Blood Pressure Trend in Children with Chronic Kidney Disease in Nigeria, Sub-Saharan African Region

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Abstract

Background: Various mechanisms are involved in the development of hypertension in children with chronic kidney disease (CKD), some of which are due to the disease process, while some are due to the treatments employed in CKD. **Aim:** The aim of the study was to determine the trend of blood pressure in Nigerian children with CKD comparable with controls. **Materials and Methods:** A cross-sectional study assessing the blood pressure of children from 6 to 17 years with CKD and selected age- and sex-matched controls who were recruited consecutively at the Paediatric Nephrology Clinic and Children Outpatient Clinic of University of Nigeria Teaching Hospital. Data obtained were analyzed using the Statistical Package for the Social Sciences (SPSS) version 17. **Results:** The mean systolic blood pressure (BBP) was 126.08 ± 37.43 mmHg for subjects compared to 105.04 ± 14.33 mmHg for controls, whereas the mean diastolic blood pressure (DBP) was 78.96 ± 22.74 mmHg for subjects compared to 66.25 ± 10.66 mmHg for controls. The distribution of SBP and DBP across the CKD stages showed more severe hypertension (Stages I and II) prevalence in the later stages of CKD (Stages 4 and 5) ($\chi_y^2 = 7.21$, P = 0.01, respectively). The mean value of the mean arterial blood pressure (MAP) was 94.67 ± 26.98 mmHg for subjects compared to 79.18 ± 10.61 mmHg for controls. The increase in MAP across the stages of CKD was significant. **Conclusion:** This study shows a high prevalence of the severe forms of high blood pressure in this group of children with CKD in the subregion.

Keywords: Blood pressure, children, chronic kidney disease, sub-Saharan Africa

NTRODUCTION

Chronic kidney disease (CKD) is defined as the evidence of functional and structural abnormalities (abnormal urinalysis, imaging studies, or histology) lasting for more than three months with or without a decreased glomerular filtration rate (GFR).^[1-5]

CKD is usually associated with hypertension in both children and adults with CKD in developing countries.^[6-9] When left untreated, CKD could cause serious complications and deterioration of clinical outcome.

Analysis by the North American Pediatric Renal Trials and Collaborative Studies had shown that hypertension presents in 48% of patients in the early stage of CKD, and this may persist in 50%–75% of children with uremia and 50%–80% of children with renal allograft recipients.^[10-12]

Several mechanisms have been explained as the pathogenesis of hypertension in children with CKD. This includes the

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activation of the renin–angiotensin–aldosterone system which leads to an increase in vascular tone and resistance. This then leads to sodium retention with fluid overload.^[12,13] There are volume-dependent factors that regulate blood pressure in these patients with CKD.^[12]

There is also the involvement of the volume-independent sympathetic nervous system over activity that plays a strong role in the development of hypertension in these children with CKD.^[12] The afferent signals from the damaged kidney lead to the activation of dopaminergic and leptin pathways in CKD.^[12-14]

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Hypertension is a notable cause of cardiovascular disease with a rising mortality. The prevalence is very high, especially in low-income countries. Due to ignorance, lack of awareness and late presentation and poor control of hypertension in the developing world, there are a reported high number of children with end-organ damage, especially the CKD variety, stemming from hypertension.^[12-14]

Hypertension is a public health emergency. It is used to prognostic the course of cardiovascular and CKDs, being an essential modifiable risk factor for these conditions. The burden of hypertension is variable, with estimated 31.1% in 2010 and approximately 1.38 billion people worldwide.^[12-14] This is even higher in sub-Saharan Africa where prevalence rates of 36.9% and 36.3% were reported in males and females, respectively.^[14]

The impact of hypertension-induced CKD in children necessitated this study which aimed to assess the trend of blood pressure in Nigerian children with CKD. The outcome of this study would become a pedestal for subsequent and more extensive longitudinal and interventional studies.

MATERIALS AND METHODS

Study area

This work was conducted at the Paediatric Nephrology and the Children Outpatient Clinics of the University of Nigeria Teaching Hospital (UNTH), Ituku-Ozalla, Enugu.

The Independent Hospital Research and Ethical Committee of UNTH, Enugu, granted the ethical clearance for this study.

Study design

This was a cross-sectional study assessing blood pressure parameters in children 6–17 years who had features of CKD lasting up to three months. Children who had CKD arising from a primary cardiovascular disease (both congenital and acquired) were excluded from the study.

Patient recruitment

Reagent strip Medi-Combi 10 urine test strips were used for the screening urinalysis of controls, whereas the subjects were children with clinical, laboratory, and radiological features suggestive of the diagnosis of CKD attending the Paediatric Nephrology Clinic. The diagnosis of CKD was made using the following parameters: past or present history suggestive of renal disease or morphological urinary tract abnormality; with unresolving abnormal urinalysis findings (proteinuria of $\geq 1+$; hematuria-microscopic or gross; red blood cell casts and granular casts on microscopy-each of at least three months duration) or radiological features, irrespective of the GFR was required.^[20-26]

Both systolic and diastolic blood pressures were separately classified into normal blood pressure, prehypertension, Stage I hypertension, and Stage II hypertension. Mean arterial pressure (MAP) was determined for each subject/ control.

