

---

**“ANTENATAL SCREENING FOR  
HEMOGLOBINOPATHIES IN A TERTIARY  
CARE CENTRE – A ONE YEAR DESCRIPTIVE  
OBSERVATIONAL STUDY”**

---

**BY**

**REG.NO: BJ0121008**

**Dissertation**

*Submitted to*

*KAHER, Belagavi, Karnataka,*

*In partial fulfilment of the requirements for the degree of*

**MASTER OF SURGERY (M.S.)  
In  
OBSTETRICS AND GYNECOLOGY**

**DEPARTMENT OF OBSTETRICS AND GYNECOLOGY  
JAWAHARLAL NEHRU MEDICAL COLLEGE, KAHER,  
BELAGAVI – 590010, KARNATAKA.**

---

**DECEMBER-2024 / JANUARY-2025**

---

KLE ACADEMY OF HIGHER EDUCATION AND RESEARCH,  
BELAGAVI, KARNATAKA.

## Endorsement

This is to certify that the dissertation entitled “Antenatal Screening For Hemoglobinopathies In A Tertiary Care Centre – A One Year Descriptive Observational Study” is a bonafide research work done by **Reg No: BJ0121008**.

  
**Dr. Yeshita Pujar** MS,FICOG

Professor and Head,  
Department of Obstetrics & Gynecology,  
J. N. Medical College,  
Nehru Nagar, Belagavi – 10

Date: 11/7/24

Place: Belagavi

  
**Dr. N.S. Mahantshetti** MD

Principal,  
J. N. Medical College,  
Nehru Nagar,  
Belagavi – 10

Date: 11/7/24

Place: Belagavi

## UNDERTAKING

I, **Reg.No. BJ0121008**, hereby declare that the information and the data mentioned in my dissertation entitled “**Antenatal Screening For Hemoglobinopathies In A Tertiary Care Centre – A One Year Descriptive Observational Study**” belongs to me and is original.

- An act or instance of using are closely imitating the language and thoughts of another author without authorization and the representation of that authors work as one’s own, as by not crediting the original author.
- A piece of writing or other work reflecting such unauthorised use or imitation.
- The deliberate or reckless representation of another’s words, thoughts, or ideas as one’s own without attribution in connection with submission of academic work, whether graded or otherwise.

I hereby declare that the dissertation prepared by me is original-one and does not involve plagiarism anywhere. In case at a later stage, it is found that I have indulged in plagiarism, then, I am solely responsible for the same and the institution is at liberty to take any disciplinary action against me including cancellation of dissertation or any other penalties imposed by the university.

Date: 10/7/2024

Place: Belagavi

  
**Reg. No. BJ0121008**

# PLAGIARISM CLEARANCE



## **JAWAHARLAL NEHRU MEDICAL COLLEGE**

(A constituent unit of KLE Academy of Higher Education & Research Deemed-to-be-University)

(Recognized by National Medical Commission, New Delhi)

Accredited 'A+' Grade by NAAC (3<sup>rd</sup> Cycle)

Placed in Category 'A' by MoE (GoI)



0831 - 2471350

0831 - 2470759

www.jnmc.edu

principal@jnmc.edu

Ref No: MDC/PG/

Date: 28-06-2024

### "ACCEPTANCE LETTER"

The softcopy of thesis entitled: "ANTENATAL SCREENING FOR HEMOGLOBINOPATHIES IN A TERTIARY CARE CENTRE - A ONE YEAR DESCRIPTIVE OBSERVATIONAL STUDY" has been submitted for anti-plagiarism check through Turnitin software. The scan has been carried out and the scanned output reveals a match percentage of 05% which is within the acceptable limits of 10% as per the guidelines given by UGC.



Guide.





Dr. (Mrs.) N.S. Mahantashetti.  
Chairperson-Antiplagiarism Committee &  
Principal,  
J. N. Medical College, Belagavi.

To,  
Reg. No. BJ0121008  
Postgraduate Student,  
2021-22 Batch,  
Department of Obstetrics & Gynaecology  
J. N. Medical College, Belagavi.

## ETHICAL CLEARANCE



K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH  
(Deemed – to- be- University)

Accredited 'A+' Grade by NAAC in (3<sup>rd</sup> Cycle) Placed in Category 'A' by MHRD (GoI)

**JNMC INSTITUTIONAL ETHICS COMMITTEE**  
**JAWAHARLAL NEHRU MEDICAL COLLEGE,**  
**NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)**

Website: <http://www.jnmc.edu>  
E-Mail : [dome@jnmc.edu](mailto:dome@jnmc.edu)

Phone: (+ 91-(0)831 Office : 2472550  
Principal: 2471701  
Fax No. +91 (0)831 – 2470759

Ref No.MDC/JNMCIEC/ 68

Date: 27/09/2022

To.

