

Identification and management of iron deficiency anaemia in hospitalised children in Durban, South Africa

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Background. Despite iron deficiency anaemia (IDA) being a global challenge, guidelines on identifying and managing children in infection-burdened areas are unclear. Little is known about the investigation and treatment of IDA for hospitalised children in HIV-endemic areas.

Objectives. To determine the prevalence of anaemia in hospitalised children and to describe the factors that impact the identification and management of IDA in an urban area of South Africa (SA).

Methods. A cross-sectional study was conducted at a referral hospital in Durban, SA, from 1 January 2019 to 31 December 2019. A chart review was performed for the clinical and laboratory data of 1 138 hospitalised children between 1 and 5 years old who had full blood count results. Standard statistical analyses were performed, including comparative analyses between those with and without anaemia.

Results. There was a 24% prevalence rate of anaemia (46.2% of whom were moderate-severe). There was a greater prevalence for anaemia in malnourished children ($p < 0.0001$) and those HIV exposed ($p < 0.0001$). Despite 65.9% of anaemic children having microcytic hypochromic anaemia, iron studies were only performed in 12/273 (4.4%), and stool samples were tested in 16/273 (5.9%). The majority (260/273, 95.2%) of all anaemic children had a Mentzer index > 13 , suggesting a high prevalence of IDA. Only 10/273 (3.7%) were provided with iron. Children with microcytic hypochromic anaemia were no different in clinical presentation or outcome to other anaemic children. High numbers (55.1%) were on antibiotics, and this high infection burden may have affected the identification and management of IDA.

Conclusion. Malnourished and HIV-exposed children have a higher prevalence of anaemia. Despite this, $< 10\%$ of children had basic investigations to identify IDA. This study highlights the urgent need to implement guidelines in identifying IDA and providing iron replacement in hospitalised children in areas with high infection burdens, including SA.

Keywords: paediatric, anaemia, iron deficiency, malnutrition, HIV

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Childhood anaemia across the globe is a major public health challenge, with prevalence rates of 20 - 60% noted in sub-Saharan African countries, including South Africa (SA).^[1-3] Iron deficiency anaemia (IDA) is the most common reason for anaemia in children and has been documented to be associated with long-term negative neurocognition.^[4,5] Multiple factors, including poor dietary diversity, especially in lower socioeconomic communities, a high parasitic burden and recurrent infections, have been postulated as possible factors contributing to this high prevalence of iron deficiency in children under 5 years.^[3,6,7]

Children requiring hospitalisation have also been documented to have a high prevalence of IDA.^[8-11] These children specifically have a high incidence of infections and IDA, and this has been documented in both malaria-endemic and non-endemic regions of sub-Saharan Africa.^[4] Hospitalised children have a higher mortality than children in the community, and infectious diseases and anaemia have been associated with this greater mortality.^[12] In sub-Saharan Africa, this is further complicated as children infected with HIV and those with malnutrition have also been shown to have higher rates of IDA.^[13] Hospitalised children generally have access to investigations for anaemia, which could potentially enable timely identification of IDA. However, using only the parameters of microcytosis and hypochromasia is not adequate in identifying all children with underlying IDA.^[8]

The complex interaction of poor nutrition and a high infection burden resulting in a functional iron deficiency has made the development of universally applicable guidelines for identifying and managing iron deficiency challenging.^[14] To bypass the compounding effect of infections on iron measurements, the use of C-reactive protein and hepcidin levels, together with iron biomarkers of serum transferrin, total iron binding capacity (TIBC), serum iron and % saturation, have been advocated.^[15-17] It is unknown whether hospitalised children are being screened for IDA and, if so, which investigations or clinical findings are utilised. This information is particularly lacking in high infection-burdened countries such as SA.

