

Severe anaemia and invasive bacterial infections in Kenyan children: a 26-year hospital surveillance observational study

Kelvin Mokaya Abuga, John Muthii Muriuki, Agnes M Mutua, Kennedy Mwai Wambui, Neema Mturi, Shebe Mohammed, John Kalama Fondo, Salim Mwarumba, Caroline J Ngetsa, Charles R Newton, Kathryn Maitland, Philip Bejon, Calman A MacLennan, Manfred Nairz, J Anthony G Scott, Thomas N Williams, Sarah H Atkinson

Summary

Background Severe anaemia is a major cause of hospital admission and mortality in children living in sub-Saharan Africa, but data on its association with invasive bacterial infection are sparse. We aimed to characterise severe anaemia and its association with invasive bacterial infection in Kenyan children.

Methods In this retrospective observational study, we systematically collected admission data for children aged 1 month to 14 years admitted to Kilifi County Hospital, Kenya, between Aug 1, 1998, and July 31, 2024. We excluded children with missing haemoglobin data. We examined prevalence, trends, and factors associated with severe anaemia and very severe anaemia, and the association between severe or moderate anaemia and odds of bacteraemia. Anaemia severity was defined using WHO age-specific thresholds.

Findings Of the 84 348 admissions, representing 64 499 unique children (28 232 [43.8%] females and 36 265 [56.2%] males), included in our analysis, 16 715 (19.8%) admissions had severe anaemia. Severe anaemia was associated with multiple, co-occurring risk factors, including sickle cell disease, malaria, HIV, and malnutrition. As malaria transmission declined (2004–09), the age distribution of severe anaemia shifted towards older children; cases in children aged 5–14 years increased from 16.4% during the high-transmission era (1998–2003) to 44.7% during the low-transmission era (2010–24). Compared with mild or no anaemia, severe anaemia was associated with higher odds of in-hospital mortality (adjusted odds ratio 2.1 [95% CI 1.9–2.3]) and bacteraemia (2.7 [2.5–3.0]), particularly with non-typhoidal *Salmonella* (6.1 [4.6–8.1]), *Escherichia coli* (5.7 [4.2–7.7]), *Haemophilus influenzae* (4.3 [3.0–6.4]), *Streptococcus pneumoniae* (3.5 [2.9–4.2]), and *Klebsiella pneumoniae* (2.4 [1.4–4.0]). Moderate anaemia was also associated with increased odds of bacteraemia and mortality.

Interpretation Severe anaemia in hospitalised Kenyan children is multifactorial, increasingly affects older children, and is strongly associated with pathogen-specific bacteraemia and increased mortality. These findings support prompt evaluation for bacteraemia and closer clinical management in severely anaemic children to improve survival.

Funding Wellcome and the Science for Africa Foundation to the DELTAS Africa programme.

Copyright © 2026 The Author(s). Published by Elsevier Ltd. This is an open access article under the CC BY license (<http://creativecommons.org/licenses/by/4.0/>).

Introduction

Severe anaemia is a preventable, life-threatening condition affecting 8.7 million children younger than 5 years globally.¹ Much of the burden is in sub-Saharan Africa, where severe anaemia is found in 9% to 40% of febrile paediatric admissions and between 6% and 30% of in-hospital deaths.^{2–4} Among children admitted with severe anaemia, post-discharge mortality is high, ranging from 2% to 19%, and survivors face an increased risk of hospital readmission and developmental disorders.^{4–6} Previous studies have reported several potential causes of severe anaemia, including infectious, genetic, and nutritional factors.^{2–4} Reductions in *Plasmodium falciparum* transmission, especially between 2000 and 2010,^{7,8} might be expected to reduce the prevalence of severe anaemia. However, epidemiological evidence remains scarce, especially from sub-Saharan Africa.¹

Bacteraemia is also a major contributor to child admission and mortality in sub-Saharan Africa, accounting for 12% to 33 of in-hospital deaths.^{9,10} Experimental evidence from animal and in-vitro studies suggests that severe anaemia might increase risk of invasive bacterial infections.^{11,12} However, relatively few studies have explored this relationship, especially in populations with high burdens of severe anaemia. Existing studies have largely focused on associations between severe anaemia and either all-cause bacteraemia or specific pathogens, such as non-typhoidal *Salmonella* (NTS).^{2,13} These studies elucidate pathophysiological elements, but do not provide a holistic epidemiological description due to small sample sizes, narrow age groups, and inconsistencies in the diagnostic criteria used to define severe anaemia.¹²

In this study, we aimed to characterise severe anaemia in the context of changing malaria epidemiology and



Lancet Glob Health 2026

Published Online
<https://doi.org/10.1016/j.langlo.2026.103976>

See [Comment](https://doi.org/10.1016/j.langlo.2026.103995) <https://doi.org/10.1016/j.langlo.2026.103995>

For the Swahili translation of the abstract see [Online](#) for appendix 1

Kenya Medical Research Institute, Centre for Geographic Medicine Research, KEMRI-Wellcome Trust Research Programme, Kilifi, Kenya (K M Abuga PhD, J M Muriuki PhD, A M Mutua MSc, K M Wambui PhD, N Mturi MRCPC, S Mohammed HND, S Mwarumba MSc, C J Ngetsa MSc, Prof C R Newton MD Res, Prof K Maitland MMed PhD, Prof P Bejon FMedSci, Prof J A G Scott FMedSci, Prof T N Williams FMedSci, Prof S H Atkinson PhD); Open University, KEMRI-Wellcome Trust Research Programme-Accredited Research Centre, Kilifi, Kenya (K M Abuga, A M Mutua); Kilifi County Referral Hospital, Kilifi, Kenya (J K Fondo MMed); Modernising Medical Microbiology, University of Oxford, Oxford, UK (Prof P Bejon); Department of Psychiatry, University of Oxford, Oxford, UK (Prof C R Newton); Centre for Tropical Medicine and Global Health UK, University of Oxford, Oxford, UK (Prof P Bejon, Prof T N Williams, Prof S H Atkinson); Jenner Institute, Nuffield Department of Medicine, University of Oxford, Oxford, UK (Prof C A MacLennan FRCP DPhil); Department of Immunology and Immunotherapy, College of Medical and Dental Sciences, University of Birmingham, Birmingham, UK (Prof C A MacLennan); Department of Internal Medicine II, Medical University

Innsbruck, Innsbruck, Austria
(M Nairz MD); Department of
Infectious Disease
Epidemiology, London School of
Hygiene & Tropical Medicine,
London, UK (Prof J A G Scott);
Institute of Global Health
Innovation, Department of
Surgery and Cancer, Imperial
College London, London, UK
(Prof K Maitland,
Prof T N Williams); Department
of Paediatrics, University of
Oxford, Oxford, UK
(Prof S H Atkinson)

Correspondence to:
Prof Sarah H Atkinson, Kenya
Medical Research Institute,
Centre for Geographic Medicine
Research, KEMRI-Wellcome Trust
Research Programme,
PO Box 230-80108, Kilifi, Kenya
satkinson@kemri-wellcome.org

Research in context

Evidence before this study

Severe anaemia is prevalent among children living in sub-Saharan Africa, yet important gaps persist in understanding its association with invasive bacterial infection. We searched PubMed and Embase from database inception to Nov 28, 2025, using the keywords ("severe anaemia" OR "severe anemia") AND ("bacteraemia" OR "bacteremia" OR "Salmonella" OR "Escherichia coli" OR "Streptococcus pneumoniae" OR "Staphylococcus aureus" OR "Haemophilus influenzae" OR "Klebsiella" OR "Acinetobacter" OR "Pseudomonas aeruginosa"), with no restrictions on language. Of the 246 non-duplicate records identified, 21 were considered directly relevant to the research question. Previous studies on severe anaemia and bacteraemia in African children have been largely limited by small sample size, restricted to children younger than 5 years, or selected data for a predominant focus on malaria. Although a link between severe anaemia and non-typhoidal *Salmonella* (NTS) bacteraemia has been established, there is limited evidence regarding the association of severe anaemia with other bacterial pathogens.

