



Original Article

Clinico-Hematological Profile and Etiological Spectrum of Hemolytic Anaemia in a Tertiary Care Centre in Western India: A Retrospective Cross-Sectional Study

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ABSTRACT

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Introduction: Hemolytic anaemia comprises a group of disorders characterized by premature destruction of red blood cells (RBCs), resulting in reduced RBC lifespan and anaemia when bone marrow compensation is inadequate. In the paediatric population, it is an important cause of morbidity and presents a considerable burden on healthcare systems, particularly in regions with a high prevalence of inherited hemoglobinopathies.

Methods: A retrospective cross-sectional study was conducted at District Hospital, Rajkot, over a period of one year. The study included 100 newly diagnosed and follow-up cases of hemolytic anaemia from different age groups. Socio-demographic details, clinical features, and laboratory findings were collected using a validated proforma. Patients were classified according to etiological profile and further categorized into acute and chronic hemolytic anaemia.

Results: Among the study subjects, females constituted 53% and males 47% of the cases. The highest prevalence was observed in patients below 20 years of age (44%). The mean haemoglobin level at presentation was 7.16 g/dL. Mild anaemia was observed in 3% of patients, while 31% had moderate anaemia and 66% presented with severe anaemia.

Among cases of acute hemolytic anaemia, inherited disorders accounted for 31% of cases, followed by infections including malaria, dengue, and viral hepatitis (17.3%), malignancies (13.8%), HELLP syndrome (13.8%), autoimmune causes (6.9%), and drug-induced haemolysis (3.4%). In chronic hemolytic anaemia, inherited hemoglobinopathies such as sickle cell disease, thalassemia, and HbD disease were the predominant causes (88.8%), followed by blood transfusion-related complications (4.2%), autoimmune causes (1.4%), and bone marrow failure syndromes (1.4%).

Sickling test and NESTROF test were done as a screening test for Sickle cell disorders and Thalassemia respectively. Reticulocyte count greater than 2% was observed in 46 patients, while 71 patients had serum bilirubin levels above 1.2 mg/dL. Massive splenomegaly and hepatomegaly were noted in 55 cases, and dark urine was present in 43 cases. Patients with thalassemia major required frequent transfusions (10–12 per year), whereas those with thalassemia intermedia and sickle beta-thalassemia required transfusions less frequently, approximately once every 1–2 years. Most sickle cell anaemia patients were managed conservatively, with transfusions reserved for severe cases.

Conclusion: Hemolytic anaemia is commonly encountered in the paediatric population, with inherited hemoglobinopathies, particularly thalassemia and sickle cell anaemia, being the most frequent causes. Haemoglobin electrophoresis and high-performance liquid chromatography (HPLC) remain essential diagnostic tools

for accurate classification. Early recognition, timely diagnosis, and appropriate management can significantly reduce complications and improve overall patient outcomes.

Keywords: Hemolytic anaemia, Sickle cell anaemia, Thalassemia, Autoimmune haemolysis, Paediatric anaemia..

INTRODUCTION

Anaemia continues to be a major global public health problem and remains a significant cause of morbidity across all age groups. Among its various forms, hemolytic anaemia represents an important subgroup characterized by premature destruction of red blood cells (RBCs), resulting in reduced erythrocyte survival and the development of anaemia when compensatory erythropoiesis by the bone marrow becomes insufficient [1]. Depending on the site of RBC destruction, hemolysis may occur intravascularly or extravascularly, mainly within the spleen and reticuloendothelial system [2].

Hemolytic anaemia encompasses a heterogeneous group of inherited and acquired disorders. Inherited causes include membrane defects, enzyme deficiencies, and hemoglobinopathies such as thalassemia and sickle cell disease, whereas acquired causes include autoimmune disorders, infections, drugs, microangiopathic processes, and systemic illnesses [3]. The underlying mechanism involves accelerated RBC destruction accompanied by compensatory erythroid hyperplasia in the bone marrow. Persistent hemolysis may eventually lead to significant clinical manifestations and systemic complications.

Patients commonly present with pallor, fatigue, jaundice, splenomegaly, dark-colored urine, and occasionally pigment gallstones due to chronic bilirubin overproduction [4]. Laboratory evaluation typically demonstrates anaemia with reticulocytosis, elevated lactate dehydrogenase (LDH), increased indirect bilirubin, and decreased serum haptoglobin levels. Peripheral blood smear examination remains a valuable initial diagnostic tool, while confirmatory investigations such as the direct antiglobulin (Coombs) test, osmotic fragility test, haemoglobin electrophoresis, and high-performance liquid chromatography (HPLC) help identify the underlying etiology [5].

Management of hemolytic anaemia depends upon the cause and severity of hemolysis. Treatment may include folic acid supplementation, corticosteroids, immunosuppressive therapy, blood transfusions, splenectomy, and disease-specific interventions [6]. Early diagnosis and timely treatment are essential to minimize complications, reduce transfusion dependence, and improve quality of life. Owing to its varied etiologies, broad clinical spectrum, and diagnostic complexity, hemolytic anaemia continues to remain an important area of study in hematology and clinical medicine.

