Diabetic with Lipodystrophy

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Abstract

Background: 18 yr-old Canadian female soccer player presented to pre-participation examination with diagnosed Type 2 Diabetes secondary to Congenital Generalized Lipodystrophy. Athlete stated she was diagnosed with Diabetes in July and Lipodystrophy at 6 months of age. Her medical records indicated at her 4-year old appointment, her insulin levels at the time were elevated at 116; and presented with acanthosis nigricans in her neck and axillac. At her 5-year old check-up, she was diagnosed with hypertrophic cardiomyopathy with trace tricuspid regurg and pulmonary insufficiency. The following year, her triglyceride levels were elevated at 2.58 and her cholesterol/HDL ratio was elevated at 4.2. At age 7, she reported with polydipsia and polyuria. Her insulin levels increased to 203 and her triglycerides levels remained elevated. She also was diagnosed with normocytic anemia with decreased levels of hematocrit; presentation of acromegaloid on the head and neck, muscular hypertrophy and phlebomegaly. At the age of 8, she showed mutations in her genetic testing of AGPAT2 gene associated with Type 1 lipodystrophy. Blood work showed increased insulin to 264. At the age of 9, she had surgery for an umbilical hernia repair. At 10 years old, she presented with venous prominence. Her bone age reports 12 years old at the age of 10. Her blood work showed her glucose levels to be 5.2 and her triglycerides level at 1.58 Athlete was discharged from the clinic at age 11. Her recent blood work for glucose screening revealed her hemoglobin levels low at 116 leading to her hematocrit levels to be low at 0.35. Her glucose was elevated at 28 and her CK was elevated 543. Her triglyceride level was high at 3.88 and her cholesterol was 5.06. Her doctor prescribed her metformin at 500mg and lantus at 22 units and Humalog at 10-12 units. **Differential Diagnosis:** Congenital Generalized Lipodystrophy with Type 2 diabetes, Centrifugal lipodystrophy, prediabetic **Treatment:** She consulted with team nutritionist and developed a diabetic diet for athletes. She was given a meal plan consisting of 6 meals a day with 100-120 g of protein and 85g of carbohydrates a day. Her glucose level should be 8-10 after meals. She is to log her food every week and report back to the nutritionist for the first month to make sure she is on track. Once a pattern is set with her eating habits, she will only log her food on game days. After a month of just game day logs, if she feels comfortable with her nutrition habits, she does not need to log her food. **Uniqueness**: Congenital Generalized Lipodystrophy is an inherited autosomal recessive disorder. This disorder is characterized with lack of fatty tissue leading to fat storage in other parts of the body. Abnormal fat storage can lead to variety of medical concerns including high levels of fats circulating in the body and insulin resistance. Athlete's doctors report she has been doing much better with management and progression than most individuals with lipodystrophy. **Conclusion**: Athletes with diabetes have to keep in mind the importance of glucose levels due to the consequences of hyperglycemic episodes and other factors leading to diabetic coma if levels are not within normal limits. Also, learning about lipodystrophy is important due to one of the correlating factors being hypertrophic cardiomyopathy, the most leading cause of cardiac death in youngathletes.

Introduction

Congenital generalized lipodystrophy (also called Berardinelli-Seip congenital lipodystrophy) is a rare case identified by a relatively complete absence of fatty tissue in the body and a very muscular body figure. Fatty tissue, also known as adipose tissue is located in various regions of the body, including underneath the skin and covering the internal organs. Adipose tissue is responsible for storing fat for the use of energy and also contributes cushioning for internal structures. A lack of adipose tissue provokes the storage of fat to move into the liver and muscles, which can lead to serious health issues. Mutations in the *AGPAT2*, *BSCL2*, *CAV1*, and *PTRF* genes produce congenital generalized lipodystrophy. The proteins created from these genes are significant in the production and activity of adipocytes. Adipocytes act as the fat-storing cells in adipose tissue. Mutations in any of these genes decrease or remove the activity of their proteins. Lack of these proteins causes damage the production, structure, or activity of adipocytes and causes the body to unsuccessfully store and use fats properly. These abnormalities of adipose tissue interrupt hormones and alter many of the body's organs, leading to the various signs and symptoms of congenital generalized lipodystrophy.

