharge she was readmitted with hypokalaemia (2.3 mmol/L) and subsequently was detained under the mental health order.

Over the next 2-year period she was admitted under psychiatry on five occasions due to low body weight and hypokalaemia, including a 14-month admission to a tertiary care hospital for specialist intervention. The patient self-discharged against medical advice on four of these occasions. Periods of discharge to the community were short lived, lasting from 5 days to 2 months. Further to this, she required seven medical admissions for intravenous management of hypokalaemia.

Therapeutic intervention has remained difficult due to limited engagement, resistance to treatment interventions and frequent absconding from the ward during periods of admission. For the past 11 months she has been managed in the community; this is the longest period out of hospital since her presentation with cardiac arrest. She continues to display the same behaviours of food restriction, vomiting and excessive exercise. Therefore, management has largely been focused on risk reduction. The risk of hypokalaemia continues and risk reduction is difficult due to the patient's inconsistency with compliance with sando-K. She would frequently miss doses, and then take an excessive dose when she developed symptoms of paraesthesiae.

Current management plan includes attending the day hospital once weekly for monitoring of her weight and electrolytes, weekly review with either her eating disorder therapist or keyworker and three-monthly review with her psychiatrist. Owing to difficulties with engagement, she continues to be managed under enhanced care planning.

DISCUSSION

It is well-established that electrolyte disturbances, namely low sodium, chloride and potassium, are associated with anorexia nervosa.[2] Potassium levels may be low secondary to use of laxatives, diuretics or as in this case, self-induced vomiting. Hypokalaemia has cardiac consequences due to the effect on repolarisation. This leads to characteristic ECG changes: flattened T waves, ST segment depression, prolonged QT interval and U waves which predispose to VF, torsades de pointes and a cardiac event. The presentation of non-traumatic cardiac arrest in a young woman must raise the suspicion of hypokalaemia. Further findings of hypokalaemia, and low urinary sodium and chloride should alert the clinician to the possibility of an eating disorder.

Cardiac complications are well described in anorexia nervosa and bulimia nervosa, but are rarely the presenting symptom. Published case reports include initial presentations with seizures,[4] episodes of collapse[5] or asthenia,[6] but this is the first to describe cardiac arrest as the first presentation of an eating disorder to services.

Cardiac complications associated with anorexia nervosa occur due to alterations in cardiac structure and rhythm. Structural changes include a decrease of left ventricular mass and reduced thickness of cardiac walls in patients with anorexia nervosa.[3] The most common rhythm change is bradycardia, which can lead to polymorphic VT. Hypokalaemia can lead to QT interval prolongation; however, the extent of QT interval prolongation in anorexia nervosa has been debated in the literature.

The predictive value of a prolonged QT interval for ventricular tachycardia or sudden death is poor.[2] A predictive link has only been shown for a substantially prolonged QT interval, >600 ms.[7] Studies have reported rates of QT interval prolongation in patients with anorexia nervosa as ranging from 0%[8] to 40%.[3] Where the study accounted for electrolyte disturbances, there was no evidence of QT interval prolongation, compared to studies which did not. This would suggest that QT interval is usually normal in anorexia, but can be increased during episodes of severe hypokalaemia.[8] The risk of severe arrhythmia could therefore be reduced by control of ion imbalances.

This highlights the importance of regularly monitoring electrolytes in patients with anorexia nervosa, particularly if they are engaging with behaviours that lead to hypokalaemia (eg, self-induced vomiting, laxative or diuretic abuse). There remains value in performing an ECG. An ECG may reveal changes in keeping with hypokalaemia while awaiting blood results, or show a QTc >600 ms which is a predictive factor for VT or sudden death.

The main cardiovascular effects on the heart caused by anorexia nervosa have been shown to be reversible by refeeding.[9] This includes bradycardia, left ventricular mass, cardiac output, heart rate variability and QT interval. Therefore efforts with risk reduction should be combined with therapy aimed towards refeeding in the management of anorexia nervosa.

Learning points

- In the non-traumatic cardiac arrest of a young woman, consider hypokalaemia secondary to an eating disorder.
- Perform regular routine monitoring of electrolytes in patients with anorexia nervosa.
- Monitor ECGs (QTc) in patients with anorexia nervosa.
- Cardiovascular changes in anorexia nervosa can be reversed by refeeding and therefore a integrated therapeutic approach should be implemented.

Contributors S-LE and PCM have contributed significantly towards the case report and approved the final version for publication.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

1 Harris EC, Barraclough B. Review. Excess mortality of mental disorder. *Br J Psychiatry* 1998;173:11–53.
2 Jauregui-Garrido B, Jauregui-Lobera I. Sudden death in eating disorders. *Vasc Health Risk Manag* 2012;8:91–8.
3 Vazquez M, Olivares JL, Fleta J, *et al*. Cardiac disorders in young women with anorexia nervosa. *Rev Esp Cardiol* 2003;56:669–73.
4 Bohmer T, Eriksen EF. Something to learn from-something to learn more from? *Tidsskr Nor Laegeforen* 2011;131:2221.
5 Buchanan R, Ngwira J, Amsha K. Prolonged QT interval in bulimia nervosa. *BMJ Case Rep* 2011;2011. Pii: bcr0120113780.
6 Macias-Robles MD, Perez-Clemente AM, Macia-Bobes CM, *et al*. Prolonged QT interval in a man with anorexia nervosa. *Int Arch Med* 2009;2:23.
7 Ravaldi C, Vannacci A, Ricca V. [Cardiac complications of anorexia nervosa]. *Recenti Prog Med* 2003;94:267–70.
8 Facchini M, Sala L, Malfatto G, *et al*. Low-K+ dependent QT prolongation and risk for ventricular arrhythmia in anorexia nervosa. *Int J Cardiol* 2006;106:170–6.
9 Mont L, Castro J, Herreros B, *et al*. Reversibility of cardiac abnormalities in adolescents with anorexia nervosa after weight recovery. *J Am Acad Child Adolesc Psychiatry* 2003;42:808–13.