MATERIALS AND METHODS

This retrospective cross-sectional observational study was conducted in the Department of Pathology at a tertiary care hospital in Rajkot, Gujarat, India, over a period of one year. The hospital caters to both urban and rural populations of Saurashtra and nearby regions. The study was undertaken to evaluate the clinico-etiological profile of acute and chronic hemolytic anaemia among patients presenting to the hospital.

Study Population

- Patients of all age groups diagnosed with hemolytic anaemia
- Both newly diagnosed and follow-up cases were included
- Total sample size: 100 cases

Clinical details, demographic characteristics, and laboratory findings of all patients were collected retrospectively from hospital records using a pre-validated proforma. Relevant investigations including complete blood count, peripheral blood smear examination, reticulocyte count, serum bilirubin, lactate dehydrogenase (LDH), haemoglobin electrophoresis, HPLC, and Coombs test wherever indicated, were reviewed to establish the diagnosis and identify the underlying etiology.

Patients were classified according to their etiological profile into inherited and acquired causes of hemolytic anaemia. Further subclassification into acute and chronic hemolytic anaemia was carried out based on clinical presentation, duration of symptoms, and laboratory findings. Clinical manifestations such as pallor, jaundice, splenomegaly, hepatomegaly, dark urine, and transfusion requirements were also evaluated. Peripheral smear findings were documented and correlated with the clinical diagnosis.

Inclusion Criteria

- Patients of all age groups diagnosed with hemolytic anaemia
- Newly diagnosed as well as follow-up cases
- Patients with adequate clinical and laboratory records available for analysis

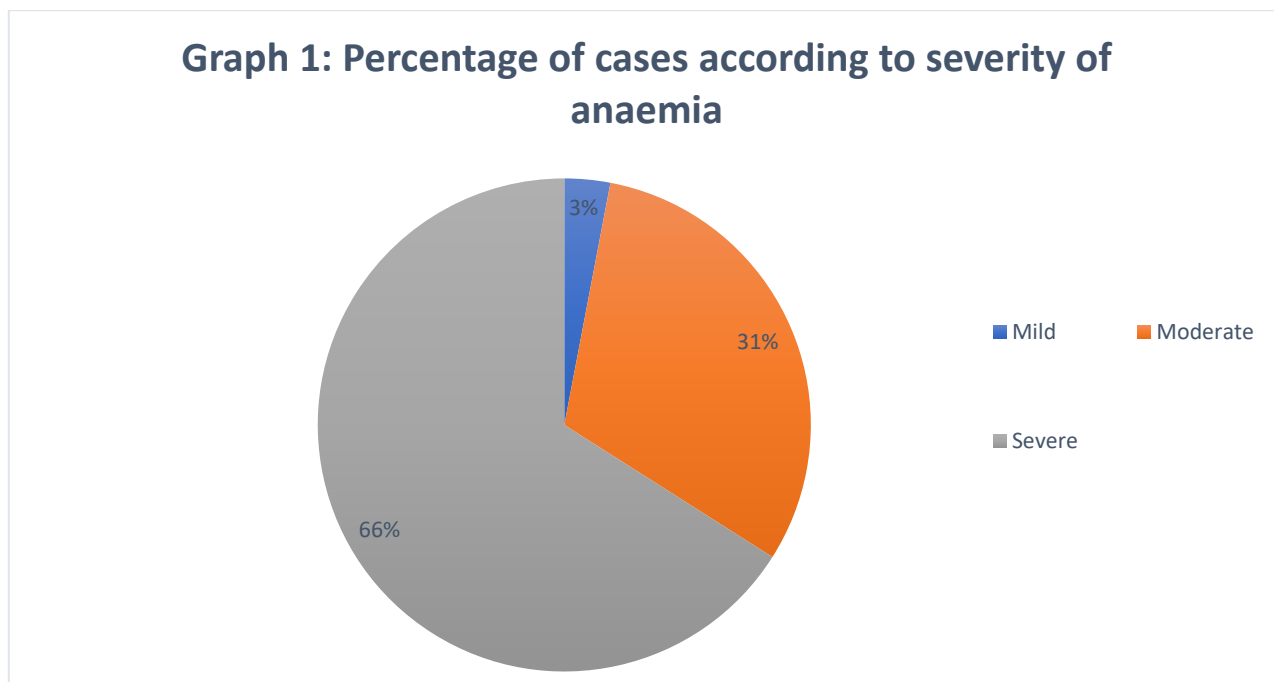
Exclusion Criteria

- Patients with incomplete clinical or laboratory records
- Cases of anaemia without evidence of hemolysis
- Nutritional anaemia and anaemia due to acute blood loss without hemolytic features

The collected data were compiled and analyzed using descriptive statistical methods. Results were expressed in the form of frequencies, percentages, and mean values wherever applicable. Institutional ethical guidelines were followed, and patient confidentiality was maintained throughout the study.

RESULTS

A total of 100 patients diagnosed with hemolytic anaemia were included in the present study. Detailed evaluation of the study population revealed that females constituted 53% of the cases, while males accounted for 47%. The majority of patients belonged to the younger age group, with 44% of cases occurring below 20 years of age. The mean haemoglobin level at presentation was 7.16 g/dL, indicating severe anaemia in a large proportion of patients.