Added value of this study

Drawing on 26 years of systematically collected data from more than 84 000 paediatric admissions aged 1 month to 14 years in coastal Kenya, we provide a comprehensive epidemiological analysis of severe anaemia and its association with bacteraemia in a high-burden setting. Our dataset includes 16 715 children with severe anaemia, and 3655 with blood culture-confirmed bacteraemia. We show that multiple, co-occurring factors are associated with severe anaemia, including malaria, malnutrition, sickle cell disease, HIV infection, and bacteraemia, and that after a decline in malaria transmission, 44·7% of severely anaemic

children are older than 5 years. Children with severe anaemia had higher odds of in-hospital mortality. We confirm a strong association between severe anaemia and NTS bacteraemia and show that severe anaemia is also associated with bacteraemia due to *Escherichia coli*, *Haemophilus influenzae*, *Streptococcus pneumoniae*, and *Klebsiella pneumoniae*. Each 1 g/dL decrease in haemoglobin levels was associated with a stepwise increase in odds of bacteraemia (except in those with very severe anaemia) and this pattern persisted whether the child had malaria, malnutrition, or HIV infection. Severe anaemia also remained strongly associated with bacteraemia during periods of both high and low malaria prevalence.

Implications of all the available evidence

The aetiology of severe anaemia in African children is multifactorial, and it has profound consequences for survival and long-term health. Our findings show that severe anaemia is strongly associated with increased odds of bacteraemia with specific pathogens. Integrating infection risk into initial assessment of severe anaemia might enhance risk stratification and improve patient outcomes. The shifting burden towards older children, and the persistently high prevalence of severe anaemia despite reductions in malaria transmission, suggest that strategies designed primarily for young children and focused on malaria alone are no longer sufficient. Comprehensive approaches that integrate improved diagnosis and management of sickle cell disease, nutritional interventions, HIV care, malaria control, and management of bacteraemia are essential to reducing the burden of severe anaemia and improving child survival in sub-Saharan Africa.

comprehensively investigate its association with a broad range of bacterial pathogens over an extended epidemiological timeline. We also aimed to investigate whether risk of bacteraemia extends across the full clinical spectrum of anaemia severity, and which underlying aetiologies of severe anaemia confer the greatest risk, using hospital surveillance data across a 26-year period.

Methods

Study background and participants

This retrospective observational study used systematically collected hospital surveillance data from all children aged 1 month to 14 years admitted to Kilifi County Hospital (KCH), Kenya, between Aug 1, 1998, and July 31, 2024. KCH is a secondary-level referral hospital located in Kilifi, a rural, malaria-endemic region approximately 3° south of the equator along the Kenyan coast. The paediatric wards at KCH admit around 4000 children annually and include a 70-bed general ward and a 15-bed high-dependency unit.

Surrounding KCH is the Kilifi Health and Demographic Surveillance System (KHDSS), established in 2000 to monitor births, deaths, and migration across an 891 km²

area.¹⁴ In this area, the prevalence of anaemia was 70·0% among children aged 0–7 years between 2005 and 2016.¹⁵ In 2007, the incidence of bacteraemia in the KHDSS was 1·45 per 1000 child-years (95% CI 1·23–1·69).¹⁶ *Haemophilus influenzae* type b (Hib) and ten-valent pneumococcal (Pneumosil PCV10) conjugate vaccines were introduced into routine immunisation schedules in November 2001 and January, 2011, respectively.¹⁷ Over the study period, insecticide-treated bednets and effective combination anti-malarial treatment became widely available in the KHDSS. Although the RTS,S and R21 malaria vaccines were tested in clinical trials within the KHDSS,¹⁸ they have not been implemented in routine immunisation programmes.

This study analysed de-identified data, with ethical approval granted by the Scientific Ethics Review Unit of the Kenya Medical Research Institute (KEMRI/SERU/CGMR-C/155/3857 and KEMRI/SERU/CGMR-C/269/4545). The study is reported in accordance with STROBE guidelines.

Procedures

Since Aug 1, 1998, children presenting to KCH have undergone systematic collection of clinical and laboratory

data, including routine complete blood counts, malaria blood films, clinical biochemistry, and blood cultures (except for children admitted with minor trauma or for elective surgery). From Feb 1, 2005, all ward admissions were routinely tested for HIV infection. Study clinicians completed a structured case record form, capturing age, sex, residence, clinical history, vital signs, and physical examination for each patient. Sex was self-reported by participant or caregiver (male or female). Additional investigations were performed as clinically indicated. Children with severe illness, such as impaired consciousness or deep breathing, were admitted to the high-dependency unit and underwent an extended biochemical evaluation, including blood gas analysis. Treatment protocols for hospitalised children followed national guidelines and WHO recommendations.¹⁹

Full blood count (Beckman Coulter, Brea, CA, USA), *P falciparum* malaria detection (microscopy examination of thick and thin Giemsa-stained blood films), and HIV-1 serological testing (rapid antibody tests) were performed using standard methods. Blood culture samples were collected in BACTEC Peds Plus bottles and processed with a BACTEC automated blood-culture system (Becton-Dickson, BD Biosciences, Franklin Lakes, NJ, USA), as previously described.¹⁰ Positive cultures were subcultured on standard media, and suspected pathogens were confirmed using serological and biochemical test kits (API, bioMérieux, Marcy-l'Étoile, France). External quality assurance was provided by the UK National External Quality Assessment Service.

Malaria was defined as the presence of *P falciparum* asexual parasites on peripheral blood film. Nutritional status was assessed using standard anthropometric indices. Wasting, stunting, and underweight were defined as Z scores of weight-for-height (WHZ), height-for-age, and weight-for-age of less than -2, respectively.²⁰ Body mass index-for-age (BAZ) of less than -2 (age ≥60 months), mid-upper arm circumference (MUAC) of less than 11.0 cm (age 6 weeks to 6 months), and MUAC of less than 12.5 cm (age 6–60 months) were also considered to indicate wasting.²¹ Severe acute malnutrition was defined as WHZ of less than -3, BAZ of less than -3 (age ≥60 months), MUAC <11.0 cm (age 6 weeks to 6 months), MUAC of less than 11.5 cm (age 6–60 months), or the presence of bilateral oedema.²² Malnutrition was defined as the presence of either wasting, stunting, underweight, or severe acute malnutrition. Patients were classified as having sickle cell disease (SCD) if they had a documented clinical diagnosis in their admission or discharge notes or were homozygous for the sickle gene through retrospective testing using electrophoresis or PCR. Children diagnosed with SCD or HIV received specialised care through dedicated follow-up clinics.

Outcomes

We first examined prevalence, trends, and factors associated with severe anaemia, defined as haemoglobin of less

than 7 g/dL (age 1–59 months) or haemoglobin of less than 8 g/dL (age 5–14 years), following the WHO²³ and Global Burden of Disease Anaemia Collaborators²⁴ thresholds. We also report prevalence and trends for very severe anaemia, defined as haemoglobin of less than 5 g/dL, a threshold applied in some studies of severe anaemia.² Moderate anaemia was defined as haemoglobin 7.0–9.9 g/dL (age 1–5 months), 7.0–9.4 g/dL (age 6–23 months), 7.0–9.9 g/dL (age 24–59 months), or 8.0–10.9 g/dL (age 5–14 years); and mild anaemia as haemoglobin 10.0–10.9 g/dL (age 1–5 months), 9.5–10.4 g/dL (age 6–23 months), 10.0–10.9 g/dL (age 24–59 months), 11.0–11.4 g/dL (age 5–11 years), or 11.0–11.9 g/dL (age 12–14 years; appendix 2 p 4).

We then examined the association between severe or moderate anaemia and odds of bacteraemia, defined as the isolation of pathogenic bacterial organisms from blood cultures. Blood cultures yielding contaminant organisms were classified as negative. Contaminants were defined as organisms not typically associated with bloodstream infections among children admitted from the KHDSS, including *Bacillus* species, *Micrococcus* species, viridans group *Streptococcus*, coagulase-negative *Staphylococcus*, and coryneform bacteria.

Statistical analysis

Data was analysed using STATA 15.1. All hospitalised children aged 1 month to 14 years during the study period were eligible. Malaria transmission in the KHDSS was categorised as high (1998–2003), declining (2004–08), and low (2009–24).⁸ We excluded admissions with missing haemoglobin data. The excluded admissions were younger and less likely to have SCD, malaria, malnutrition, or HIV (appendix 2 p 5). In-hospital mortality calculations included only patients with a documented outcome.

