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Original Research Article

# Clinico-hematological profile of hemolytic anaemia at the SAMS medical college hospital

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ABSTRACT

**Background:** Hemolytic anaemia is caused due to the higher rate of destruction of red cells (membrane) than the rate of their generation. It can be inherited from or acquired. Membrane destruction is majorly caused due to the defects of membrane and enzymes. HA can be diagnosed with laboratory test like complete blood count test, urine test etc.
**Aims and Objectives**: To study the clinical and pathological profile of hemolytic anaemia among the patients at Sri Aurobindo Institute of Medical Sciences (SAMS) medical college hospital.
**Materials and Methods:** This study included 150 patients diagnosed with HA for 12 months from Feb 2019 to March 2020. Clinico-Hematological profile of each patients was recorded.
**Results:** Current study observed that the 39.33% had beta thalassemia trait, sickle cell anaemia in 18.7%, beta thalassemia in 167.7%, malaria in 12%, sickle beta thalassemia 4.7%, and sickle cell trait. HA was more prevalent among male subjects (59%). Mean haemoglobin was least in thalassemia major (5.2gm/dl) and highest in sickle cell trait (9.2gm/dl). Mean total serum bilirubin was highest in beta thalassemia major patients and highest in beta thalassemia major (1821.4ng/dl). jaundice (57%), splenomegaly (47%) and hepatomegaly (34%) were common clinical manifestations.
**Conclusion**: HA is more prevalent among younger male population; beta thalassemia trait and sickle cell anaemia are the most common HA. Other than the hereditary causes malaria is the major cause HA.



## 1. Introduction

Anemia is caused due to the decreased levels of hemoglobin in the blood. The World Health Organization (WHO) criteria for anemia in men is less than 13 g/dL, whereas it is less than 12 g/dL for women. Hemolytic anemia (HA) is a form of low hemoglobin due to the destruction of red blood cells (RBC), increased hemoglobin catabolism and an increase in efforts of bone marrow. Hemolytic anemia (HA) is classified as normocytic anemia with mean corpuscular volume (MCV) of 80 to 100 fL.[1]

Various types of hemolytic anemia are defined based on ways and the underlying causes. Immune, non-immune mediated, intravascular, extravascular, inherited, acquired, intracorpuscular and extracorpuscular are known categories of HA.

Intracorpuscular causes are the internal abnormalities of the RBC. It can be internally damaged due to the changes in the solubility of hemoglobin (hemoglobinopathy), changes in the structure of the membrane or cytoskeleton (membranopathy), or due to the decreased metabolic abilities (enzymopathy) of the cell. Sickle cell disease (SCD) and thalassemia's are hemoglobinopathies, SCD is caused by a beta-globin gene mutation. Hereditary HA is mainly attributed to thalassemia, which is caused due the partial or complete lack of synthesis of alpha or beta globin chains of hemoglobin.[2]

RBC enzymopathies alter the shape of RBCs and causes the non-spherocytic HA's like G6PD (process the

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carbohydrates) deficiency and pyruvate kinase (rate limiting enzyme) deficiency (PKD).[3] This study attempts to reveal the clinical and hematological profile of patients with hemolytic anaemia attending the in SAMS Medical College Hospital, Indore, India.

## 2. Materials and Methods

Present observational study was conducted for 12 months at medical college hospital from Feb 2019 to March 2020. Post receiving the approval of ethics committee and patient's consent, 150 patients diagnosed with HA were included in this study. All the subjects were underwent the hemoglobin estimation, peripheral blood smear, reticulocyte count, serum bilirubin and serum ferritin tests. Specific tests like sickling test, osmotic fragility test, G6PD screening test. Ultrasonography of abdomen was done to detect organomegaly, gall stones or any other abnormality. Rapid diagnostic test for malaria was also done. Other secondary investigations done were CT scan, hepatitis markers, antinuclear antibody, etc were also considered. Physical examination included hepatomegaly, splenomegaly, hemolytic facies, jaundice, anthropometric measurements and history of blood transfusions, consanguinity were also noted.

SPSS ver. 20 was used to perform the data analysis. Frequency distribution and cross tabulation was used to prepare the tables. Quantitative data was expressed as mean whereas categorical data is expressed as number and percentage. No further statistical analysis was performed.