Data analysis

The data analysis was done using the Statistical Package for the Social Sciences (SPSS) version 17 (Chicago). Simple frequencies and proportions were used to compare demographic data. Systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP) were compared among the study population using the Chi-squared test corrected by Yates' method. The level of significance was set at <0.05 at 95% confidence interval.

RESULTS

Over six months, 9419 children aged 6-17 years were seen in the entire Department of Paediatrics of the University of Nigeria Teaching Hospital, Ituku-Ozalla, Enugu, with 24 cases of CKD seen within this period. This gives an incidence rate of approximately five new cases per million child population per year and a prevalence of 18.4 per million children population. The mean age was 12.33 ± 4.24 years, with 16 males and 8 females each in both study and control groups, giving a male-to-female ratio of 2:1 [Table 1]. Twenty-one (87.5%) of the subjects (21) were in socioeconomic class 3-5, whereas all the controls were of socioeconomic class 1–4 [Figure 1]. Nine (37.5%) subjects were in CKD Stage I, whereas 4 (16.7%) were in Stage 5. The least subjects (2) were seen in CKD Stage 3 [Table 2]. Most of the subjects (10) belonged to the age group of 15-17 years. The least subjects (3) were seen in the age group of 9–11 years [Table 2]. At enrollment, the subjects had a mean SBP of 126.08 ± 37.43 mmHg compared to 105.04 ± 14.33 mmHg for controls, whereas the mean DBP was 78.96 ± 22.74 mmHg for subjects compared to 66.25 ± 10.66 mmHg for controls. The distribution of SBP and DBP of the study population is depicted in Figure 2. The distribution of SBP and DBP across the CKD stages also showed more severe HTN (Stages I and II) prevalence in the later stages of CKD (Stages 4 and 5), with a statistically significant difference in this distribution ($\chi_{p}^{2} = 7.21$, P = 0.01, respectively). This distribution across CKD stages is similar for SBP and DBP, and thus, an example using SBP is depicted in Figure 3. The mean value of the mean arterial blood pressure (MAP) was 94.67 ± 26.98 mmHg for subjects



Figure 1: Distribution of social class in the study population

| Table 1: Age and sex spread of subjects and controls | | | | | | | | | |
|------------------------------------------------------|--------------------|----------------------|---------------------|--------------------|----------------------|---------------------|--|--|--|
| Age range (years) | | Subject | | Control | | | | | |
| | Male, <i>n</i> (%) | Female, <i>n</i> (%) | Total, <i>n</i> (%) | Male, <i>n</i> (%) | Female, <i>n</i> (%) | Total, <i>n</i> (%) | | | |
| 6–8 | 2 (8.3) | 5 (20.7) | 7 (29.1) | 2 (8.3) | 5 (20.7) | 7 (29.1) | | | |
| 9–11 | 2 (8.3) | 1 (4.2) | 3 (12.5) | 2 (8.3) | 1 (4.2) | 3 (12.5) | | | |
| 12-14 | 3 (12.5) | 1 (4.2) | 4 (16.7) | 3 (12.5) | 1 (4.2) | 4 (16.7) | | | |
| 15-17 | 9 (37.5) | 1 (4.2) | 10 (41.7) | 9 (37.5) | 1 (4.2) | 10 (41.7) | | | |
| Total | 16 (66.7) | 8 (33.3) | 24 (100.0) | 16 (66.7) | 8 (33.3) | 24 (100.0) | | | |



Figure 2: Distribution of systolic blood pressure and diastolic blood pressure in subjects and controls. $\chi_{y}^{2} = 0.08$, P = 0.78; df = 1. SBP: Systolic blood pressure, DBP: Diastolic blood pressure

compared to 79.18 \pm 10.61 mmHg for controls. Similar analysis on MAP shows 8 (33.3%) subjects with values in the hypertensive range, with MAP severity distribution across the stages of CKD in subjects typified in Figure 4. This noted more proportion of subjects as hypertensive as stage of CKD progresses to end-stage renal disease (Stage 5), with a sharp increase from CKD Stage 3. The increase in MAP across the stages of CKD was significant.

DISCUSSION

The documented incidence rate of CKD in children aged 6–17 years in this study is comparable to that reported by Odetunde *et al.*^[27] Our documented prevalence is, however, lower when compared to the reports from the Chilean 42.5 per MARP,^[28] Jordan 51 per MARP,^[1,29] Italian 74.7 per MARP,^[30] and the United States 82 per MARP^[31,32] reports. These other studies had a wider spread from all over the index country, giving a better representation of the country's population, unlike the skewed coverage of this hospital-based study.