Categorical variables were summarised as counts and percentages, and continuous variables as medians and IQRs. We used cluster-robust univariable and multivariable logistic and linear regression models with standard errors clustered at the child level to determine factors associated with moderate or severe anaemia, and to examine associations with bacteraemia and all-cause in-hospital mortality. Multivariable models adjusted for age, sex, number of hospital admissions, and year of admission. Because bacteraemia prevalence did not differ between mild and no anaemia (appendix 2 p 6), these categories were combined as the reference group. Secondary analyses stratified admissions by age group, putative aetiology of severe anaemia, consecutive 5-year intervals, or malaria transmission period. Sensitivity analyses were restricted to first admissions and used standard logistic regression.

Population attributable fractions (PAF) were from multivariable regression models using the punaf STATA package, which estimates PAFs directly from model predictions.²⁵ Temporal trends were assessed using locally estimated scatterplot smoothing. Proportional Venn diagrams illustrated overlapping causes of severe or moderate

See Online for appendix 2

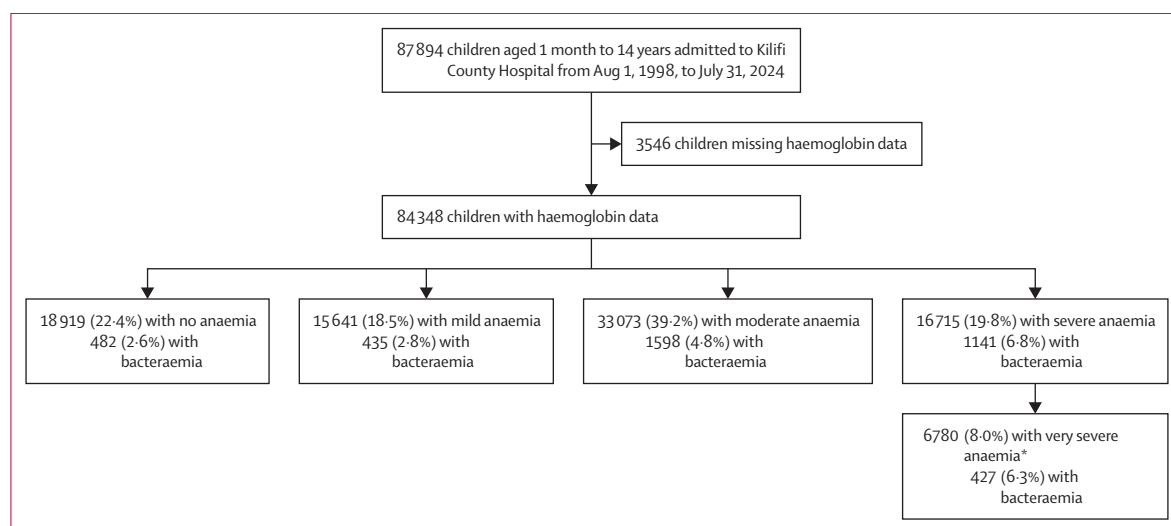


Figure 1: Study flow chart

*Very severe anaemia is a subset of severe anaemia.

anaemia and their associated in-hospital mortality. Data completeness exceeded 95% for most variables, except HIV status (routinely measured from 2005; appendix 2 p 7). Missing data were assumed to be missing at random, and no imputation was performed. All reported p values are two-sided.

Role of the funding source

The funders of the study had no role in study design, data collection, data analysis, data interpretation, or writing of the report.

Results

Between Aug 1, 1998, and July 31, 2024, 87 894 children aged 1 month to 14 years were admitted to KCH. After excluding 3546 (4%) admissions due to missing haemoglobin data (figure 1), 84 348 admissions were included in the analysis, representing 64 499 unique children (28 232 [43.8%] females, 36 265 [56.2%] males, and two [<0.1%] with missing data): 52 259 with a single admission and 12 240 with multiple admissions (appendix 2 p 8). Anaemia was identified in 77.6% (n=65 429) of admissions: 16 715 (19.8%) had severe anaemia, 33 073 (39.2%) had moderate anaemia, and 15 641 (18.5%) had mild anaemia (table). Children with severe anaemia were older, with a median age of 28.6 months (IQR 13.0–64.8), than children without severe anaemia (21.4 months [9.4–46.1]; $p<0.0001$). The proportion of severe anaemia admissions spanned from 14.9% in infants aged 1–11 months to 27.8% in children older than 5 years (appendix 2 p 18). The probability of transfusion rose progressively from 1.1% among children with mild and no anaemia on admission to 68.7% among children with very severe anaemia.

Overall, in-hospital mortality was 5.5% and remained relatively stable throughout the study period (appendix 2 p 19). In-hospital mortality increased with anaemia severity:

4.3% in children with mild and no anaemia, 5.6% in moderate, and 7.8% in severe anaemia. Severe anaemia (adjusted odds ratio [aOR] 2.1 [95% CI 1.9–2.3]) and moderate anaemia (1.4 [1.3–1.5]) were associated with greater odds of in-hospital mortality compared with mild or no anaemia. The association between severe anaemia and mortality remained robust in sensitivity analyses stratified by 5-year intervals (appendix 2 p 19) and malaria transmission eras (appendix 2 p 9).

The proportion of hospitalised children with severe anaemia declined from 34.7% in 1998 to 11.1% in 2024 (figure 2A), coinciding with a decline in severe malarial anaemia (figure 2B). The pattern of in-hospital mortality by grade of anaemia remained similar throughout the study period (figure 2C). The age profile of children with severe anaemia shifted towards older ages as malaria has declined (figure 2D), a trend likely driven by the increasing age of children with malnutrition and SCD (appendix 2 p 20), and a reduction in malaria-associated severe anaemia over time (figure 3). Children admitted with severe anaemia were more likely to present with severe anaemia again in subsequent hospital admissions compared with children admitted with moderate or mild and no anaemia (appendix 2 p 10). The proportion of severe anaemia admissions of children aged 5–14 years has increased over time, being 16.4% in the period of high malaria transmission (1998–2003), 21.6% during the period of declining transmission (2004–09), and 44.7% in the period of low malaria transmission (2010–24; appendix 2 p 11).

Severe anaemia was associated with multiple, overlapping factors, each associated with different in-hospital mortality rates (figure 3A). Malaria, malnutrition, and SCD were associated with most severe anaemia admissions, but 8.3% (n=1383) of cases were not associated with any of these three conditions. The relative contributions of these factors have also shifted over time. Because malaria

