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**CASE REPORT**

# DYSLIPIDAEMIA ASSOCIATED WITH DAILY CONSUMPTION OF FRIED-CHICKEN EGGS IN A 22-MONTH-OLD BOY WITH TYPE 1 DIABETES MELLITUS

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## Abstract

**Background/Aim:** Management of toddlers with type 1 diabetes poses a challenge not only to their families but also to the healthcare professionals involved in their care. The aim of this report is to highlight the potential adverse effect of daily consumption of excess amount of fried- chicken eggs on the serum lipid profile of a toddler with type 1 diabetes and the usefulness of computing the serum non-HDL-C in the detection of dyslipidaemia. **Case report:** We report a case of a 22-month-old boy on follow up in our Endocrinology and Metabolism Clinic for type 1 diabetes. Maternal grandmother was feeding him daily with two fried chicken eggs with the aim of providing adequate nutrition as well as satisfying the perceived child's likeness for eggs. Four months after commencement of daily consumption of two fried-chicken eggs, the child's serum cholesterol (223mg/dl) and HDL-C (103mg/dl) increased by 2-folds. The patient's serum triglyceride, low-density lipoprotein cholesterol, non-high-density lipoprotein cholesterol, and very-low-density lipoprotein cholesterollevels were all found to be borderline high, using the National Heart, Lung and Blood Institute 2011 criteria. The serum lipid profile normalized after adjusting the child's dietary practice. **Conclusion:** Daily consumption of excess amount of fried chicken eggs can potentially lead to dyslipidaemia in a toddler with type 1 diabetes and this is easily detectable by computing the serum non-HDL-C concentration.

**Keywords:** Dyslipidaemia, Egg Consumption, Type 1 Diabetes, Non-HDL-C
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## Introduction

Eggs are an inexpensive and highly nutritious functional food, providing both macro- and micronutrients essential for health, particularly in growing children.According to nutritional experts and health agencies, eggs are considereda controversial food because of its saturated fat and cholesterolcontent [1]. One boiled- or fried-chicken egg contains approximately 200mg and 240mg of cholesterol,

respectively [2]. With regard to fatty acid content of egg, the approximate concentrations per egg of saturated-, polyunsaturated- and monosaturated-fatty acids are 1.5g, 0.7g and 1.9g, respectively [2]. Chakrabarty et al [3], demonstrated that there was a large variability in individual response to dietary cholesterol. In this regard, dietary cholesterol has been shown to increase plasma cholesterol in hyperresponders [4,5] but no effect was documented among hyporesponders [3,6]. The pattern of individualserum cholesterol changes in relation to dietary cholesterol consumption is influenced by several factors such as ethnicity, genetic makeup, hormonal factors and the nutritional status (body mass index) of the consumer [7,8]. Adamopoulos et al [9] demonstrated that diet enriched with egg yolk was associated with elevated plasma glucose compared with control diet in rats. In the same study, it was noted that all-cause mortality was affected by egg consumption. The American Heart Association recommendation for primary prevention of cardiovascular disease (CVD) in children and adolescents is an intake of ≤ 10% of total calories from saturated fat with ≤ 30% of the calories from total fat and intake of 300mg per day dietary cholesterol with adequate energy to support growth and development [10].

Disorders of lipoprotein metabolism are one of the key risk factors involved in atherogenesis, accounting for approximately 50% of the population-attributable risk of developing CVD [11]. Therefore, estimation of cardiovascular disease risk has become the cornerstone for prevention of CVDs. Viewed from this perspective, several lipoprotein ratios (also called atherogenic ratios) have been defined with the aim of optimizing the predictive capacity of the conventional lipid levels, such as serum levels of triglyceride (TG), low density lipoprotein cholesterol (LDL-C), total cholesterol (TC) and high-density lipoprotein cholesterol (HDL-C). Some of the atherogenic ratios suggested for predicting CVD risk include Castelli's Risk Index (CRI) I and II and Atherogenic Coefficient (AC) [12,13]. These ratios can provide information on risk factors which are difficult to quantify by routine analyses of serum lipid levels and could be a better reflection of metabolic and clinical interactions between lipid fractions [14]. In 2011, the American Academy of Pediatrics redefined dyslipidaemia by adopting the non-HDL-C level (instead of LDL-C level), triglyceride (TG) level and HDL-C level as the three major criterions [15]. It has been stated that at no additional cost, non-HDL-C measures all atherogenic apolipoprotein B-containing lipoproteins, including LDL-C, very lowdensity lipoprotein cholesterol (VLDL-C), and to lesser extent intermediate density lipoprotein cholesterol (IDL-C), lipoprotein(a), chylomicrons and chylomycron remnants [16]. Thus, making estimation of non-HDL-C in any individual a useful screening tool. According to National Heart, Lung and Blood Institute (NHLBI) guideline in 2011, serum non-HDL-C levels 120-144mg/dl and ≥ 145mg/dl represent borderline- and abnormally-high levels, respectively [15].