Elevated blood pressure values (systolic, diastolic, and mean arterial blood pressures) increased in severity as CKD progressed to end-stage renal disease, especially in those on suboptimal doses or no antihypertensive therapy at the time of presentation. The finding is corroborated by other authors on similar subjects.^[6-8,33-38] Recent data from a cohort of patients with CKD indicated that masked hypertension (defined as ambulatory hypertension in the presence of casual blood



Figure 3: Distribution of systolic blood pressure for chronic kidney disease stages in subjects. $\chi_{y}^{2} = 7.21$; $P = 0.01^{*}$. *Significant. CKD: Chronic kidney disease, HTN: Hypertension

pressure <95th percentile)^[39] is present in 25% of children with Stage 2-4 CKD, which is often associated with a two-fold increase in development of complications such as left ventricular hypertrophy.^[39] This validates the need to use ambulatory blood pressure monitoring device for similar studies and even for routine care of this group of children. Changes in indexed SBP are more contributory compared to other blood pressure parameters in causing certain cardiovascular complications in Western children with CKD,^[40] which will require studies to establish such in African children. Furthermore, this study clearly showed that higher severity of blood pressure levels (Stage I and II hypertension) was statistically significant in children with CKD Stages 4 and 5. These are stages of CKD where cardiovascular complications are expected to peak. Hypertension from any cause (primary or secondary) could initiate CKD.[41,42] It could also develop from the disease process of CKD.[12-19] No matter the etiology, if hypertension is not well controlled, it may lead to the progression of CKD to end-stage kidney disease.^[31]



Figure 4: Distribution of MAP for chronic kidney disease stages in subjects. $\chi^2 = 12.88$; $P = 0.01^*$.*Significant. CKD: Chronic kidney disease

This is corroborated in this study by documenting statistically significant levels of the severe forms of hypertension in children with terminal stages of CKD.

Finally, severe forms of hypertension noted as prevalent in African children with CKD could propend or worsen the course of CKD in these children. Careful assessment of all blood pressure parameters with prompt and adequate blood pressure control using appropriate antihypertensive medication is thus highly recommended.

CONCLUSION

This study shows a high prevalence of the severe forms of high blood pressure in this group of children with CKD in the subregion.

Limitations of the study

The low sample size of this study which is limited to a tertiary center may not reflect the true situation in the entire country's population, and thus, a multicenter replication of the same study is recommended. As a cross-sectional study, causal relationships cannot be established. In addition, the study cannot draw conclusions about long-term effects or treatment strategies.

Line of future research

While this article addresses the relationship between CKD and hypertension in children, there is a need for further research and more advanced studies that could explore mechanisms and treatment strategies in more depth. There may be a need to modify the methodology and design a longitudinal cohort study that will follow up these children with CKD, give interventions, and monitor changes in them. More biochemical parameters could also be looked at during the study.

MAP = Mean arterial blood pressure.

Mean arterial blood pressure (MAP) is defined as the average blood pressure during a single cardiac cycle.^[43] It reflects

| Table 2 | 2: Di | stribution | of | chronic | kidney | disease | stages | for |
|---------|-------|------------|----|---------|--------|---------|--------|-----|
| age in | subj | jects | | | | | | |

| Age group (years) (n) | | | | | |
|-----------------------|------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|--|--|
| 6–8 | 9–11 | 12–14 | 15–17 | | |
| 2 | 1 | 1 | 5 | | |
| 2 | 2 | 1 | 1 | | |
| 2 | 0 | 0 | 0 | | |
| 0 | 0 | 2 | 1 | | |
| 1 | 0 | 0 | 3 | | |
| 7 | 3 | 4 | 10 | | |
| | 6–8 2 2 2 0 1 7 | Age grou 6-8 9-11 2 1 2 2 2 0 0 0 1 0 7 3 | Age group (years) (n) 6-8 9-11 12-14 2 1 1 2 2 1 2 0 0 0 0 2 1 0 0 7 3 4 | | |

CKD: Chronic kidney disease

the hemodynamic perfusion pressure of the vital organs. It is believed that a MAP of >60 mmHg is enough to perfuse the internal organs, whereas MAP is hypertensive for value \geq 105 mmHg. Mean arterial blood pressure (MAP) was determined for each subject/control by substituting the average value for both SBP and DBP in the formula: mean blood pressure (MAP) = {DBP + (SBP-DBP)/3}.^[43]

eGFR = Estimated GFR

The GFR in ml/min/1.73 m² was estimated using the Haycock–Schwartz formula^[44] as follows:

$$\frac{K \times Height(cm)}{serum\ creatinine(\frac{mg}{dl})}$$

Where:

 $eGFR = Estimated GFR in ml/min/1.73 m^2$

K = Empirically derived constant relating height to muscle mass

Values for "K" (when plasma or serum creatinine is in mg/dl)

- K = 0.33 for low birth weight infants <one year
- = 0.45 for term AGA infants <one year
- = 0.55 for boys and girls (2–12 years)
- = 0.55 for adolescent females (13–21 years)
- = 0.70 for adolescent males (14–21 years)

Single sample was collected each for all the tests done during the study.

