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Original Research Article

Clinical, Haematological and Aetiological Profile and Short-term Outcome of Severe Anaemia in Children Aged 5–12 Years at a Tertiary Care Hospital in South Gujarat

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Abstract:

Background: Severe anaemia is a frequent cause of paediatric hospitalisation in India and can lead to cardiac failure and death. Data on its clinical presentation, haematological pattern and causes in school-age children (5–12 years) are limited.

Objectives: To describe the clinical profile, haematological profile, aetiology, treatment and short-term outcome of severe anaemia in children aged 5–12 years.

Methods: This prospective observational study included 80 children aged 5–12 years admitted with newly diagnosed severe anaemia (haemoglobin ≤7 g/dL) to the paediatric department of a tertiary care hospital in Surat between March 2023 and June 2024. Clinical features were recorded on a structured proforma. Complete blood count, red cell indices, peripheral smear, reticulocyte count, serum iron studies, vitamin B12, folate and stool examination were performed in all children, with haemoglobin electrophoresis, HPLC, sickling and Coombs tests when indicated. Data were analysed with SPSS version 23.

Results: Weakness (100%), fever (75%), anorexia (75%) and breathlessness (62.5%) were the leading complaints. Pallor was universal, koilonychia was present in 31.25% and hepatosplenomegaly in 18.75%; 12.5% had congestive cardiac failure. Sepsis (20%), gastroenteritis (15%), viral infections (15%) and malaria (10%) were the commonest associated illnesses. Haemoglobin was <5 g/dL in 18.75%. Microcytic hypochromic anaemia was the predominant morphology (52.5%) and RDW was raised in 72.5%. Iron-deficiency anaemia was the leading cause (56.25%), followed by megaloblastic anaemia (18.75%), anaemia of chronic disease (12.5%), combined iron and B12 deficiency (6.25%) and aplastic anaemia (6.25%). Blood transfusion was required in 31.25%, and 57.5% were discharged within 7 days. Mortality was 7.5%. Clinical and haematological parameters did not differ significantly by sex.

Conclusion: Nutritional deficiencies, chiefly of iron and vitamin B12, underlie three-quarters of severe anaemia in school-age children in South Gujarat, often worsened by infection. A systematic work-up with red cell indices, peripheral smear and targeted biochemical tests allows accurate aetiological diagnosis and rational, cause-specific treatment.

Keywords: Severe anaemia; Iron-deficiency anaemia; Megaloblastic anaemia; Red cell indices; Blood transfusion; Children.

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Introduction

Anaemia is a reduction in haemoglobin concentration or red cell mass below the normal range for age and sex. It impairs oxygen delivery to tissues. WHO defines anaemia in children aged 5–11 years as haemoglobin below 11.5 g/dL, and grades it as severe when haemoglobin falls below 8 g/dL [1].

Anaemia affects roughly one-third of the world's population. Most of the burden falls on low- and middle-income countries, where iron deficiency, infections and haemoglobinopathies are the main causes [2]. In India, NFHS-5 (2019–21) found anaemia in 67.1% of children under five [3]. The Comprehensive National Nutrition Survey documented substantial anaemia and micronutrient

deficiency among school-age children and adolescents as well [4].

Anaemia is not a disease in itself but a sign of an underlying disorder, so the cause must be found before treatment is chosen. Iron deficiency is the most common cause worldwide. It results from inadequate intake, increased requirements during growth, blood loss (often from hookworm infestation) or malabsorption [5]. Vitamin B12 and folate deficiency cause megaloblastic anaemia and are particularly common in India, where a large share of the population follows a vegetarian diet [6,7]. Acute and chronic infections, including malaria, sepsis and tuberculosis, cause anaemia of inflammation through cytokine-mediated iron sequestration, reduced erythropoietin response and shortened red cell survival [8]. Less often, marrow failure, haemolysis and malignancy are responsible.