	Mild and no anaemia (n=34 560; reference)*	Moderate anaemia (n=33 073)*	Severe anaemia (n=16 715)*	Very severe anaemia (n=6780)*	p value (vs mild and no anaemia)†		
					Moderate anaemia	Severe anaemia	Very severe anaemia
Clinical characteristics							
Haemoglobin, g/dL	11·0 (10·3–11·8)	8·8 (8·1–9·4)	5·4 (4·1–6·4)	3·8 (3·0–4·4)	<0·0001	<0·0001	<0·0001
Age, months	21·8 (9·7–46·4)	20·9 (9·0–45·7)	28·6 (13·0–64·8)	28·8 (13·1–62·5)	0·059	<0·0001	<0·0001
Sex							
Female	14 958/34 560 (42·3%)	14 049/33 073 (42·5%)	7641/16 715 (45·7%)	3240/6780 (47·8%)	Ref	Ref	Ref
Male	19 601/34 560 (56·7%)	19 022/33 073 (57·5%)	9074/16 715 (54·3%)	3540/6780 (52·2%)	0·0053	0·0005	<0·0001
Fever‡	17 270/34 560 (50·0%)	20 125/33 073 (60·9%)	10 337/16 715 (61·9%)	3889/6780 (57·4%)	<0·0001	<0·0001	<0·0001
Vomiting	10 426/30 426 (30·6%)	8796/32 478 (27·1%)	4907/16 403 (29·9%)	2110/6649 (31·7%)	<0·0001	0·20	0·11
Jaundice§	209/34 560 (0·6%)	565/33 073 (1·7%)	1181/16 715 (7·1%)	569/6780 (8·4%)	<0·0001	<0·0001	<0·0001
Coma¶	2121/31 544 (6·7%)	2589/28 659 (9·0%)	1637/13 752 (11·9%)	612/5507 (11·1%)	<0·0001	<0·0001	<0·0001
Severe pneumonia	7994/34 560 (23·1%)	8175/33 073 (24·7%)	3104/16 715 (18·6%)	1268/6780 (18·7%)	<0·0001	0·11	0·49
Very severe pneumonia**	3544/34 560 (10·3%)	3328/33 073 (10·1%)	1944/16 715 (11·6%)	875/6780 (12·9%)	0·88	<0·0001	<0·0001
Malnutrition††	17 855/34 560 (51·7%)	19 106/33 073 (57·8%)	11 503/16 715 (68·8%)	4831/6780 (71·3%)	<0·0001	<0·0001	<0·0001
Wasting‡‡	10 565/33 553 (31·5%)	11 210/31 981 (35·1%)	6813/16 093 (42·3%)	2828/6582 (43·0%)	<0·0001	<0·0001	<0·0001
Stunting§§	10 384/32 597 (31·9%)	12 053/31 088 (38·8%)	7616/15 780 (48·3%)	3279/6472 (50·7%)	<0·0001	<0·0001	<0·0001
Underweight¶¶	11 971/33 020 (36·3%)	13 495/31 694 (42·6%)	8612/16 044 (53·7%)	3658/6557 (55·8%)	<0·0001	<0·0001	<0·0001
Severe acute malnutrition	5922/34 560 (17·1%)	7084/33 073 (21·4%)	4260/16 715 (25·5%)	1788/6780 (26·4%)	<0·0001	<0·0001	<0·0001
Transfused	396/34 560 (1·1%)	783/33 073 (2·4%)	6088/16 715 (36·4%)	4659/6780 (68·7%)	<0·0001	<0·0001	<0·0001
In-hospital mortality	1475/34 560 (4·3%)	1849/33 073 (5·6%)	1294/16 715 (7·8%)	556/6780 (8·2%)	<0·0001	<0·0001	<0·0001
Laboratory characteristics							
Bacteraemia	917/34 560 (2·7%)	1598/33 073 (4·8%)	1141/16 715 (6·8%)	427/6780 (6·3%)	<0·0001	<0·0001	<0·0001
<i>Plasmodium falciparum</i> malaria	4792/34 560 (13·9%)	9226/33 073 (27·9%)	8086/16 715 (48·4%)	3417/6780 (50·4%)	<0·0001	<0·0001	<0·0001
Sickle cell disease	135/34 560 (0·4%)	1536/33 073 (4·6%)	2824/16 715 (16·9%)	1229/6780 (18·1%)	<0·0001	<0·0001	<0·0001
HIV status, positive***	516/17 163 (3·0%)	989/14 261 (6·9%)	519/6165 (8·4%)	174/2484 (7·0%)	<0·0001	<0·0001	<0·0001
Microcytosis†††	9070/34 560 (26·3%)	14 574/33 073 (44·2%)	6069/16 715 (36·4%)	2050/6780 (30·3%)	<0·0001	<0·0001	<0·0001
Macrocytosis‡‡‡	154/34 560 (0·4%)	134/33 073 (0·4%)	597/16 715 (3·6%)	440/6780 (6·5%)	0·44	<0·0001	<0·0001
Hypochromasia§§§	12 635/34 360 (36·8%)	19 639/32 942 (59·6%)	10 540/16 635 (63·4%)	4150/6740 (61·6%)	<0·0001	<0·0001	<0·0001
Thrombocytopaenia¶¶¶	2800/33 841 (8·3%)	6038/31 984 (18·9%)	6315/15 780 (40·0%)	2810/6351 (44·2%)	<0·0001	<0·0001	<0·0001
Red blood cells, ×10 ⁶ /μL	4·7 (4·3–5·1)	4·0 (3·5–4·6)	2·4 (1·7–3·0)	1·6 (1·2–2·0)	<0·0001	<0·0001	<0·0001
MCV, fL	74·0 (68·0–80·0)	70·0 (62·0–77·0)	73·0 (63·0–82·0)	76·0 (66·0–85·0)	<0·0001	<0·0001	<0·0001
MCHC, g/dL	32·4 (31·5–33·2)	31·6 (30·4–32·7)	31·1 (29·4–32·8)	31·1 (29·0–33·0)	<0·0001	<0·0001	<0·0001
White blood cells, ×10 ³ /μL	12·2 (9·0–16·5)	12·7 (9·1–17·9)	14·1 (9·0–22·6)	15·1 (9·3–25·7)	<0·0001	<0·0001	<0·0001
Platelets, ×10 ⁶ /μL	401 (286–535)	372 (201–536)	199 (97–382)	172 (92–325)	<0·0001	<0·0001	<0·0001
Data are median (IQR) or n/N (%). MCHC=mean corpuscular haemoglobin concentration. MCV=mean cell volume. MUAC=mid-upper arm circumference. *Mild anaemia was defined as haemoglobin 10·0–10·9 g/dL (age 1–5 months), 9·5–10·4 g/dL (age 6–23 months), 10·0–10·9 g/dL (age 24–59 months), 11·0–11·4 g/dL (age 5–11 years), or 11·0–11·9 g/dL (age 12–14 years); moderate anaemia as haemoglobin 7·0–9·9 g/dL (age 1–5 months), 7·0–9·4 g/dL (age 6–23 months), 7·0–9·9 g/dL (age 24–59 months), or 8·0–10·9 g/dL (age 5–14 years); severe anaemia (includes very severe anaemia) as haemoglobin of less than 7 g/dL (age 1–59 months) or haemoglobin of less than 8 g/dL (age 5–14 years); and very severe anaemia as haemoglobin of less than 5·0 g/dL. †p values were derived from cluster-robust logistic (binary outcomes) and linear (continuous outcomes) regression models adjusting for age, sex, number of hospital admissions, and year of hospitalisation. ‡Axillary temperature more than 37·5°C. §Diagnosed based on clinical recognition by the admitting clinician. ¶Blantyre coma score of less than 3. Cough or difficulty breathing plus lower chest wall indrawing. **Cough or difficulty breathing plus either prostration, lethargy, hypoxia, loss of consciousness, or a history of convulsions. ††The presence of wasting, stunting, underweight, or severe acute malnutrition. †††Weight-for-height Z score of less than –2 or MUAC less than 11·0 cm in children aged 6 weeks to 6 months, or MUAC less than 12·5 cm in children older than 6 months using WHO child growth standards. §§Height-for-age Z score of less than –2. ¶¶Weight-for-age Z score of less than –2. Weight-for-height Z score of less than –3 or MUAC less than 11·5 cm in children older than 6 months or presence of bilateral oedema. ***Data available from Feb 1, 2005. †††MCV of less than 67 fL for children younger than 24 months, MCV less than 73 fL for age 24–60 months, and MCV less than 65 fL for children older than 60 months. ²⁶ ††††MCV of more than 100 fL. §§§MCHC of less than 32 g/dL. ¶¶¶¶Platelets less than 150×10 ⁶ /μL.							
Table: Characteristics of all children aged 1 month to 14 years admitted to Kilifi County Hospital from Aug 1, 1998, to July 31, 2024							

