A case of refeeding syndrome in pregnancy with anorexia nervosa

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Abstract

Refeeding syndrome very rarely develops during pregnancy. A 35-year-old primiparous woman pregnant with twins complained of severe fatigue at 19 weeks' gestation. She was admitted to our hospital in a malnourished condition because of repeated self-induced vomiting due to anorexia nervosa. Just after hospitalization, she voluntarily increased her caloric intake significantly above the recommended prescribed diet, without medical permission. Nine days later, she developed refeeding syndrome. Electrolyte replacement and calorie restriction were started and her condition gradually improved. The healthy twin babies were born by cesarean section at 36 weeks' gestation. Acute increases in caloric intake by previously malnourished pregnant women with anorexia nervosa may induce refeeding syndrome. Women with the binge eating/purging subtype of anorexia nervosa may be at additional risk due to alternating phases of starvation and overeating.

Keywords

Anorexia nervosa, high-risk pregnancy, nutrition, refeeding syndrome

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Introduction

Refeeding syndrome (RFS) is a potentially lethal condition that can lead to heart failure, cardiac arrest, respiratory failure, and encephalopathy. RFS occurs as a result of the acute shift in fluids and electrolytes caused by rapid refeeding after a prolonged period of starvation.¹ The mechanism of RFS is that during refeeding, glycogen, fat, and protein synthesis are stimulated by insulin which was activated by glycemia.² This process needs minerals such as phosphate and magnesium and cofactors including thiamine. Insulin stimulates the absorption of potassium and glucose into the cells. Intracellular migration of magnesium and phosphate also occurs. This process results in a decrease in the serum levels of phosphate, potassium, and magnesium, all of which are already depleted. Particularly, phosphate plays a key role in several biological processes affecting every physiological system.³

There are only two prior reports of RFS during pregnancy, both caused by severe hyperemesis; however, neither of these papers reported neonatal or obstetric outcomes in detail.^{4,5} Therefore, the obstetric outcomes following the development of RFS during pregnancy remain unclear. We present a rare case of a pregnant woman with anorexia nervosa who developed RFS.

Case

A 35-year-old nulliparous woman conceived dichorionic, diamniotic twins after gonadotropin therapy. She had been irregularly undergoing non-pharmacological psychiatric treatment for anorexia nervosa since the age of 24. Before pregnancy, her body weight and body mass index (BMI) were 50 kg and 19.0 kg/m², respectively. At 19 weeks of gestation, she complained of general fatigue and weakness and was admitted to our hospital. Her body weight and BMI were 48.0 kg and 18.3 kg/m², respectively. Before hospitalization, she had experienced repeated self-induced vomiting. At admission, urinalysis showed trace proteinuria and no ketones. The blood tests showed mild hypoalbuminemia (31 g/L) but severe hypokalemia (1.6 mmol/L) and hypochloremia (70 mmol/L), and hypophosphatemia (0.55 mmol/L). The electrocardiogram revealed ST segment depression only. She was carefully started on a treatment program, comprising peripheral intravenous fluid infusion with replacement of electrolytes and vitamin B complex

including thiamine as well as prescribed oral intake of food. On the second day of admission, she was noted to have hypophosphatemia (0.26 mmol/L).

By the ninth day of admission, her electrolyte imbalance had improved. However, her body weight and BMI had rapidly increased to 63.8 kg and 24.3 kg/m², respectively. She developed severe generalized edema and hypoalbuminemia (18 g/L). It was subsequently discovered that she had been overeating by bingeing on the food brought in by her family, in addition to the prescribed diet. Management was with restriction of calorie intake according to the following protocol: initially, the patient was administered 1000 kcal/day, and calories were increased by 500 kcal/10 days to the maximum of 2000 kcal/day. The changes in body weight, electrolytes, and albumin during the hospitalization are shown in Figure 1. The body weight gradually decreased, and the edema subsided. Other complications such as heart failure did not occur. By the 25th day, her body weight and BMI had decreased to 59.8 kg and 22.8 kg/m², respectively. After she was discharged home, she was administered 2000 kcal/day and was required to keep a record of daily dietary intake. She was followed up by her obstetrician and psychiatrist every two weeks. We confirmed daily calorie intake, body weight, fetal condition, and her mental condition.