Financial support and sponsorship Nil.

Conflicts of interest

There are no conflicts of interest.

Definition of terms and list of abbreviations

NKF-KDOQI = National Kidney Foundation-Kidney Disease Outcome Quality Initiative

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Relationship between Iron Deficiency Anaemia and Intestinal Helminthiasis among School Age Children in Abakaliki Metropolis

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Abstract

Background: In a world plagued by poverty, ignorance, malnutrition and disease especially in developing countries, children are particularly vulnerable to anaemia. A 2015 report by the WHO reveals that iron deficiency is a major cause of anaemia accounting for about 50% of cases worldwide. The importance of iron in growing children cannot be overemphasised especially with regards to neurologic and cognitive development. Iron deficiency has a negative impact on childhood growth and development. It also affects neuronal energy metabolism, the metabolism of neurotransmitters, myelination, and memory function. These undesirable consequences can be irreversible; thus, early detection through periodic screening exercise will enable prompt intervention. Aims: This community-based cross-sectional study aimed at assessing the prevalence of iron deficiency anaemia (IDA) among primary school children in Abakaliki, Nigeria, and investigating its association with intestinal parasitic infections. Materials and Methods: A total of 372 children aged 6-12 years were enrolled in the study. Sociodemographic data were collected, and stool samples were examined for intestinal parasites. Haemoglobin concentration, serum on, and total iron-binding capacity were measured to determine anaemia and iron deficiency status. Result: The results showed a high prevalence of intestinal parasitic infections had a significantly higher prevalence of IDA compared to those without infections. The study also revealed a significant association between IDA and lower socioeconomic status, as well as maternal and paternal educational attainment. These findings highlight the substantial burden of IDA among primary school children in Abakaliki and the role of intestinal parasitic infections in contributing to iron deficiency. Conclusion: The study emphasises the need for integrated interventions, including health education, improved school meal provision, iron supplementation, regular deworming programmes, and measures to control and prevent intestinal parasitic infections. By addressing these actors, efforts can be made to reduce the prevalence of IDA and its associated health consequences in the population.

Keywords: Intestinal helminthiasis, iron deficiency anaemia, parasitic infection, total iron-binding capacity

INTRODUCTION

The World Health Organisation estimated the prevalence of iron deficiency anaemia (IDA) among school-age children in developed countries to be 5.9% against 48.1% in developing countries.^[1] Iron is one of the most common elements in the earth's crust, yet iron deficiency is the most common cause of anaemia, affecting over 500 million people worldwide.^[2] Iron deficiency is caused by insufficient dietary intake, poor iron absorption, or loss from bleeding.^[3]

Iron status can be considered as a spectrum, with one end being normal iron status with good stores and the opposite end being IDA. In between both ends of the spectrum is iron deficiency without

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anaemia.^[4] Iron deficiency is the result of long-term negative iron balance, which usually goes unnoticed till the stores are depleted, finally leading to anaemia. Iron deficiency anaemia is, therefore, a more severe stage of iron deficiency.^[5] However the deleterious effect on the body begins even before anaemia develops.^[5]

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School age is a vital period of childhood during which rapid neuro-cognitive development occurs.^[3] It is also a period of transition from childhood to puberty. During this phase of life, every part of the body is growing, maturing, and preparing the body for adult life.^[4] All macro and micronutrients are required at high levels to maintain normal homeostasis.^[6] Macronutrient deficiency manifests faster, and attention is more often focused on it.^[3] Micronutrient deficiencies are usually not so obvious, but the consequences can be very disastrous.^[3] Iron deficiency is the most common micronutrient deficiency in many developing countries and has major health, social and economic consequences.^[4]

Aside from anaemia, iron deficiency has non-haematological consequences, including decreased physical work capacity,^[7] decreased athletic performance,^[6,8] depressed immune function,^[9] decreased concentration, impaired cognitive function, and poor school performance.^[10] Others include prematurity and foetal growth retardation in a newborn.^[11] Furthermore, iron-deficient individuals have increased absorptive capacity of divalent heavy metals, such as lead and cadmium, thus increasing the risk of heavy metal poisoning.^[12]

A low serum iron and ferritin with an elevated total iron-binding capacity (TIBC) are diagnostic of iron deficiency.^[13] Although low serum ferritin remains a very sensitive and specific test for assessing iron deficiency, a normal or high level may be an unreliable marker because ferritin is an acute phase reactant and levels might be increased in inflammatory or neoplastic conditions.^[14]

Helminthiasis can cause chronic blood loss and lead to IDA.^[15] Furthermore, intestinal parasites cause inflammation of the gastrointestinal tract and inhibit food absorption which will lead to decreased bioavailability of dietary source of iron.^[15] Stoltzfus *et al.*^[15] assessed the importance of hookworms on the epidemiology of iron deficiency anaemia in Zanzibari school children. The study revealed that infection with malaria, *Trichuris trichiura, Ascaris lumbricoides*, and hookworms were all associated with poor iron status, the association with hookworms being the strongest.^[15] Similar findings were also reported in other studies.^[16,17] It is, therefore, imperative that parasitic infection should be assessed for and treated in order to prevent IDA and make treatment of IDA more efficient.

