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Case Report

Re-feeding syndrome and alcoholic cardiomyopathy: A case of interacting diagnoses

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ABSTRACT

Re-feeding syndrome is an uncommon clinical entity of fluid and electrolyte disorders that typically occurs after re-initiation of enteral nutrition following prolonged fasting. This disorder can be complicated by left ventricular (LV) dysfunction, arrhythmias, and death. Alcohol abuse and anorexia nervosa are independently associated with similar complications. The interaction between these diagnoses can result in significant, but reversible, LV dysfunction. We present the case of a 69-year-old woman with a history of significant alcohol abuse and anorexia nervosa. The patient was admitted to hospital for the management of re-feeding syndrome, which was complicated by significant LV dysfunction. Her LV function normalized following a combination of electrolyte replacement, re-institution of feeding, and abstinence from alcohol. Re-feeding syndrome, anorexia nervosa, and alcohol abuse are conditions that commonly co-exist. These conditions may have a synergistic relationship, potentially resulting in a profound cardiomyopathy. Careful monitoring and aggressive electrolyte replacement may be helpful in identifying this complication and minimizing its potential harm.

<**Learning objective:** Re-feeding syndrome can be complicated by significant myocardial dysfunction, particularly in patients with a history of alcohol abuse or anorexia nervosa, which independently cause cardiac dysfunction. Physicians should be aware of the risk of new cardiomyopathy in patients with these overlapping diagnoses. We review the case of a patient with these conditions who developed a significant reversible cardiomyopathy managed with re-institution of feeding and electrolyte replacement.>

Introduction

Anorexia nervosa (AN) is a common cause of protein-calorie malnutrition with several potential cardiovascular complications including brady-arrhythmias, hypotension, and mitral valve prolapse [1]. The development of left ventricular (LV) dysfunction is relatively uncommon in patients with isolated AN [2], although reduced LV dimensions have been observed [3]. In addition to cardiovascular complications, patients with AN are prone to development of metabolic disturbances, collectively referred to as re-feeding syndrome, with re-initiation of enteral nutrition [4]. This syndrome typically occurs within 4 days of feeding and is characterized by cardiac, neurologic, pulmonary, neuromuscular, and hematologic effects. The cardiac effects of re-feeding syndrome, which are primarily attributed to hypophosphatemia and hypokalemia, may include arrhythmias, hypotension, LV dysfunction, and sudden cardiac death [4].

Chronic alcohol abuse increases risk of developing re-feeding syndrome, and is independently associated with hypophosphatemia and hypokalemia [4]. The co-existence of re-feeding syndrome and chronic alcohol abuse may have a potential negative synergistic relationship that enhances the risk and severity of cardiac complications, such as significant LV dysfunction, due to the abnormal metabolic milieu [4]. However, correction of the electrolyte abnormalities may result in reversibility [4]. We describe the case of a patient with re-feeding syndrome and chronic alcohol abuse, who developed a profound, but reversible, cardiomyopathy.

Case report

A 59-year-old woman was brought to the emergency department by her daughter for concerns regarding significant weight loss and immobility. The patient had been restricting her food intake for several weeks, resulting in a 10 kg weight loss over that

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same period. Her weight upon presentation to the emergency department was 38 kg with a body mass index of 14 kg/m^2. Additionally, she had been unable to get out of bed for approximately 4 weeks with several recent falls. The current situation had been triggered by a recent divorce with her husband, with significant decline in her mood, inability to sleep, and decreased appetite.

Her past medical history was significant for AN, depression, and chronic macrocytic anemia. The patient's social history was significant for alcohol abuse with a 30-year history of drinking 6–7 ounces of rum per day. Prior to her current illness, the patient had no significant functional limitations or other cardiac symptoms. She did not endorse recent infectious symptoms, exposures to cardiotoxic drugs, and denied a family history of cardiomyopathy. The patient had no previous cardiac investigations.

The patient's initial laboratory investigations are outlined in Table 1. These investigations were significant for a potassium level of 2.6 mmol/L, a bicarbonate of 32 mmol/L, magnesium of 0.34 mmol/L, and phosphate 0.84 mmol/L. The patient's alcohol level was not detectable.

The patient was initially admitted to the hospital for re-institution of enteral nutrition. Due to persistent tachycardia an electrocardiogram (ECG) and transthoracic echocardiogram (TTE) were ordered. The ECG showed sinus tachycardia without other significant changes. The TTE, shown in Fig. 1 and Supplemental Video 1, was performed 2 days following admission and showed that the LV systolic function was moderately reduced with a LV ejection fraction of approximately 30–40%. The right ventricular (RV) size and function were grossly normal, and there was no significant valve disease. The LV wall thickness was small with intraventricular septal thickness of 0.57 cm (normal 0.6–1.0 cm). At that time, cardiology was consulted regarding the etiology of her LV dysfunction.