**REG.NO: BJ0121008**

PG Student in Obstetrics & Gynecology,  
J. N. Medical College,  
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled  
“ANTENATAL SCREENING FOR HEMOGLOBINOPATHIES IN A TERTIARY CARE  
CENTRE – ONE YEAR DESCRIPTIVE OBSERVATIONAL STUDY .” is ethical and  
justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics  
Committee.

**(Dr. Smita Sonoli)**  
Member Secretary  
JNMC Institutional Ethics Committee  
J.N.Medical College, Belagavi.

**(Dr. Harsha Hegde)**  
Chairman,  
JNMC Institutional Ethics Committee  
J.N.Medical College, Belagavi

## **LIST OF ABBREVIATIONS USED**

HPLC	High Performance Liquid Chromatography
WHO	World Health Organisation
NFHS	National Family Health Survey
RBC	Red Blood Cell
HbA	Hemoglobin A
HbS	Hemoglobin S
HbC	Hemoglobin C
HbS	Hemoglobin S
HbE	Hemoglobin E
HbD	Hemoglobin D
CVS	Chorionic Villus Sampling
SCD	Sickle Cell Disease
IUGR	Intra Uterine Growth Restriction
BTT	Beta Thalassemia Trait
SCT	Sickle Cell Trait
NESTROFT	Naked Eye Single Tube Redcell Osmotic Fragility Test
LMP	Last Menstrual Period
EDTA	Ethyl Diamine Tetra Acetic acid

PCV	Packed Cell Volume
MCV	Mean Corpuscular Volume
MCH	Mean Corpuscular Hemoglobin
MCHC	Mean Corpuscular Hemoglobin Concentration
SD	Standard Deviation
CM	Chorionic Villus Sampling
IDA	Iron Deficiency Anemia
ICMR	Indian Council of Medical Research

## **ABSTRACT**

### **Antenatal Screening for Hemoglobinopathies in a Tertiary Care Centre – A One Year Descriptive Observational Study**

**Background:** Hemoglobinopathies are inherited genetic disorders affecting the structure or production of hemoglobin, imposing significant public health concern. It is no more an endemic problem because of globalization of migration. In India, about 10,000 -15,000 babies are born with thalassemia major every year and the cost of optimal treatment for each child is over a lakh per year, which only few can afford. Implementing antenatal screening program can significantly reduce the burden as it facilitates early identification of carriers, enabling timely genetic evaluation and prenatal diagnosis and allows for informed decision-making by prospective parents regarding termination of pregnancy.

**Objectives:** The study aimed to perform early antenatal screening among pregnant women to find out the prevalence of hemoglobinopathies in them.

**Methods:** This descriptive observational study was conducted over 12 months. Pregnant women with a gestation period of  $\leq 20$  weeks were recruited through convenient sampling. Detailed history, general and obstetric examinations, and blood sample analyses (complete hemogram, peripheral blood smear, and HPLC) were performed. Couples at risk were identified and offered prenatal diagnosis.

**Results:** The study included 389 pregnant women, with 35 (8.99%) diagnosed with hemoglobinopathies, of them 94.29% had thalassemia minor and 5.71% had sickle cell trait. 74.2% of hemoglobinopathy cases were newly diagnosed and 25.7% were already known cases. Majority of the participants with thalassemia minor (78.7%) and

all the sickle cell trait (100%) had moderate anemia, with Thalassemia minor showed predominantly microcytic hypochromic anemia (78.7%), whereas Sickle cell trait had a mix of normocytic hypochromic and microcytic hypochromic anemia. 73% husbands of newly diagnosed participants with hemoglobinopathy opted for screening. Of the total high risk couple (11), 72.7% underwent genetic evaluation with amniocentesis and 37.5% of the tested fetus detected with hemoglobinopathy.

**Conclusion:** This study highlights a significant prevalence of hemoglobinopathies among pregnant women, predominantly Thalassemia Minor, with moderate anemia. Implementing comprehensive screening program is crucial for early detection and management, which can significantly improve maternal and fetal health outcomes.