At 36 weeks of gestation, she underwent cesarean section after presenting in spontaneous labor. The first twin weighed 2140 g (−1.3 SD, compared with Japanese population data) at birth and had Apgar scores of 8 and 10 at 1 and 5 min, respectively. The second twin weighed 2506 g (−0.1 SD) at birth and had Apgar scores of 8 and 9 at 1 and 5 min, respectively. There were no postpartum complications, and her anorexia nervosa improved under psychiatric treatment.

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Figure 1. Change in body weight (●), phosphorus (■), and albumin (▲) during the hospitalization. P: phosphorus (normal range: 0.74–1.45 mmol/L); Alb: albumin (normal range: 38–52 g/L)

Discussion

To date, RFS has been recorded as a complication of severe hyperemesis in two pregnant women.^{4,5} As with our case, both of these previous reports' patient involved repeated vomiting. One patient was admitted to the intensive care unit for coma.⁴ Both cases resulted in preterm delivery and one case necessitated cesarean section.^{4,5} Our case was associated with selective intrauterine growth restriction of twin 1. RFS causes the cardinal features of hypophosphatemia and thiamine deficiency. It is unclear whether these maternal conditions adversely affect fetus. However, phosphate is transferred across the placenta for fetal skeletal mineralization.⁶ Therefore, it is theorized that prolonged hypophosphatemia might affect fetal growth. Also, chronic thiamine deficiency generally induces Wernicke's encephalopathy in adults, and so it is assumed to have some adverse effect on the fetus. Thus, we should carefully treat RFS during pregnancy to ensure the well-being of both the mother and the fetus. The trigger of RFS onset in all previous cases was the rapid increase in calorie intake following commencement of nasogastric feeding and total parenteral nutrition.^{4,5} Our case is the first reported to have developed RFS from spontaneous overeating. This case demonstrates that malnourished pregnant women with eating disorders, such as anorexia nervosa, are at risk of RFS during enforced feeding or during binge eating episodes.

To prevent the onset of RFS, awareness and proactive management are very important. The National Institute for Health and Clinical Excellence published that risk factors for RFS are a useful tool for identifying patients at risk of RFS.² Once RFS occurs, careful treatment, including calorie control, is essential. Most pregnant women have greater basal calorie intake than do non-pregnant women; therefore, the appropriate calorie restriction level to treat RFS in pregnant women is unclear. In the present case, our patient started refeeding 1000 kcal/day based on the recommendation of one report.⁷ However, another study recently suggested that refeeding with higher calories (≥ 1400 kcal/day) may be appropriate under careful medical monitoring with electrolyte correction.⁸ In addition, high doses of multivitamins for pregnant women with RFS have been shown to be

appropriate because of increased requirements for vitamins during pregnancy.¹ Further studies are needed to investigate the best treatment for pregnant women with RFS.

In conclusion, pregnant women who have experienced prolonged malnourishment induced by condition such as anorexia nervosa are at risk for RFS. RFS can be prevented by identification of at risk patients and careful reintroduction of a calorie-controlled diet under medical and dietetic supervision. Once RFS has developed, appropriate treatment, including electrolyte infusion and careful nutrition support, is essential.

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Ethical approval

Written consent was obtained from patient for publication. This study was approved by the Institutional Review Board of the National Defense Medical College.

Guarantor

H Soyama

Contributorship

H Soyama and MM designed the study. Case summary by TN and MM, HS wrote the first draft of the article. H Sasa and MT conducted critical revision of the manuscript. KF supervised all processes of this study. All authors accepted responsibility for the paper as published.

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