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ISSN: 2550-794X

International Digital Organization for Scientific Research IDOSRJSR1.1.2936.100
IDOSR JOURNAL OF SCIENTIFIC RESEARCH 9(1) 29-36, 2024.
https://doi.org/10.59298/IDOSRJSR/2024/1.1.2936.100

Abnormal Lipid Profile Level in Neonates and Possible Development of Atherosclerosis in Adulthood: Mini Review

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ABSTRACT

The lipid profile during the neonatal period plays a pivotal role in determining the future risk of atherosclerosis and cardiovascular diseases in adulthood. Abnormal lipid levels in neonates have been associated with an increased propensity for the development of atherosclerosis later in life. This review aims to explore the significance of abnormal lipid profiles in neonates and their potential implications for the development of atherosclerosis in adulthood. The discussion encompasses the mechanisms underlying lipid metabolism in early life, the impact of various factors on lipid levels in neonates, and the long-term consequences of abnormal lipid profiles. Understanding the relationship between neonatal lipid profiles and the risk of atherosclerosis in adulthood is crucial for developing preventive strategies and interventions aimed at reducing the burden of cardiovascular diseases in later life. This review synthesizes idea from existing literature to provide insights into the importance of early lipid assessment and its implications for long-term cardiovascular health.

Keywords: Abnormal Lipid Profile, Neonates, Atherosclerosis, Cardiovascular Risk, Lipid Metabolism, Early Life, Long-term Health, Preventive Strategies, Cardiovascular Diseases

INTRODUCTION

It was exactly a century ago that cord lipids were measured for the first time by Herrman and Neuman in 1912 AD who found them to be considerably lower than those found in normal women. Recently interest in cord lipids has increased because it is thought that adult serum lipid disorders have their roots in neonate and atherogenic changes are postulated to originate early in life [1-3]. Observations from both epidemiological and clinical studies have suggested that the pathological process of coronary artery disease like atherosclerosis begins in neonate. The aortas of children as young as 3-4 years age often contain intimal lipid deposits, commonly called "fatty streaks" [4]. These aortic fatty streaks increase in extent rapidly during the second decade of life and similar lesions begin to appear in the coronary arteries in the latter part of second decade. Risk reduction would likely be of greatest importance if appropriate intervention were to begin early in life because there is compelling evidence that atherosclerosis has its origins in Neonate [5-8]. A relatively small reduction in mean cholesterol levels for a population of neonates, if continued into adult life could significantly reduce the incidence of risk of coronary heart disease. For men, results from the previous cohort studies have shown that a decrease of serum cholesterol concentration of 0.6 mmol/l (about 10%) was associated with a decrease in incidence of ischaemic heart disease of 54% at age 40 years, 39% at age 50, 27% at 60, 20% at 70, and 19% at 80. The data for women were found to be limited but indicate a similar effect. The significant physiological change in

lipid profile values post birth is also well documented from previous report. Previous studies on tracking of lipid profile among newborns have at the earliest observed changes only 6 months post birth [9-10]. Here, there is need to review the lipid parameters of neonates and their different values based on causative factors like gestational age, sex, weight and also to track how the values change over the first week of life to see how fast the lipid profile parameters are altered after birth.

Overview of Lipid Profile Parameters

Lipids are essential for energy homeostasis, reproductive and organ physiology, and numerous aspects of cellular biology. They are also linked to many pathological processes, such as obesity, diabetes, heart disease, and inflammation. To meet the different demands from a variety of tissues, the human body has evolved a sophisticated lipoprotein transport system to deliver cholesterol and fatty acids to the periphery. Lipoproteins are composed of triglycerides (TG), cholesterol esters, phospholipids, and apolipoproteins, which modulate lipoprotein catabolism. In the forward transport system, TG-rich very low-density lipoprotein (VLDL) released by the liver delivers fatty acids to adipocytes for storage and to cardiac and skeletal muscle for energy consumption. Lipoprotein lipase (LPL), secreted by the adipocyte, muscle, and macrophage, plays an important role in VLDL fatty acid release, and its subsequent conversion to low-density lipoprotein (LDL). Cholesterol ester-rich LDL, on the other hand, delivers cholesterol to peripheral tissues for steroidogenesis and maintaining cell membrane integrity. Conversely, in the reverse transport system, high-density lipoprotein (HDL) transports excess cholesterol from extra hepatic cells, such as macrophages at the vessel wall, to liver, where it can be recycled or catabolized to bile acid [11]. Disturbances in this system are integral components of life-threatening diseases, best exemplified by the metabolic syndrome, or syndrome X, which refers to patients who are insulin-resistant (hyperinsulinemic), dyslipidemic (elevated TG and decreased HDL-cholesterol levels), frequently hypertensive and at high risk for developing coronary artery disease (CAD) and atherosclerosis [12]. Furthermore, Lipid Profile is a **combination of blood tests** performed to check the cholesterol levels and the level of triglycerides in the blood. Lipids are any of a class of organic compounds that are fatty acids or their derivatives that are insoluble in polar solvents but soluble in organic solvents, hence blood lipids are nothing but fat content present in the blood.