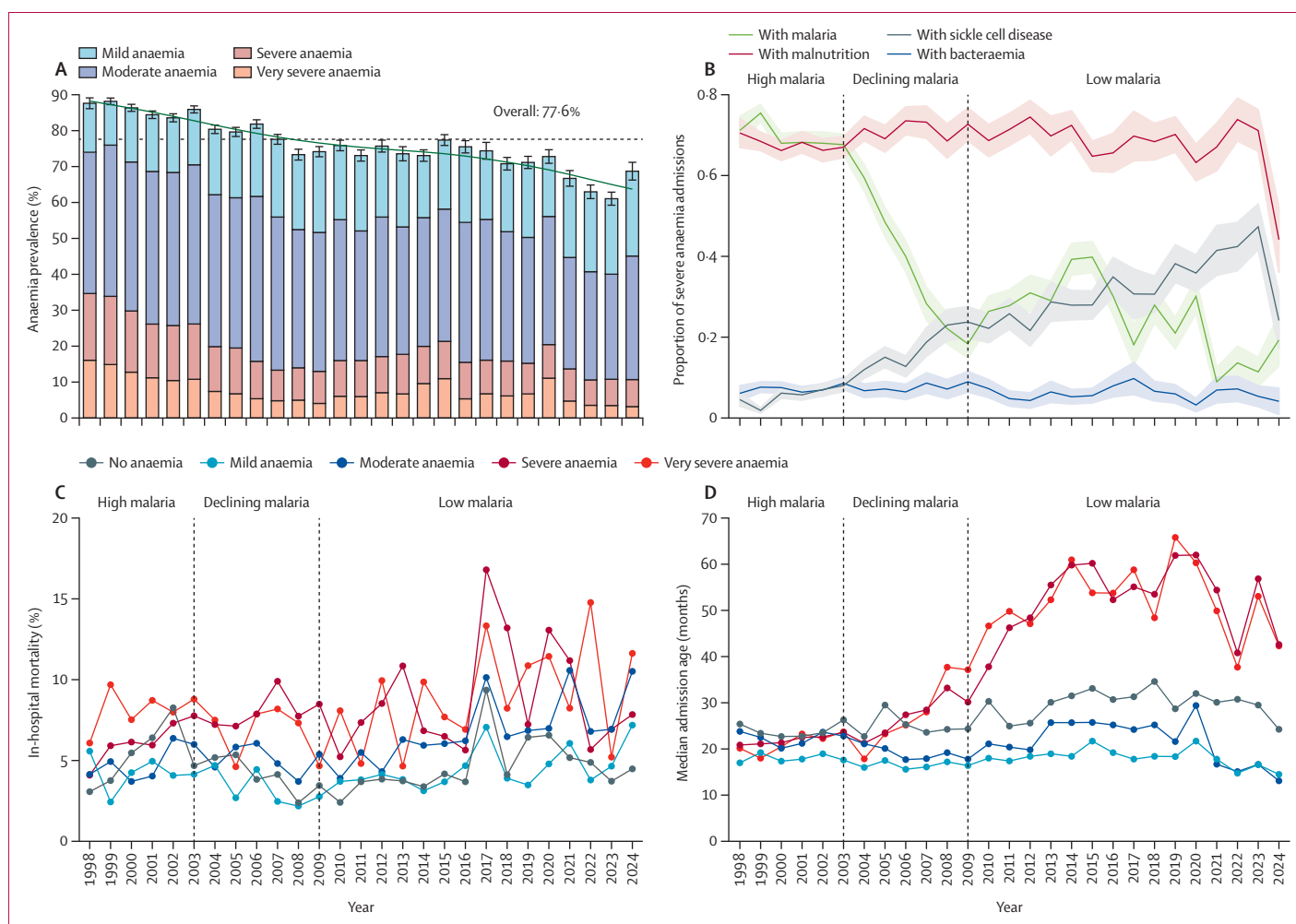


Figure 2: Severe anaemia trends in children admitted to Kilifi County Hospital between Aug 1, 1998, and July 31, 2024

The study period is divided into high (1998–2003), declining (2004–09), and low (2010–24) malaria transmission eras. (A) Annual prevalence of anaemia by severity category: mild, moderate, severe, and very severe. The solid green line represents the smoothed trend (locally estimated scatterplot smoothing), the dotted black line denotes the overall mean anaemia prevalence across the study period (mean 77.6%). (B) Proportion of severe anaemia admissions with malaria, malnutrition, sickle cell disease, and bacteraemia. Shading indicates 95% CIs. (C) In-hospital mortality stratified by anaemia severity: no anaemia, mild, moderate, severe (includes very severe anaemia), and very severe. (D) Median age at admission by anaemia severity category: no anaemia, mild, moderate, severe (includes very severe anaemia), and very severe.

transmission has declined in the study area, malnutrition and SCD have emerged as increasingly important factors associated with severe anaemia (figure 3B–D). A similar trend was seen among admissions with moderate anaemia, where malnutrition was associated with 57.7% ($n=8335$) of admissions in the period of low malaria transmission (appendix 2 p 21). In-hospital mortality ranged from 3.3% in severely anaemic children with SCD alone to 14.3% in children with malnutrition alone (figure 3A).

SCD (aOR 73.0 [95% CI 59.1–90.2; $p<0.0001$]) and *P. falciparum* malaria (4.7 [4.5–5.0]; $p<0.0001$) were strongly positively associated with severe anaemia admission. Bacteraemia, HIV infection, and malnutrition were associated with greater odds of severe anaemia (appendix 2 p 12). The PAF of severe anaemia due to SCD was 12.2% (95% CI 11.8–12.6), malnutrition was 24.3% (22.7–25.8), and *P. falciparum* malaria was 27.1% (26.2–28.1). Restricting

analyses to first admissions gave similar estimates (appendix 2 p 12). The strength of these associations varied by age, and SCD, HIV, and malnutrition were identified as more important factors in children aged 5–14 years (appendix 2 p 22).

Bacteraemia was an important cause of hospitalisation, accounting for 4.3% ($n=3656$) of all paediatric admissions. The highest burden was observed among infants (appendix 2 p 13). A reduction in admissions with *H. influenzae* and *Streptococcus pneumoniae* was observed after introduction of the Hib and PCV10 vaccines into routine immunisation schedules in 2001 and 2011, respectively (appendix 2 p 23). Among children with severe anaemia, the predominant pathogens were *S. pneumoniae*, NTS, *Escherichia coli*, and *H. influenzae* (appendix 2 p 14). Overall in-hospital mortality among admissions with bacteraemia was 24.8%; although, this proportion varied

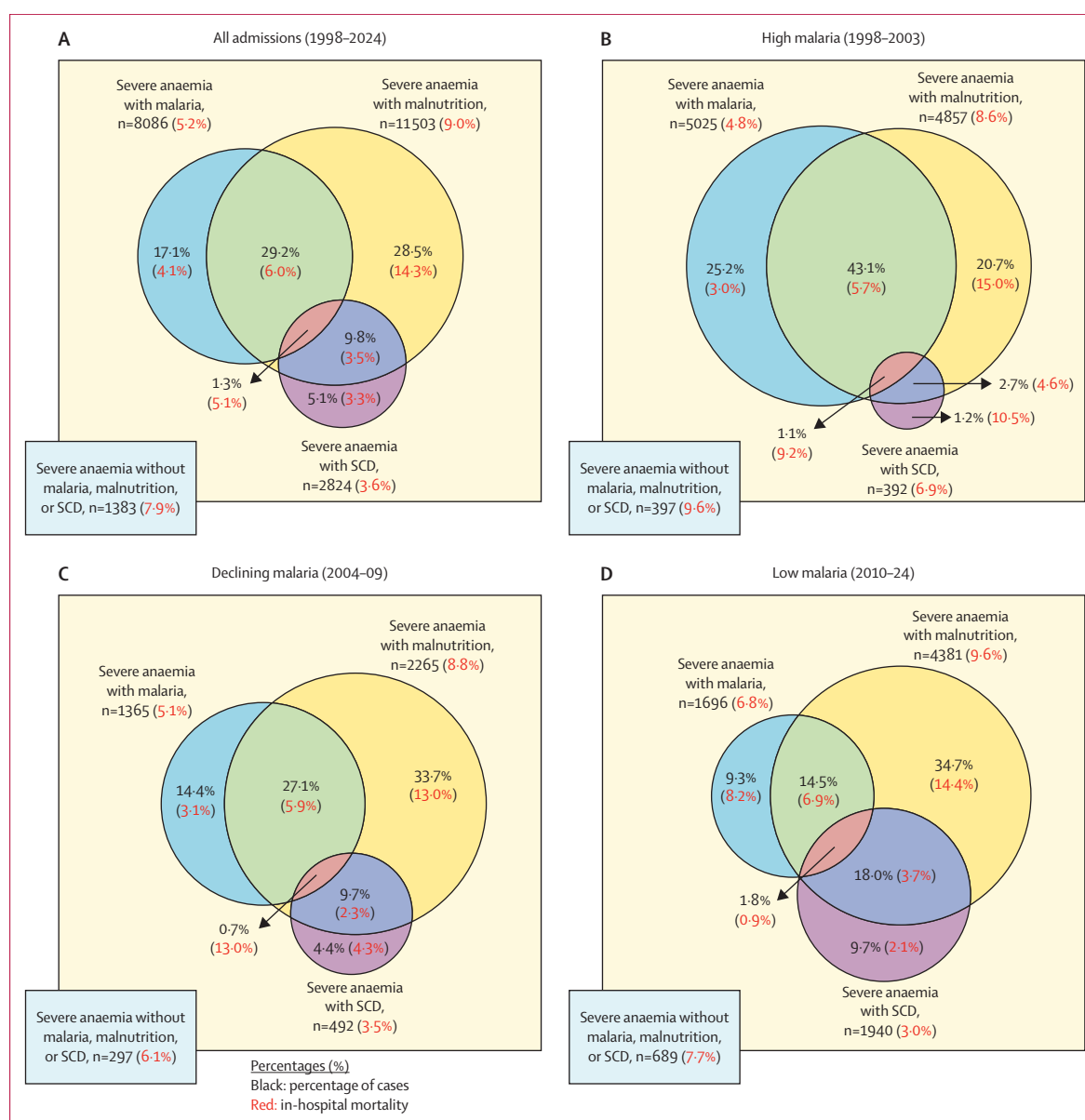


Figure 3: Prevalence, overlap, and mortality for groups of children aged 1 month to 14 years with severe anaemia across all admissions (August, 1998, to July, 2024; A), during high malaria transmission period (1998–2003; B), during declining malaria transmission period (2004–09; C), and during a low malaria transmission period (2010–24; D)

The percentage of children in each group (with respect to all severe anaemia cases) are in black. The in-hospital mortality is in red (calculated as the number of severely anaemic children who died in the group divided by all severe anaemia admissions in the group). SCD=sickle cell disease.

by bacterial pathogen and anaemia severity. In-hospital mortality was highest in *Pseudomonas aeruginosa* bacteraemia (appendix 2 p 24), and in severe anaemia compared with mild or no anaemia (27.6% vs 22.4%, aOR 1.4 [95% CI 1.2–1.8]; $p=0.0008$).