## 3. Result

As part of this study we have recorded the demographic parameters and results of various clinical-hematological tests of all 150 subjects. Beta Thalassemia Trait was the most common recorded HA which was prevalent among 39.33% subjects, followed by Sickle Cell Anaemia (18.67%) subjects and more prevalent among female subjects, Beta Thalassemia (major and intermedia) recorded in 16.67% subjects (Tables 1 and 2).

Out of 150, 81 (54%) subjects were in the age group 0-15 years followed by 57(38%) in age group 16-30 years. Our results show that HA is more prevalent among younger population (below 15 years) than the elders. Incidences of HA diminishes with increasing age (Table 1).

Male preponderance was seen in most the HA types, except the sickle cell anaemia which was recorded in 61% females. Over all 59% subjects were males and 41% were females.

Mean haemoglobin was found to be least in thalassemia major (5.2gm/dl) followed by Autoimmune Haemolytic Anaemia (6.8gm/dl), thalassemia intermedia (6.9gm/dl) and highest mean haemoglobin was found to be in sickle cell trait (9.2gm/dl). Jaundice (57%) was the most common finding followed by splenomegaly (47%), hepatomegaly (34%), gall stones (15%), growth retardation (12%). Direct Coomb's Test was only positive for AIHA.

Present study recorded the haematological parameters of the subjects as in Table 3. Mean total serum bilirubin was highest in beta thalassemia major patients as 3.75mg/dl and 3.35 mg/dl in G6PD HA patients. Similarly, highest mean of S. ferritin was observed in beta thalassemia major (1821.4ng/dl) followed by sickle cell anaemia (1386.2ng/dl) and malaria (1110.6ng/dl).

The mean Hb was least in beta thalassemia as 5.26gm/dl, followed by Autoimmune HA as 6.9. Sickle cell trait patients have significant higher mean haemoglobin as 8.9mg/dl. Two third of cases were had haemoglobin in the range of 5.2 to 8.2gm/dl, where 25 patients had low haemoglobin i.e. less than 5gm/dl and 10 subjects recorded higher levels of haemoglobin i.e. above 8.5gm/dl.

Other clinical manifestation include jaundice (56%), splenomegaly (48%), hepatomegaly (36%), gall stones (13%), haemolytic facies (2%), growth retardation (9%) and edema (5%). Splenomegaly was recorded in all the patients of beta thalassemia. No deaths were recorded during study.

## 4. Discussion

Hemolytic anemia is unevenly prevalent in India, which could be due to the factors like the demography and geography. Western and eastern parts have higher incidences of HA compared to southern parts of India. Central and north India have 7 to 8% incidence of HA among the patients of anemia.[4]

Present study observed the male to female ratio as 1.45 (59% male and 41% female), similar sex ratio of 1.51 (179 male and 118 female) is reported by to Chatopadhyay et al.[5] Another study by Anusha R et al, reported 0.9 as male to female which could be due the study setup and sample section.[6]

Current study noted the common HA's as beta thalassemia trait in 39.33%, followed by sickle cell anemia in 18.7% and beta thalassemia 16.67% subjects, similar observation were made in the study by Venkateshwary et al., they recorded thalassemia trait in 28.26% and thalassemia major in 16.45%.[7]

Another study by Ambekar SS et al. also found similar results where beta thalassemia trait was the most common HA.[8] Shivashankara et al. in similar study recorded most common HA as the beta thalassemia major followed by thalassemia trait, sickle cell trait and sickle cell thalassemia.[9]

Current study recorded the mean Hb as 7.2gm/dl and mean HbA2 of 6% among the beta thalassemia trait HA patients, it is similar to the results of Venkataswamy et al. where it observed the mean Hb and HbA2 of 13.3gm/dl and 6.2%.[7]

**Table 1:** HA distribution according to age

| HA Diagnosis | Age group in years | | | | Total | % |
|---|---|---|---|---|---|---|
| | 0-15 | 16-30 | 31-45 | 46-60 | | |
| Beta Thalassemia Trait | 46 | 11 | 2 | 0 | 59 | 39.33 |
| Beta Thalassemia | 17 | 8 | 0 | 0 | 25 | 16.67 |
| Sickle Cell Anaemia | 8 | 16 | 3 | 1 | 28 | 18.67 |
| Sickle Beta Thalassemia | 1 | 4 | 1 | 1 | 7 | 4.67 |
| Sickle Cell Trait | 1 | 1 | 1 | 0 | 3 | 2.00 |
| Autoimmune HA | 2 | 1 | 1 | 0 | 4 | 2.67 |
| G6PD deficiency | 4 | 1 | 1 | 0 | 6 | 4.00 |
| Malaria | 2 | 15 | 1 | 0 | 18 | 12.00 |
| Total | 81 | 57 | 10 | 2 | 150 | 100 |
| % | 54.00 | 38.00 | 6.67 | 1.33 | 100 | |

