

A Case of Anorexia Nervosa with Cardiac Arrest Managed With a Multidisciplinary Approach That Included Anabolic Steroid Treatment

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Abstract: The patient, a 54-year-old woman who could independently carry out her activities of daily living, was found in an unconscious state at home by her mother. She had suffered from malnutrition since she was a teenager. When emergency medical technicians checked her, she was in a state of cardiopulmonary arrest; thus, they performed basic cardiopulmonary resuscitation. Spontaneous circulation was obtained after one cycle of chest compression. When she was examined by medical personnel who were dispatched by a physician-staffed helicopter, she was in a hypoglycemic state (glucose level of 10 mg/dl). Glucose infusion was administered after securing a venous route. On arrival, her height was 157 cm, and her weight was 28 kg, which corresponded to a body mass index of 13. She was diagnosed with anorexia nervosa with severe malnutrition, hypoxic and hypoglycemic encephalopathy, Takotsubo cardiomyopathy, and liver dysfunction due to kwashiorkor after examinations. The post-admission course was eventful and treated by multidisciplinary treatment that included the administration of anabolic steroids. Her body weight eventually increased to 33 kg, and her anabolic steroid dose was tapered. She was transported to a local medical facility for rehabilitation and mental support on the 91st hospital day. Multidisciplinary treatment including the administration of anabolic steroids may help improve a critically ill condition in patients with anorexia nervosa.

Keywords: anorexia nervosa; cardiac arrest; anabolic steroids.

INTRODUCTION

Anorexia nervosa (AN) is a psychiatric disorder that can result in life-threatening medical complications and multifaceted endocrine abnormalities [1]. The treatment of AN is difficult and requires a multidisciplinary efforts. However, the overall prognosis is quite poor, with only 40–50% of AN patients progressing to a complete recovery [1]. In some cases, AN has a fatal outcome due to multiple reasons[2]. We herein report the case of a severely malnourished middle-aged woman who was suffering from long-standing AN and who was hospitalized with hypoglycemia and cardiac arrest.

CASE REPORT

The patient, a 54-year-old woman whose activity of daily living was independent, was found in an unconscious state at home by her mother. She had suffered from malnutrition since she was a teenager. When emergency medical technicians checked her, she was in cardiopulmonary arrest; thus, they performed basic cardiopulmonary resuscitation. Spontaneous circulation was obtained after one cycle of chest compression. When she was examined by medical personnel who were dispatched by a physician staffed helicopter, she was in a hypoglycemic state (glucose level of 10mg/dl). A glucose infusion was administered

after securing a venous route. On arrival, her Glasgow Coma Scale level was E2V1M4. A physical examination revealed the following findings: blood pressure, 106/72 mmHg; heart rate, 70 beats per minute; a respiratory level, 16 breaths per minute; and an SpO₂ of 100% under an oxygen mask with 10 L/min of oxygen. Her height was 157 cm and her weight was 28 kg, which corresponded to a body mass index of 13. An arterial gas analysis (FiO₂ 1.0) revealed the following: pH, 7.50; PCO₂, 43 mmHg; PO₂, 379 mmHg, HCO₃⁻, 33.3 mmol/l; base excess, 9.8 mmol/l; and lactate, 0.8 mmol/l. The main results of a biochemical analysis of the blood were as follows: white blood cell count, 5,800/μl; hemoglobin, 11.8 g/dl; platelet count, 15.0×10⁴/μl; total protein, 5.0 g/dl; albumin, 0.8 g/dl; glucose, 128 mg/dl; total bilirubin, 1.0 mg/dl; aspartate aminotransferase, 225 IU/L; alanine aminotransferase, 110 IU/L; blood urea nitrogen, 49.7 mg/dl; creatinine, 0.56 mg/dl; creatinine phosphokinase, 367 IU/l; amylase, 334 IU/l; sodium, 131mEq/l; potassium, 3.5 mEq/l; chloride, 92 mEq/l; calcium, 7.5 mg/dl; phosphate, 5.2 mg/dl; c-reactive protein level, 0.3 mg/dl; activated partial thromboplastin time, 29.7 (26.0); prothrombin time, 18.6 (11.8) s; fibrinogen, 236 mg/dl; and fibrin degradation products, 9.4 μg/m. Chest roentgenography was negative and electrocardiography showed incomplete right bundle branch block. Cardiac

