Assessment of blood pressure and lipid profile in 1–5 years stunted children attending a tertiary care hospital

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ABSTRACT

Introduction: The prevalence of coronary artery disease (CAD) is rapidly increasing worldwide, particularly affecting low- and middle-income countries with high burden of malnutrition. Childhood stunting signifies chronic undernutrition in early life. There is an increased risk of metabolic alterations, namely, poor glycemic 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. **Aim:** This study aims to assess the blood pressure (BP) and lipid profile in stunted children in the age group of 1–5 years. **Materials and Methods:** The present study was conducted in a tertiary care hospital in eastern region of India. The study comprised 65 stunted children (with height for age [HFA] <−2 standard deviation [SD]) in the cases group and 65 children with normal height and weight as control in the age group of 12 months. Detailed history and physical examination were recorded in a pretested pro forma. BP was recorded with a mercury sphygmomanometer with proper cuff. A 4-h fasting blood sample was collected for analysis of serum lipid profile. **Results:** Among the cases, 70% of children had HFA <−2SD and 30% had HFA<−3SD. The prevalence of elevated BP (particularly diastolic) among the study group was significantly higher than the controls (28% and 6%, respectively) (p=0.006). The mean serum triglyceride was significantly higher (p<0.001) and mean high-density lipoprotein was significantly lower in the case group (p<0.001) than the control group. **Conclusion:** The metabolic changes associated with stunting pose a threat for future cardiovascular disease. Early detection and prevention of stunting and its consequences will decrease the risk of cardiovascular morbidity and mortality in future life.

Key words: *Blood pressure, Lipid profile, Risk of cardiovascular disease, Stunting*

The prevalence of coronary artery disease (CAD) is rapidly increasing worldwide, particularly affecting low- and middle-income countries, where the condition is coexistent with childhood undernutrition and early life infections [1]. The predisposing risk factors to CAD include hypertension (HTN), obesity, diabetes mellitus, and hyperlipidemia [2]. Recent studies have found that children exposed to prenatal and early postnatal undernutrition may develop atherogenic metabolic changes in early life [3-7]. Atherogenic changes in the intimal layer of vessels may begin as early as in the 1st year of life [8]. Consequently, these children are predisposed to the increased risk of CAD in future life.

As per the Global Nutrition Report, 2018, childhood stunting signifying chronic undernutrition in early life, is a significant global health issue, affecting 149 million children under 5 years of age [9]. Stunting is largely the irreversible outcome of inadequate nutrition, infections, and lack of psychosocial care during the first 1000 days of a child's life. Children with stature deficit lose the potential for physical growth, have reduced neurodevelopmental and cognitive function and increased risk of metabolic alterations. These alterations include poor glycemic control, HTN, and altered lipid profile that pose a risk of cardiovascular disease in future life [10]. To bring about a primary prevention of CAD in adulthood, identification and detection of these risk factors and intervention must begin at an early age [11].

There is scanty literature regarding blood pressure (BP) in preschool children with growth retardation and norms of lipid profile in normal preschool children [12,13]. This study aims to assess and compare the BP and lipid profile in stunted children and with normal children in the age group of 1–5 years.

MATERIALS AND METHODS

The present study was conducted in a tertiary care hospital in eastern region of India. A total of 80 children in the age group of 1–5 years with height for age (HFA) <−2 standard deviation (SD) were included as cases and an equal number of children with HFA between mean and +1SD were randomly selected for the control group. Children with weight <−2SD and above +2SD were not included in the study as well as in the control group. Parents were counseled about the study and those children whose parents consented for the study were included in the study.

The final study population comprised 65 (nutritionally) stunted children in the age group of 12–60 months (with HFA<−2SD) and a control group of 65 children with normal HFA. The sample size was calculated with the following formula, $n=z^2\times\sigma^2\div d^2$, where n is the sample size, z is the standard normal variate (at 5% Type 1 error [p<0.05] it is 1.96), σ is the SD, and d is the confidence interval (95%).

Children with non-nutritional causes of stunting were excluded from the study group. Children with diseases such as nephrotic syndrome, chronic glomerulonephritis, chronic renal failure, endocrinopathies (family history of diabetes), and cardiopathies, overweight and obese children, and children with moderate-to-severe malnutrition were excluded from the study, as lipid profile and BP were different in those children. None of the children included in the study had known family history dyslipidemia.