**Table 2:** HA diagnosis according to gender

| HA Diagnosis | Gender | | | | Total | % |
|---|---|---|---|---|---|---|
| | Male | % | Female | % | | |
| Beta thalassemia trait | 38 | 64.41 | 21 | 35.59 | 59 | 39.33 |
| Beta thalassemia | 14 | 56.00 | 11 | 44.00 | 25 | 16.67 |
| Sickle cell anaemia | 11 | 39.29 | 17 | 60.71 | 28 | 18.67 |
| Sickle beta thalassemia | 4 | 57.14 | 3 | 42.86 | 7 | 4.67 |
| Sickle cell trait | 1 | 33.33 | 2 | 66.67 | 3 | 2.00 |
| Autoimmune HA | 3 | 75.00 | 1 | 25.00 | 4 | 2.67 |
| G6PD deficiency | 6 | 100.00 | 0 | 0.00 | 6 | 4.00 |
| Malaria | 12 | 66.67 | 6 | 33.33 | 18 | 12.00 |
| Total | 89 | | 61 | | 150 | 100.00 |
| % | 59.33 | | 40.66 | | 100 | |

**Table 3:** Mean Haematological parameters distribution of various types of HA.

| Diagnosis | Total Serum Bilirubin (mg/dl) | Serum Ferritin (ng/dl) | Hb (gm/dl) |
|---|---|---|---|
| Beta thalassemia trait | 2.1 | 569.9 | 7.15 |
| Sickle cell anaemia | 3.05 | 1400 | 8.18 |
| Beta thalassemia | 3.75 | 1825 | 5.25 |
| Sickle beta thalassemia | 3.1 | 209.9 | 6.95 |
| Sickle cell trait | 1.75 | 280.9 | 8.9 |
| Autoimmune HA | 2.15 | 704 | 6.9 |
| Malaria | 2.85 | 1109.9 | 7.65 |
| G6PD deficiency | 3.35 | 136 | 7.78 |

Serum bilirubin was observed to be elevated in most of the patients which is similar to the observations were made by Preethi et al.[10]

In present study sickle cell anemia recorded in 18.7% subjects which is higher than the observation of 5.6% by Venkataswamy et al., this difference could be due to the number of subjects in different age groups.[7]

Beta thalassemia had 16.7% cases with mean hemoglobin and HbF of 5.25gm/dl and 91% similar observation were made by Preethi et al., where the Hb ranged from 3-8.2 gm/dl, and HbF mean was 75.2%. Total serum bilirubin in the study was 3.75mg/dl which is higher than 2.7mg/dl recorded by Anusha R et al.[6]

In present study sickle cell trait cases were 2% of with mean Hb of 9.1 gm/dl. In Venkataswamy et al, sickle cell trait comprised of 1.55% of all cases with mean values of Hb as 10.8gm/dl. The mean HbS recorded was 41.2% which is similar to 40% recorded by Venkataswamy et al.[7]

Present study based on peripheral smear findings recorded 6 cases of G6PD and 4 cases of Autoimmune HA were noted based on the G6PD estimation assay.

In present study malaria was the commonest acquired HA i.e. among 18 (12%) subjects, the mean Hb malarial subjects was 7.65gm/dl. Almost similar observations were made by Anusha R et al., where 21.9% patients were reported with malaria and the mean hemoglobin of these patients was 6.3gm/dl.[6]

## 5. Conclusion

Present study conclude that the HA is more prevalent among younger male population, among all types of HA, beta thalassemia trait and sickle cell anemia are the most common. Other than the hereditary causes malaria is the major cause of acquired HA. There is lack of awareness about HA which can be detected by normal hematological investigations. To reduce the disease burden of HA medical community should spread the awareness about it and conduct the hematological anemia patients to ascertain the risk of HA.

## 6. Source of Funding

None.

## 7. Conflict of Interest

The authors declare that there is no conflict of interest.

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