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Evaluation of lipid profile in stunted children's of age below 5 years from North Bihar Region

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Abstract
Lipid disorders include high levels of low-density lipoprotein (LD) cholesterol, or fats called triglycerides, or both in blood. High levels of these lipoproteins and fats increase the risk for developing heart disease. The early onset of lipid disorders including elevated total or low-density lipoprotein (LDL) cholesterol levels, low levels of high-density lipoprotein (HDL) cholesterol, and high levels of triglycerides (fats) is alarming as these conditions in childhood are predictive of elevated risk for cardiovascular disease in adulthood. Hence based on above findings the present study was planned for Evaluation of Lipid Profile in Stunted Children's of Age Below 5 years from North Bihar Region.
The present study was planned in Department of Paediatrics, Darbhanga Medical College & Hospital, Laheraisarai, Bihar, India. Total 100 children's of age below 5 years were enrolled in the present study between July 2019 to December 2019.Out of that the 50 Stunted children's were enrolled in the Group A as Cases and remaining 50 cases were enrolled in Group B as Control patients.
The data generated from the present study concludes that there is an increased risk of metabolic alterations, namely, poor glycaemic control, hypertension, and altered lipid profile that occur in children with stature deficit. Long-term effect of these metabolic alterations may predispose these undernourished children to an increased risk of CAD in future life.

Keywords: lipid profile, stunted children's, below 5 years, North Bihar Region, etc

Introduction
Stunting causes irreversible physical and mental damage to children. A stunted child is too short for their age, does not fully develop and stunting reflects chronic under nutrition during the most critical periods of growth and development in early life. It is defined as the percentage of children, aged below 5 years, whose length/height for age is below minus two standard deviations (moderate and severe stunting) and minus three standard deviations (severe stunting) from the median of the WHO Child Growth Standards. In India, 35 per cent of children younger than five years of age are stunted, a manifestation of chronic under nutrition. Stunting and other forms of under-nutrition are thought to be responsible for nearly half of all child deaths globally.
Stunted growth is a reduced growth rate in human development. It is a primary manifestation of malnutrition (or more precisely under nutrition) and recurrent infections, such as diarrhea and helminthiasis, in early childhood and even before birth, due to malnutrition during fetal development brought on by a malnourished mother. The definition of stunting according to the World Health Organization (WHO) is for the "height for age" value to be less than two standard deviations of the WHO Child Growth Standards median [1].
As of 2012 an estimated 162 million children under 5 years of age, or 25%, were stunted in 2012. More than 90% of the world's stunted children live in Africa and Asia, where respectively 36% and 56% of children are affected [2]. Once established, stunting and its effects typically become permanent. Stunted children may never regain the height lost as a result of stunting, and most children will never gain the corresponding body weight. Living in an environment where many people defecate in the open due to lack of sanitation, is an important cause of stunted growth in children, for example in India [3].
Stunted growth in children has the following public health impacts apart from the obvious impact of shorter stature of the person affected: greater risk for illness and premature death [1], may result in delayed mental development and therefore poorer school performance and later on reduced productivity in the work force, reduced cognitive capacity.
Women of shorter stature have a greater risk for complications during child birth due to their smaller pelvis, and are at risk of delivering a baby with low birth weight. Stunted growth can even be passed on to the next generation (this is called the "intergenerational cycle of malnutrition") [1].
The impact of stunting on child development has been established in multiple studies [4]. If a child is stunted at age 2 they will have higher risk of poor cognitive and educational achievement in life, with subsequent socio-economic and inter-generational consequences [5, 4]. Multi-country studies have also suggested that stunting is associated with reductions in schooling, decreased economic productivity and poverty [6]. Stunted children also display higher risk of developing chronic non-communicable conditions such as diabetes and obesity as adults. If a stunted child undergoes substantial weight gain after age 2, there is a higher chance of becoming obese. This is believed to be caused by metabolic changes produced by chronic malnutrition, that can produce metabolic imbalances if the individual is exposed to excessive or poor-quality diets as an

adult. This can lead to higher risk of developing other related non-communicable diseases such as hypertension, coronary heart disease, metabolic syndrome and stroke [5, 6]. At societal level, stunted individuals do not fulfill their physical and cognitive developmental potential and will not be able to contribute maximally to society. Stunting can therefore limit economic development and productivity, and it has been estimated that it can affect a country's GDP up to 3% [4, 6].