Details of the study were submitted to and approved by the ethical committee. In all children, the measurements, clinical examination, and blood sampling were done in the presence of mothers or guardians with proper consent. Children were weighed on an electronic scale with precision of ±10 g. The lengths of the children younger than 2 years were measured with the aid of an infantometer to the nearest 0.1 cm. The height of children more than 24 months was measured using a wall-mounted vertical stadiometer. The values were compared to those of the WHO child growth standards and expressed as SD score (Z-score).

BP was measured with a mercury sphygmomanometer with an appropriate cuff size. The width of the cuff was approximately 40% of the midarm circumference and length of the cuff covered 80–100% of the arm circumference. During BP measurement, the lower end of the cuff was around 2–3 cm above the antecubital fossa. BP was measured on the right arm of subjects at the heart level in a relaxed, sitting position with uncrossed legs, and after a resting period of 3–5 min. Three measurements were made at 2 min interval and the mean was taken for the study [14]. None of the children had their BP measured previously.

BP values were compared to those of the American Academy of Pediatrics guidelines for high BP in children, 2017 which related BP to age, gender, and height. Children with BP <90th percentile; >90th percentile to <95th percentile;>95th percentile to 95th percentile +12 mmHg, and >95th percentile +12 mmHg were considered to have normal BP, elevated BP, Stage I HTN, and Stage II HTN, respectively [15].

Blood samples were collected aseptically through venipuncture after 4 h of fasting, from children for the estimation of lipid profile. Children were allowed water till 2 h before drawing the blood samples. Estimation of the following was done for the assessment of lipid profile, namely, serum total cholesterol (TC), serum triglyceride (TG), serum high-density lipoprotein (HDL), and serum low-density lipoprotein (LDL) cholesterol. Serum lipid profile estimation was done by calorimetric method. Autoanalyzer used in this tertiary center is Vitros 5600. LDL was calculated by Fredrickson-Friedewald's formula according to which LDL=TC–HDL+VLDL. Values for lipid profiles were considered as per lipid profile norms in the Indian children [12] and the NCEP guidelines [16] as shown in Table 1.

RESULTS

In the study group, there were 38 (58.4%) males and 27 (41.5%) females. Male-to-female ratio was 1.40:1. In the control group, there were 39 (60%) males and 26 (40%) females. Male-to-female ratio was 1.5:1. The mean age of the study group and control group was 36.73±3.64 months and 38.18±3.75 months, respectively. The study and control population belonged to lower socioeconomic status as per modified Kuppuswamy scale.

The mean age and height of the two groups are shown in Fig. 1. Forty-six children (70%) in the study group had HFA<−2SD and 19 children (30%) in the study group had HFA<−3SD. The mean height of the study and control groups was 85.23±2.939 cm and 94.8±2.71 cm, respectively.

The distribution of systolic BP (SBP) and diastolic BP (DBP) is shown in Table 2.

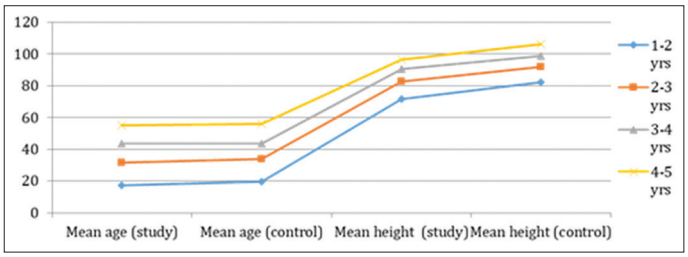

Figure 1: Age-wise distribution of height

Table 1: Normal range of parameters of lipid profile and cutoff limits

Lipid profile	Normal range (mg/dl)	Cutoff limits (mg/dl)
TC	134.5±27.1	>190
TG	91.1±29.85	≥150
LDL	80.1±21.65	≥130
HDL	34.15±13.05	≤20

Statistical calculations were done with SPSS 25 software. TC: Total cholesterol, TG: Triglyceride, LDL: Low-density lipoprotein, HDL: High-density lipoprotein

Table 2: Distribution of BP

Age group (years)	Study group		Control group	
	Mean SBP (mmHg)	Mean DBP (mmHg)	Mean SBP (mmHg)	Mean DBP (mmHg)
1–2	94.14±3.436	53.8±2.225	92.88±3.16	52.9±2.12
2–3	98.57±2.533	58.857±4.111	96.25±2.33	56.63±4.111
3–4	102.4±2.683	62.38±4.610	98.86±2.76	60.5±4.610
4–5	106.18±3.480	66.7±4.690	102.56±2.98	62.6±4.690
Total	100.32±3.03	59.93±3.909	97.63±2.80	58.15±3.57