Neonates Lipid metabolism

Lipids are crucial macromolecules needed for growth in animals and man alike. They are involved in the formation of steroid hormones (testosterone, estrogen), signaling functions and forms a key structural component in all cellular membrane. Lipid metabolism is the cellular basis of essential substances; the main components are Triglycerides and Total Cholesterol. Lipid metabolism plays an important role in maintaining the body's normal physiological function. In the fetal period, the normal lipid metabolism is essential to maintain normal fetal growth and development thus, they are required for neonatal development. Embryos has two major sources of cholesterol, endogenous and exogenous as do neonates and adult. The endogenous cholesterol comes from de novo synthesis, while the exogenous cholesterol is obtained from the maternal circulation after being transported across the placenta and possibly the secondary yolk sac. The characteristic distribution of TG in neonates, especially the relatively high LDL concentration, may partly be explained by a reduction in hepatic lipase activity. However, lipase activity, even including lipoprotein lipase (LPL), cannot fully explain the mechanism of Triglyceride distribution because Triglyceride concentrations were markedly low. Another possible mechanism is low lipid transfer across the placenta and low lipogenesis in the liver. Furthermore, in human fetuses, increases in hepatic LDL receptor activity are positively correlated with gestational age and negatively correlated with LDL concentration. The impact that dietary cholesterol has on sterol metabolism has also been studied. The fetus appears to be somewhat protected from down regulation of sterol biosynthesis. In contrast, neonates, like adults, can suppress sterol synthesis rates [13]. In one study, infants were fed breast milk versus cow milk-based formula. After 4 months of diets with different cholesterol concentrations, total-C and LDL-C levels are higher in infants consuming more dietary cholesterol. Unlike fetal tissues, the fractional synthetic rate (FSR) of cholesterol is lower in infants consuming more cholesterol demonstrating the ability to regulate sterol biosynthesis in the neonate.

Prenatal Epigenetic Programming and Atherosclerosis Risk

Fetal development represents a crucial period for epigenetic reprogramming, imparting lifelong impacts on gene expression and disease susceptibility [14]. The body's lipid metabolic activities regulate epigenetics by contributing to the availability of molecules like fatty acids, cholesterol, and other lipid

derivatives. These lipid molecules can be used as substrates for enzymes that are involved in epigenetic modifications like DNA methylation and histone acetylation [15]. This can change how genes are expressed during development, and epigenetics controls metabolism by controlling the expression of metabolic genes [15]. These bidirectional relationships between body metabolic activities and epigenetics emphasize the interplay between these processes. The thrifty phenotype hypothesis, postulated by Hales and Barker, proposes that during prenatal development, fetuses undergo metabolic adaptations to optimize survival in resource-limited environments [16]. These adjustments may involve altered lipid metabolic processes to prioritize the survival of vital organs [16]. Although these adaptations have been beneficial, they can have detrimental effects when the scarce resource becomes abundant, potentially leading to metabolic disorders in adulthood. Also, Intrauterine life restriction leads to metabolic programming that can raise the likelihood of developing metabolic syndrome and, subsequently, cardiovascular morbidity during adulthood [17]. These adverse events can have a lasting impact on the growth of organs and increase the likelihood of developing diseases later in life. Therefore, factors that could influence fetal lipid metabolism can impact epigenetic programming in adulthood. Hence, fetal metabolic programming affected by dyslipidemia can set the stage for atherosclerosis development in late childhood. This metabolic derangement can introduce fetal long-term, relatively irreversible changes in blood vessels, increasing the risk of atherosclerosis in adulthood.