Based on severity grading (3%) of patients had mild anaemia, (31%) had moderate and (66%) had severe anaemia. The mean haemoglobin at presentation was 7.16 g/dl

Among the total study population, 29 patients were diagnosed with acute hemolytic anaemia, whereas 71 patients had chronic hemolytic anaemia. Inherited causes formed the major etiological group in both acute and chronic hemolytic anaemia. Among acute hemolytic anaemia cases, inherited disorders such as G6PD deficiency and acute sickle cell crisis accounted for the highest proportion, followed by infections including malaria, dengue, and viral hepatitis. Autoimmune causes, HELLP syndrome, malignancies, drug-induced hemolysis, and other miscellaneous causes were also observed. In chronic hemolytic anaemia, inherited hemoglobinopathies including sickle cell disease, thalassemia, and HbD constituted the predominant etiological category.

Table 1: Distribution of subjects according to etiological profile of Acute hemolytic anaemia

Sr. No.	Etiological Profile	Number	Percentage (%)
1	Autoimmune	2	6.9
2	Inherited (G6PD, acute sickle crisis)	9	31
3	Infections (Malaria, Dengue, Viral hepatitis)	5	17.3
4	Drugs (Primaquine)	1	3.4
5	HELLP Syndrome	4	13.8
6	Malignancy	4	13.8
7	Others (Burns, Leukemoid reaction, HDN, other causes)	4	13.8
8	Total	29	100

Inherited causes such as G6PD deficiency and acute sickle cell crisis were the most common causes of acute hemolytic anaemia (31%), followed by infections including malaria, dengue, and viral hepatitis (17.3%). HELLP syndrome,

malignancy, and miscellaneous causes each accounted for 13.8% of cases, while autoimmune and drug-induced hemolysis were less frequent.

Table 2: Distribution of subjects according to etiological profile of Chronic hemolytic anaemia

Sr. No.	Etiological Profile	Number	Percentage (%)
1	Autoimmune (SLE)	1	1.4
2	Inherited (Hemoglobinopathies)	63	88.8
3	Blood transfusion complication	3	4.2
4	Bone marrow failure	1	1.4
5	Others (Cardiac valve disease, DIC)	3	4.2
6	Total	71	100

Inherited hemoglobinopathies, including sickle cell disease, thalassemia, and HbD, were the predominant causes of chronic hemolytic anaemia (88.8%). Blood transfusion-related complications and other causes such as cardiac valve disease and DIC each accounted for 4.2% of cases, while autoimmune hemolytic anaemia and bone marrow failure were uncommon (1.4% each).

Screening tests like Sickling was positive in all the 29 cases of sickle cell disease and 5 cases of sickle cell trait. NESTROF test was positive in all the 12 cases of thalassemia major and 5 cases of thalassemia minor.

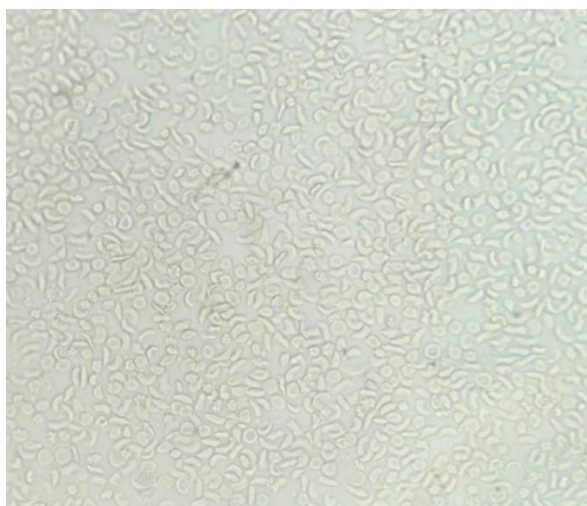


Figure 1: Sickling test positive in a case of sickle cell disease

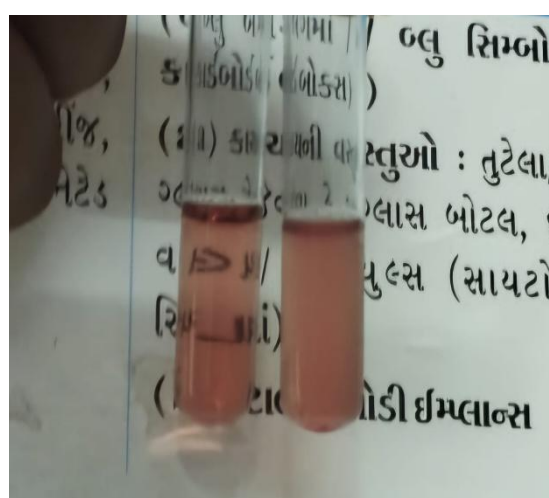


Figure 2: NESTROF test positive in a case of Thalassemia

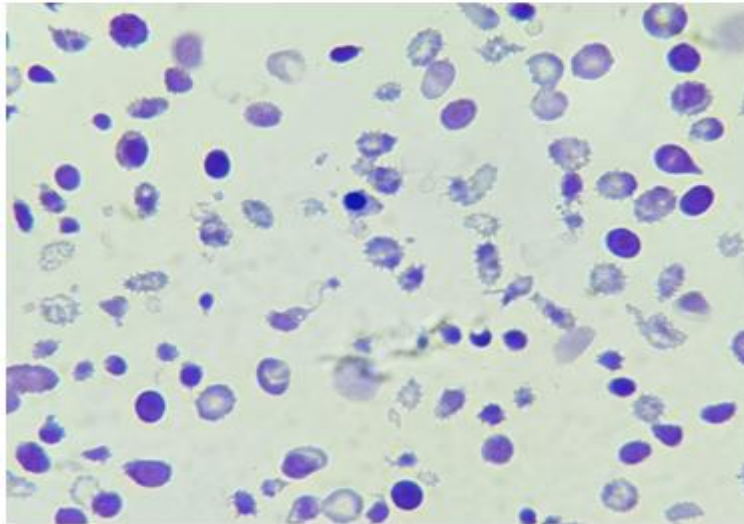


Figure 3: Peripheral smear showing Nucleated RBC and fragmented RBC's in a case of Hemolytic anaemia