Another dilemma raised is that oral iron supplementation to treat IDA in the presence of concurrent infection is generally not considered useful, as hepcidin upregulation inhibits iron uptake in the gut.^[14] However, a large meta-analysis, conducted as a Cochrane review on iron supplementation in children in malaria-endemic areas, concluded that iron supplementation was safe if malaria prevention and treatment were guaranteed.^[18] As a result, the World Health Organization (WHO) adjusted their recommendations, now advising that daily iron supplementation should be given to all children living in areas with a high prevalence of anaemia.^[19]

Worldwide, there is a concern that iron identification, investigation and replacement in children remains a challenge.^[20] We aim in this study to report the prevalence of anaemia in hospitalised children in an urban area of SA, where there is a low malaria burden but high rates of HIV and malnutrition. We describe further the factors that impact the identification of IDA and the investigation and management of iron deficiency in hospitalised children.

Methods

A cross-sectional study was conducted at a referral tertiary hospital (Victoria Mxenge Hospital (King Edward VIII Hospital)) in Durban, KwaZulu-Natal Province, SA, from 1 January 2019 to 31 December 2019. This 920-bed tertiary/regional-level hospital caters for referred paediatric patients. The HIV antenatal seroprevalence of the population served by this hospital is high at 44.3% (confidence interval (95% CI) 41.6 - 46.7), reflecting a high burden of both HIV-exposed and infected children.^[21] Nearly 10% of all hospitalised children at this hospital have severe acute malnutrition as per WHO classification.^[22] At admission, children typically have routine laboratory tests that include a full blood count (FBC), and this result includes haemoglobin (Hb), mean cell volume (MCV), mean haemoglobin concentration (MCH), a total red cell count (RCC) red cell distribution (RDW), platelet and white cell count. The hospital utilises the National Health Laboratory Service (NHLS) laboratory information data system (TrakCare) as an electronic medical portal to document these requests and results. Children between the ages of 12 months and 5 years, admitted specifically to the paediatric medical wards (thus excluding children admitted for surgical and other non-medical reasons), and who had a FBC test result, were included. Children aged <12 months were specifically excluded, as ex-premature infants are frequently admitted in the first year in such referral hospitals. Children with known inherited haemoglobinopathies and malignancies were excluded. For children with repeat admissions, only the first FBC was recorded. The primary investigator and a study assistant collected clinical and sociodemographic data from patients' medical records. Appendix 1 (<http://coding.samedical.org/file/2330>) is the data collection tool utilised to extract the data.

Anaemia was defined as Hb below the fifth percentile for age. According to WHO criteria, in a child aged 12 months - 5 years, this is haemoglobin <11 g/dL.^[20] The severity of anaemia was classified as mild, moderate and severe when Hb levels were between 10 and 10.9 g/dL, 7 and 9.9 g/dL, and <7 g/dL, respectively. Children with a Hb <11 g/dL were further categorised as having microcytic hypochromic anaemia if the MCV was <80 femtolitres and an MCH <27 picograms. We specifically categorised those children with anaemia into groups with microcytic hypochromic anaemia (MHA) compared with those without MHA. This was done as a low MCV is generally used as a surrogate marker for identifying the need to investigate iron deficiency. Additionally, a Mentzer index (MCV/RCC) of >13 was used as a proxy indicator of presumed iron deficiency, and compared with the serum ferritin level measurement.^[21,22]

The primary investigator verified and entered all data from the completed data collection tools into an Excel (Microsoft, USA) spreadsheet. Statistical analysis was performed using the R Statistical computing software, version 3.6.3 (R Core Team, Austria). The categorical variables were described as counts and percentage frequencies. Comparative analyses were done to determine relationships between clinical characteristics and laboratory measurements. The χ^2 or Fisher's exact test (small frequencies) were used to determine the association between categorical data. The prevalence of anaemia was calculated as a proportion. Medians

and interquartile ranges were used to report continuous variables. Statistical significance was defined as $p < 0.05$.