echo showed dysfunction of the apical portion of the left ventricle with compensatory hyperkinesis of the basal walls producing ballooning of the apex with systole. Whole body computed tomography revealed brain atrophy and a lack of fatty tissue. She was diagnosed with anorexia nervosa with severe malnutrition, hypoxic and hypoglycemic encephalopathy, Takotsubo cardiomyopathy, and liver dysfunction due to kwashiorkor. She underwent nasogastric tube feeding with pulse intravenous alimentation. Furthermore, anabolic steroids were administered with minerals including potassium, magnesium and phosphate. The following day, she regained consciousness; however, she could not eat. On the 6th hospital day, she developed lung edema and pneumonia. Pressure-assisted ventilation did not improve her respiratory dysfunction; thus, tracheal intubation was performed followed by mechanical ventilation with the administration of antibiotics and a diuretic. On the 12th hospital day, her respiratory dysfunction improved and she was extubated. However, she could not expel sputum; thus, tracheal intubation was reperfomed and mechanical ventilation was reinitiated on the same day. On the 16th hospital day, tracheostomy was performed and mechanical ventilation with sedation was withdrawn on the following day. On the 18th hospital day, she had an appetite and could eat food; however, she required assistance because of complicated tetraparesis due to disuse atrophy and/or critical illness polyneuropathy and myopathy. On the 28th hospital day, her respiratory function deteriorated when her tracheal tube was exchanged due to the aspiration of supraglottic secretion; thus, oral intake was temporally stopped for three days. On the 40th hospital day, after switching to a speech cannula to secure the airway, she developed pneumonia again and oral intake was temporally stopped. Oral intake was restarted again on the 46th hospital day, after the improvement of her aspiration pneumonia. The improvement of her cardiac motion was confirmed by ultrasound. With the improvement of her extremity muscle power through rehabilitation, she became able to feed and expel sputum by herself. On the 70th hospital day, the tracheal tube was removed; she did not develop pneumonia. Her body weight had increased to 33 kg and her anabolic steroid dose was tapered. She could walk unassisted. She was transported to a local medical facility for rehabilitation and mental support on the 91st hospital day.

DISCUSSION

This is the first reported case in which a patient showed a complete recovery from hypoglycemic cardiac arrest and multiple complications, including Takotsubo cardiomyopathy, liver dysfunction, lung edema, pneumonia and tetraparesis, with multidisciplinary treatment that included the administration of anabolic steroids. We focused on anabolic steroid treatment.

Anabolic steroids have two main effects: namely, androgenic and anabolic [3]. The androgenic effects are achieved by the modulation of the androgen receptor expression, while the anabolic effects are achieved by interference with the glucocorticoid receptor expression, which results in an anticatabolic effect. The most common androgen is testosterone, which exerts its effects by directly binding with the androgen receptors. Many synthetic or designer anabolic steroids modify the testosterone structure to maximize their anabolic properties. Such changes enhance the protein anabolic effects and thus may be useful as therapeutic options for the restoration of fat-free muscle mass and improvement in strength in patients with chronic illnesses or conditions such as critical illness-related myopathy. These effects might have helped improve the critically ill condition of the present patient.

Miller previously reported the results of 33 randomized women with AN who were treated with transdermal testosterone [4]. At baseline, the free testosterone level was correlated with the L4 bone density, body mass index, depressive symptoms, and spatial cognition. The C-terminal propeptide type 1 collagen levels during testosterone treatment were higher than those in patients who received a placebo. The depressive state of patients receiving testosterone improved from severe to moderate depression. The improvement of the mental state may improve appetite, similar to the present case.

CONCLUSION

Multidisciplinary treatment that included the administration of anabolic steroids may help improve a critically ill condition in patients with anorexia nervosa.

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