The signs and symptoms of congenital generalized lipodystrophy are generally noticeable at birth or early childhood. Due to the lack of adipose tissue and exceeded muscular tissue, individuals appear very muscular. Veins present prominent due to the lack of adipose tissue underneath the skin. Those who are affected by lipodystrophy have a large chin, prominent orbital ridges, large hands and feet, and a prominent umbilicus. One of the most common factors is insulin resistance. Insulin resistance occurs when the body's tissues are incapable of recognizing insulin. Insulin is a hormone that is needed to help regulate blood sugar levels. Insulin resistance can lead to a more serious disease called diabetes mellitus if not treated properly.

Case Report Background

Subject: female soccer player with diabetes secondary to lipodystrophy
Design: one study case study Statistical analysis: Congenital generalized lipodystrophy has an estimated predominance of 1 in 10 million people around the world. It turns out to be more prevalent in certain regions of Lebanon and Brazil. Screening: complete blood count, physical appearance, serum concentrations of electrolytes, insulin, creatinine, triglycerides, cholesterol, echocardiogram, skeletal survey, renal & liver ultrasound. Nutritional log: proteins, carbohydrates, fats, cholesterol, chromium.
Carbohydrates are important to athletes because they are the only macronutrient that can be metabolized anaerobically, meaning they are the "master fuel" of the body. They are important nutrient for nervous system as well because carbohydrates are used as the primary energy source. An adequate consumption of carbohydrates helps to spare muscle tissue. Carbohydrates must be present in order to metabolize fats. Low intake of carbohydrates leads to break down of muscle tissue.
Proteins provide the structure of muscles and tissues, regulate cell functions, assists in maintaining fluid acid-base, transportation of fluids, and serve as an energy. Athletes should strive for a positive nitrogen balance. A nitrogen balance is when a greater amount of protein is being assimilated greater than they are being broken down. Athletes should stick to consuming high quality complete proteins. These complete proteins supply all essential amino acids.
The mineral Chromium has an impact on managing Type 2 Diabetes and insulin sensitivity. Its major function is to improve the action of insulin by increasing receptors. Foods rich in Chromium are mushrooms, prunes, nuts, whole grains, yeast, broccoli, wine, cheese, egg yolks, asparagus and some dark chocolate. A deficiency in chromium can lead to alterations in carbohydrate and protein metabolism. This alteration can lead to decrease endurance performance and the body's ability to build and repair muscle. Chromium deficiency leads to high blood glucose, which overtime leads to type 2 diabetes and lipid abnormalities. However, too much chromium can lead to interference of iron and zinc absorption.

Method 1: A reduction on the amount of food you consume may also be beneficial by consulting with a dietitian to arrange your ideal meal plan. Athletes are to consume 55-70% carbohydrates of total calories. About 4-24 hours prior to activity, an athlete is recommended to consume foods high in carbohydrates (60-70%) About 0-4 hours before activity, foods should be in the low glycemic level and easily digested. Athletes should consume approximately 1-4.5 grams of carbohydrates. During exercise, carbohydrate consumption has been reported to delay fatigue in both short- and long duration activity. Athletes should consume 30-90 grams per hour of activity. After activity, an athlete's primary focus should be replenishment of muscle and liver glycogen stores. Consumption of carbohydrates should be within 2 hours as soon as activity is completed and every 2 hours of 1.2 grams per kilogram of bodyweight for 6 hours. Athletes are not recommended to consume protein before activity, however, if protein should be consumed it should be within 3 hours before activity. Consumption during activity should be primary focused on carbohydrates rather than protein. After activity is when protein consumption is important. Athletes should consume 6-20 grams of protein in post exercise meal within 3 hours. Adding a good amount of carbohydrates with protein intake is highly recommended for recovery due to its insulin response. Method 2: keeping your total fat intake between 20% and 30% of total dietary energy controls normal triglyceride serum concentration. Most lipodystrophy individuals also have high amounts of fats called triglycerides streaming in the bloodstream. congenital generalized lipodystrophy causes an abnormal accumulation of fats in the liver called hepatic steatosis. This fatty liver can develop into an enlarged liver called hepatomegaly and further leads to liver failure. Method 3: Physical activity is one of the primary treatments for lowering blood glucose levels in type 2 diabetes. Mild- moderate intensity exercise decreased blood glucose levels and this affect is continued into the post-exercise period The American College of Sports Medicine (ACSM) recommend aerobic exercises at 50-85% of the maximal volume of oxygen that can be consumed for promoting cardiorespiratory fitness and one set of eight to twelve repetitions using eight to ten resistance exercises for developing muscular fitness and strength in healthy adults. ACSM and ADA recommend aerobic exercise at an intensity of 40-70% pf VO2 and anaerobic exercise for a minimum of one set of ten to fifteen repetitions using eight to ten resistance exercise for most patients with type 2 diabetes.