Ethical approval

Ethics approval from the University of KwaZulu-Natal (UKZN) Biomedical Research Ethics Committee (ref. no. BREC/00003034/2021), with a waiver for informed consent, was obtained to analyse anonymised, routinely collected data. Gatekeeper permissions from the KwaZulu-Natal Department of Health, Victoria Mxenge (King Edward VIII) Hospital and NHLS were obtained before data collection and verification. Adherence to ethical guidelines was ensured throughout the research process.

Results

Of all the hospitalised children with a FBC ($n=1\ 138$), 24% ($n=273$) had a Hb <11 g/dL. Fig. 1 illustrates this breakdown. Of these children, 180 (65.9%) had MHA. Of the 93 (34.1%) anaemic children who did not have MHA, 11 had macrocytic anaemia, and the remainder ($n=82$) had normochromic normocytic anaemia.

Table 1 compares the clinical characteristics of children with and without anaemia. Of all the analysed admissions, 659 (57.9%) were male and 479 (42.1%) were female children ($p=0.298$). Of 1 120 with documented WHO nutritional classifications, 70 (25.6%) anaemic children were malnourished, compared with only 76 (9%) of those without anaemia ($p < 0.0001$). More anaemic children (42.1%, 109/259) than non-anaemic children (25.6%, 210/821) were born to mothers who were HIV positive ($p > 0.0001$). There were no significant differences in the numbers of laboratory-verified HIV-positive children with and without anaemia ($p=0.319$). Utilising the three most common diagnoses, there was no significant difference in the proportions of children admitted with seizures, lower respiratory tract infections and acute gastroenteritis ($p=0.653$).

To determine whether anaemic children with MHA could be identified clinically and whether they were prioritised for investigations for IDA, we compared anaemic children with and without MHA. Table 2 provides the clinical comparisons, and Table 3 the laboratory comparisons. Children with or without MHA had similar ages ($p=0.704$), gender ($p=0.293$) and history of being born prematurely at <36 weeks' gestation ($p=0.165$). These two groups of anaemic children also had similar proportions of malnourished ($p=0.660$) and HIV-exposed ($p=0.727$) children. Only 27.9.9% of all children with anaemia were documented to have pallor. Both anaemic groups also required similar in-hospital management, with 30/273 (11%) requiring invasive ventilation and 150 (55.1%) requiring intravenous antibiotics. Children with and without MHA had similar average length of stays at 4 days ($p=0.420$). The mortality in the two groups was similar ($p=0.768$).

Evaluating the laboratory investigations 147/273 (53.8%) had mild anaemia, and 126 (46.2%) had severe anaemia. There was a difference between the RDW in children with and without MHA ($p < 0.001$). The median albumin was similar ($p=0.200$).

Table 3 provides a comparison of the laboratory investigations.

We analysed the Mentzer index (MCV/RCC) of children with anaemia to identify possible IDA. The mean Mentzer index of all anaemic children was 18.41, with only 13/273 having an MCV/RCC ratio <13, indicating that (260/273, 95.2%) had a high Mentzer index.

Table 4 indicates the investigation done for screening for IDA among all children with anaemia. Only (16/273, 5.9%) had a documented stool investigation done for parasites, and only (12/273, 4.4%) had formal iron studies requested. There was no difference between the requests for both stool ($p=0.806$) and iron studies ($p=0.757$).

