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Case Report

High Lipemic Serum (Milky Serum): A Case Report

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Abstract

Background: Hypertriglyceridemia characterized by elevated serum levels of triglycerides which, can be primary (Genetic/familial) or secondary. According to Fredrickson's classification (WHO), Primary hypertriglyceridemia includes type 1, 2b, 3, 4 and 5. In this case report, we present a case of seven years, Girl with milky serum detected coincidentally. The serum triglycerides were raised and standing plasma test was positive. On the other hand, the child did not have the typical signs and symptoms of hypertriglyceridemia. Her parents presented her to the department of childs nutrition complaining, as they claim, from growth failure. At time of presentation, a familial hypertriglyceridemia at such age is seriously considered.

Keywords: *Hypertriglyceridemia; Milky White Serum; Young Children; Fredrickson's Classification; Growth Failure; Simvastatin*

Introduction

Fasting plasma triglyceride levels that are, typically above the 95th percentile for age and sex is known as hypertriglyceridemia [1,2]. This is has risky impacts on the cardiovascular system [2]. Plasma triglycerides have two main sources: the 1st is exogenous which is carried in chylomicrons and the 2nd is endogenous carried on very-low-density lipoprotein (VLDL) particles [3]. These lipoproteins and chylomicrons undergoes hydrolysis by an enzyme called lipoprotein lipase into free fatty acids in capillaries of adipose and muscle tissues [3].

According to WHO (Fredrickson's classification), hypolipoproteinaemia is classified into five types. This is based on serum lipid concentration, appearance of serum after centrifugation, and lipoprotein electrophoresis.

Type I is characterized by hyperchylomicronaemia and hypertriglyceridemia, confirmed by lipoprotein lipase deficiency; and Type V by higher levels of VLDL, chylomicrons, cholesterol and triglycerides [4,5].

There are some interfering factors like ingestion of fatty meals, drugs (e.g., Cholestyramine, estrogens and oral contraceptives etc.), alcohol and pregnancy which may cause elevated TG level. Some drugs which may cause decreased level of TGs are Ascorbic acid, asparaginase, colestipol and clofibrate [6].

In the pediatrics field, high levels of triglyceride, in more than 90%, is secondary to conditions such as obesity and type 2 diabetes mellitus. Secondary causes or acquired causes include high fatty diet, obesity, diabetes, hypothyroidism and certain medications [2].

Triglyceride in the body serves as depots of energy. The primary sources of TG is the fat consumed in the diet [3]. When energy is required TGs are broken into its components glycerol and fatty acids which are released into the blood. TGs do not travel free in the blood stream, but transported in particles called lipoproteins which also contain cholesterol, proteins and phospho- lipids. Certain lipoproteins have higher triglyceride content and are called triglyceride-rich lipoproteins, for instance, Chylomicrons and Very Low Density Lipoproteins (VLDL). These TG rich lipoproteins act as a transporter to carry TG and Cholesterol throughout the blood stream.

TGs are also present in Low density lipoproteins (LDL) and High-Density Lipoproteins (HDL) but in much smaller quantities [4]. Hypertriglyceridemia is frequently associated with other lipid abnormalities and the metabolic syndrome (abdominal obesity, insulin resistance, low high-density lipoprotein (HDL), high triglyceride, and hypertension), which are linked to coronary artery disease [5].

Case Report

A seven years old girl child was presented by her parents to the department of child's nutrition with growth faltering (her weight and height was >-3 SD according to normal growth standards charts). The following investigations X-ray of both wrists for bone age assessment and blood tests, namely serum calcium, phosphorous, alkaline phosphatase, CBC, T4 and TSH, Blood urea and serum creatinine, IGF (Insulin like growth factor) were ordered.