Results

Congenital lipodystrophy has no effect on her soccer training or physical activity. She meets with the nutritionist on site to set up a meal plan to manage her diabetes. She was advised to add more protein into her diet to slow down her carbohydrate metabolism. She eats 85 grams of carbohydrates and 100-120 grams of protein everyday. Her blood glucose levels should be at 7mg in the morning and 8-10mg after each meal. [8] Her food preferences for healthy grains are whole grain bread, whole grain cereal, and fig bars. She enjoys getting her fruits in with fresh and dried products. She also enjoys yogurt and milk. Due to her increase in protein intake, she is starting to use protein supplements to help achieve her required consumption. Once she is familiar with this amount of intake, we will start eliminating supplementation and add in high quality complete whole food proteins. . Student athlete states that she is lactose intolerant so she was recommended to drink lactose free milk with her cereal instead of almond milk. She is also slightly over the carbohydrate percentage recommended by her nutritionist. She discussed with her nutritionist eating less cereal and bagels almost everyday and increasing her protein intake by eating more chicken and other meats. She is low in Vitamin C, D, E, and K. She was advised to add more fruits and vegetables.

Nutrient	Consumption	Target
Carbohydrates	343 g	340 g
Protein	266 g	400-420 g
Potassium	1838 mg	4700 mg
Calcium	563 mg	1000 mg
Vitamin C	53 mg	75 mg
Vitamin D	6 ug	15 ug
Vitamin K	72 ug	90 ug
Vitamin E	5 mg AT	15 mg AT

Discussion and Summary

Lipodystrophy is a very rare congenital condition and has a lot of serious correlating factors. Diabetes is one of the most common factors of lipodystrophy. Diabetes is a abnormal rise in blood glucose levels that is also called hyperglycemia. Type 2 diabetics do not use insulin properly leading to insulin resistance. The worst of the factors is hypertrophic cardiomyopathy, which the myocardium of the heart is abnormally thick. Hypertrophic cardiomyopathy is the number 1 leading cause of sudden death of young athletes. As you can see with the case study, lipodystrophy is not a ineligible condition in sports but the relating factors can lead to serious conditions leading to ineligibly

Other possible treatments for lipodystrophy are leptin treatment has been proven profitable in managing hypertriglyceridemia and diabetes mellitus. In lipodystrophy, the leptin levels and cellular leptin sensitivity is reverse. In lipodystrophy, leptin levels are low and cells are susceptible to it; therefore, leptin replacement therapy is used as the primary treatment of choice. Currently, the 4-month leptin-replacement therapy consisting of twice-daily injection protocol was stated to enhance glucose levels and lipid metabolism in nine female patients with lipodystrophy in the United States. Considering the results of the leptin-replacement therapy, it is very certain that leptin deficiency is the primary effect of the metabolic abnormalities correlated with lipodystrophy. Under harsh discipline of changes in lifestyle and a massive high compliance of leptin injection, we establish that the leptin-replacement therapy enhances both insulin sensitivity and insulin secretion adequately and briskly improves glucose and lipid metabolism in patients with generalized lipodystrophy. Its results are managed for up to 36 months without any adverse effects. Leptin-replacement therapy is valuable to diabetic obstacles and lipodystrophic complications. The once-daily leptin injection is beneficial to manage glucose and lipid metabolism for a long period of time. Research shows that leptin-replacement therapy is a sufficient and safe treatment for long-term improvement of glucose and lipid metabolism and obstacles in generalized lipodystrophy.

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