Severe anaemia is a paediatric emergency. Children may present with fatigue, breathlessness, tachycardia or features of congestive cardiac failure. In resource-limited settings, severe anaemia is an important contributor to hospital mortality [9]. Hospital-based studies from India have described the clinical and haematological profile of children with anaemia, but most included mild and moderate cases or focused on children under five [10–13]. Comparatively few studies have examined severe anaemia specifically in children aged 5–12 years. In this age group, school attendance, a changing diet and helminth exposure may alter the pattern of causes. A systematic laboratory approach underpins accurate diagnosis. The complete blood count with red cell indices (MCV, MCH, MCHC) and red cell distribution width (RDW) allows morphological classification. A raised RDW with microcytosis strongly suggests iron deficiency [14]. Peripheral smear examination, reticulocyte count, serum iron, ferritin, total iron-binding capacity (TIBC), vitamin B12 and folate levels, and stool examination then identify the specific cause. Treatment ranges from oral haematinics to parenteral iron and blood transfusion. Transfusion practices vary widely between centres because transfusion thresholds differ [15,16]. Unnecessary transfusion exposes children to infection and transfusion reactions, whereas delayed transfusion in children with cardiac decompensation can be fatal. This study was undertaken at a tertiary care hospital in South Gujarat. It describes the clinical presentation, haematological profile, aetiology, treatment and short-term outcome of severe anaemia in children aged 5–12 years.

Materials and Methods

Study design, setting and ethics: This prospective observational study was carried out in the Department of Paediatrics, Government Medical College and New Civil Hospital, Surat, a tertiary referral centre for South Gujarat. Data were collected from March 2023 to June 2024 after approval from the Institutional Ethics Committee. Written informed consent was obtained from the parents or guardians of all participants.

Participants: Children aged 5–12 years admitted to the paediatric wards with newly diagnosed severe anaemia were included. Severe anaemia was operationally defined as haemoglobin ≤7 g/dL.

We excluded children whose parents declined consent, children on chronic regular transfusion for leukaemia, bleeding disorders or established haemolytic anaemia, children with mild or moderate anaemia, and those who left against medical advice or were not followed until discharge.

Sample Size: Using $n = 4pq/l^2$, with a reference prevalence of 72.7% and an allowable error of 10%, the required sample was 80.

Consecutive eligible children were enrolled by convenience sampling until this number was reached.

Clinical Evaluation: Each child was stabilised on arrival. A detailed history was then taken and recorded on a pre-designed proforma. It covered presenting complaints (weakness, fever ≥38 °C, anorexia, breathlessness, palpitation, cough, diarrhoea, dysentery, vomiting, abdominal pain, convulsions and altered sensorium), past illness and treatment. General examination looked for pallor of the conjunctiva and nail beds, icterus, oedema, lymphadenopathy, koilonychia, platonychia, hair changes, glossitis, bald tongue and angular stomatitis. Systemic examination covered the respiratory, cardiovascular, abdominal and central nervous systems. Congestive cardiac failure was diagnosed clinically by tachycardia, gallop rhythm, basal crepitations and tender hepatomegaly. Associated illnesses such as sepsis, malaria, dengue, gastroenteritis, respiratory and urinary infections were diagnosed by standard clinical and laboratory criteria.

Laboratory Investigations: About 3 mL of venous blood was collected from the median cubital vein under aseptic precautions (70% alcohol followed by povidone-iodine) into EDTA tubes by trained staff. The following methods were used:

Investigation	Method
Haemoglobin	Automated analyser; cross-checked by Sahli method
PCV, MCV, MCH, MCHC, RDW, platelet count	Automated haematology cell counter
Peripheral blood smear	Leishman stain
Reticulocyte count	Brilliant cresyl blue supravital stain
Serum iron and TIBC	Colorimetric methods
Serum ferritin, vitamin B12, folic acid	Chemiluminescent immunoassay (Architect platform)
Stool	Microscopy for ova, cysts and occult blood

Haemoglobin electrophoresis, high-performance liquid chromatography (HPLC), sickling test and direct and indirect Coombs tests were performed when clinically indicated. Bone marrow examination was done when marrow failure was suspected.