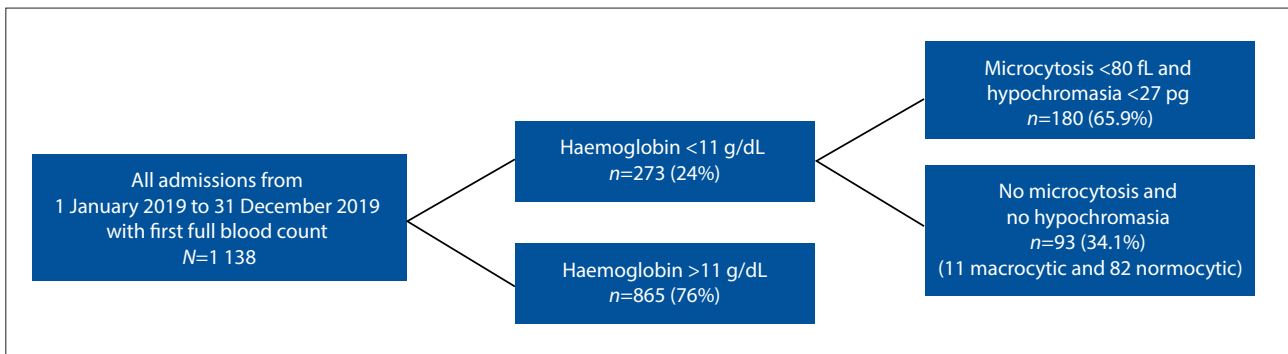


Fig. 1. Breakdown of hospitalised children analysed in the study (N=1 138) (fL =femtolitres; pg = picograms.).

Table 1. Comparison of clinical characteristics of hospitalised children with and without anaemia

Clinical characteristic	No anaemia, n=865, Hb >11 g/dL, n (%)	Anaemia, n=273, Hb <11 g/dL, n (%)	p-value
Gender			
Male, n=659	509 (58.8)	150 (54.9)	0.298
Female, n=479	356 (41.2)	123 (45.1)	
WHO nutritional classification, n=1 120			
OWFA	37 (4.40)	6 (2.2)	>0.0001
NWFA	734 (86.6)	197 (72.2)	
MAM	34 (4)	50 (18.3)	
SAM	42 (5)	20 (7.3)	
HIV exposure, n=1 080			
HIV exposed	210 (25.6)	109 (42.1)	>0.0001
HIV not exposed	611 (74.4)	150 (57.9)	
HIV result, n=323			
HIV negative	106 (83.5)	168 (85.7)	0.319
HIV positive	21 (16.5)	28 (14.3)	
Admission diagnosis*			
Seizures	177 (39.8)	51 (33.1)	0.0653
LRTI	136 (30.6)	53 (34.4)	
AGE	132 (29.7)	50 (32.5)	

*Top three most common.
WHO = World Health Organization; OWFA = overweight for age; NWFA = normal weight for age; MAM = moderate acute malnutrition; SAM = severe acute malnutrition;
LRTI = lower respiratory tract infections; AGE = acute gastroenteritis.

Only 10/273 (3.7%) children were provided with iron treatment, and 50 (18.3%) were provided with multivitamins at discharge. There were no differences among children regarding the provision of iron treatment (p=0.750).

Discussion

In this cohort of hospitalised children between the ages of 1 and 5 years, the prevalence of anaemia was 24%. This is lower than the 42.8%, 42.5% and 38.6% seen in Botswana, India and Vietnam, respectively, in similar-sized paediatric referral hospitals.^[4,8,9] The proportion of anaemic children presenting with moderate to severe anaemia (46.2%) in our study was, however, similar to these cohorts in Botswana (43.9%) and Vietnam (50%).^[4,9] The setting of our study differs from most other countries in terms of much higher HIV exposure rates, and the widescale uptake of antiretrovirals in those children infected with HIV. The prevalence identified in this study was similar to the point prevalence of anaemia (23.4%) seen in slightly older children in a peri-urban disadvantaged community in the same province in SA.^[1]

Our study shows that children with poor nutrition (severe and moderate malnutrition) had a higher prevalence of anaemia, similar to Brazil.^[10] This close association with poor nutrition has

been found previously, largely due to a poor dietary diversity of foods given to children aged >1 year in socioeconomically poor countries.^[6] Our cohort reflects children largely from a lower socioeconomic environment, and the high rates and severity of anaemia demonstrated require urgent attention that should include evaluation and management of IDA in malnourished children. This study also highlights an additional complexity in the increased prevalence of anaemia in HIV-exposed children, most of whom are HIV negative. With the high HIV antenatal seroprevalence, HIV-exposed negative children account for a large pool of children, and further studies are required to establish the aetiology of this anaemia. In our study, HIV-positive children did not have a higher prevalence of anaemia than HIV-negative children, contrary to an earlier study finding.^[12] We postulate that the care of HIV-infected children who receive regular antiretroviral drugs often includes access to micronutrients, and this may account for these findings.