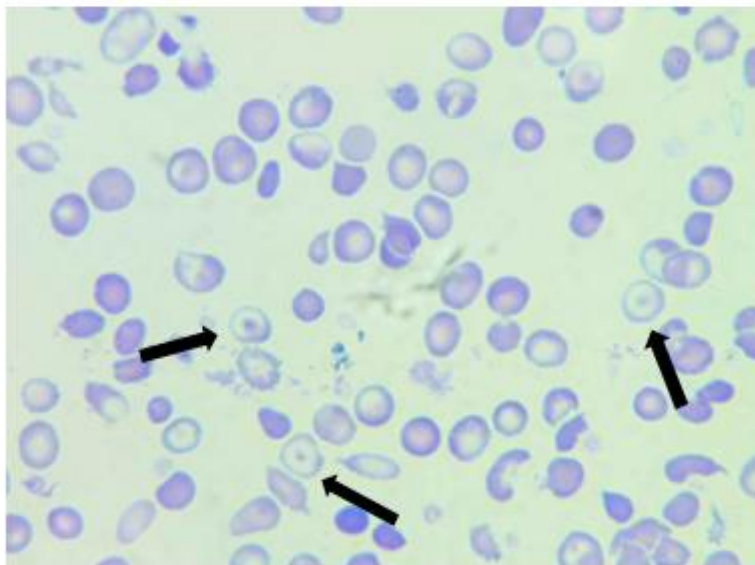


Figure 4: Peripheral smear showing Target cells and fragmented RBC's in a case of Thalassemia major

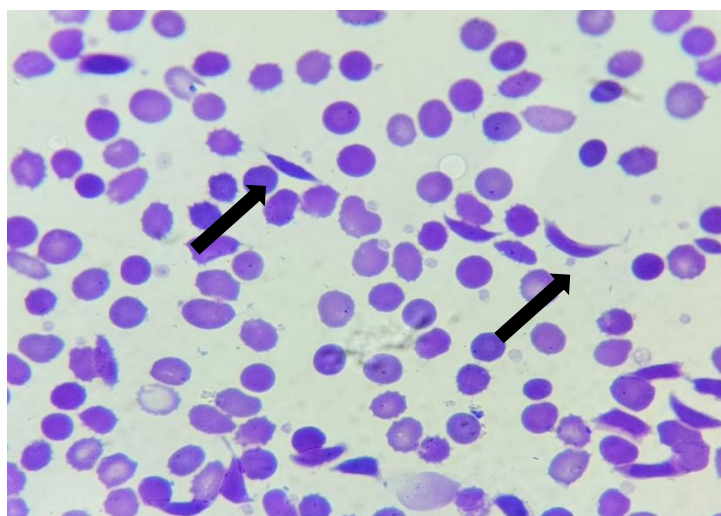


Figure 5: Peripheral smear showing Sickle cells in a patient with Sickle cell anaemia

Manual reticulocyte count using supravital stain demonstrated reticulocytosis (>2%) in 46 patients, suggesting increased marrow activity secondary to hemolysis. The highest reticulocyte counts were observed in sickle cell homozygous disease followed by thalassemia major. Hyperbilirubinemia was also a common laboratory finding, with 71 patients

showing serum bilirubin levels greater than 1.2 mg/dL. The highest bilirubin values were noted in hemolytic disease of the newborn and viral hepatitis-associated hemolysis.

Raised ESR values were observed in several cases, with the highest ESR documented in disseminated intravascular coagulation (DIC), followed by acute myeloid leukemia, systemic lupus erythematosus, and thalassemia major. Serum ferritin estimation performed in selected thalassemia major patients revealed elevated ferritin levels suggestive of transfusional iron overload, and iron chelation therapy was initiated in indicated cases.

Mean TSB was highest in autoimmune followed by thalassemia major, BT complication, sickle beta thalassemia, sickle cell disease. Erythrocyte sedimentation rate (ESR), measured by Wintrobe's method, was elevated in several patients. The highest ESR value was observed in disseminated intravascular coagulation (88 mm/hr), followed by acute myeloid leukemia, systemic lupus erythematosus, and thalassemia major.