Classification: Anaemia was classified morphologically as microcytic hypochromic, normocytic normochromic, normocytic hypochromic, macrocytic or dimorphic, using red cell indices and smear findings. Aetiological diagnoses were assigned on combined clinical and laboratory criteria:

- **Iron-deficiency anaemia (IDA):** microcytic hypochromic picture with low serum iron and ferritin and raised TIBC.
- **Megaloblastic anaemia:** macrocytosis with low serum vitamin B12 or folate.
- **Combined deficiency:** dimorphic smear with evidence of both deficiencies.
- **Anaemia of chronic disease:** normocytic or mildly microcytic anaemia with underlying infection or inflammation and preserved iron stores.
- **Aplastic anaemia:** pancytopenia with hypocellular marrow.

Treatment and outcome: All children were managed according to WHO and national F-IMNCI guidelines. Packed red cells were transfused to all children with Hb <5 g/dL, and to those with Hb 5–7 g/dL who had features of cardiac failure.

Others received parenteral iron (iron sucrose), intramuscular vitamin B12, or oral iron, folic acid and nutritional rehabilitation, together with albendazole and treatment of associated infections. Primary outcomes were duration of hospital stay and use of investigations for diagnosis. Secondary outcomes were discharge or death.

Statistical Analysis: Data were entered in Microsoft Excel and analysed with SPSS version 23. Categorical data are expressed as frequencies and percentages. Differences between boys and girls were tested using the chi-square test, and $p < 0.05$ was considered significant.

Results

Eighty children with severe anaemia were enrolled out of 644 anaemic admissions (12.42%). The most common age group was 7–8 years (47.5%), and 49 (61.25%) were girls.

Table 1: Presenting complaints (n = 80)

Symptom	n	Percentage (%)
Weakness / easy fatigability	80	100
Fever (≥38 °C)	60	75.00
Anorexia	60	75.00
Breathlessness	50	62.50
Palpitation	30	37.50
Cough	20	25.00
Diarrhoea	20	25.00
Abdominal pain	18	22.50
Dysentery	15	18.75
Vomiting	15	18.75
Altered sensorium	3	3.75
Convulsions	3	3.75

Every child had weakness. Fever, anorexia and breathlessness were the next most frequent complaints, and over a third reported palpitations.

Table 2: General and systemic examination findings (n = 80)

Finding	n	Percentage (%)
General examination		
Pallor (conjunctiva and nail beds)	80	100
Koilonychia	25	31.25
Angular stomatitis	20	25.00
Icterus	15	18.75
Glossitis	15	18.75
Pedal oedema	10	12.50
Lymphadenopathy	10	12.50
Platonychia	10	12.50
Dry, sparse hair	10	12.50
Bald, smooth tongue	10	12.50
Cyanosis / clubbing	0	0
Systemic examination		
Hepatosplenomegaly	15	18.75
Basal crepitations	10	12.50
Haemic murmur	10	12.50
Gallop rhythm	10	12.50
Altered sensorium	3	3.75
Convulsions	3	3.75

Pallor was universal. Signs of nutritional deficiency (koilonychia, angular stomatitis, glossitis) were common. One in eight children had gallop rhythm and basal crepitations, consistent with cardiac decompensation.

Table 3: Associated illnesses (n = 80)

Associated illness	n	Percentage (%)
Sepsis	16	20.00
Gastroenteritis	12	15.00
Viral infection	12	15.00
Congestive cardiac failure	10	12.50
Malaria	8	10.00
Lower respiratory tract infection	6	7.50
Dengue	4	5.00
Urinary tract infection	2	2.50

Sepsis was the most frequent associated condition. Infections of all types together were present in most children. Malaria (10%) and dengue (5%) reflect the regional burden of vector-borne disease.