We also looked specifically at all anaemic children with and without microcytosis and hypochromasia, to determine whether specific characteristics may suggest early identification of IDA. We found no specific clinical or laboratory findings other than the RDW that differed between anaemic children with and without MHA. In the context of suspected high rates of infection or

Table 2. Comparison of clinical characteristics of anaemic children

Clinical characteristic	Microcytic hypochromic anaemia, n=180, n (%)*	All other anaemia, n=93, n (%)*	p-value	Overall
Age, months				
Median (Q1 - Q3)	24 (17.8 - 35.0)	26 (18.0 - 36.0)	0.704	24 (18.0 - 35.0)
n (min - max)	180 (1.00 - 58.0)	93 (1.00 - 59.0)		273 (1.00 - 59.0)
Gender				
Female	77 (42.8)	46 (49.5)	0.293	123 (45.1)
Male	103 (57.2)	47 (50.5)		150 (54.9)
Premature birth (<36 weeks' gestation)				
No	171 (95.0)	84 (90.3)	0.165	255 (93.4)
Yes	8 (4.4)	9 (9.7)		17 (6.2)
MUAC				
Median (Q1 - Q3)	15.2 (14.3 - 16.1)	15 (14.0 - 16.0)	0.193	15 (14.0 - 16.0)
n (min - max)	156 (9.00 - 19.0)	78 (9.50 - 22.0)		234 (9.00 - 22.0)
WHO nutritional classification				
OWFA	5 (2.8)	1 (1.1)	0.660	6 (2.2)
NWFA	144 (80)	70 (75.1)		214 (78.4)
MAM	19 (10.6)	14 (15.1)		33 (12.1)
SAM	12 (6.7)	8 (8.6)		20 (7.3)
HIV exposure				
Exposed	69 (38.3)	40 (43.0)	0.727	109 (39.9)
Unexposed	102 (56.7)	48 (51.6)		150 (54.9)
Unknown	9 (5.0)	5 (5.4)		14 (5.1)
Presence of pallor				
No	164 (91.1)	82 (88.2)	0.441	246 (90.1)
Yes	16 (8.9)	11 (11.8)		27 (9.9)
Antibiotics (intravenous)				
Yes	102 (56.7)	48 (52.2)	0.481	150 (55.1)
No	78 (43.3)	44 (47.8)		122 (44.9)
Mortality				
No	172 (95.6)	88 (94.6)	0.768	260 (95.2)
Yes	8 (4.4)	5 (5.4)		13 (4.8)

*Unless otherwise indicated.

MUAC = mid upper arm circumference; WHO = World Health Organization; OWFA = overweight for age; NWFA = normal weight for age; MAM = moderate acute malnutrition; SAM = severe acute malnutrition.

inflammation, the role of a wide RDW (anisocytosis) in identifying IDA requires further exploration. These findings add evidence that neither clinical signs nor utilising microcytosis and hypochromasia are adequate in determining the need to investigate IDA. This is similar to other studies where iron studies identified that 69% of children with normochromic anaemia and 80% of all anaemic children had IDA.^[8,26] These findings reiterate the need to review current practices in identifying IDA in high infection disease-burdened countries. This includes reviewing the reliance on utilising the ubiquitous parameters of a low MCV and a low MCH as key gatekeeping laboratory measures to identify the need for investigating IDA, and considering the Mentzer index and other indices as triggers for investigation.