SBP: Systolic blood pressure, DBP: Diastolic blood pressure

Table 3: Prevalence of elevated BP

Study group				Control group			
Normal BP (<90th p)		Elevated BP ≥90th–<95th percentile		Normal BP (<90th p)		Elevated BP ≥90th–<95th percentile	
Number	%	Number	%	Number	%	Number	%
47	72	18	28	61	94	4	6

BP: Blood pressure

Table 4: Distribution of lipid profile

Parameters of lipid profile	Study group		Mean value	Control group	p-value
	<−2SD	<−3SD			
TC (mg/dl)	151.37±14.81	140.4±10.8	148±14.53	142±12.6	p=0.0263
LDL (mg/dl)	93.96±8.35	88.8±7.72	92.48±8.41	80.6±12.8	p<0.0001
HDL (mg/dl)	35.7±7.06	32.5±4.91	34.8±6.58	43.2±6.8	p<0.0001
TG (mg/dl)	107.71±13.45	96.60±9.59	104.182±13.30	94.1±15.33	p=0.0007

TC: Total cholesterol, TG: Triglyceride, LDL: Low-density lipoprotein, HDL: High-density lipoprotein

The mean SBP and DBP in the study group were 100.32±3.03 mmHg and 59.93 mmHg and in the control group were 97.63 mmHg and 58.15 mmHg, respectively. In the study group, 28% of children had elevated DBP (90^{th}–95^{th} percentile) and 4% of children had both elevated SBP and DBP. In the control group, 6% of children had elevated BP. The prevalence of elevated BP in study group and control group are shown in Table 3.

The distribution of parameters of lipid profile among the study and control groups is shown in Table 4.

DISCUSSION

The knowledge of altered lipid profile and BP in stunted children may help in anticipation, prevention, and understanding the cardiovascular risk factors associated with growth retardation in children. In the study group, 28% of children had elevated DBP (90^{th}–95^{th} percentile) and 4% of children had both elevated SBP and DBP. In the control group, 6% of children had elevated BP. No child in the study group had Stage I or Stage II HTN. Thus, a significant proportion of stunted children has elevated BP, particularly diastolic (p=0.006). This is similar to the studies done by Febba *et al*. and Sawaya *et al*. and lower than the results obtained by Sesso *et al*. [17-19]. Febba *et al*. found that nutritional stunting is associated with high angiotensin-converting enzyme activity in childhood that may contribute to higher BP in these children [17]. Sesso *et al*. found that 42% of children had elevated BP and 29% of children had BP ≥95^{th} percentile which were remarkably greater than those in controls (2%) (p<0.001) [19].

The studies of Lauer and Clarke and Bao *et al*. have shown that adult BP is correlated with childhood BP and that adult essential HTN and cardiovascular diseases begin in childhood [20,21]. Thus, the high prevalence of elevated BP (diastolic) in this study is a relevant finding. Although these subjects cannot be considered hypertensive as yet, they may be more prone to have HTN, cardiovascular, and renal diseases in future life.

In the present study, a mean serum cholesterol 142±12.6 mg/dl, TG=94.1±15.33 mg/dl, HDL=43.2±10.36 mg/dl, and LDL=80.6±12.8 mg/dl were seen in the control group. There is scarce literature on lipid profile among normal Indian children. Khalil *et al*. studied the lipid profile of normal Indian children (3–12 years) and found the mean plasma cholesterol level as 134.5±27.1 mg/dl, mean TG 91.1±29.85 mg/dl, mean HDL 34.15±13.05 mg/dl, and mean LDL 80.1±21.65 mg/dl. No significant difference was noted between the two sexes [12].

The mean serum cholesterol and HDL were found to be much lower in Indian children as compared to Western data as observed by Freedman *et al*. (170 mg/dl and 72.9 mg/dl, respectively) [22]. In a similar study, Chandar *et al*. found that healthy Indian male children (1–4 years) have mean TC, HDL, LDL, and TG levels of 137.0±20.02 mg/dl, 51.8±14.17 mg/dl, 69.1±25.43 mg/dl, and 77.7±20.80 mg/dl, respectively. The mean values of same parameters in the study in female children were 126.0±22.04 mg/dl, 46.8±13.66 mg/dl, 66.4±18.92 mg/dl, and 63.0±12.82 mg/dl, respectively [13].

The mean cholesterol and HDL levels found in the present study were higher than those observed by Khalil *et al*. [12]. However, the HDL levels were lower than those observed by Chandar *et al*. [13]. Lipid levels vary widely with geographical areas, dietary profile, and other sociocultural habits. The TG level in the present study group (94.1±15.33 mg/dl) was similar to that of Khalil *et al*. and higher compared to that found by Chandar *et al*., while the level of LDL found in the present study (80.6±12.8 mg/dl) which was similar to the study of Khalil *et al*. and Chandar *et al*.