Table 3: Hematological profile of study population

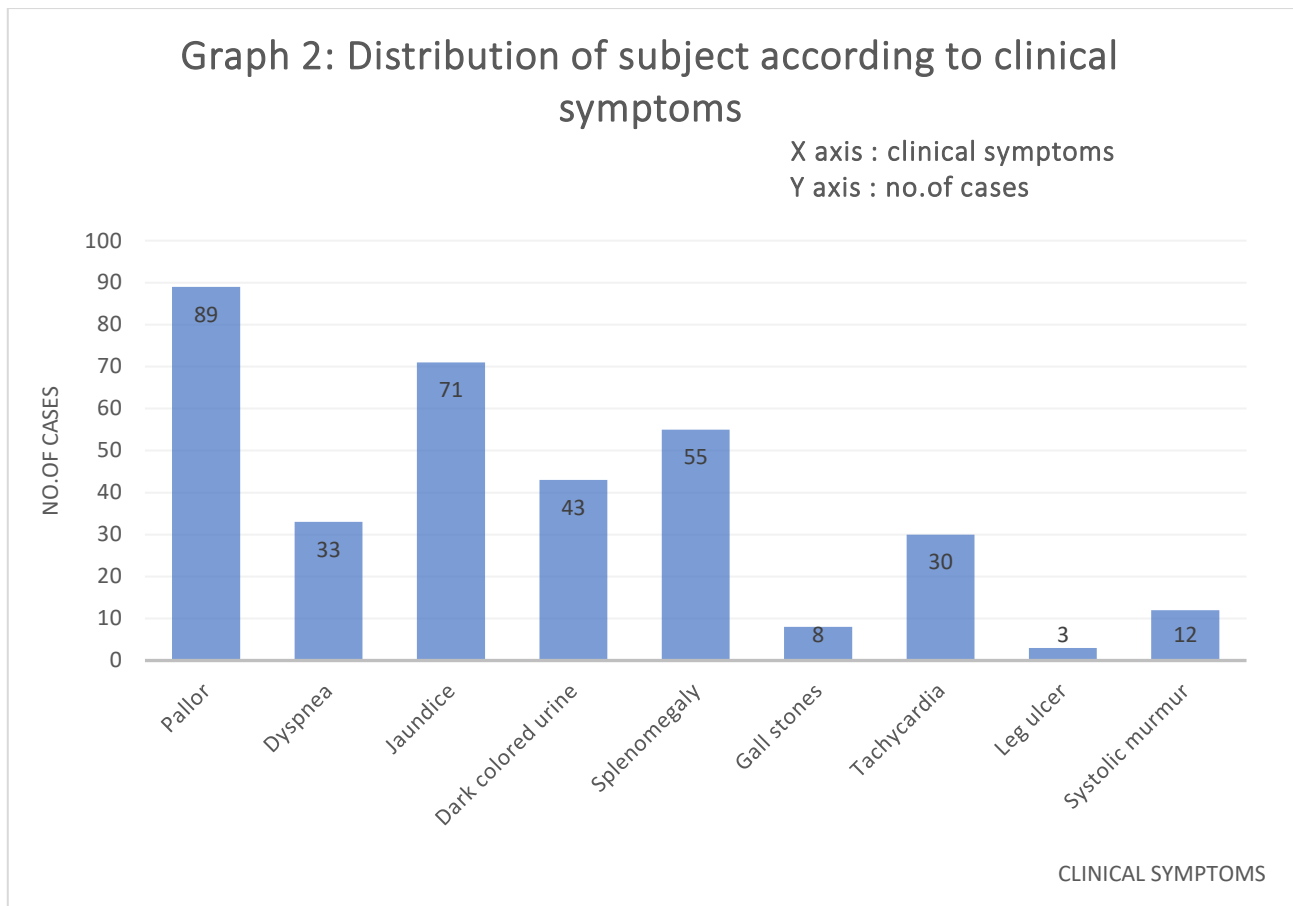
Causes	Mean Hb	Mean Reticulocyte Count	Mean Total Serum Bilirubin
Beta Thalassemia Major	5.3	7.5	4.9
Thalassemia Minor	8.4	3	2.4
Thalassemia Intermedia	6.7	5	3.1
Sickle Beta Thalassemia	6.4	7	4.2
Sickle Cell Disease	7.1	9	4
Sickle Cell Trait	9.8	2	1.9
HbD Punjab	8.2	3	2.1
Autoimmune	6.9	7	5.3
HELLP Syndrome	6.9	6	3.7
Infections	7.6	4	3.1
Malignancy	6.5	1.5	2.2
Bone Marrow Failure	5.9	0.8	1.8
BT Complication	7.1	6	4.4
Others	7.5	4.5	3

The highest mean reticulocyte count was observed in sickle cell disease followed by beta thalassemia major, indicating marked compensatory marrow activity. Mean total serum bilirubin levels were highest in autoimmune hemolytic anaemia followed by beta thalassemia major and blood transfusion-related complications. Patients with bone marrow failure showed the lowest reticulocyte counts

Patients with thalassemia major required frequent blood transfusions, averaging 10–12 transfusions per year, whereas patients with thalassemia intermedia and sickle beta-thalassemia required less frequent transfusion support. Most patients with sickle cell anaemia were managed conservatively, and blood transfusions were administered only during severe crises or complications.

Serum ferritin estimation was performed in 12 patients with thalassemia major, with a mean ferritin level of 1344 ng/mL. Iron chelation therapy was initiated in four patients with evidence of iron overload. The mean haemoglobin level among thalassemia major patients was 5.3 g/dL.