X ray report of the bone age evaluation was 5 years old which is below normal. The serum sample sent to the biochemistry laboratory for analysis was found to be lipemic. Lipid profile was done as the sample was lipemic, which showed high levels of serum triglycerides (1265 mg/dl) and IGF was 88 ng/ml (reference range: 95.0 - 242.0). The standing plasma test was positive thus, showing the presence of chylomicrons. Other required investigations were recorded normal.

The patient was a refugee and she belongs to the Yazidis religion of Iraqis origin and she is belonging to a very low socioeconomic status. There were no signs of hyperlipidemia like xanthoma or hepatosplenomegaly. Serum triglycerides of both parents were within reference range Dietary restriction of fats was advised and simvastatin tab 10mg once daily was recommended for life, and the patient was asked to review again for further work-up. After six weeks, serum triglyceride was 190 mg/dl which shows great result as a consequence of simvastatin therapy.

Discussion

In general, the treatment of hypertriglyceridemia begins with Therapeutic Lifestyle Changes (TLC). Specially, a low fat, carbohydrate-controlled diet should be adopted. Saturated fat should not make up more than 7% of total daily calories, carbohydrates should be restricted to 50.0% to 60.0% of daily calories, and simple sugars like sucrose should be avoided. In adults, alcohol should be greatly reduced or stopped altogether, along with smoking cessation if indicated. Discontinuation of any offending medications should be considered as well [9].

Primary hypertriglyceridemia includes familial chylomicronemia (Type 1), familial combined hyperlipoproteinaemia (Type 2b) familial dysbetalipoproteinaemia (Type 3), familial hypertriglyceridemia (Type 4) and primary mixed hyperlipidemia (Type 5) [7].

In the present case, a provisional diagnosis of either type 1 or type 5 hyperlipidemia was given as, the serum was lipemic, development of a creamy supernatant layer when the sample was refrigerated overnight (presence of chylomicrons) and elevated levels of serum triglycerides. Both type 1 and 5 have common clinical features of eruptive xanthomata, lipemia retinalis, enlargement of liver and spleen, focal neurological deficits and recurrent pain abdomen with risk of pancreatitis [3].

Although, in this case the child needs to be evaluated further. Standing plasma test positive and elevation of serum triglycerides are also common to both types. While the differences between type 1 and 5 include presentation during childhood in type 1 and in adulthood in type 5. There is a deficiency of lipoprotein lipase, APO CII activity or homozygous gene mutations in type 1 while a less severe functional deficiency in type 5.

The prevalence of type 1 (1:106) being much lower than type 5 (1:103). Secondary factors are associated with type 5. Total cholesterol is elevated to a greater extent in type 5 relative to that in type 1.3 The presence of markedly increased triglyceride concentrations should prompt consideration of an inherited condition either detected by inspection of milky appearing serum or during routine screening [8].

Basic treatment in this case is dietary restriction of fats to 20 g/day or less or to 15% or less of the total energy intake [9]. Medical treatment with drugs like fibrates niacin is rarely indicated [10,11]. However, treatment is recommended in this case for life with great

result after six weeks of commencing therapy with simvastatin tab 10 mg. However, initiating a combination of fibrates, niacin and or fish oil to lower triglyceride levels to below 500 mg/dL is the primary goal [7].

Moreover, lipemic serum was a purely coincidental finding in this case. The serum triglycerides were elevated, but not to such an extent so as to cause the appearance of clinical manifestations. Dietary restriction of fats will prevent elevation of serum triglycerides further, thereby preventing complications of hypertriglyceridemia like pancreatitis, cardiovascular disease.

Thus, genetic diagnosis of hyperlipidaemia and gene based therapies should be made easily available so that, these patients can be diagnosed earlier and managed more effectively.

Conclusion

Familial congenital hypertriglyceridemia can be diagnosed in the same existing time as the child is being investigated for other diagnostic purposes. Typical clinical symptoms and signs not necessarily to appear in all cases of congenital hypertriglyceridemia as this appears clinically when serum triglycerides reach certain extent. However, treatment with simvastatin as illustrated in this case, has great clinical therapeutic implementation.

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