Table 4: Haematological and biochemical findings (n = 80)

Parameter	Finding	n	Percentage (%)
Haemoglobin	<5 g/dL	15	18.75
	5–7 g/dL	65	81.25
PCV, MCV, MCH	Decreased	42	52.50
MCV	Increased	10	12.50
MCHC	Decreased	42	52.50
	Normal	28	35.00
RDW	Increased	58	72.50
	Normal	22	27.50
Platelet count	Decreased	17	21.25
	Normal	63	78.75
Serum iron	Decreased	42	52.50
Serum ferritin	Decreased	42	52.50
Serum TIBC	Increased	42	52.50
Serum vitamin B12	Decreased	10	12.50
Serum folate	Decreased	0	0
Stool	Occult blood positive	5	6.25
	Parasites (ova/cysts) seen	5	6.25

Nearly one in five children had haemoglobin below 5 g/dL. Low red cell indices with low iron and ferritin and raised TIBC were found in 52.5% of children. RDW was raised in 72.5%, and vitamin B12 was low in 12.5%. No child had folate deficiency.

Table 5: Morphological and aetiological classification of severe anaemia (n = 80)

Classification	Type	n	Percentage (%)
Morphological	Microcytic hypochromic	42	52.50
	Macrocytic	10	12.50
	Normocytic normochromic	10	12.50
	Dimorphic	10	12.50
	Normocytic hypochromic	8	10.00
Aetiological	Iron-deficiency anaemia	45	56.25
	Megaloblastic anaemia	15	18.75
	Anaemia of chronic disease	10	12.50
	Combined (iron + B12) deficiency	5	6.25
	Aplastic anaemia	5	6.25

Microcytic hypochromic anaemia was the predominant morphology. Nutritional anaemias (iron, B12 or both) accounted for 81.25% of cases. Anaemia of chronic disease and aplastic anaemia made up the rest.

Table 6: Treatment, hospital stay and outcome (n = 80)

Variable	Category	n	Percentage (%)
Treatment	Oral haematinics + nutritional rehabilitation	45	56.25
	Packed red cell transfusion	25	31.25
	Parenteral iron / vitamin B12	10	12.50
Hospital stay	≤7 days	46	57.50
	8–15 days	22	27.50
	>15 days	12	15.00
Outcome	Discharged	74	92.50
	Died	6	7.50

More than half the children were managed with oral therapy and diet alone, while about a third needed transfusion. Most were discharged within a week; six children (7.5%) died.

Table 7: Comparison of clinical and haematological parameters by sex

Parameter	Category	Male (n = 31)	Female (n = 49)	p-value
Haemoglobin	<5 g/dL	7 (22.6%)	8 (16.3%)	0.485
	5–7 g/dL	24 (77.4%)	41 (83.7%)	
Red cell indices	Microcytic hypochromic	21 (67.7%)	21 (42.9%)	0.084
	Macrocytic	2 (6.5%)	8 (16.3%)	
	Normal indices	8 (25.8%)	20 (40.8%)	
Platelet count	Decreased	8 (25.8%)	9 (18.4%)	0.428
	Normal	23 (74.2%)	40 (81.6%)	
General examination signs	—	—	—	0.729
Systemic examination signs	—	—	—	1.000
Associated illnesses	—	—	—	0.432

Chi-square test; $p < 0.05$ considered significant.

No clinical or haematological parameter differed significantly between boys and girls. Microcytic indices were more frequent among boys and macrocytosis among girls, but these differences did not reach significance ($p = 0.084$).

Discussion

In this cohort of school-age children with severe anaemia, nutritional deficiency was the dominant cause. Iron deficiency alone accounted for 56.25% of cases, and iron, vitamin B12 or both together for 81.25%. Infection was a frequent companion, and one child in thirteen died.