Severe and moderate anaemia were associated with greater odds of bacteraemia compared with mild or no anaemia (aOR 2.7 [95% CI 2.5– 3.0]; $p<0.0001$ and 1.8 [1.7–2.0]; $p<0.0001$, respectively). Among individual pathogens, severe anaemia was associated with increased

odds of NTS (6.1 [4.6–8.1]), *E coli* (5.7 [4.2–7.7]), *H influenzae* (4.3 [3.0–6.4]), *S pneumoniae* (3.5 [2.9–4.2]), and *Klebsiella pneumoniae* (2.4 [1.4–4.0]; figure 4). Severe anaemia remained strongly associated with bacteraemia in sensitivity analyses stratified by 5-year intervals (appendix 2 p 25), during epidemiological periods of high, declining, and low malaria prevalence across the study period (appendix 2 p 9), and in analyses restricted to each child's first hospital admission (appendix 2 p 26). The aORs of bacteraemia differed by underlying severe anaemia

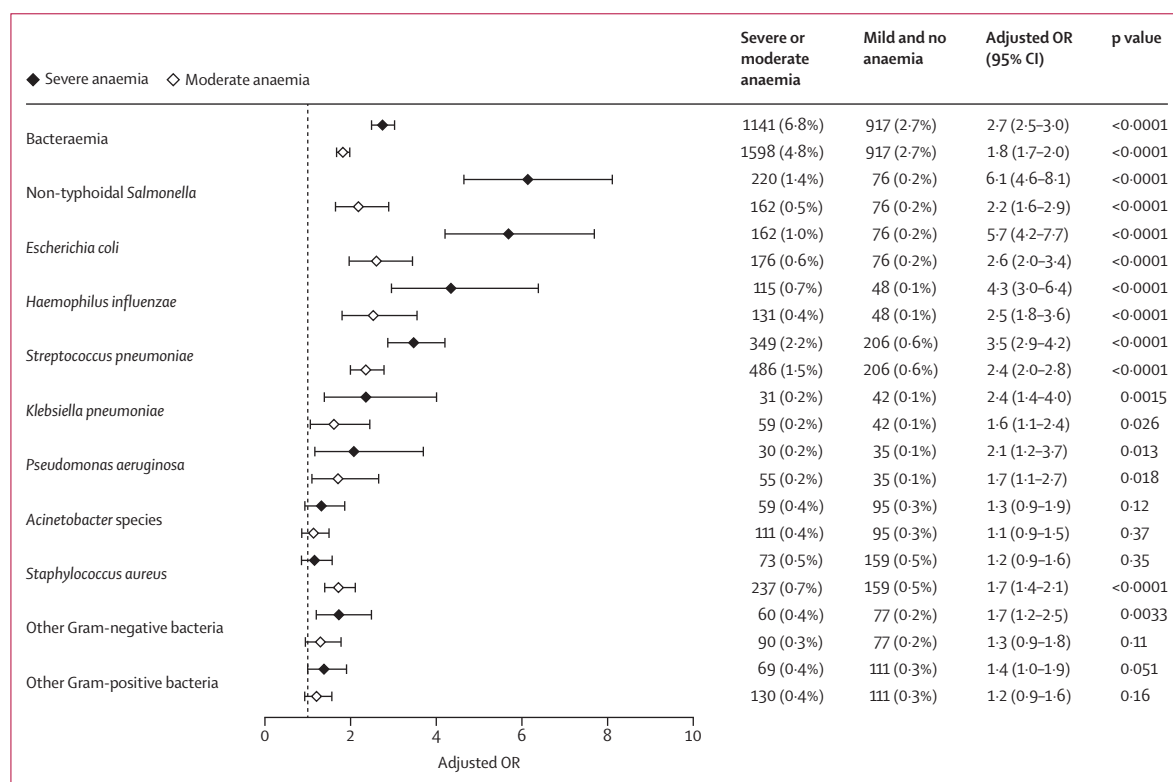


Figure 4: Association between severe anaemia or moderate anaemia and odds of bacteraemia among children aged 1 month to 14 years admitted to Kilifi County Hospital between Aug 1, 1998, and July 31, 2024

ORs and 95% CIs were derived from cluster-robust logistic regression models adjusting for age, sex, number of hospital admissions, and year of hospitalisation. Odds of bacteraemia did not differ between children with mild anaemia and those without anaemia (adjusted OR 1.0 [95% CI 0.9–1.2]; $p=0.49$). Children with mild or no anaemia were the reference group. OR=odds ratio.

aetiology, ranging from 1.2 (1.1–1.4) in severe malarial anaemia to 11.0 (8.5–14.2) in HIV-associated severe anaemia (appendix 2 p 15).

Decreasing haemoglobin was associated with increased odds of all-cause bacteraemia (aOR 1.14 [95% CI 1.12–1.15]; $p<0.0001$), with pathogen-specific effects: NTS (aOR 1.31 [95% CI 1.26–1.35]), *E coli* (1.30 [1.25–1.35]), *H influenzae* (1.17 [1.13–1.18]), and *S pneumoniae* (1.15 [1.13–1.18]; appendix 2 p 27).

Using haemoglobin of 11 g/dL or more as the reference, each 1 g/dL decrease in haemoglobin was associated with increased odds of bacteraemia above 4 g/dL (figure 5; appendix 2 pp 16–17), a trend observed across different severe anaemia aetiologies (appendix 2 p 28). A linear trend of increasing odds of bacteraemia with decreasing haemoglobin levels was particularly evident for NTS and *E coli*. The odds of *S pneumoniae* also increased as haemoglobin levels decreased, peaking at around 5 g/dL, before decreasing thereafter. No consistent trend was observed for *P aeruginosa*, *K pneumoniae*, *Staphylococcus aureus*, and *Acinetobacter* species (figure 5).

Discussion

Severe anaemia remains an important cause of paediatric admissions and mortality among Kenyan children living in Kilifi, and its prevalence has remained persistently high

over time. Severe anaemia was associated with multiple and overlapping aetiologies, including malaria, malnutrition, SCD, bacteraemia, and HIV infection. As malaria transmission has declined between 2004 and 2009, the burden of severe anaemia has shifted to children aged 5–14 years. Severe anaemia was associated with greater odds of mortality and bacteraemia. We confirmed that severe anaemia was strongly associated with an increased odds of NTS bacteraemia,² and found that severe anaemia was also associated with increased odds of *E coli*, *S pneumoniae*, *H influenzae*, and *K pneumoniae* bacteraemia. The association followed a severity gradient; each 1 g/dL decrease in haemoglobin was associated with increased odds of bacteraemia. Consistent with our findings, severe anaemia was associated with a five-fold increased risk of bacteraemia in Malawian children.² Similar observations have also been reported for severe anaemia and NTS in Mozambique¹³ and severe malarial anaemia and bacteraemia in Uganda.²⁷

The prevalence of severe anaemia remains unacceptably high globally, and progress to meet global targets remains suboptimal.²⁸ In our setting, the aetiology of severe anaemia is multifactorial, and the burden of severe anaemia has shifted from younger children to older age groups over time. This trend might be partly attributed to the declining prevalence of malaria, which predominantly causes severe anaemia in children younger than 5 years in endemic