Clinically, splenomegaly and hepatomegaly were common findings, observed in 55 patients, while dark-colored urine was present in 43 patients. All patients with thalassemia major had splenomegaly, and a few required splenectomy due to massive splenic enlargement and associated symptoms. Jaundice was noted in the majority of cases, and leg ulcers were observed in some patients with sickle cell disease. Hyperbilirubinemia (total serum bilirubin >1.2 mg/dL) was seen in 71 cases, with the highest bilirubin levels recorded in hemolytic disease of the newborn (15 mg/dL), followed by viral hepatitis-associated hemolysis (10 mg/dL).



Splenomegaly was seen in 55 cases out of which 5 cases had massive splenomegaly (>20cm) causing abdominal discomfort. All cases of thalassemia major had splenomegaly. Splenectomy was undertaken in 2 cases. Jaundice was seen in 71 cases and leg ulcer was seen in 3 cases.

Sickle cell disease was the most common hemolytic disorder observed in the study population, followed by beta thalassemia major. Male predominance was noted in sickle cell disease and beta thalassemia major, whereas certain disorders such as HbD Punjab and DIC showed female predominance in this study. Positive family history was consistently observed in inherited hemoglobinopathies including thalassemia major, sickle cell disease, and sickle beta-thalassemia, highlighting their genetic basis. The majority of patients belonged to the pediatric and young age groups, particularly between 1–10 years of age. A history of similar illness among siblings and parental consanguinity was also commonly observed in inherited disorders

Table 4: Distribution of hemolytic anaemia according to sociodemographic variables

Type of Hemolytic Anaemia	Female	Male	Total	Family History		1–10	11–20	21–30	31–40	41–50	51–60	>60
				Yes	No							
Thalassemias												
Beta Thalassemia Major	4	8	12	12	0	2	3	1	3	1	1	1
Thalassemia Minor	3	3	6	0	6	1	1	1	2	1	0	0
Thalassemia Intermedia	1	0	1	0	1	1	0	0	0	0	0	0
Sickle cell disorders												
Sickle Cell Disease	12	17	29	29	0	15	3	4	3	3	0	1
Sickle Cell Trait	4	4	8	5	3	3	1	2	1	1	0	0
Sickle Beta	3	1	4	4	0	2	0	1	1	0	0	0

Thalassemia												
Others												
HbD Punjab	3	0	3	0	3	0	0	3	0	0	0	0
Autoimmune	2	1	3	0	3	0	1	1	0	0	1	0
DIC	2	0	2	0	2	0	0	2	0	0	0	0
Infections	2	4	6	0	6	1	2	1	1	1	0	0
Malignancy	3	1	4	0	4	1	0	0	1	1	1	0
BT Complication	0	3	3	0	3	0	0	0	2	0	1	0
Others	14	5	19	0	19	6	1	4	5	1	1	1
Total	53	47	100	50	50	32	12	20	19	9	5	3

Inherited hemoglobinopathies, particularly sickle cell disease and beta thalassemia major, were the most common hemolytic disorders observed. Male predominance and positive family history were commonly noted in these inherited conditions. Most patients belonged to the pediatric and young age groups, especially 1–10 years, with frequent history of similar illness among siblings and parental consanguinity.

Peripheral smear examination revealed anisopoikilocytosis as the most common RBC morphological abnormality, followed by hypochromic microcytic RBC's and fragmented RBC's. These findings were predominantly associated with inherited hemolytic disorders such as sickle cell disease and thalassemia. Target cells and sickle cells were characteristic findings in hemoglobinopathies. RBC inclusions were mainly seen in infection-related hemolysis, whereas RBC aggregates were observed in autoimmune hemolytic anaemia. Peripheral smear evaluation proved to be a valuable and cost-effective diagnostic tool in identifying the underlying etiology of hemolytic anaemia.

Table 5: Distribution of cases according to peripheral smear RBC morphology

RBC Morphology	Inherited Disorders	Autoimmune	Infections (Malaria ,Dengue , Viral hepatitis)	Malignancy & Blood transfusion complications	Others	Total
Hypochromic Microcytic RBCs	56	2	2	4	4	68
Macrocytic	3	0	2	1	1	7
Anisopoikilocytosis	70	1	1	2	2	76
Fragmented RBCs	50	2	4	6	5	67
Target Cells	25	0	1	2	4	32
Sickle Cells	41	0	0	0	0	41
RBC Inclusions	0	0	3	0	0	3
RBC Aggregates	0	3	0	0	0	3

Peripheral smear examination showed anisopoikilocytosis as the most common RBC abnormality, followed by fragmented RBCs and hypochromic microcytic RBCs, predominantly in inherited hemolytic disorders such as sickle cell disease and thalassemia. Target cells and sickle cells were characteristic of hemoglobinopathies. Peripheral smear evaluation proved to be a valuable and cost-effective tool in identifying the underlying etiology of hemolytic anaemia.

Leukocytosis was the most common WBC abnormality observed on peripheral smear examination, predominantly associated with inherited hemolytic disorders such as sickle cell disease and thalassemia. A shift to the left was also frequently noted, reflecting increased marrow activity and associated inflammatory response. Leukopenia was observed in a smaller number of cases, particularly in autoimmune and inherited disorders. Blast-like morphology was identified in one case associated with malignancy. Peripheral smear examination of the WBC series provided supportive diagnostic information regarding associated infections, inflammatory states, and hematological malignancies.