Clinical profile: Weakness was universal. Fever (75%), anorexia (75%) and breathlessness (62.5%) were the next most common complaints. Ramana Sastry reported weakness in 81.8% of children of the same age group, but fever in only 40.9% [11]. Janjale et al. recorded fever in 66.1% [10]. Our higher rate of fever reflects the large share of children with sepsis, viral illness and malaria. Pallor of the conjunctiva and nail beds was present in every child, as in most earlier series [11,17]. Pallor at these sites remains the most reliable bedside sign of severe anaemia. Koilonychia (31.25%) was close to the 36.3% reported by Ramana Sastry [11]. Angular stomatitis, glossitis

and bald tongue were more frequent in our children, which fits the high rate of vitamin B12 deficiency seen in this cohort. Hepatosplenomegaly (18.75%) was similar to earlier reports (17–18%) [10,11]. Twelve and a half per cent of children had congestive cardiac failure and 3.75% had neurological features, showing how quickly severe anaemia can decompensate.

Associated illnesses: Sepsis (20%), gastroenteritis (15%), viral infections (15%) and malaria (10%) were common. Infection worsens anaemia in several ways. Malaria destroys red cells directly, and severe dengue can cause bleeding.

Systemic inflammation raises hepcidin and traps iron in macrophages, producing anaemia of inflammation [8]. Infection also suppresses appetite and impairs nutrient absorption, so pre-existing iron and B12 deficiency is deepened.

Haematological profile: Haemoglobin was below 5 g/dL in 18.75% of children, slightly lower than the 23.7% reported by Janjale et al. [10]. Microcytic hypochromic anaemia was the most common morphology (52.5%). This is lower than the 81.8% in Ramana Sastry's study [11] but similar to Janjale et al. (47.5%) [10]. RDW was raised in 72.5% of our children. A high RDW with microcytosis is a sensitive early marker of iron deficiency and helps separate it from thalassaemia trait [14]. Macrocytic (12.5%) and dimorphic (12.5%) pictures were more frequent than in most Indian series [10–12]. This matches the low vitamin B12 levels we found and the predominantly vegetarian diet of our population [6,7]. Saba et al. similarly found dimorphic anaemia to be the second most common type among hospitalised children in Bangalore [12].

Aetiology: Iron-deficiency anaemia was the leading cause, in agreement with Ramana Sastry (58%) [11], Zareen et al. [17] and other Indian studies [12,13]. Megaloblastic anaemia was the second most common cause (18.75%), much higher than the 5% reported by Ramana Sastry [11].

Vitamin B12 deficiency is common among Indian vegetarians and is easily missed when iron and folic acid are prescribed empirically [6,7]. Aplastic anaemia (6.25%) and anaemia of chronic disease (12.5%) show that not all severe anaemia is nutritional. They underline the need for a full work-up before starting haematinics.

Treatment and outcome: Transfusion was required in 31.25% of children. This is comparable to Ogunlesi et al. (35.5%) [15], lower than Lundblad et al. (55.6%) [16] and higher than Jenifer et al. (14%) [18]. The variation largely reflects different transfusion thresholds. We transfused all children with Hb <5 g/dL and those with Hb 5–7 g/dL with cardiac failure, because many children came from remote areas without transfusion facilities. More than half the children (56.25%) recovered with oral therapy and diet, and most were discharged within a week. The mortality of 7.5% is consistent with the high case-fatality of severe anaemia complicated by sepsis and cardiac failure reported in resource-limited settings [9].

Conclusion

Severe anaemia in children aged 5–12 years in South Gujarat presents mainly with weakness, fever, anorexia and breathlessness, with universal pallor and frequent signs of nutritional deficiency. Iron-deficiency anaemia is the leading cause, but vitamin B12 deficiency, anaemia of chronic disease and aplastic anaemia together account for over 40% of cases.

Associated infections, particularly sepsis and malaria, are common and contribute to cardiac failure and death. A stepwise work-up should be completed before treatment. It should combine careful clinical examination with red cell indices, RDW, peripheral smear, iron studies, vitamin B12 levels and stool examination. Empirical iron–folic acid therapy should be avoided, and transfusion should be reserved for children with very low haemoglobin or cardiac decompensation. Early detection of iron and B12 deficiency through school-based screening is likely to prevent progression to severe disease.

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