**KEY WORDS:** Antenatal women, Hemoglobinopathies, Anemia, Thalassemia, Sickle cell anemia, genetic counselling

## **TABLE OF CONTENTS**

<b>SI NO</b>	<b>PARTICULARS</b>	<b>PAGE NO</b>
<b>1.</b>	<b>INTRODUCTION</b>	<b>1-3</b>
<b>2.</b>	<b>AIMS AND OBJECTIVES</b>	<b>4</b>
<b>3.</b>	<b>REVIEW OF LITERATURE</b>	<b>5-25</b>
<b>4.</b>	<b>MATERIALS AND METHODS</b>	<b>26-32</b>
<b>5.</b>	<b>RESULTS</b>	<b>33-52</b>
<b>6.</b>	<b>DISCUSSION</b>	<b>53-63</b>
<b>7.</b>	<b>CONCLUSION</b>	<b>64</b>
<b>8.</b>	<b>SUMMARY</b>	<b>65-66</b>
<b>9.</b>	<b>BIBLIOGRAPHY</b>	<b>67-83</b>
<b>10.</b>	<b>ANNEXURES</b>	<b>84-94</b>
	<b>ANNEXURE: I –INFORMED CONSENT FORM</b>	<b>84-87</b>
	<b>ANNEXURE: II – PROFORMA</b>	<b>88-93</b>
	<b>ANNEXURE: III – MASTER CHART</b>	<b>94</b>

## LIST OF TABLES

SL NO.	TABLE	PAGE NO
1	Prevalence of hemoglobinopathies	34
2	Distribution of participants according to type of hemoglobinopathies	35
3	Distribution of participants – Newly diagnosed vs Already known cases	36
4	Age group wise distribution of subjects according to prevalence of hemoglobinopathies	37
5	Distribution of participants according to Gestational age at enrollment	38
6	Association between degree of consanguinity and hemoglobinopathy	39
7	Distribution of participants based on degree of consanguinity	40
8	Distribution of participants according to proportion of anemia	41
9	Distribution of participants according to proportion of anemia among various Hemoglobinopathies	43
10	Distribution of participants based on Peripheral Smear	45
11	Distribution of participants according to Hematological Profile in Hemoglobinopathies	47
12	Association between Anemia and Hemoglobinopathy	49
13	Association between Anemia and Mentzer index	50
14	Association between Hemoglobinopathy and Mentzer index	51

## LIST OF FIGURES

<b>SL NO</b>	<b>FIGURES/GRAPHS</b>	<b>PAGE NO</b>
<b>1</b>	<b>HPLC equipment, Chromatogram and Report</b>	<b>29</b>
<b>2</b>	<b>Amniocentesis Report</b>	<b>30</b>
<b>3</b>	<b>Prevalence of hemoglobinopathies</b>	<b>34</b>
<b>4</b>	<b>Distribution of participants according to type of hemoglobinopathies</b>	<b>35</b>
<b>5</b>	<b>Distribution of participants - Newly diagnosed vs Already known cases</b>	<b>36</b>
<b>6</b>	<b>Age group wise distribution of subjects according to prevalence of hemoglobinopathies</b>	<b>37</b>
<b>7</b>	<b>Distribution of participants according to Gestational age at enrollment</b>	<b>38</b>
<b>8</b>	<b>Distribution of participants based on consanguinity</b>	<b>39</b>
<b>9</b>	<b>Distribution based on degree of consanguinity and hemoglobinopathy</b>	<b>40</b>
<b>10a</b>	<b>Distribution of participants according to proportion of anemia among subjects without hemoglobinopathies</b>	<b>41</b>
<b>10b</b>	<b>Distribution of participants according to proportion of anemia among subjects with hemoglobinopathies</b>	<b>41</b>
<b>11a</b>	<b>Distribution of participants according to proportion of anemia among Thalassemia Minor</b>	<b>44</b>

<b>11b</b>	<b>Distribution of participants according to proportion of anemia among Sickle cell trait</b>	<b>44</b>
<b>12</b>	<b>Distribution of participants based on Peripheral Smear</b>	<b>46-47</b>
<b>13</b>	<b>Distribution of participants according to Hematological Profile in Hemoglobinopathies</b>	<b>48</b>
<b>14</b>	<b>Association between Anemia and Hemoglobinopathy</b>	<b>49</b>
<b>15</b>	<b>Association between anemia and Mentzer index</b>	<b>50</b>
<b>16</b>	<b>Association between Hemoglobinopathy and Mentzer index</b>	<b>51</b>
<b>17</b>	<b>STROBE diagram</b>	<b>52</b>

## **INTRODUCTION**

Anaemia in pregnancy is emerging as one of the most important causes of maternal morbidity and offspring mortality in almost all the developing countries including India.<sup>1</sup> India contributes to about 80 % of the maternal deaths due to anemia in South Asia.<sup>2</sup> In 2021, the WHO has estimated that the prevalence of anemia in pregnant women is 37 % globally, with more prevalence in developing countries.<sup>3</sup> According to the latest NFHS-5 (December 2020), the prevalence of anemia in pregnant women in India is 52.2%.<sup>4</sup> The most common causes of anemia among pregnant women are iron deficiency and inherited hemoglobinopathies.