With the lack of universal guidelines for investigation for iron deficiency, tailor-made options to identify which patients to investigate and what investigations to use have been suggested, especially in infection-burdened contexts.^[27] Hepcidin levels and C-reactive protein adjusted levels have been suggested, in addition to the more commonly available iron studies, including serum iron, ferritin and TIBC.^[13,27] Our study, however, indicates that there seems to be an overall lack of recognition of the need to investigate IDA. The availability of specialised iron studies may not be a factor in the poor investigation

of IDA. This low propensity to identify the need to investigate for IDA in hospitalised children with anaemia, whether it is MHA or not, is of particular concern in the context of the high HIV exposure and malnutrition rates. An expedited review of investigational algorithms is required to determine if this pattern is seen across different contexts in the developing world, and the possible reasons for this failure to investigate IDA in vulnerable populations. Discharge checklists to support the provision of iron for children with anaemia on discharge require widescale uptake and oversight.

Despite the 2016 WHO recommendations for routine iron supplementation in countries with high burdens of iron deficiency, it is widely conducted practice not to start iron among hospitalised children due to the high infection burden.^[9,14,19,26] This seems to be the case in our cohort, where <5% of anaemic children were provided with iron on discharge, and <20% were provided with multivitamins. Over half of this cohort were on intravenous antibiotics during the hospitalisation, suggesting a high infection disease burden. This common practice could be a factor in the poor provision of iron on discharge.

While this study evaluates children admitted to one hospital in SA, and requires additional evaluations in other centres to corroborate these findings, it does reflect a vulnerable population that has not been assessed adequately regarding IDA and anaemia.

Table 3. Comparison of laboratory investigations of children with anaemia

Clinical characteristic	Microcytic hypochromic anaemia, n (%)*	All other anaemia, n=93, n (%)*	p-value	Overall
Severity of anaemia, n=273				
Mild	92 (51.1)	55 (59.1)	0.207	147 (53.8)
Moderate-severe	88 (48.9)	38 (40.9)		126 (46.2)
Red cell distribution width				
Median (Q1 - Q3)	16.6 (15.2 - 18.9)	15.3 (14.3 - 17.2)	<0.001	16.3 (14.9 - 18.4)
n (minimum - maximum)	180 (5.6 - 10.9)	93 (5.50 - 10.9)		273 (0 - 34.8)
Albumin				
Median(Q1 - Q3)	35 (29.3 - 38)	33.5 (23.5 - 38)	0.200	34.0 (28.0 - 38.0)
n (minimum - maximum)	122 (11.0 - 48.0)	62 (9.00 - 45)		184 (9.00 - 48.0)

*Unless otherwise indicated.

Table 4. Investigations and management for iron deficiency in children with anaemia

Clinical characteristic	Microcytic hypochromic anaemia, n (%)	All other anaemia, n (%)	p-value	Overall, n (%)
Stool specimen				
Not requested	169 (93.9)	88 (94.6)	0.806	257 (94.1)
Requested	11 (6.1)	5 (5.4)		16 (5.9)
Iron studies				
Not requested	170 (95)	90 (96.8)	0.757	260 (95.6)
Requested	9 (5.0)	3 (3.2)		12 (4.4)
Iron treatment started during admission or provided at discharge				
Not provided	177 (98.3)	86 (92.5)	0.481	263 (96.3)
Provided	3 (1.7)	7 (7.5)		10 (3.7)
Multivitamins provided at discharge				
Not provided	148 (82.2)	75 (80.6)	0.750	223 (81.7)
Provided	32 (17.8)	18 (19.4)		50 (18.3)

Further research is required, including qualitative studies into the factors influencing decisions to recognise and investigate anaemic children for IDA in disease-burdened contexts. The need for education of healthcare workers is also an intervention highlighted by this study.

Conclusion

A high prevalence of anaemia was identified in hospitalised children between the ages of 1 and 5 years. Malnourished and/or HIV-exposed children have a higher prevalence of anaemia on admission, while HIV-positive children in the post-antiretroviral era seem to have no additional risk. Less than 10% of anaemic children have basic screening for IDA, and very few are provided with iron replacement even on discharge. An urgent review of the implementation of guidelines in the investigation of children for IDA, and for the provision of iron replacement in hospitalised children in SA, is required. Further studies are required in HIV-exposed, HIV-negative children regarding the prevalence of anaemia and possible causes.