Possible outcome of Abnormal Lipid profile in neonates

Abnormal levels of blood lipids and their lipoprotein carriers play a central role in the development of CVD. Elevated serum levels of total cholesterol and its main carrier, low-density lipoprotein (LDL), have the strongest causal evidence with atherosclerosis [18]. Cholesterol is a key component in the atherosclerotic plaque, while LDL is likely to start the lipid deposition in the endothelium by moving from the blood into the vessel wall. Both in serum and in the vessel wall, LDL is oxidised through actions of free radicals or leukocyte activity and becomes a key player in the development of foam cells. When accumulated over time, these foam cells are significant for the evolution of the atherosclerotic plaque. Elevated plasma apolipoprotein (APO) B is also a strong risk marker, as it represents the total atherogenic particle number. Moreover, high-density lipoprotein (HDL) and its principal apolipoprotein carrier, APO A1, have apparent cardioprotective effects and functions, with multiple mechanisms identified such as reverse cholesterol transport from foam cells to liver, and anti-inflammatory, anti-oxidative, and anti-thrombogenic effects as well as regression of atherosclerotic plaques [18]. However, the causal relationship between low HDL and increased risk of CVD is unclear, and it is rather suggested that low HDL levels are a secondary phenomenon occurring alongside with high triglyceride levels [19], which in turn have been shown to have a close association with the disease [20]. Similarly, human umbilical cord blood (UCB) has been studied for the heterogeneity of plasma lipoproteins at birth by employing density gradient ultracentrifugation gel electrophoresis [21]. However, these methodologies are tedious, labor-intensive, and sparsely available and generally have sample size too small to make a clinical impact. Nonetheless, these innovations underscore the importance of identifying high risk neonates and offer an opportunity to commence preventive interventions. Report has shown that pre-term neonates had a higher levels of serum TC, TG, LDL-C and VLDL-C compared to full term neonates. This is because the preterm neonates remain deprived of the opportunity of energy storage in late gestation period: thus, lack both, hepatic carbohydrate (Glycogen and subcutaneous adipose tissues (triglycerides) [22]. The rise in human umbilical cord blood (UCB cholesterol levels may reflect the metabolic adaptation to provide adequate energy, especially to the "essential " Organs like brain (Fetal brain sparring" Phenomenon) [23].

Cardiovascular disease and its possible risk factors

Cardiovascular diseases (CVDs) are considered multifactorial conditions that especially affect the essential components of the circulatory system of the human body such as heart, blood vessels, and blood itself [24]. CVDs can be congenital or acquired throughout people's lifespan. Atherosclerosis, rheumatic heart disease and cardiovascular inflammation are the main and more prevalent cardiovascular acquired problems.

Traditional Risk Factors and Novel Biomarkers

Hypertension

A large number of observational studies have demonstrated a continuous relationship between both systolic and diastolic blood pressure and cardiovascular morbidity and mortality [25]. This association is partly explained by the pathophysiological link between hypertension and

inflammation (i.e. through angiotensin II). Endothelial injury and vascular cell proliferation induced by increased pressure are other effects that exacerbate the atherosclerotic process [24].

Hyperglycaemia and hyperinsulinaemia

Diabetes is a metabolic disease resulting from defects of insulin secretion and/or insulin action, and a prominent risk factor for CVD. The hyperglycaemia characterising both type 1 and type 2 diabetes plays a central pathophysiological role in the atherosclerotic process. Hyperglycaemia may, for example, cause protein glycosylation and accumulation of advanced glycation end products (AGEs), a decrease in endothelium-derived NO availability, and an increase in oxidative stress; it affects vascular function mainly through overproduction of reactive oxygen species (ROS), and increases the endothelial expression of various adhesion molecules, which all results in endothelial dysfunction and vascular inflammation. Hyperinsulinaemia, which in most instances occurs as a reflection of insulin resistance, is another important cardiovascular risk factor. Insulin is involved in the process of atherosclerosis via stimulation of smooth muscle cell proliferation and enhancement of lipoprotein metabolism in arterial tissue [24].

Clustering of metabolic and vascular risk factors

CVD risk factors tend to cluster and interact multiplicatively. In line with the causal pie model described above, the presence of a single risk factor will yield a lower probability for disease in comparison tomultiple metabolic abnormalities in an individual. Various risk prediction scores (for example the Framingham formula or SCORE) have therefore been developed toestimate absolute CVD risk via multivariable assessment of known risk factors. The metabolic syndrome (MetS) is another assessment to evaluate clustering of metabolic risk factors, proven to be a strong risk for CVD morbidity and mortality [27].

Lifestyle-related and other factors

Several lifestyle-related factors have been shown to have a close, independent association with CVD morbidity and mortality. Moreover, clustering of healthy lifestyle-related factors is associated with a major risk reduction for incident CVD [28-29]. As these factors are related to the individual's chosen lifestyle, they are modifiable. The negative consequences of an unhealthy lifestyle have been shown to be potentially reversible, thus reducing the complication of atherosclerosis [30-32]. Decline in risk factors has been related to a remarkable decline in acute myocardial infarctions [33-44]. Other established risk factors such as age, gender, ethnicity, and genetic predisposition are not modifiable, but their influence on disease risk can be mitigated by a healthy lifestyle.