Hemoglobinopathies are group of diseases characterized by abnormalities in both quantitative and qualitative production of the hemoglobin. This affects the hemoglobin molecule in its structure, function or production.<sup>5</sup> Being an important cause of morbidity and mortality, they impose a heavy burden on families and the health sector in our country. Worldwide, around 80-90 million people are carriers for the hemoglobinopathy genes<sup>3</sup>. An estimated 7% of the world population carry an abnormal hemoglobin gene, while about 3,00,00 -5,00,000 are born annually with significant hemoglobin disorders.<sup>6</sup> India has the largest number of children with Thalassemia major in the world – about 1 to 1.5 lakhs and almost 42 million carriers of  $\beta$  (beta) thalassemia trait.<sup>7</sup>

The Government of India is presently engaged in implementing a prevention and control program for haemoglobinopathies at the national level and the guidelines have been formulated under the terms of the National Health Mission and published by the Ministry of Health and Family Welfare (2016), with a final policy currently under deliberation.<sup>7</sup>

The major concerned hemoglobinopathic disorders in India are  $\beta$ - thalassemia and sickle cell anemia.<sup>8</sup> Thalassemia is a hereditary blood disorder caused by mutations in the genes (alpha or beta) responsible for hemoglobin production. This condition leads to the production of abnormal hemoglobin and consequent reduction in the number of healthy red blood cells, resulting in anemia.<sup>9</sup> Thalassemia is classified into several types based on the specific globin chain affected ( alpha or beta), most commonly beta chain leading to Beta-Thalassemia Major and Intermedia and Minor.<sup>10</sup> The severe forms of thalassemia are prevalent in the Mediterranean, Middle Eastern, South Asian, and Southeast Asian populations.<sup>11,12</sup> The high frequency of carriers in these regions underscores the importance of screening to manage and reduce the incidence of these disorders.<sup>13</sup> Also, Sickle cell disease is caused by a mutation in the beta-globin gene, leading to the production of abnormal hemoglobin known as hemoglobin S (HbS). Under low oxygen conditions, HbS polymerizes, causing red blood cells to become rigid and sickle-shaped. They cause a high degree of morbidity, moderate to severe hemolytic anemia among vulnerable segments of the society like infants and children, pregnant women leading to several deaths in India.<sup>14</sup> The prevalence varies across different regions and communities, with higher rates observed in tribal populations and certain states such as Gujarat, Maharashtra, and Tamil Nadu and North East states.<sup>15</sup>

***Need for the study:***

The prevalence of hemoglobinopathies in antenatal women in India is ranging from 4 -12% in different geographical locations. Hemoglobinopathies with carrier traits are usually unaware of their condition and future prospects. If a couple carry a clinically significant hemoglobinopathy trait, there is 1 in 4 chance with each pregnancy that their children will inherit a major hemoglobinopathy and this forms the

basis and major objective of antenatal screening.<sup>16</sup> In India, about 10,000 -15,000 babies with thalassemia major are born every year.<sup>17</sup> The cost of optimal transfusion and chelation treatment for each child is around Rs.1,20,000-1,40,000 per year, which only 10-15% could afford.<sup>18</sup> The most effective approach to reduce the burden of society is to reduce the incidence by implementation of a carrier screening program.

Antenatal screening for hemoglobinopathies is a crucial aspect of antenatal care, aiming to identify these conditions early in pregnancy by thorough family history assessment and blood investigations to detect abnormal hemoglobin variants.<sup>19,20</sup>

Early antenatal detection of these hemoglobinopathies is essential for several reasons<sup>21,22</sup>:

- a) Enables timely genetic evaluation, there by the couple gets the option of prenatal diagnosis for testing their fetus for hemoglobinopathy
- b) Allows for informed decision-making by prospective parents regarding termination of pregnancy if required.
- c) Facilitates the management of maternal and fetal risks associated with these disorders
- d) Facilitates in planning the delivery at a higher centre, equipped to handle high risk pregnancies with multi-disciplinary approach.