Declaration. The data supporting the findings of this study are available from the author(s) on request.

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- Gwetu TP, Taylor M, Chhagan M, Kauchali S, Craib M. Health and educational achievement of school-aged children: The impact of anaemia and iron status on learning. *Health SA* 2019;24:a1101. <https://doi.org/10.4102/hsag.v24i0.1101>
- Safiri S, Kolahi AA, Noori M, et al. Burden of anemia and its underlying causes in 204 countries and territories, 1990 - 2019: Results from the Global Burden of Disease Study 2019. *J Hematol Oncol* 2021;14(1):185. <https://doi.org/10.1186/s13045-021-01202-2>
- Lemoine A, Tounian P. Childhood anemia and iron deficiency in sub-Saharan Africa - risk factors and prevention: A review. *Arch Pediatr* 2020;27(8):490-496. <https://doi.org/10.1016/j.arcped.2020.08.004>
- Siamisang AB, Gezmu AM, Slone JS, et al. Prevalence and associated risk factors of anemia among hospitalised children in a tertiary level hospital in Botswana. *Glob Pediatr Health* 2023;10:2333794X231156059. <https://doi.org/10.1177/2333794X231156059>
- Jáuregui-Lobera I. Iron deficiency and cognitive functions. *Neuropsychiatr Dis Treat* 2014;10:2087-2095. <https://doi.org/10.2147/NDT.S72491>
- Visser M, van Zyl T, Hanekom SM, et al. Associations of dietary diversity with anaemia and iron status among 5- to 12-year-old schoolchildren in South Africa. *Public Health Nutr* 2021;24(9):2554-2562. <https://doi.org/10.1017/S1368980020000543>
- Mamabolo RL, Alberts M. Prevalence of anaemia and its associated factors in African children at one and three years residing in the Capricorn District of Limpopo Province, South Africa. *Curationis* 2014;37(1):1160. <https://doi.org/10.4102/curationis.v37i1.1160>
- Maiti D, Acharya S, Basu S. Recognising missed opportunities to diagnose and treat iron deficiency anemia: A study based on prevalence of anemia among children in a teaching hospital. *J Family Med Prim Care* 2019;8(3):899-903. https://doi.org/10.4103/jfmpc.jfmpc_81_19
- Souvanlasy B, Wiangnon S, Jetsrisuparb A, et al. Iron deficiency anemia among hospitalised children with microcytic anemia hospitalised in national children's hospital in Vientiane, Lao PDR. *Hematol Transfus Med* 2019;29:35-46. <https://he01.tci-thaijo.org/index.php/JHematolTransfusMed/article/view/159485/127421> (accessed 13 February 2024).
- Dos Santos RF, Gonzalez ES, de Albuquerque EC, et al. Prevalence of anemia in under five-year-old children in a children's hospital in Recife, Brazil. *Rev Bras Hematol Hemoter* 2011;33(2):100-104. <https://doi.org/10.5581/1516-8484.20110028>