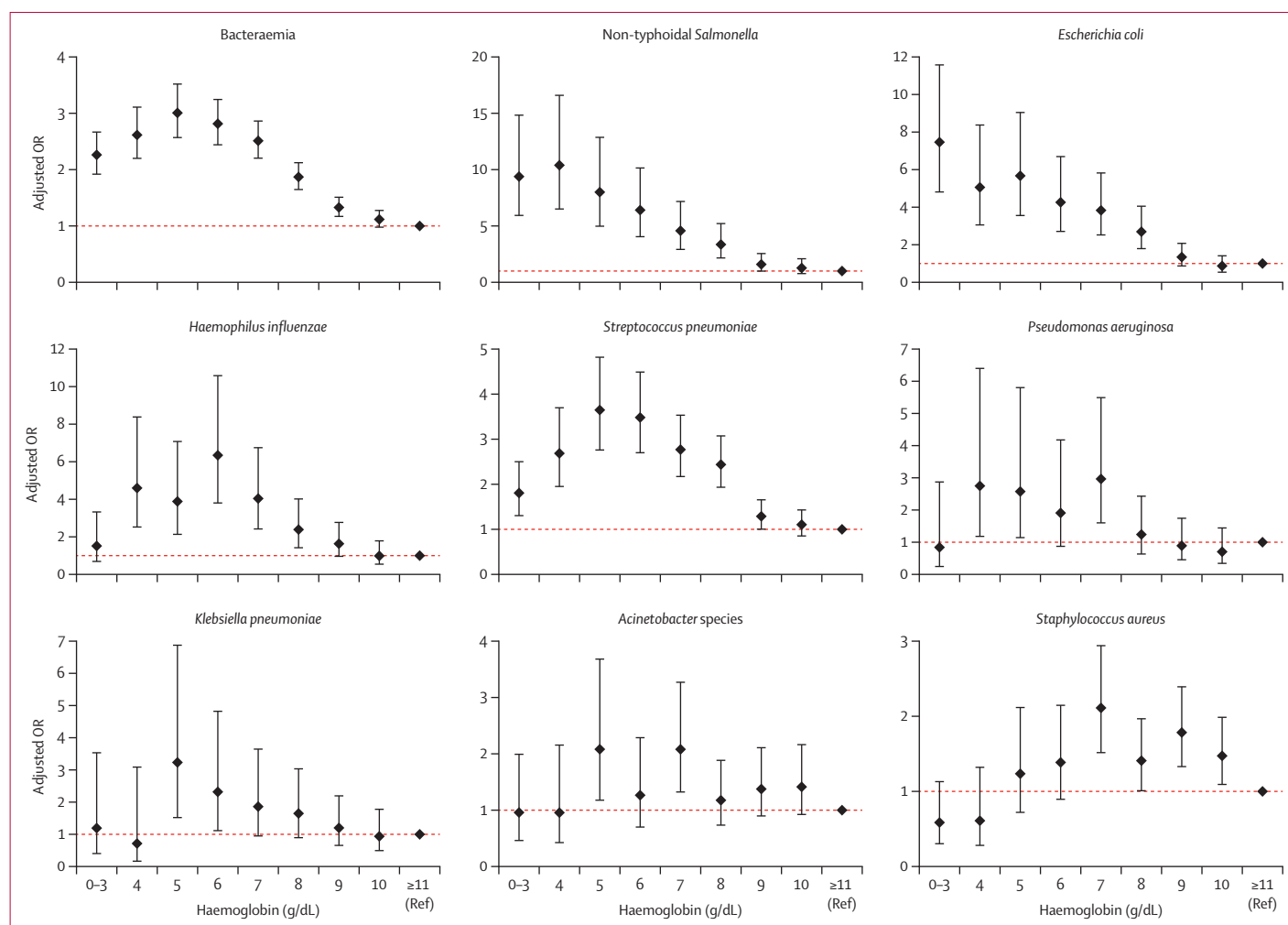


Figure 5: Association between haemoglobin levels and odds of bacteraemia in children aged 1 month to 14 years admitted to Kilifi County Hospital between Aug 1, 1998, and July 31, 2024. Each haemoglobin level (by integer) was compared to the reference group with haemoglobin levels of 11 g/dL or more. ORs and 95% CIs were derived from cluster-robust logistic regression models adjusting for age, sex, number of hospital admissions, and year of hospitalisation. Note that the scales on the y-axes are different in each plot. OR=odds ratio.

areas.⁸ We found that the overall burden of severe anaemia declined from the late 1990s to 2010, a trend that parallels reductions in malaria in Kilifi over the same period.⁸ Other settings across Africa also experienced a reduction in malaria incidence over this period.⁷ However, malaria accounted for 48.4% of severe anaemia admissions and frequently overlapped with malnutrition, HIV infection, and SCD. The shift in distribution of severe anaemia to children aged 5–14 years might also reflect improved survival among children with SCD, including children attending the specialist clinic at KCH, where hydroxyurea (introduced in 2015) is used by approximately 30% of patients. Global efforts to understand and curb severe anaemia have primarily focused on children younger than 5 years and women of reproductive age,¹ and our findings suggest that this focus could potentially overlook a substantial population of older children at risk.

Decreasing haemoglobin levels were strongly associated with bacteraemia. These findings remained robust across

malaria transmission periods and were pathogen-specific, supporting a biological relationship rather than an epidemiological phenomenon. Several mechanisms might underlie the observed association between low haemoglobin and bacteraemia, including (1) increased iron availability for bacterial pathogens, (2) impaired immune function, and (3) increased gut microbiota dysbiosis and intestinal permeability.¹² Anaemia is associated with dysregulated immunity,¹¹ which might be more pronounced in more severe cases. Severe anaemia is also characterised by reduced hepcidin and elevated iron recycling through increased erythrophagocytosis,¹² which transiently increases non-transferrin-bound iron. Siderophilic bacteria, such as *E coli*, might benefit from this iron disruption, as shown in in-vitro and animal model studies.^{29,30} These studies found less pronounced effects of iron dysregulation on *S aureus*,³⁰ consistent with our findings that severe anaemia was not associated with this pathogen. Nonetheless, reverse causality cannot be ruled out, because bacterial infections

might themselves contribute to severe anaemia through increased haemolysis, anaemia of inflammation, or iron malabsorption, and mechanistic studies are needed to confirm our observations. Our findings support prioritisation of early diagnostic evaluation for bacterial infection, close clinical monitoring, and consideration of empiric antimicrobial therapy alongside standard management in children with severe anaemia. Clinical management of severe anaemia should, therefore, integrate infection risk with haemoglobin concentration to inform triage, antimicrobial use, and transfusion strategies.

The strengths of the study are that we utilised a large 26-year dataset (n=84 348), which enabled detailed evaluation of severe anaemia aetiologies, pathogen-specific associations, and was adequately powered for subgroup and stratified analyses. The extended surveillance period allowed assessment of associations across major epidemiological transitions, including declining malaria transmission, vaccine introductions (Hib and PCV10), and improvements in clinical care. Although these shifts might have introduced temporal heterogeneity in exposure and outcome distributions, the consistency of effect estimates across time strata supports the internal validity of our findings. However, some limitations should be considered when interpreting our data. First, blood cultures have low sensitivity and in Kenya antibiotics are available without prescription, so blood cultures are more likely to be negative on admission. Thus, the true association between severe anaemia and bacteraemia might be higher than our estimates. Second, our study was observational, and, as such, the associations might be subject to unmeasured confounders, including collider bias arising from restricting analyses to hospitalised children, and reverse causality. Also, the study was conducted at a single site. Excluding admissions without haemoglobin measurements might have introduced bias, because children with missing values differed in age and comorbidity profile compared with children included in the analysis. It is also likely that a proportion of children with severe anaemia and bacteraemia died outside the hospital. Previous estimates indicate that only one-third of children dying in the KHDSS area present to a hospital.¹⁰ KCH does not routinely screen for hookworm infections, or biomarkers of iron (eg, ferritin, transferrin saturation, or soluble transferrin receptor), vitamin A, or vitamin B12 status, which are important causes of anaemia in sub-Saharan Africa.² SCD was also mostly clinically diagnosed, and some cases might have been missed as a result. In the absence of cause-specific mortality data from autopsy, we could not distinguish deaths directly attributable to anaemia from those due to bacterial infections or other comorbid conditions. Nonetheless, the size of our study, the systematic nature of the hospital surveillance, and the extensive period of the study (1998–2024) offer strong support to our conclusions. Taken collectively, these observations suggest that severe anaemia is prevalent among paediatric admissions, its burden is shifting to children aged 5–14 years, and it is an

important risk factor for bacteraemia and mortality in this region.