MATERIALS AND METHODS

The study was a community-based cross-sectional study carried out in Abakaliki, the capital city of Ebonyi State in the southeastern part of Nigeria, between October 25, 2017, and December 8, 2017. The study participants comprised 372 children aged 6–12 years attending primary schools in *Abakaliki metropolis*. Those with a history of haemoglobinopathy, those on iron supplements, those that had received blood transfusion within the previous three months, and those whose parents did not give consent for the study were excluded from the study. Sociodemographic data were obtained using a pretested structured questionnaire, and the socioeconomic class was

determined in accordance with the method described by Olusanya *et al.*^[18] Stool samples were collected and taken to the laboratory of Alex Ekwueme Federal University Teaching Hospital Abakaliki within 2 h for examination. However, if examination could be not done immediately, the stool was preserved with 10% aqueous formalin and polyvinyl alcohol, which were used as fixative. Saline and iodine wet mount of the stool were prepared on a slide. Saline mounts generally demonstrate worm eggs, larvae, trophozoites, and cyst, whereas iodine mounts demonstrate mainly cysts.

All selected participants had their haemoglobin concentration measured on-site using a portable haemoglobinometer (VERI-Q RED haemoglobin meter, Korea). Anaemia was reported as haemoglobin concentration below age, defining values as <11.5 g/dL in children 5–11 years and <12 g/dL in children 12–14 years.^[4] For those with anaemia, further evaluation to ascertain iron deficiency was done. From them, 4 mL of venous blood was collected through the peripheral vein aseptically and dispensed into a plain bottle. The sample was transported to the laboratory within 2 h of collection for analysis. The iron assay and TIBC were analysed using the TECO diagnostic Iron/TIBC reagent set kit (TECO USA, 2013). Iron deficiency anaemia was defined in this study as the presence of anaemia plus low serum iron (<50 µg/dL) and increased TIBC (>450 µg/dL).^[19,20]

Data were entered and analysed using SPSS version 22 (IBM, New York, USA). Results were presented as ratios, means, and proportions, while comparisons were made with the Chi-square test or Fisher's exact applicable. A P < 0.005 was accepted as statistically significant.

RESULTS

A total of 372 (50.8% male and 49.2% female; male: female of 1:0.97) children were enrolled in the study, with a mean age of 8.1 ± 1.6 years. Majority (80.4%) were under 10 years of age. A higher proportion, 146 (39.2%), were from the low socioeconomic class. This is shown in Tables 1 and 2.

The number of children found to be infected with at least one intestinal parasite was 94, with a prevalence of 25.3%. Hookworm was the most common 40 (39.6%) followed by Ascaris species 23 (22.8%). Other intestinal parasites seen with decreasing frequency were Entamoeba histolytica (10.9%), *Trichuris trichuria* (8.9%), Schistosoma Species (6.9%),

| Table 1: Age | and gende | r distributio | n of study | y participants |
|--------------|-----------|---------------|------------|----------------|
| Age (years) | Male | Female | Total | Percentage |
| 6 | 31 | 40 | 71 | 19.1 |
| 7 | 42 | 34 | 76 | 20.4 |
| 8 | 38 | 35 | 73 | 19.6 |
| 9 | 40 | 39 | 79 | 21.3 |
| 10 | 23 | 19 | 42 | 11.3 |
| 11 | 7 | 8 | 15 | 4 |
| 12 | 8 | 8 | 16 | 4.3 |
| Total | 189 | 183 | 372 | 100 |

Taenia Species (5.9%), and Strongyloides species (5%). Table 3 shows the age distribution of intestinal parasitic infestation among study participants. Parasitic infestation was more in children <10 years (78.6%); the most common (29.8%) in those nine years of age, and more among the males, 50 (53.2%).