Diabetes mellitus diagnosed during the first 2 years of life differs from the disease in older children regarding its causes, clinical characteristics, treatment options and needs in terms of education and psychosocial support [17]. Although hypoglycaemia is a major acute complication of diabetes mellitus in children less than 5years old, hyperglycaemia and blood glucose variability is a cause for concern because of its association with microvascular

complications, even in prepubertal children and those who have diabetes for only 1 to 2 years [18]. Therefore, the presence of dyslipidaemia in a toddler with diabetes mellituscould worsen theblood glucose variability, thereby potentially increasing the risk of microvascular complications (retinopathy and nephropathy). In addition, the presence of severe hypertriglyceridaemia can complicate diabetic ketoacidosis by its association with development of pancreatitis, leading to increased morbidity and mortality [19,20]. Djousséet al [21], found that daily consumption of eggs was associated with increased risk of type 2 diabetes. In that report, the authors postulated that excessive egg consumption may potentiate the risk of cardiovascular disease by inducing impaired glucose metabolism and insulin resistance [21]. In literature, there are no reports on the health effects of daily egg consumption in children with either types 1 or 2 diabetes. To the best of our knowledge, this is the first report on the subject. The aim of this report is to highlight the potential adverse effect of excessive daily consumption of fried-chicken eggs on the serum lipid profile of a toddler with type 1 diabetes and the usefulness of computing the serum non-HDL-C in detection of dyslipidaemia.

**Case report**

We report a case of a 22-month-old Nigerian boy who was successfully managed for new-onset type 1 diabetes complicated by diabetic ketoacidosis (DKA). He was diagnosed at the age of 18 months based on polyuria, polydipsia, and weight loss, all of one week duration. In addition, he had increased tendency to fall asleep. He also had four episodes of vomiting associated with weakness and tendency to fall asleep. His biochemical parameters showed hyperglycaemia (blood glucose 497mg/dl), ketonuria (2 pluses), acidosis (10mmol/L), and HbA1C 8.5%. Glutamic acid decarboxylase antibody profile was positive. He is currently being followed up in the endocrinology and metabolism clinic of the University of Benin Teaching Hospital (UBTH). After his treatment for DKA, he was discharged home on basal-bolus regimen with long-acting insulin (glargine) and rapidly-acting insulin (aspart). His glycaemic control was satisfactory. At home, grandmother was worried about the adequacy of the child's nutritional intake and decided to feed him with two fried chicken eggs daily. This daily consumption of fried eggs was for a period of 4 months. This was revealed during an interaction with the grandmother when the child presented with a febrile illness (malaria). Subsequent evaluation of his serum lipid profile revealed dyslipidaemia despite being normal at the time of initial presentation with DKA. His serum lipid profiles are summarized in Table 1. When the total as well as high density lipoprotein cholesterol levels at presentation and after 4 months on daily fried chicken eggs are compared, there was a 2-fold increase.

**Table 1. Serum lipid profiles at presentation and follow-up visits**

| Serum lipids | Reference intervals for age and sex [15] | At presentation | After 4 months on daily fried-egg consumption | 6 months after cessation of daily fried-egg consumption |
|---|---|---|---|---|
| TCh(mg/dl) | < 200 | 115 | 223 | 120 |

| | | | | |
|---|---|---|---|---|
| HDL-C(mg/dl) | 35-65 | 43 | 103 | 70 |
| LDL-C(mg/dl) | ≤100 | 52 | 100 | 58 |
| TG (mg/dl) | ≤100 | 55 | 102 | 63 |

TCh = Total cholesterol; HDL-C = High density lipoprotein cholesterol; LDL-C = Low density lipoprotein cholesterol; TG = Triglyceride

Using the appropriate formulae [13], the atherogenic lipid ratios were computed and the cut-off points suggested by Bonito et al [22] and Lloyd-Jones et al [23], respectively were applied to define normal values. The results of the computed atherogenic lipid ratios are summarized in Table 2.