Contributors

Conceptualisation: KMA, JMM, TNW, and SHA. Formal analysis: KMA, JMM, AMM, KMW, and SHA. Supervision: PB, CAM, MN, JAGS, TNW, and SHA. Investigation: NM, SMO, JFK, SMw, CJN, CRN, KM, PB, JAGS, TNW, and SHA. Writing (original draft): KMA, JMM, KMW, and SHA. Writing (review and editing): KMA, JMM, AMM, KMW, NM, SMO, JFK, SMw, CJN, CRN, KM, PB, CAM, MN, JAGS, TNW, and SHA. KMA, JMM, AMM, KMW, and SHA accessed and verified the data. All authors had full access to the data. All authors critically reviewed and approved the manuscript and had final responsibility for the decision to submit for publication.

Equitable Partnership Declaration

The authors of this paper have submitted an Equitable Partnership Declaration (appendix 3). This statement allows researchers to describe how their work engages with researchers, communities, and environments in the countries of study. This statement is part of The Lancet Group's broader goal to decolonise global health.

Declaration of interests

We declare no competing interests.

Data sharing

All data and analyses underlying this article are available in Harvard Dataverse repository (<https://doi.org/10.7910/DVN/RUDR3T>) and applications for data access can be made through the Kilifi Data Governance Committee (cgmrc@kemri-wellcome.org).

Acknowledgments

This work was supported by Wellcome (224315 to KMA; 226014 to SHA; and 203077, <https://doi.org/10.35802/203077>, a core award to the KEMRI-Wellcome Trust Research Programme); and the Science for Africa Foundation to the Developing Excellence in Leadership, Training, and Science in Africa (DELTAS Africa) programme (DEL-22-012 to KMA and AMM), with support from Wellcome Trust and the UK Foreign, Commonwealth & Development Office and is part of the EDCPT2 programme supported by the EU. The authors would like to thank the children whose data was used in this study and their parents or guardians. We would also like to thank the clinical and laboratory staff at the Kilifi County Hospital and KEMRI-Wellcome Trust Research Programme who collected the data used in this study.

References

- 1 Stevens GA, Paciorek CJ, Flores-Urrutia MC, et al. National, regional, and global estimates of anaemia by severity in women and children for 2000–19: a pooled analysis of population-representative data. *Lancet Glob Health* 2022; **10**: e627–39.
- 2 Calis JC, Phiri KS, Faragher EB, et al. Severe anemia in Malawian children. *N Engl J Med* 2008; **358**: 888–99.
- 3 Moraleda C, Aguilar R, Quintó L, et al. Anaemia in hospitalised preschool children from a rural area in Mozambique: a case control study in search for aetiological agents. *BMC Pediatr* 2017; **17**: 63.
- 4 Cannon R, George EC, Olupot-Olupot P, et al, and the TRACT trial group. Incidence and predictors of hospital readmission in children presenting with severe anaemia in Uganda and Malawi: a secondary analysis of TRACT trial data. *BMC Public Health* 2021; **21**: 1480.
- 5 Kwambai TK, Mori AT, Nevitt S, et al. Post-discharge morbidity and mortality in children admitted with severe anaemia and other health conditions in malaria-endemic settings in Africa: a systematic review and meta-analysis. *Lancet Child Adolesc Health* 2022; **6**: 474–83.
- 6 Phiri KS, Calis JC, Faragher B, et al. Long term outcome of severe anaemia in Malawian children. *PLoS One* 2008; **3**: e2903.
- 7 Snow RW, Sartorius B, Kyalo D, et al. The prevalence of *Plasmodium falciparum* in sub-Saharan Africa since 1900. *Nature* 2017; **550**: 515–18.
- 8 Njuguna P, Maitland K, Nyaguara A, et al. Observational study: 27 years of severe malaria surveillance in Kilifi, Kenya. *BMC Med* 2019; **17**: 124.

See Online for appendix 3

- 9 Sigaúque B, Roca A, Mandomando I, et al. Community-acquired bacteremia among children admitted to a rural hospital in Mozambique. *Pediatr Infect Dis J* 2009; **28**: 108–13.
- 10 Berkley JA, Lowe BS, Mwangi I, et al. Bacteremia among children admitted to a rural hospital in Kenya. *N Engl J Med* 2005; **352**: 39–47.
- 11 Mooney JP, Galloway LJ, Riley EM. Malaria, anemia, and invasive bacterial disease: A neutrophil problem? *J Leukoc Biol* 2019; **105**: 645–55.
- 12 Abuga KM, Nairz M, MacLennan CA, Atkinson SH. Severe anaemia, iron deficiency, and susceptibility to invasive bacterial infections. *Wellcome Open Res* 2023; **8**: 48.
- 13 Mandomando I, Bassat Q, Sigaúque B, et al. Invasive *Salmonella* infections among children from rural Mozambique, 2001–2014. *Clin Infect Dis* 2015; **61** (suppl 4): S339–45.
- 14 Scott JA, Bauni E, Moisi JC, et al. Profile: The Kilifi Health and Demographic Surveillance System (KHDSS). *Int J Epidemiol* 2012; **41**: 650–57.
- 15 Muriuki JM, Mentzer AJ, Webb EL, et al. Estimating the burden of iron deficiency among African children. *BMC Med* 2020; **18**: 31.
- 16 Scott JA, Berkley JA, Mwangi I, et al. Relation between falciparum malaria and bacteraemia in Kenyan children: a population-based, case-control study and a longitudinal study. *Lancet* 2011; **378**: 1316–23.
- 17 Adetifa IMO, Bwanaali T, Wafula J, et al. Cohort profile: the Kilifi vaccine monitoring study. *Int J Epidemiol* 2017; **46** (3): 792.
- 18 Bejon P, Lusingu J, Olotu A, et al. Efficacy of RTS,S/AS01E vaccine against malaria in children 5 to 17 months of age. *N Engl J Med* 2008; **359**: 2521–32.
- 19 WHO. Pocket book of hospital care for children: guidelines for the management of common childhood illnesses, Second edition. World Health Organization, 2013.
- 20 WHO Multicentre Growth Reference Study Group. WHO child growth standards based on length/height, weight and age. *Acta Paediatr Suppl* 2006; **450**: 76–85.
- 21 WHO. WHO child growth standards: head circumference-for-age, arm circumference-for-age, triceps skinfold-for-age and subscapular skinfold-for-age: methods and development. World Health Organization, 2007.
- 22 WHO. WHO child growth standards and the identification of severe acute malnutrition in infants and children: a joint statement by the World Health Organization and the United Nations Children's Fund. World Health Organization, 2009.
- 23 WHO. Haemoglobin concentrations for the diagnosis of anaemia and assessment of severity. World Health Organization, 2011.
- 24 Anaemia Collaborators GBD, and the GBD 2021 Anaemia Collaborators. Prevalence, years lived with disability, and trends in anaemia burden by severity and cause, 1990–2021: findings from the Global Burden of Disease Study 2021. *Lancet Haematol* 2023; **10**: e713–34.
- 25 Newson RB. Attributable and unattributable risks and fractions and other scenario comparisons. *Stata J* 2013; **13**: 672–98.
- 26 Aldrimer M, Ridefelt P, Röddö P, Niklasson F, Gustafsson J, Hellberg D. Population-based pediatric reference intervals for hematology, iron and transferrin. *Scand J Clin Lab Invest* 2013; **73**: 253–61.
- 27 Church J, Maitland K. Invasive bacterial co-infection in African children with *Plasmodium falciparum* malaria: a systematic review. *BMC Med* 2014; **12**: 31.
- 28 Atkinson SH, Suchdev PS, Bode M, et al. Getting back on track to meet global anaemia reduction targets: a *Lancet Haematology* Commission. *Lancet Haematol* 2025; **12**: e717–67.
- 29 Stefanova D, Raychev A, Deville J, et al. Hepcidin protects against lethal *Escherichia coli* sepsis in mice inoculated with isolates from septic patients. *Infect Immun* 2018; **86**: e00253–18.
- 30 Prentice S, Jallow AT, Sinjanka E, et al. Hepcidin mediates hypoferrremia and reduces the growth potential of bacteria in the immediate post-natal period in human neonates. *Sci Rep* 2019; **9**: 16596.