Table 6: Distribution of cases according to peripheral smear WBC morphology

WBC Morphology	Leukocytosis	Leukopenia	Shift to Left	Blast-like Morphology
Inherited Disorders	42	8	28	0
Autoimmune	0	2	0	0
Infections	3	0	0	0
Malignancy & BT Complication	5	1	1	1
Others	17	1	5	0
Total	67	12	34	1

Leukocytosis was the most common WBC abnormality observed, predominantly in inherited hemolytic disorders such as sickle cell disease and thalassemia, with frequent left shift indicating increased marrow activity and inflammation. Leukopenia was less common and mainly associated with autoimmune and inherited disorders, while blast-like morphology was identified in one malignancy case. Peripheral smear examination of the WBC series provided useful supportive diagnostic information regarding infections, inflammatory states, and hematological malignancies.

DISCUSSION

Hemolytic anaemia remains an important hematological disorder with diverse etiologies and clinical manifestations. In the present study, inherited hemoglobinopathies were the most common cause of chronic hemolytic anaemia, while infections and acute sickling crises contributed significantly to acute hemolytic anaemia. Similar observations have been reported from studies conducted in Gujarat, other parts of India, and various international populations [8,9].

The present study demonstrated a slightly higher prevalence among females (53%) compared to males (47%). However, most Indian studies have reported a male predominance, likely due to sociocultural differences in healthcare utilization and referral patterns [8,10]. The majority of patients in our study belonged to the younger age group (<20 years), which is comparable to studies by Mohanty et al. and Weatherall et al., where inherited hemoglobinopathies presented predominantly during childhood and adolescence [9,11].

The mean haemoglobin level at presentation in the present study was 7.16 g/dL, with severe anaemia observed in 66% of cases. Similar findings have been documented in studies from western India and Mediterranean countries involving transfusion-dependent thalassemia patients [9,12]. Severe anaemia at presentation may be attributed to chronic hemolysis, delayed diagnosis, recurrent crises, and inadequate transfusion support.

Inherited causes accounted for 88.8% of chronic hemolytic anaemia cases in the present study, with sickle cell disease and thalassemia being the predominant disorders. Gujarat is recognized as a high-prevalence region for sickle cell disease, especially among tribal communities, which explains the increased burden observed in our study population [8,13]. Similar findings were reported by Balgir et al., who highlighted the significant prevalence of hemoglobinopathies in western and central India [10].

Among acute hemolytic anaemia cases, infections such as malaria, dengue, and viral hepatitis constituted an important etiological group. Tropical infections are well-known triggers of hemolysis in developing countries and have also been described in studies from Africa and Southeast Asia [14]. Autoimmune hemolytic anaemia contributed to a smaller proportion of cases in the present study, which is consistent with observations from other Indian studies [15].

Reticulocytosis and hyperbilirubinemia were common laboratory findings, indicating active hemolysis and compensatory marrow response. Massive splenomegaly and hepatomegaly were predominantly observed in patients with thalassemia major and sickle cell disease. Comparable findings have been reported in studies from Gujarat and neighbouring states due to chronic extravascular hemolysis and extramedullary hematopoiesis [8,13].

Peripheral smear examination continues to play a significant role in the diagnosis of hemolytic anaemia. In the present study, anisopoikilocytosis, fragmented RBCs, target cells, and sickle cells were common findings. Similar morphological abnormalities have been described by Bain et al. and Firkin et al., emphasizing the diagnostic importance of peripheral smear evaluation in differentiating inherited and acquired hemolytic disorders [16,17].

The need for frequent blood transfusions among thalassemia major patients in the present study was comparable to previous Indian and international studies [12,18]. Elevated serum ferritin levels in transfusion-dependent patients underline the importance of regular monitoring and initiation of iron chelation therapy to prevent long-term complications associated with iron overload.

Overall, the present study confirms that inherited hemoglobinopathies remain the leading cause of hemolytic anaemia in Gujarat and other parts of India, whereas infections and autoimmune disorders contribute significantly to acute hemolytic episodes. Early diagnosis using haemoglobin electrophoresis and HPLC, along with timely management and genetic counselling, can substantially reduce morbidity and improve patient outcomes.

Table 7: Comparison of Demographic and Clinical Profile with Other Studies

Study	Region	Sample Size	Common Age Group	Male:Female Ratio	Most Common Etiology	Mean Hb (g/dL)
Present Study, 2026	Rajkot, Gujarat, India	100	<20 years	0.9:1	Sickle cell disease/Thalassemia	7.16
Patel et al. [7]	Gujarat, India	82	Pediatric age group	1.2:1	Sickle cell disease	6.2

Mohanty et al. [8]	Odisha, India	120	<30 years	1.3:1	Hemoglobinopathies	6.5
Balgir et al. [9]	Central India	95	Childhood	1.1:1	Sickle cell anaemia	5.8
Weatherall et al. [10]	United Kingdom	150	Childhood	1:1	Thalassemia	6.0

Table 8: Comparison of Etiological Profile of Hemolytic Anaemia

Study	Inherited Causes (%)	Autoimmune (%)	Infection-related (%)	Transfusion-related (%)
Present Study	88.8	1.4	17.3	4.2
Patel et al. [7]	84.0	3.0	10.0	2.0
Mohanty et al. [8]	79.0	5.0	14.0	2.0
Balgir et al. [9]	86.0	2.0	8.0	1.0
Akinyanju et al. [13]	82.0	4.0	18.0	3.0