The prevalence of anaemia among study participants was 40.3%. Of those aged 6–11 years (356), 142 were anaemic while 214 had normal haemoglobin levels (\geq 11.5 g/dL). For those aged 12 years (16), 8 had haemoglobin concentration <12 g/dL and were anaemic by definition. Table 4 shows the age and sex distribution of anaemia among the study participants. Out of the 150 with anaemia, 82 (54.7%) were males and was commoner in those aged 10 years (54.8%). The anaemic children (150) were analysed for serum iron and TIBC, and the results are shown in Table 5. The mean serum iron level and TIBC among the

Table 2: School type, socioeconomic class, and family union type

| Variables | Frequency (%) |
|---------------------------------------------------|---------------|
| Type of school | |
| Public | 195 (52.4) |
| Private | 177 (47.6) |
| Socioeconomic class | |
| Upper | 112 (30.1) |
| Middle | 114 (30.6) |
| Lower | 146 (39.2) |
| Fathers highest educational status | |
| Primary | 64 (17.2) |
| Secondary | 129 (34.7) |
| Tertiary and above | 179 (48.1) |
| Fathers occupation score | |
| 1 (professionals, top civil servants/businessmen) | 58 (15.6) |
| 2 (mid-level civil servants, skilled workers) | 240 (64.5) |
| 3 (unskilled workers earning below minimum wage) | 74 (19.9) |
| Mothers highest educational status/score | |
| Primary (0) | 111 (29.8) |
| Secondary (1) | 161 (43.3) |
| Tertiary (2) | 100 (26.9) |
| Family union type | |
| Monogamy | 356 (95.7) |
| Polygamy | 16 (4.3) |

Table 3: Age and gender distribution of parasitic infestation among study participants

| Age | Freque | Percentage | | |
|---------|--------|------------|-------|------|
| (years) | Male | Female | Total | |
| 6 | 6 | 8 | 14 | 14.9 |
| 7 | 8 | 10 | 18 | 19.2 |
| 8 | 6 | 7 | 13 | 13.8 |
| 9 | 18 | 10 | 28 | 29.8 |
| 10 | 8 | 2 | 10 | 10.6 |
| 11 | 2 | 2 | 4 | 4.3 |
| 12 | 2 | 5 | 7 | 7.4 |
| Total | 50 | 44 | 94 | 100 |

anaemic children were $48.2 \pm 23.3 \,\mu$ g/dL and $479.1 \pm 85.9 \,\mu$ g/dL, respectively. Table 6 shows the analysis of serum iron and TIBC in those with anaemia. A total of 114 had low serum iron (<50 μ g/dL) and 127 had elevated TIBC (>450 μ g/dL). One hundred and one (67.3%) had low serum iron and high TIBC, and these are the subjects who, by definition, in this study had IDA. Therefore, out of the 372 participants, 101 had IDA, giving a prevalence of 27.2%. Iron deficiency anaemia was found more among the eight years olds (34.2%) and the lowest among the 12-year-old (18.8%). About 57% of the children with IDA were male.

Table 7 shows the relationship between sociodemographic characteristics and IDA among study participants. The mean age of children with IDA was 8.09 years \pm 1.56, with male predominance 58 (57.4%). A greater proportion of them attended public schools 57 (56.4%). Over 50% of these children belonged to low socioeconomic class. There was a significant association between the mother's educational attainment and the presence of IDA (*P* = 0.009). Furthermore, the same was seen with father's educational attainment (*P* = 0.048).

Table 8 shows a significant relationship between intestinal parasitic infection and IDA (P = 0.002). About 39% of children with intestinal parasitic infection had IDA.

DISCUSSION

Anaemia is endemic in developing countries, and iron deficiency is the most common micronutrient deficiency, leading to anaemia worldwide.^[21] This is the first study to provide information on the prevalence of IDA in school-aged children in Abakaliki, Ebonyi State, and it clearly demonstrated a high burden of anaemia and iron deficiency among school-aged children in the study area.

This study demonstrated a high burden of anaemia among school-aged children in Abakaliki metropolis. A similar study done in a rural community in Abia state showed a very high prevalence (82%) among school-aged children.^[22] This is clearly a much higher prevalence compared to the current study, and this disparity may be due to its rural community setting as opposed to the current study that was carried out in an urban metropolis. In contrast, a study conducted in Edo state by Osazuwa et al.^[23] reported a lower prevalence of 38%. However, the study involved a wider age range of 1–15 years. In Africa, a study was carried out in six African countries by Hall et al.^[24] and a study done in Kinshasa Zaire by Hedberg et al.^[25] reported a similar prevalence to the index study. Stoltzfus et al.^[15] reported a higher prevalence of 62% among school children in Tanzania. In most developed countries, the prevalence of anaemia is much lower compared to developing countries.^[24] Yip et al.^[26] supported this assumption by reporting a decline in the prevalence of anaemia among American children from 7.8% in 1975 to 2.9% in 1985.