Hence, the present study was conducted with the aim to perform early antenatal screening for Hemoglobinopathies among women attending KLE's Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi.

**AIMS AND OBJECTIVES**

To find the prevalence of hemoglobinopathies in pregnant women in early antenatal period at or before 20 weeks of gestation

## **REVIEW OF LITERATURE**

Anemia in pregnancy is a prevalent and significant health concern, affecting a remarkable number of pregnant women globally. It is characterized by a reduction in the number of red blood cells or hemoglobin concentration, which diminishes the blood's ability to carry oxygen to the body's tissues.<sup>23</sup> The most common cause of anemia in pregnancy is iron deficiency, which accounts for approximately 75% of cases. This deficiency occurs due to the increased iron demands of the growing fetus and placenta, expanded maternal blood volume, and often inadequate dietary intake. Other causes include deficiencies in folate and vitamin B12, and genetic conditions like hemoglobinopathies as sickle cell disease and thalassemia, which affect hemoglobin production and there by function.<sup>24,25</sup> Pregnant women are particularly susceptible to anemia due to these increased nutritional demands.<sup>26</sup> A cross sectional observational study on antenatal women over one year found a high prevalence of anemia with 81.8% with a mean hemoglobin concentration of 9.06g/dl.<sup>27</sup> Another study of anemia in young pregnant Indian women found moderate to severe anemia cases, with prevalent iron deficiency in 65%. Dimorphic anemia was observed in 18.3%, and 11.6% showed hemolytic anemia, half of which were thalassemia trait.<sup>28</sup>

Anemia during pregnancy is linked to several adverse outcomes, such as preterm birth, intercurrent infections, heart failure during antepartum, postpartum hemorrhage, cardiac failure, shock during labour and delayed healing, subinvolution, lactation failure, puerperal sepsis, pulmonary embolism during puerperium leading to an elevated risk of maternal and perinatal mortality. It has also been associated with low birth weight, long-term negative effects on the cognitive and physical development of infants.<sup>29,30</sup>

Management of anemia in pregnancy involves early routine screening to detect and diagnose anemia. Nutritional supplementation, particularly with iron and folic acid, is a standard preventive measure. In cases where anemia is due to deficiencies in other nutrients, appropriate supplements are administered.<sup>26,31</sup> For hemoglobinopathies, specialized care and treatment plans are necessary. Effective management and prevention strategies are crucial to improving maternal and fetal health outcomes, emphasizing the importance of regular prenatal care and monitoring.<sup>32,33</sup>

During pregnancy, the complications of hemoglobinopathies can arise from physiological changes or the complications inherent to the condition. The increased physiological demands of pregnancy, such as increased oxygen requirements, can exacerbate existing anemia or disrupt hemoglobin production, particularly in women with pre-existing hemoglobinopathies.<sup>34</sup> Additionally, complications like placental insufficiency or hormonal changes can further complicate the condition, potentially worsening maternal anemia or triggering vaso-occlusive crises.<sup>35</sup> Iron deficiency, common in pregnancy, may exacerbate anemia in women with hemoglobinopathies. Genetic inheritance, where abnormal hemoglobin genes are passed down from parents, predispose offspring to conditions like sickle cell disease or thalassemia. Recognizing these causes is essential for comprehensive prenatal care, including genetic counselling and multidisciplinary management, to optimize outcomes for both mother and the fetus.<sup>36,37</sup>

### **Introduction to Hemoglobinopathies:**

Hemoglobinopathies are a group of inherited blood disorders characterized by abnormalities in the structure, function, or production of hemoglobin, the protein molecule in red blood cells (RBCs) responsible for carrying oxygen throughout the

body.<sup>38</sup> These disorders result from genetic mutations or variations affecting the genes encoding the alpha or beta globin chains of hemoglobin, leading to the production of abnormal hemoglobin variants.<sup>39</sup>

## **Hemoglobin Structure and Function:**

Hemoglobin is a crucial protein found in red blood cells that plays a central role in transporting oxygen from the lungs to tissues throughout the body and facilitating the removal of carbon dioxide. Understanding its structure and function is essential for comprehending its physiological significance in human body.