The causes for stunting are principally very similar if not the same as the causes for malnutrition in children. Most stunting happens during the 1,000-day period that spans from conception to a child's second birthday. [citation needed] The three main causes of stunting in South Asia, and probably in most developing countries, are poor feeding practices, poor maternal nutrition, and poor sanitation.

Inadequate complementary child feeding and a general lack of vital nutrients beside pure caloric intake is one cause for stunted growth. Children need to be fed diets which meet the minimum requirements in terms of frequency and diversity in order to prevent undernutrition [7]. Exclusive breastfeeding is recommended for the first six months of life and complementary feeding of nutritious food alongside breastfeeding for children aged six months to 2-years-old. Prolonged exclusive breastfeeding is associated with under nutrition because breast milk alone is nutritionally insufficient for children over six months old [8, 9]. Prolonged breastfeeding with inadequate complementary feeding leads to growth failure due to insufficient nutrients which are essential for childhood development. The relationship between undernutrition and prolonged duration of breastfeeding is mostly observed among children from poor households and whose parents are uneducated as they are more likely to continue breast-feeding without meeting minimum dietary diversity requirement [10].

Poor maternal nutrition during pregnancy and breastfeeding can lead to stunted growth of their children. Proper nutrition for mothers during the prenatal and postnatal period is important for ensuring healthy birth weight and for healthy childhood growth. Prenatal causes of child stunting are associated with maternal under nutrition. Low maternal BMI predisposes the fetus to poor growth leading to intrauterine growth retardation, which is strongly associated with low birth weight and size. Women who are underweight or anemic during pregnancy, are more likely to have stunted children which perpetuates the inter-generational transmission of stunting. Children born with low birthweight are more at risk of stunting [7]. However, the effect of prenatal under nutrition can be addressed during the postnatal period through proper child feeding practices [11].

There is most likely a link between children's linear growth and household sanitation practices. The ingestion of high quantities of fecal bacteria by young children through putting soiled fingers or household items in the mouth leads to intestinal infections. This affect children's nutritional status by diminishing appetite, reducing nutrient absorption, and increasing nutrient losses.

The diseases recurrent diarrhoea and intestinal worm infections (helminthiasis) which are both linked to poor sanitation have been shown to contribute to child stunting. The evidence that a condition called environmental enteropathy also stunts children is not conclusively available yet, although the link is plausible and several studies are underway on this topic. Environmental enteropathy is a syndrome causing changes in the small intestine of persons and can be brought on due to lacking basic sanitary facilities and being exposed to faecal contamination on a long-term basis [12].

Research on a global level has found that the proportion of stunting that could be attributed to five or more episodes of diarrhoea before two years of age was 25% [13]. Since diarrhoea is closely linked with water, sanitation and hygiene (WASH), this is a good indicator for the connection between WASH and stunted growth. To what extent improvements in drinking water safety, toilet use and good hand washing practices contribute to reduce stunting depends on the how bad these practices were prior to interventions.

Growth stunting is identified by comparing measurements of children's heights to the World Health Organization 2006 growth reference population: children who fall below the fifth percentile of the reference population in height for age are defined as stunted, regardless of the reason. The lower than fifth percentile corresponds to less than two standard deviations of the WHO Child Growth Standards median.

As an indicator of nutritional status, comparisons of children's measurements with growth reference curves may be used differently for populations of children than for individual children. The fact that an individual child falls below the fifth percentile for height for age on a growth reference curve may reflect normal variation in growth within a population: the individual child may be short simply because both parents carried genes for shortness and not because of inadequate nutrition. However, if substantially more than 5% of an identified child population have height for age that is less than the fifth percentile on the reference curve, then the population is said to have a higher-than-expected prevalence of stunting, and malnutrition is generally the first cause considered.

The Lancet has published two comprehensive series on maternal and child nutrition, in 2008 [6] and 2013 [5]. The series review the epidemiology of global malnutrition and analyze the state of the evidence for cost-effective interventions that should be scaled-up to achieve impact and global targets. In the first of such series [6], investigators define the importance of the 1000 day and identify child malnutrition as being responsible for one third of all child deaths worldwide. This finding is key in that it points at malnutrition as a key determinant of child mortality that is often overlooked. When a child dies of pneumonia, malaria or diarrhea (some of the causes of child mortality in the world), it may well be that malnutrition is a key contributing factor that prevents the body from successfully fighting the infection and recovering from the disease [6]. In the follow up series in 2013, the focus on under nutrition is expanded to the increasing burden of obesity in both high, middle- and low-income countries. Several countries with high levels of child stunting and under nutrition are starting to display worrisome increasing trends of child obesity concurrently, due to increased wealth and the persistence of significant inequalities [5]. The challenges these countries face are particularly difficult as they require intervening on two levels on what has come to be called “double burden of malnutrition”. As an example, in India 30% of children under 5 years of age are stunted, and 20% are overweight. Neglecting these nutritional problems is not an option anymore if countries are to escape poverty traps and provide

opportunities to their people to live fulfilling productive lives without stunting [5].