The notable contributor to this high prevalence of anaemia in the index study was iron deficiency, as 67% of the children with anaemia in this study were iron deficient. Another explanation

| Age | Anaemia* | | | | No anaemia | | | Percentage of |
|-------|----------|--------|-------|------|------------|-------|-----|--------------------------|
| | Male | Female | Total | Male | Female | Total | | anaemia per age group |
| 6 | 19 | 14 | 33 | 12 | 26 | 38 | 71 | 46.5 |
| 7 | 16 | 13 | 29 | 26 | 21 | 47 | 76 | 38.2 |
| 8 | 16 | 14 | 30 | 22 | 21 | 43 | 73 | 41.1 |
| 9 | 11 | 12 | 23 | 29 | 27 | 56 | 79 | 29.1 |
| 10 | 15 | 8 | 23 | 8 | 11 | 19 | 42 | 54.8 |
| 11 | 2 | 2 | 4 | 5 | 6 | 11 | 15 | 26.7 |
| 12 | 3 | 5 | 8 | 5 | 3 | 8 | 16 | 50 |
| Total | 82 | 68 | 150 | 107 | 115 | 222 | 372 | |

*Anaemia=Hb <11.5 g/dL for children 6-11 years and Hb <12 g/dL for children 12 years. Hb: Haemoglobin

Table 5: Mean serum iron and total iron-binding capacity values among anaemic children across different age groups (n=150)

| Age (years) | Mean serum iron (ug/dL) | Mean TIBC (ug/dL) |
|-------------|-------------------------|-------------------|
| 6 | 54.1±26.2 | 461.8±104.8 |
| 7 | 40.5±17.5 | 493.4±120.2 |
| 8 | 43.4±21.9 | 479.8±54.7 |
| 9 | 48.2±26.2 | 488.7±59.9 |
| 10 | 57.8±22.6 | 490.0±40.7 |
| 11 | 37.5±5.8 | 460.5±6.0 |
| 12 | 48.3±21.5 | 445.5±113.7 |

TIBC: Total iron-binding capacity

for the high prevalence of anaemia in this study was the high prevalence of intestinal parasitic infection (25%). Most of these participants with iron deficiency did not have iron deficiency anaemia; which is the most severe consequences of iron deficiency. However, if this deficiency is not replenished, over time, iron deficiency anaemia occurs.^[4] Onimawo et al.^[22] in their study among school-aged children reported a much higher prevalence of IDA. Aside from the fact that the study was carried out in a rural community, they had a higher prevalence of intestinal parasitic infection among the children (40% vs. 25%). This high prevalence of helminthiasis might explain the high prevalence of IDA because of the effects of intestinal parasites on the gut leading to blood loss and IDA.^[15] Ughasoro et al.^[27] and Ekwochi et al.^[16] reported a higher prevalence of IDA among preschool children (<six years) in Enugu. In contrast to these, Akodu et al.[28] in Lagos reported a significantly lower prevalence (10%) of IDA. Other African and developing countries also reported higher prevalence compared to that in the current study.^[15,17,29] In developed countries, the trend is different, with a striking contrast in prevalence rate. Halterman et al.^[30] reported a prevalence of 3% of IDA in the United States of America, with peak seen in adolescent girls (8.7%). In Spain, Salas et al.[31] reported a prevalence of 1.7% among children aged six-nine years. These findings reveal that iron deficiency, although lower in developed countries, remains a global problem seen more in developing countries, and the burden is not limited to preschool children. The majority of the children with IDA (82%) in this study were under 10 years old. This is similar to the findings of similar studies.^[17,29,32] This could be attributed to the increased body demands for iron and other trace elements for body development, including immunity, especially during the early childhood periods.[33]

There was a significant relationship between iron deficiency and intestinal parasitic infection in this study. About a quarter of the study population were infected with intestinal parasites, hookworm being the most culpable. About 40% of the children infected with intestinal parasites had iron deficiency anaemia in contrast to 23% of those without intestinal parasitic infection that also had iron deficiency anaemia. Intestinal parasites are known to predispose children to iron deficiency, especially hookworm.^[15] These parasites feed on blood from the gut and cause chronic blood loss.^[15] They can also cause gut inflammation thereby reducing the absorptive ability of the gut.^[34] They also compete for the micronutrients available for absorption, all leading to micro-nutrient deficiencies, including iron.^[34] Stoltzfus et al., in their study, reported a relationship between iron status and malaria, Trichuris trichiura, Ascaris lumbricoides, and hookworms infections.^[15] Hookworm infection was found to have the strongest association with IDA.^[15] Many other studies on iron deficiency also showed a similar association between iron deficiency anaemia and intestinal parasitic infections.[1,17,28]

These findings emphasise the importance of intestinal parasites in the epidemiology of iron deficiency anaemia. Before any interventional programme for the eradication of iron deficiency will succeed, there must be concurrent treatment or prevention of intestinal helminthiasis in school-age children. This was demonstrated by Stoltzfus et al.[35] in a de-worming programme in Zanzibar where they observed that there was a significant reduction in the burden of iron deficiency and moderate-to-severe anaemia in school children in its first year of implementation.