The lipid levels found in this study may be representative of the local pediatric population of the same age group. However, various confounding factors could not be excluded. Compared to the control group, the lipid profile in the study group was characterized by higher TG, LDL, and cholesterol levels (p<0.01) and lower HDL levels (p<0.01). Florĕncio *et al*. found that individuals with short stature had higher levels of serum TC, LDL, and TG than adults with normal height [23].

A high prevalence of dyslipidemias (77%) represented by elevated TG level and low HDL level was seen in the present study group. This finding is consistent with similar studies on stunted children by Veiga *et al*. and Alves *et al*. [24,25]. Veiga *et al*. found that almost all children (98.9%) with chronic undernutrition (aged between 12 and 71 months) had dyslipidemia characterized by the predominance of low HDL levels (86.1% of the children) together with hypertriglyceridemia [24]. Similarly, Alves *et al*. found higher TG and low HDL in children from 1 to 6 years with moderate-to-severe stunting [25].

The study by Rohrer *et al*. found that both increased TG levels and low HDL level are correlated, and low HDL is caused by acceleration in catabolism and not by a decrease in the synthesis of these particles [26]. The increased TG level observed among stunted children may be due to the fact that malnourished children have decreased body mass, which leads to a reduction in the amount of LDL, favoring a lower clearance of circulating

TG [27]. It is assumed, therefore, that the high frequency of dyslipidemia (elevations in TC and low HDL) observed in the present study was a consequence of the adaptation to chronic undernutrition and may predict future risk of CAD among stunted children in this region. From this perspective, further studies are necessary to better explain the changes in lipid levels. Lipids and glucose levels were measured from 4 h fasting blood samples in our study. Studies have found that while HDL independent of fasting time [28], TG is influenced by timings [29]. However, new evidence suggests that the impact of fasting on lipids is modest as concluded by Mora [30].

The study design had a few limitations. It was a cross-sectional study with a small sample size. There was a lack of information on dietary pattern. Residual confounding factors such as unmeasured genetic and environmental factors could not be excluded that may have been associated with the study variables.

CONCLUSION

Stature deficit in early childhood is associated with increased risk of elevated BP and metabolic changes that pose a major threat to cardiovascular health in adults. The need for monitoring of BP in malnourished children for timely detection of HTN should be emphasized. Malnutrition should be more often recognized as a factor associated with increased BP in childhood. Awareness and early intervention such as lifestyle and dietary modification will help to prevent this risk.