Lipid disorders include high levels of low-density lipoprotein (LDL) cholesterol, or fats called triglycerides, or both in blood. High levels of these lipoproteins and fats increase the risk for developing heart disease. The early onset of lipid disorders including elevated total or low-density lipoprotein (LDL) cholesterol levels, low levels of high-density lipoprotein (HDL) cholesterol, and high levels of triglycerides (fats) is alarming as these conditions in childhood are predictive of elevated risk for cardiovascular disease in adulthood. Hence based on above findings the present study was planned for Evaluation of Lipid Profile in Stunted Children's of Age Below 5 years from North Bihar Region.

Methodology

The present study was planned in Department of Paediatrics, Darbhanga Medical College & Hospital, Laheraisarai, Bihar, India. Total 100 children's of age below 5 years were enrolled in the present study between July 2019 to December 2019.Out of that the 50 Stunted children's were enrolled in the Group A as Cases and remaining 50 cases were enrolled in Group B as Control patients.

The collected blood samples were centrifuged at 4000 revolutions per minute (rpm) during 5 minutes, and then serums were decanted. Decanted serums were used on the same day to measure lipid parameters. On each blood sample, the following measurements were performed: total cholesterol using cholesterol oxidase method [12], HDL cholesterol by precipitation with phosphotungstic acid in the presence of magnesium ions [13], triglycerides by glycerol phosphate oxidase method [14]. LDL cholesterol was estimated by calculation using the formula of Friedwald *et al*. [15] if triglycerides do not exceed 3.4 g/L. Variation in lipid profile was determined using usual values proposed by Bingen *et al*. [16]

All the patients were informed consents. The aim and the objective of the present study were conveyed to them. Approval of the institutional ethical committee was taken prior to conduct of this study.

Results & Discussion

Diet and activity patterns of children are influenced by family environment and parental obesity has been considered as a major risk for child obesity. Therefore, lifestyle intervention to combat nutrition transition is necessary. Arterial thickening and formation of fatty streaks can be the outcome of metabolic syndrome and if diagnosed at an early stage in childhood, these changes are reversible in children. Thus, early identification of the markers of sub-clinical atherosclerosis is warranted. Moreover, atherosclerotic risk is also more in obese individuals with metabolic syndrome.

Although atherosclerosis manifests clinically in middle and late adulthood, it is known to have a long asymptomatic phase of development, that begins early in life, often during childhood, and is significantly related to dyslipidaemias. Dyslipidemia, characterised by elevated total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), non-high-density lipoprotein cholesterol (non-HDL-C) and triglyceride (TG) levels as well as low high-density lipoprotein cholesterol (HDL-C) concentration, is well-known cardiovascular disease (CVD) risk factor [17].

With respect to lipid profiling for CVD risk assessment, LDL-C levels are widely targeted for primary prevention and intervention. At present, however, some investigators have suggested that non-HDL cholesterol may be superior to LDL cholesterol alone as a predictor of CVD risk factors in adolescents [18] and adults [19], largely because cholesterol-enriched very-low-density lipoprotein and intermediate-density lipoprotein have been shown to be atherogenic in addition to LDL. As for TG, the relationship between TG and CVD risk factors is controversial. In some studies, the relationship is not statistically significant after controlling for other lipids, particularly HDL-C [20]. However, several meta-analyses have concluded that TG is a CVD risk factor independent of HDL-C and other risk factors [21].