Diet

Both total energy intake and the qualitative aspects of diet (nutrients, patterns, etc.) have an important independent impact on disease development. For example, a high intake of saturated fats of animal origin is generally considered unfavourable, while vegetable-based diets containing mainly unsaturated fats (such as the Mediterranean diet) are seen as protective [33-34]. Similar comparisons have been made between diets with low versus high proteins and carbohydrates [31-32]. In recent years, high consumption of soda, sweets, and fast food with high levels of sugar and empty carbohydrates and low levels of nutrients has probably played an important role in the dramatic increase of overweight and obesity worldwide. Food culture and habits vary considerably between different global regions, and the variation is partly reflected by the distribution of disease burden. There is also an important interaction between PA, and diet [37-38].

Atherosclerosis

Atherosclerosis is defined as a chronic condition occurring in the blood vessels. It is characterised by the decrease in elasticity as a result of the narrowing and rigidity of the blood vessels' walls caused by deposition of fats, cholesterol, calcium and other substances (minerals and cellular debris among others) in the inner layer of medium and large-sized arteries. Arteriosclerosis increases blood pressure, diminishes the good blood flow to different body structures and produces serious tissue damage. Additionally, the plaque formed in the blood vessels walls may break and result in the formation of blood clots (thrombus) which in turn can produce serious blockages in situ or elsewhere. Ischemic heart disease or coronary heart disease (CHD) (inadequate supply of oxygen-rich blood to the heart muscle) and stroke (reduction in blood flow to the brain tissue by blood vessel blockage or intracranial bleeding) are the most serious health consequences of arteriosclerosis disease [38-44].

Pediatric Atherosclerosis

Atherosclerotic vascular changes can begin early in neonatal life, setting the stage for cardiovascular disease (CVD) events in adulthood. If premature development of cardiovascular disease can be

anticipated during childhood, the disease might be prevented. For most children, atherosclerotic vascular changes are minor and can be minimized or even prevented with adherence to a healthy lifestyle. However, in some children, the process is accelerated because of the presence of identifiable risk factors (e.g. Obesity, dyslipidemia, and hypertension) or specific diseases that are associated with premature CVD (e.g. diabetes mellitus). The death rate from cardiovascular disease is lowest in children with lower cholesterol levels and in individuals who exercise regularly [35-39]. Research has shown that Atherosclerosis begins at an early stage of life, reveals childhood and adolescence as critical periods for the detection of risk factors for cardiovascular disease and the prevention of future complications. Monitoring these factors would help ascertain the symptoms that when modified can mitigate or even reverse the progression of those dysfunctions. A range of risk factors, including genetic factors, hypertension, dyslipidemia, obesity, metabolic syndrome (MS), an atherogenic diet, and physical inactivity, is associated with cardiovascular disease, and the prevalence of these factors is increasing among children and adolescents [40-44]. Lifestyle and eating habits are fundamentally important for protection against the manifestation and progression of Atherosclerosis risk factors. Atherosclerosis being the main is causative for cardiovascular disease, and therefore should be a key target of heart disease-prevention programs. The emphasis is on hypercholesterolemia, hypertriglyceridemia, overweight, hyperglycemia, hypertension, and physical inactivity. Correlations between the plasma levels of cholesterol and a decrease or delay in Atherosclerosis progression by means of diet and lifestyle changes have been documented, with the hypothesis that for each 1% reduction in total cholesterol, a decrease of 2% in the occurrence of coronary artery disease was observed. Studies have also reported that the degree of atherosclerosis in children and young adults can be correlated with the same risk factors that have been identified in adults [30-40].

CONCLUSION

Hypercholesterolemia is an important risk factor for atherosclerotic cardiovascular disease, including cerebrovascular disease, coronary heart disease, and peripheral arterial disease; it is usually symptomatically quiescent until significant atherosclerosis has developed. Complications of hypercholesterolemia and atherosclerosis include myocardial infarction, ischemic cardiomyopathy, sudden cardiac death, ischemic stroke, erectile dysfunction, claudication, and acute limb ischemia. Pre-term Neonates with dyslipidemia are more prone to atherosclerotic cardiovascular disease (ACVD) and there is an increase in its probability when living a sedentary lifestyle and a diet characterized by the excessive consumption of saturated fats, trans-fatty acids, and cholesterol. Other associations include diabetes, excess body weight mainly in the abdominal region, hypothyroidism, nephrotic syndrome, and cholestatic liver disease. Low HDL-C levels are associated with smoking and abdominal obesity.

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CITE AS Mathias Abiodun Emokpae, Timothy God-Giveth Olusegun, Oluwasina Moses Egbeyemi and Emmanuel Ifeanyi Obeagu (2024). Abnormal Lipid Profile Level in Neonates and Possible Development of Atherosclerosis in Adulthood: Mini Review. IDOSR JOURNAL OF SCIENTIFIC RESEARCH 9(1) 29-36. https://doi.org/10.59298/IDOSRJSR/2024/1.1.2936.100