Table 9: Comparison of Clinical Features

Clinical Feature	Present Study (%)	Indian Studies (%)	International Studies (%)
Jaundice	71	65–78 (Mohanty et al.[8], Balgir et al. [9])	60–75 (Weatherall et al.[10], Akinyanju et al. [13])
Splenomegaly	55	50–70 (Patel et al.[7], Balgir et al. [9])	48–68 (Weatherall et al.[10], Akinyanju et al. [13])
Hepatomegaly	46	40–62 (Patel et al. [7], Mohanty et al [8])	38–60 (Weatherall et al. [10])
Dark urine	43	35–48 (Mohanty et al [8])	30–45 (Akinyanju et al. [13])
Severe anaemia	66	58–72 (Patel et al. [7], Balgir et al. [9])	55–70 (Weatherall et al. [10], Borgna et al.[11])

CONCLUSION

The present study highlights that hemolytic anaemia remains an important cause of morbidity across all age groups, particularly among the pediatric and young population. Inherited hemoglobinopathies, especially sickle cell disease and thalassemia, constituted the major etiological group in chronic hemolytic anaemia, while infections and acute hemolytic crises were common causes of acute hemolysis. Clinical manifestations such as pallor, jaundice, splenomegaly, hepatomegaly, and severe anaemia were frequently observed.

Peripheral smear examination, sickling test, NESTROF test, reticulocyte count, haemoglobin electrophoresis, and HPLC proved to be valuable diagnostic tools in identifying the underlying etiology. Early diagnosis, appropriate transfusion support, iron chelation therapy, infection control, and genetic counselling play a crucial role in reducing disease-related complications and improving patient outcomes. Increased awareness and implementation of screening programs are essential, particularly in regions with a high prevalence of inherited hemoglobinopathies.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest regarding the publication of this study.

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REFERENCES

1. Phillips J, Henderson AC. Hemolytic anemia: evaluation and differential diagnosis. *Am Fam Physician.* 2018;98(6):354-361.

2. Berentsen S, Barcellini W. Autoimmune hemolytic anemias. *N Engl J Med.* 2021;385(15):1407-1419. doi:10.1056/NEJMra2033982
3. Baldwin C, Pandey S. Hemolytic anemia. StatPearls. Treasure Island (FL): StatPearls Publishing; 2023.
4. Hill A, Hill QA. Autoimmune hemolytic anemia. *Hematology Am Soc Hematol Educ Program.* 2018;2018(1):382-389. doi:10.1182/asheducation-2018.1.382
5. Jäger U, Barcellini W, Broome CM, et al. Diagnosis and treatment of autoimmune hemolytic anemia in adults: recommendations from the First International Consensus Meeting. *Blood Rev.* 2020;41:100648. doi:10.1016/j.blre.2019.100648
6. Michalak SS, Olewicz-Gawlik A, Rupa-Matysek J, et al. Autoimmune hemolytic anemia: current knowledge and perspectives. *Immun Ageing.* 2020;17:38. doi:10.1186/s12979-020-00208-7
7. Patel AP, Naik MR, Shah NM, Sharma NP, Parmar PH. Prevalence of common hemoglobinopathies in Gujarat: an analysis of a large population screening program. *Natl J Community Med.* 2012;3(1):112-116.
8. Mohanty D, Colah RB, Gorakshakar AC, et al. Prevalence of β -thalassemia and other haemoglobinopathies in six cities in India: a multicentre study. *J Community Genet.* 2013;4(1):33-42. doi:10.1007/s12687-012-0114-0
9. Balgir RS. The burden of haemoglobinopathies in India and the challenges ahead. *Curr Sci.* 2000;79(11):1536-1547.
10. Weatherall DJ, Clegg JB. Inherited haemoglobin disorders: an increasing global health problem. *Bull World Health Organ.* 2001;79(8):704-712.
11. Borgna-Pignatti C, Cappellini MD, De Stefano P, et al. Survival and complications in thalassemia. *Ann N Y Acad Sci.* 2005;1054:40-47. doi:10.1196/annals.1345.006
12. Colah R, Italia K, Gorakshakar A. Burden of thalassemia in India: the road map for control. *Pediatr Hematol Oncol J.* 2017;2(4):79-84. doi:10.1016/j.phoj.2017.10.002
13. Akinyanju OO. A profile of sickle cell disease in Nigeria. *Ann N Y Acad Sci.* 1989;565:126-136. doi:10.1111/j.1749-6632.1989.tb24160.x
14. Barcellini W. New insights in autoimmune hemolytic anemia: from pathogenesis to therapy. *J Clin Med.* 2020;9(12):3859. doi:10.3390/jcm9123859
15. Bain BJ. *Blood Cells: A Practical Guide.* 5th ed. Wiley-Blackwell; 2015.
16. Firkin F, Chesterman C, Penington D, Rush B. *de Gruchy's Clinical Haematology in Medical Practice.* 6th ed. Blackwell Science; 2003.
17. Cappellini MD, Porter JB, Viprakasit V, Taher AT. A paradigm shift on beta-thalassaemia treatment: how will we manage this old disease with new therapies? *Blood Rev.* 2018;32(4):300-311. doi:10.1016/j.blre.2018.02.001