Table 1: Demographic Details

Groups	**Group A**	**Group B**
Group of	**Cases**	**Control**
Type of Children's	**Stunted Children's**	**Normal Children's**
Total Cases	**50**	**50**
Parameters		
Age (Years):		
0 – 1 years	7	11
1 – 2 years	15	8
2 – 3 years	16	13
3 – 4 years	5	9
4 – 5years	7	10
Sex		
Males	28	24
Females	22	26

Table 2: Lipid Profile

Groups	**Group A**	**Group B**
Group of	**Cases**	**Control**
Type of Children's	**Stunted Children's**	**Normal Children's**
Total Cases	**50**	**50**
Cholesterol (mg/dl)	172.1 ± 24.5	182.4 ± 29.5
Triglycerides: TG (mg/dl)	67.5 ± 22.6	128.3 ± 31.3
High Density Lipid: HDL (mg/dl)	42.1 ± 8.4	46.2 ± 6.5
Very Low-Density Lipid: VLDL (mg/dl)	51.4 ± 21.7	38.4 ± 7.6
Low Density Lipid: LDL (mg/dl)	81.2 ± 17.6	69.8 ± 16.3

Recent studies involving subjects of different age groups have also shown the importance of non-HDL-C as a reliable, less costly parameter that is strongly correlated with cardiovascular risk because non-HDL-C includes all atherogenic lipid sub fractions [22]. Data from the Bogalusa Heart Study suggest that childhood non-HDL-cholesterol levels persist and best predict adult dyslipidemia and other CVD risks [23]. Another recent study, using data from the Framingham Heart Study, showed that non-HDL-C was a better predictor of cardiovascular disease risk than LDL-C

[24]. These findings are consistent with the findings of the present study, in which non-HDL-C was shown to outperform LDL-C. Another major advantage of non-HDL-C is that it can be accurately calculated in a non-fasting state and is therefore very practical to obtain in clinical practice. In 2011, the Expert Panel on Integrated Guidelines for Cardiovascular Health and Risk Reduction in Children and Adolescents released its summary report, which recommends non-HDL-C as a predictor of CVD risk [25]. In addition, non-HDL-C is included in the diagnostic criteria of metabolic syndrome by Chinese Society of Pediatrics [26].

Lipid profile abnormalities at birth indicate that initial genetic inheritance is already at risk of developing a CVD [27]. For instance, newborn genetic inheritance at birth mentioned by Hales *et al.* [28] for the first time in 1991 would significantly determine the occurrence of CVD. Studies have shown that serum cholesterol level in adult is associated with stunted growth in late pregnancy [29]. There are controversies about many studies conducted on newborns' cord blood lipid profile. Vaziri Esfarjani *et al.* [30] reported an increase in total cholesterol, HDL cholesterol and triglycerides and a decline in HDL cholesterol compared to reference values among Iranian term newborns For Alinaghi Kazemi and Sadeghzadeh [31] there was a relative increase in triglycerides' total cholesterol among Iranian term newborns. Increased total cholesterol and HDL cholesterol have been observed by Pardo *et al.* in Brazilian near-term newborns [32].

Relationship between coronary artery disease and serum cholesterol levels is difficult to evaluate in children because clinically significant coronary artery disease does not occur. On the other hand, early appearance of coronary artery disease in patients with homozygous familial hypercholesterolemia proves that young arteries are not resistant to atherogenic effects of high cholesterol. Coronary artery disease detectable by angiography may occur as early as 18 and 25 years of age in male and female heterozygotes respectively, and as early as 6 years of age in patients of homozygous familial hyper cholestero-lemia [33, 34].

It is the primary responsibility of governments to ensure that policies and actions address the obesogenic environment and to provide guidance and support for optimal development at each stage of the life-course. By focusing attention on these sensitive periods of the life-course, interventions can address specific risk factors, both individually and in combination. Such an approach can be integrated into other components of the maternal neonatal-child health agenda, and to the broader effort to tackle non communicable diseases across the whole population. Nutrition information can be confusing and thus poorly understood by many people. Given that individuals and families choose their diets, the population needs to be empowered to make healthier choices about what to eat and provide their infants and children. This is not possible unless nutrition literacy is universal and provided in a manner that is useful, understandable and accessible to all members of society. It is not sufficient to rely on nutrient labeling or simple codes such as traffic light labels or health star ratings. All governments must lead in developing and disseminating appropriate and context-specific food based dietary guidelines for both adults and children. The necessary information should be provided through media and educational outlets and public health messaging in ways that reach all segments of the population, such that all of society is empowered to make healthier choices. As children enter school, health and nutrition literacy should be included in the core curriculum and supported by a health-promoting school environment

Conclusion

The data generated from the present study concludes that there is an increased risk of metabolic alterations, namely, poor glycaemic control, hypertension, and altered lipid profile that occur in children with stature deficit. Long-term effect of these metabolic alterations may predispose these undernourished children to an increased risk of CAD in future life.

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