Supplementary Video 1 related to this article can be found, in the online version, at http://dx.doi.org/10.1016/j.jccase.2016.04.006.

Her physical examination at time of cardiology consultation revealed a heart rate of 133 bpm, respiratory rate 18 breaths per minute, blood pressure 133/85 mmHg, and oxygen saturation of 94% on room air. Auscultation of the chest revealed crackles at the bases bilaterally. Jugular venous pressure was elevated at 5 cm above sternal angle with positive abdominal jugular reflux. Auscultation of the heart revealed normal first and second heart sounds, with the presence of a third heart sound. Repeat laboratory investigations at that time are outlined in Table 1. Of note, there was normal thyroid stimulating hormone, elevated transferrin saturation, and significantly reduced phosphate at 0.30 mmol/L, which was consistent with the development of re-feeding syndrome.

At that time, it was felt that there were several potential causes of the patients LV dysfunction. The leading diagnoses were alcoholic cardiomyopathy, re-feeding syndrome, Takotsubo cardiomyopathy, hemochromatosis, or other cause of pre-existing cardiac dysfunction with new symptoms related to increased intake. Cardiac magnetic resonance imaging was arranged to help

Table 1 Laboratory results throughout the course of the hospitalization. – indicates no lab value obtained on that particular day.

	Normal values	Day 0	Day 2	Day 5	Day 8
Hemoglobin (g/L)	123–157	119	108	106	102
Mean corpuscular volume (fL)	80–100	107	109	108	108
Sodium (mmol/L)	135–145	137	142	138	139
Potassium (mmol/L)	3.5–5.0	2.6	4.8	4.4	4.8
Bicarbonate (mmol/L)	24–30	32	26	28	30
Magnesium (mmol/L)	0.75–0.95	0.34	0.89	0.63	0.60
Phosphate (mmol/L)	0.80–1.50	0.84	–	0.30	1.41
Calcium (mmol/L)	2.18–2.58	1.85	–	2.18	2.30
Creatinine (μmol/L)	50–90	38	38	46	37
Random glucose (mmol/L)	3.8–11.1	4.8	5.5	6.0	5.7

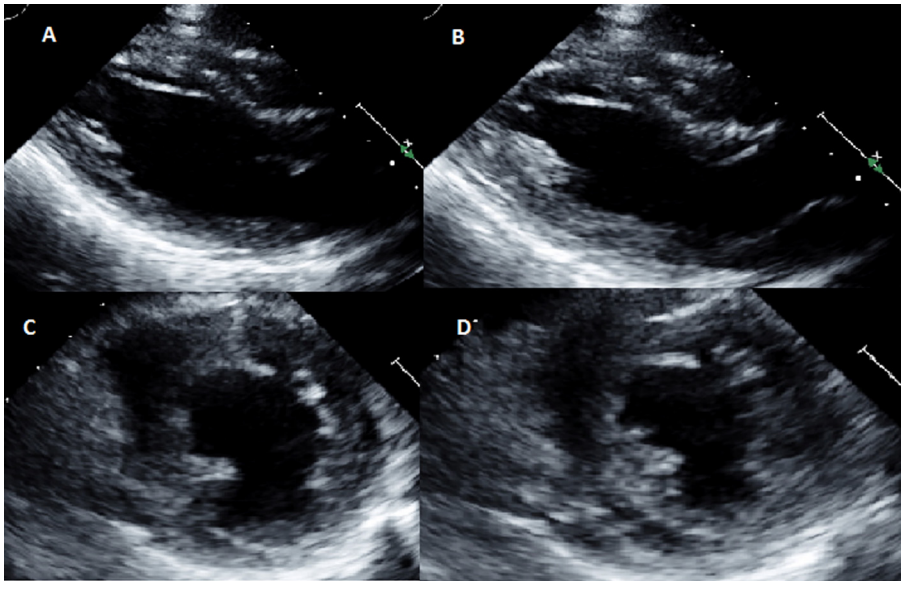

Fig. 1. Initial transthoracic echocardiogram showing moderate left ventricular dysfunction. Para-sternal long-axis view during end-diastole (A) and end-systole (B) and para-sternal short-axis views during end-diastole (C) and end-systole (D).

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