Recommendation and Conclusion

This study has portrayed the burden of IDA among primary school children in Abakaliki. It is evident that iron deficiency is endemic in this age group because the prevalence of IDA, which is the most severe spectrum of iron deficiency, is 27%. Oduneye, et al.: Assessment of iron status and intestinal helminthiasis among school age children

| Table | Table 6: Age distribution of iron deficiency anaemia and analysis of serum iron and total iron-binding capacity ($n=150$) | | | | | | | | |
|--------|-----------------------------------------------------------------------------------------------------------------------------|--------------------|----------------------|-------------------------|-------------------|----------------|-----------------|--------------------------------------------------------|--|
| Age | Frequency of anaemia | Reduced serum iron | Normal serum iron | Increased serum iron | Increased TIBC | Normal TIBC | Reduced TIBC | IDA (anaemia + reduced serum iron + increased TIBC) | |
| 6 | 33 | 22 | 11 | 0 | 24 | 7 | 2 | 18 | |
| 7 | 29 | 26 | 3 | 0 | 24 | 3 | 2 | 21 | |
| 8 | 30 | 26 | 4 | 0 | 27 | 2 | 1 | 25 | |
| 9 | 23 | 19 | 3 | 1 | 21 | 2 | 0 | 18 | |
| 10 | 23 | 12 | 11 | 0 | 21 | 2 | 0 | 12 | |
| 11 | 4 | 4 | 0 | 0 | 4 | 0 | 0 | 4 | |
| 12 | 8 | 5 | 3 | 0 | 6 | 2 | 0 | 3 | |
| Total | 150 | 114 | 35 | 1 | 127 | 18 | 5 | 101 | |
| Normal | range of serum | iron: 50–150 ug/c | IL, Normal range | of TIBC: 250-45 | 50 ug/dL. TIBC: | Total iron-bi | nding capacity | , IDA: Iron deficiency anaemia | |

Table 7: Relationship between sociodemographic factors and iron deficiency anaemia among study participants

| Variable | Presence of IDA, frequency (%) | | | χ^2 | Р |
|---------------------------------------------|--------------------------------|---------------------|------------------------|----------|--------|
| | Yes (<i>n</i> =101) | No (<i>n</i> =271) | Total (<i>n</i> =372) | | |
| Sex | | | | | |
| Male | 58 (30.7) | 131 (69.3) | 189 (100.0) | 2.081 | 0.149 |
| Female | 43 (23.5) | 140 (76.5) | 183 (100.0) | | |
| Type of school | | | | | |
| Public | 57 (29.2) | 138 (70.8) | 195 (100.0) | 0.689 | 0.406 |
| Private | 44 (24.9) | 133 (75.1) | 177 (100.0) | | |
| Father's highest educational status | | | | | |
| Primary | 23 (35.9) | 41 (64.1) | 64 (100.0) | 5.688 | 0.048* |
| Secondary | 39 (30.2) | 90 (69.8) | 129 (100.0) | | |
| Tertiary and above | 39 (21.8) | 140 (78.2) | 179 (100.0) | | |
| Mother's highest educational status | | | | | |
| Primary | 42 (37.8) | 69 (62.2) | 111 (100.0) | 9.456 | 0.009* |
| Secondary | 39 (24.2) | 122 (75.8) | 161 (100.0) | | |
| Tertiary and above | 20 (20.0) | 80 (80.0) | 100 (100.0) | | |
| Family union type | | | | | |
| Monogamous | 97 (27.2) | 259 (72.8) | 356 (100.0) | 0.04 | 0.842 |
| Polygamous | 4 (25.0) | 12 (75.0) | 16 (100.0) | | |
| Social class | | | | | |
| Low | 54 (37.0) | 92 (63.0) | 146 (100.0) | 12.14 | 0.002* |
| Middle | 26 (22.8) | 88 (77.2) | 114 (100.0) | | |
| Upper | 21 (18.8) | 91 (81.2) | 112 (100.0) | | |
| *Statistically significant. IDA: Iron defic | iency anaemia | | | | |

| Table 8: Association between intestinal parasitic infection and iron deficiency anaemia | | | | | | | | |
|-----------------------------------------------------------------------------------------|----------------------|--------------------------------|------------------------|--------|--------|--|--|--|
| Variable Intestinal parasites | Pi | Presence of IDA, frequency (%) | | | Р | | | |
| | Yes (<i>n</i> =101) | No (<i>n</i> =271) | Total (<i>n</i> =372) | | | | | |
| Yes | 37 (39.4) | 57 (60.6) | 94 (100.0) | 9.4828 | 0.002* | | | |
| No | 64 (23.0) | 214 (77.0) | 278 (100.0) | | | | | |

IDA: Iron deficiency anaemia, *Statistically Significant

Therefore, it can be inferred that a larger proportion of the children without anaemia may actually be iron deficient. This calls for a Multi-disciplinary collaboration to fight this preventable nutritional disorder. This includes health education and community awareness on the significance and burden of iron deficiency anaemia, its causes, consequences and preventive measures. Also, there is need to enforce use of iron supplementation among primary school children, at least once in every academic session; regular de-worming among school children; and institute measures to eradicate infection reservoirs of the helminths.

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Conflicts of interest

There are no conflicts of interest.

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