11. Akin F, Solak ES, Kilicaslan C, Boke SB, Arslan S. Iron deficiency anemia among hospitalised children in Konya, Turkey. *Anemia* 2013;2013:514801. <https://doi.org/10.1155/2013/514801>
12. Jonker FAM, te Poel E, Bates I, Boele van Hensbroek M. Anaemia, iron deficiency and susceptibility to infection in children in sub-Saharan Africa, guideline dilemmas. *Br J Haematol* 2017;177(6):878-883. <https://doi.org/10.1111/bjh.14593>
13. Goosen C, Baumgartner J, Mikulic N, et al. Anaemia, iron and vitamin A status among South African school-aged children living with and without HIV. *S Afr J Child Health* 2022;16(2):105-110. <https://doi.org/10.7196/sajch.2022.v16i2.1897>
14. Kroot JJ, Tjalsma H, Fleming RE, Swinkels DW. Hepcidin in human iron disorders: Diagnostic implications. *Clin Chem* 2011;57(12):1650-1669. <https://doi.org/10.1373/clinchem.2009.140053>
15. Wander K, Shell-Duncan B, McDade TW. Evaluation of iron deficiency as a nutritional adaptation to infectious disease: An evolutionary medicine perspective. *Am J Hum Biol* 2009;21(2):172-179. <https://doi.org/10.1002/ajhb.20839>
16. Thejpal R. Iron deficiency in children. *S Afr Med J* 2015;105(7):607-607. <https://doi.org/10.7196/SAMJNEW.7781>
17. World Health Organization. Use of ferritin concentrations to assess iron status in individuals and populations. Geneva: WHO, 9 August 2023. <https://www.who.int/tools/elena/interventions/ferritin-concentrations#:~:text=A%20ferritin%20concentration%20exceeding%20150,based%20on%20previous%20WHO%20recommendation> (accessed 13 February 2024).
18. Casals-Pascual C, Huang H, Lakhal-Littleton S, et al. Hepcidin demonstrates a biphasic association with anemia in acute *Plasmodium falciparum* malaria. *Haematologica* 2012;97(11):1695-1698. <https://doi.org/10.3324/haematol.2012.065854>
19. Neuberger A, Okebe J, Yahav D, Paul M. Oral iron supplements for children in malaria-endemic areas. *Cochrane Database Syst Rev* 2016;2(2):CD006589. <https://doi.org/10.1002/14651858.CD006589.pub4>
20. World Health Organization. Executive summary. Guideline: Daily iron supplementation in infants and children. Geneva: WHO, 2016. https://iris.who.int/bitstream/handle/10665/204712/9789241549523_eng.pdf?sequence=1 (accessed 13 February 2024).
21. Özdemir N. Iron deficiency anemia from diagnosis to treatment in children. *Türk Pediatri Ars* 2015;50(1):11-19. <https://doi.org/10.5152/tpa.2015.2337>
22. Hoque M, Hoque ME, van Hal G, Buckus S. Prevalence, incidence and seroconversion of HIV and syphilis infections among pregnant women of South Africa. *S Afr J Infect Dis* 2021;36(1):296. <https://doi.org/10.4102/sajid.v36i1.296>
23. Ndlovu S, David-Govender C, Tinarwo P, Naidoo KL. Changing mortality amongst hospitalised children with Severe Acute Malnutrition in KwaZulu-Natal, South Africa, 2009 - 2018. *BMC Nutr* 2022;8(1):63. <https://doi.org/10.1186/s40795-022-00559-y>
24. World Health Organization/United Nations Children's Fund/United Nations University. Iron deficiency anaemia: Assessment, prevention, and control. A guide for programme managers. Geneva: WHO, 2001. https://cdn.who.int/media/docs/default-source/2021-dha-docs/ida_assessment_prevention_control.pdf?sfvrsn=fb8c459c_1&download=true (accessed 13 February 2024).
25. Lawrie D, Glencross DK. Use of the Mentzer index will assist in early diagnosis of iron deficiency in South African children. *S Afr Med J* 2015;105(9):702-703. <https://doi.org/10.7196/samjnew.8589>
26. Sherali A, Ahad A, Tikmani SS, Sohail S. Screening of iron deficiency anemia in children using Mentzer index in Pakistan: A cross sectional study. *Glob Pediatr Health* 2023;10:2333794X221130986. <https://doi.org/10.1177/2333794X221130986>
27. Aguilar R, Moraleda C, Quintó L, et al. Challenges in the diagnosis of iron deficiency in children exposed to high prevalence of infections. *PLoS One* 2012;7(11):e50584. <https://doi.org/10.1371/journal.pone.0050584>

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