REFERENCES

1. DeBoer MD, Lima AA, Oría RB, Scharf RJ, Moore SR, Luna MA, *et al*. Early childhood growth failure and the developmental origins of adult disease: Do enteric infections and malnutrition increase risk for the metabolic syndrome? Nutr Rev 2012;70:642-53.
2. Lauer RM, Connor WE, Leaverton PE, Reiter MA, Clarke WR. Coronary heart disease risk factors in school children: The Muscatine study. J Pediatr 1975;86:697-706.
3. Barker DJ. Fetal origins of coronary heart disease. BMJ 1995;311:171-4.
4. Law CM, de Swiet M, Osmond C, Fayers PM, Barker DJ, Cruddas AM, *et al*. Initiation of hypertension *in utero* and its amplification throughout life. BMJ 1993;306:24-7.
5. Hales CN, Barker DJ, Clark PM, Cox LJ, Fall C, Osmond C, *et al*. Fetal and infant growth and impaired glucose tolerance at age 64. BMJ 1991;303:1019-22.
6. Barker DJ, Gluckman PD, Godfrey KM, Harding JE, Owens JA, Robinson JS. Fetal nutrition and cardiovascular disease in adult life. Lancet 1993;341:938-41.
7. Stein CE, Fall CH, Kumaran K, Osmond C, Cox V, Barker DJ. Fetal growth and coronary heart disease in South India. Lancet 1996;348:1269-73.
8. McGill HC Jr., McMahan CA, Zieske AW, Sloop GD, Walcott JV, Troxclair DA, *et al*. Associations of coronary heart disease risk factors with the intermediate lesion of atherosclerosis in youth. The pathobiological determinants of atherosclerosis in youth (PDAY) research group. Arterioscler Thromb Vasc Biol 2000;20:1998-2004.
9. Fanzo J, Hawkes C, Udomkesmalee E, Afshin A, Allemandi L, Assery O, *et al*. Global Nutrition Report: Shining a Light to Spur Action on Nutrition. Bristol, UK: Development Initiatives Poverty Research Ltd.; 2018.
10. de Onis M, Branca F. Childhood stunting: A global perspective. Matern Child Nutr 2016;12 Suppl 1:12-26.
11. Stamler J. Epidemiology of coronary heart disease. Med Clin North Am 1973;57:5-46.
12. Khalil A, Gupta S, Madan A, Venkatesan M. Lipid profile norms in Indian children. Indian Pediatr 1995;32:1177-80.
13. Chandar V, Gidvani CH, Gupta AK, Wilson CG, Sharma YV. Lipid profile in normal healthy children. Med J Armed Forces India 1994;50:101-4.
14. Update on the 1987 task force report on high blood pressure in children and adolescents: A working group report from the national high blood pressure education program. National high blood pressure education program working group on hypertension control in children and adolescents. Pediatrics 1996;98:649-58.
15. Flynn JT, Kaelber DC, Baker-Smith CM, Blowey D, Carroll AE, Daniels SR, *et al*. Clinical practice guideline for screening and management of high blood pressure in children and adolescents. Pediatrics 2017;140:e20171904.
16. National Cholesterol Education Program (NCEP): Highlights of the report of the expert Panel on blood cholesterol levels in children and adolescents. Pediatrics 1992;89:495-501.
17. Febba A, Sesso R, Barreto GP, Liboni CS, Franco MC, Casarini DE. Stunting growth: Association of the blood pressure levels and ACE activity in early childhood. Pediatr Nephrol 2009;24:379-86.
18. Sawaya AL, Sesso R, Florêncio TM, Fernandes MT, Martins PA. Association between chronic undernutrition and hypertension. Matern Child Nutr 2005;1:155-63.
19. Sesso R, Barreto GP, Neves J, Sawaya AL. Malnutrition is associated with increased blood pressure in childhood. Nephron Clin Pract 2004;97:c61-6.
20. Lauer RM, Clarke WR. Childhood risk factors for high adult blood pressure: The Muscatine study. Pediatrics 1989;84:633-41.
21. Bao W, Threefoot SA, Srinivasan SR, Berenson GS. Essential hypertension predicted by tracking of elevated blood pressure from childhood to adulthood: The Bogalusa heart study. Am J Hypertens 1995;8:657-65.
22. Freedman DS, Shear CL, Srinivasan SR, Webber LS, Berenson GS. Tracking of serum lipids and lipoproteins in children over an 8-year period: The Bogalusa heart study. Prev Med 1985;14:203-16.
23. Florêncio TT, Ferreira HS, Cavalcante JC, Stux GR, Sawaya AL. Short stature, abdominal obesity, insulin resistance and alterations in lipid profile in very low-income women living in Maceió, North-Eastern Brazil. Eur J Cardiovasc Prev Rehabil 2007;14:346-8.
24. Veiga GR, Ferreira HS, Sawaya AL, Calado J, Florêncio TM. Dyslipidaemia and undernutrition in children from impoverished areas of Maceió, state of Alagoas, Brazil. Int J Environ Res Public Health 2010;7:4139-51.
25. Alves JF, Britto RP, Ferreira HS, Sawaya AL, Florêncio TM. Evolution of the biochemical profile of children treated or undergoing treatment for moderate or severe stunting: Consequences of metabolic programming? J Pediatr (Rio J) 2014;90:356-62.
26. Rohrer L, Hersberger M, von Eckardstein A. High density lipoproteins in the intersection of diabetes mellitus, inflammation and cardiovascular disease. Curr Opin Lipidol 2004;15:269-78.
27. Brunzell JD, Hazzard WR, Porte D Jr., Bierman EL. Evidence for a common, saturable, triglyceride removal mechanism for chylomicrons and very low density lipoproteins in man. J Clin Invest 1973;52:1578-85.
28. Craig SR, Amin RV, Russell DW, Paradise NF. Blood cholesterol screening influence of fasting state on cholesterol results and management decisions. J Gen Intern Med 2000;15:395-9.
29. Folsom AR, Kuba K, Leupker RV, Jacobs DR, Frantz ID Jr. Lipid concentrations in serum and EDTA-treated plasma from fasting and nonfasting normal persons, with particular regard to high-density lipoprotein cholesterol. Clin Chem 1983;29:505-8.
30. Mora S. Nonfasting for routine lipid testing: From evidence to action. JAMA Intern Med 2016;176:1005-6.

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