**Table 2. Summary of serum atherogenic lipid ratios of our patient**

| Serum atherogenic lipid ratios | Normal values | Patient's results |
|---|---|---|
| TG/HDL-C [22] | <2.0 | 0.99 |
| Castelli's Risk Index I (CRI-I) [23] | <4.4 | 2.17 |
| Castelli's Risk Index II (CRI-II) [23] | <2.9 | 0.97 |
| Atherogenic Coefficient (AC) [23] | 3.01±0.16 | 1.17 |

TG = Triglyceride; HDL-C = high density lipoprotein cholesterol

As shown in Table 2, based on adult values, his TG/HDL-C ratio, CRI-I, CRI-II and AC were apparently normal. Normative values are not available for children. In the index case, we computed the non-HDL-C (total cholesterol minus HDL-C) and it was 120mg/dl(3.1mmol/L), representing borderline high serum level based on the most recent American Academy of Pediatrics definition of dyslipidaemia [15]. In sum, our patient's serum non-HDL-C level was clearly elevated. The patient's serum very-low-density lipoprotein cholesterol (VLDL-C) level was borderline high (20.4mg/dl) [15].

The grandmother was advised against this practice. She now gives boiled egg to the child three times a week. His first presentation at UBTH followed referral from a private hospital. At the onset of the symptoms, he was taken to the first private hospital where some blood tests were performed and he was said to be "fine" and so, was taken home. The next day, with persistence of the vomiting, he was taken to another private hospital where his random blood glucose was found to be 497mg/dl. He was then commenced intravenous fluid (4.3% Dextrose in 0.18% saline) and referred to UBTH, Benin City. There is no family history of diabetes mellitus or previous illness requiring a hospital admission. Birth weight is unknown to the grandmother. He was fed with breast milk for the first four months of life. Thereafter, artificial formula was added. Guinea-corn pap with milk as well as other family diet were added at the age of eight months. He is on cereals and family diet at the time of presentation. The child has been living with his grandparents since the age of four months when his mother left the country (Nigeria). On examination at presentation, the child was found to be acutely ill-looking, afebrile, not pale or icteric or cyanosed. He

was dehydrated (sunken eyes, loss of skin turgor and slow capillary refill). Anthropometric measurement showed weight 11kg ($50^{th}$ percentile), length 86cm ($50^{th}$ percentile) and occipitofrontal circumference 49cm ($75^{th}$ percentile). He had tachycardia with pulse rate 146 beats per minute. His blood pressure was normal for age, sex and height. Respiratory rate was 40 cycles per minute. Other examinations were unremarkable. At first presentation in UBTH, the biochemical findings were blood glucose 533mg/dl, serum bicarbonate 14mmol/L, ketonuria (3+), glycosuria (2+), urea 44mg/dl, HbA1C 8.5%. The results of his full blood count and differentials were: WBC 24.8 x $10^3$/µL with lymphocyte 28.7%; granulocyte 63.2%;and monocyte 8.1%. His platelet count was 468x$10^3$/µL and haematocrit was 40.1%. The concentrations of serum sodium, potassium, chloride and creatinine were within normal limits. A diagnosis of new-onset type 1 diabetes with ketoacidosis was made. Using ISPAD 2014 Guidelines [24], he was successfully treated for diabetic ketoacidosis (DKA). Intravenous cefuroxime (100mg/kg/day) was added to the therapy for suspected bacterial infection. The patient responded well to treatment and was discharged to the clinic for follow up.

Over a 4-year period (2014-2017), only five children below the age of five years with type 1 diabetes were seen in our hospital (UBTH), representing an incidence of approximately one case per annum. Of the five patients, one was below 2 years old.

**Discussion**

In the index case, the diagnosis of dyslipidaemia was based on the presence of hypercholesterolaemia (≥ 200mg/dl) and high HDL-C, hyperalphalipoprotein (≥ 65mg/dl), indicating atherogenic dyslipidaemia. In addition, the LDL-C and triglycerides were slightly elevated in our patient. All pointing to dyslipidaemia in our patient. Both the serum total cholesterol and HDL-C demonstrated a two-fold increase from the levels before commencement of daily consumption of excess amount of fried-chicken eggs. The serum levels of total cholesterol (TC) of 223mg/dl represents hypercholesterolaemia in children, based on NHLBI criteria [15]. The high serum HDL-C level (103mg/dl) represents hyperalphalipoproteinaemia [15].Studies in adults indicate that very high HDL-C is associated with adverse cardiovascular health consequences [25,26]. The observed rapid and dramatic rise in serum total and HDL-C levels over a short period of 4 months is also a cause for concern. The dyslipidaemia in the index case is most probably due to the daily consumption of excess amount of fried-chicken eggs. According to the American Heart Association, consumption of two fried-chicken eggs daily for 4 months was well above the recommended intake for both cholesterol and saturated fat in children and adolescents for primary prevention of cardiovascular disease [10]. This view is supported by the fact that the abnormal serum lipid levels normalized following reduction in frequency of egg consumption and change from fried to boiled eggs. Secondly, dyslipidaemia associated with diabetes most commonly manifests as elevated triglycerides and low levels of HDL-C [27]. In our patient, the HDL-C was markedly elevated while the triglyceride showed only slight increase above normal for age and gender, further supporting the view that the dyslipidaemia observed in the index case was associated with the excessive fried-egg consumption. Dietary macronutrient intakes are strongly linked to

blood lipid levels. Mensick et al [28] in a meta-analysis of 60 controlled trials, revealed that diets high in saturated fat increase total cholesterol as well as LDL-C whereas diets high in mono- and polyunsaturated fats increase HDL-C. Diets high in carbohydrate intake increase serum triglyceride level [28]. Fried chicken eggs contain cholesterol, saturated fat, mono- and polyunsaturated fat, all of which are known to influence serum lipid levels [1,2].

The serum non-HDL-C level in our patient was remarkably high. In this regard, his serum level of non-HDL-C (which measures all atherogenic apolipoprotein B-containing lipoproteins) was nearly twice the cutoff point (≥ 65mg/dl) that defines high HDL-C [15]. Serum level of non-HDL-C varies inversely with age. Such an unfavourable lipid profile in childhood has been linked to future hypertension and atherosclerotic disease in adulthood [29]. Goff et al [30], reported that mean serum total cholesterol levels tended to be steady during prepubertal period, dropped during puberty in both sexes, with the drop being more profound in boys, and then rise again in late adolescence.

Madsen et al [31] reported that HDL-C level greater than 77mg/dl in adults was associated with adverse cardiovascular health. Our patient had HDL-C level of 103mg/dl, suggesting a potential risk for cardiovascular disease. The adverse health effects of very high serum HDL-C levels has also been noted by Ko et al [26]. In the index case, the reason for the markedly elevated serum HDL-C level may be multifactorial (dietary and genetic). The results of a meta-analysis of cholesterol feeding studies using a variety of sources of dietary cholesterol (including eggs) showed that for every 100mg per day increase in dietary cholesterol intake, circulating high-density lipoprotein increased by 0.01mmol/L (0.4mg/dl) [32]. Alternatively, the markedly elevated serum HDL-C level in our patient may be due to the presence of genetic variants of HDL-C. Such variants are found in mutations due to cholesterol ester transfer protein(CETP), ATP-binding cassette transporter A1 (ABCA1), hepatic lipase (LIPC) and scavenger receptor B1 (SCARB1) [33,34]. We could not investigate for these HDL-C variants because of inadequate laboratory facility in our centre. This is an important finding because it implies that non-HDL-C is sensitive screening tool for detecting abnormal serum lipid levels, in the face apparently normal lipid ratios. Based on the lipid ratios, the index case was not at risk of cardiovascular disease but surveillance is indicated.

In our patient, some clinical data negate the diagnosis of monogenic diabetes mellitus. Such negative findings include age 18 months at first diagnosis, and presence of ketoacidosis as well as glutamic acid antibodies at first presentation. More importantly, absence of family history of diabetes mellitus makes monogenic form of diabetes less likely [35]. Typically, maturity onset diabetes of youth (MODY) (excluding neonatal diabetes) is characterized by early onset between the ages of 9 and 30 years [35].

In conclusion, daily consumption of excess amount of fried-chicken eggs can potentially lead to dyslipidaemia in a toddler with type 1 diabetes and this is easily detectable by computing the serum non-HDL-C concentration. We advocate intensification of education regarding medical nutrition therapy in parents/caregivers.

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