### ***1. Hemoglobin Structure:***

Hemoglobin is a complex protein composed of four polypeptide subunits, each bound to a heme group. The heme group contains an iron ion ( $\text{Fe}^{2+}$ ) at its centre, which is crucial for oxygen binding. The protein portion consists of two pairs of globin chains: two alpha chains and two beta chains in adult hemoglobin (HbA). The specific arrangement of these subunits forms the quaternary structure of hemoglobin, known as a tetramer.<sup>40,41</sup>

### ***2. Oxygen Binding and Release:***

The primary function of hemoglobin is to bind oxygen in the lungs and release it to tissues throughout the body. This process involves a reversible binding interaction between oxygen and the iron ions within the heme groups.<sup>42</sup> When oxygen binds to one heme group, it induces a conformational change in the hemoglobin molecule, making it easier for subsequent oxygen molecules to bind. This phenomenon is known as cooperative binding, allowing hemoglobin to efficiently load and unload oxygen as needed.<sup>43</sup>

### ***3. Cooperativity and the Bohr Effect:***

Hemoglobin exhibits cooperativity, meaning that the binding of one oxygen molecule enhances the affinity of hemoglobin for subsequent oxygen molecules. Additionally, factors such as pH, carbon dioxide concentration, and temperature influence the affinity of hemoglobin for oxygen.<sup>44</sup> The Bohr effect describes how increased acidity (lower pH) and higher levels of carbon dioxide in tissues promote the release of oxygen from hemoglobin, facilitating oxygen delivery to metabolically active tissues.<sup>45</sup>

### ***4. Role in Carbon Dioxide Transport:***

In addition to oxygen transport, hemoglobin plays a crucial role in carrying carbon dioxide away from tissues to the lungs for exhalation. Carbon dioxide binds to specific amino acid residues on the globin chains of hemoglobin, forming carbamino hemoglobin. This reversible binding allows hemoglobin to transport carbon dioxide from tissues to the lungs, where it can be released and exhaled.<sup>46</sup>

### ***5. Hemoglobin Variants and Genetic Disorders:***

Genetic mutations affecting the structure or function of hemoglobin can lead to various hemoglobinopathies, such as sickle cell disease and thalassemia. Sickle cell disease results from a single amino acid substitution in the beta globin chain, causing hemoglobin molecules to polymerize and distort red blood cells into a sickle shape. Thalassemia is characterized by reduced synthesis of one or more globin chains, leading to abnormal hemoglobin production and anemia.<sup>47,48</sup>

## **6. Regulation of Hemoglobin Synthesis:**

The synthesis of hemoglobin is tightly regulated at the genetic level. Transcription factors and regulatory elements control the expression of globin genes, ensuring the proper balance of alpha and beta globin chains. Imbalances in globin chain synthesis can lead to hemoglobinopathies and associated clinical manifestations.<sup>49,50</sup>

### **Types of Hemoglobinopathies:<sup>51</sup>**

#### ***Sickle Cell Disease (SCD):***

Sickle cell disease is one of the most well-known hemoglobinopathies. It is caused by a mutation in the HBB gene, leading to the production of abnormal hemoglobin known as hemoglobin S (HbS). This abnormal hemoglobin causes red blood cells to become stiff and sickle-shaped, leading to vaso-occlusive crises, anemia, organ damage, and other complications.<sup>48,52</sup>

#### ***Thalassemia:***

Thalassemia is a group of inherited blood disorders characterized by reduced or absent production of one of the globin chains that make up hemoglobin.<sup>53,54</sup> There are two main types of thalassemia:

***Alpha Thalassemia:*** Alpha thalassemia results from mutations in the HBA1 and HBA2 genes, which code for alpha-globin chains. Depending on the number of affected genes, alpha thalassemia can be classified as silent carrier, alpha thalassemia trait, hemoglobin H disease, or hydrops fetalis.<sup>55</sup>

***Beta Thalassemia:*** Beta thalassemia results from mutations in the HBB gene, which codes for beta-globin chains. Depending on the severity of the mutation, beta

thalassemia can be classified as beta thalassemia minor (trait), beta thalassemia intermedia, or beta thalassemia major (Cooley's anemia).<sup>56</sup>

***Hemoglobin C Disease (HbC Disease):*** Hemoglobin C disease is caused by a mutation in the HBB gene, resulting in the production of abnormal hemoglobin C (HbC). Individuals with hemoglobin C disease may experience mild hemolytic anemia and have red blood cells with characteristic HbC crystals.<sup>57</sup>

***Hemoglobin E Disease (HbE Disease):*** Hemoglobin E disease is caused by a mutation in the HBB gene, resulting in the production of abnormal hemoglobin E (HbE). Depending on the specific genetic mutation, individuals with hemoglobin E disease may have mild to moderate hemolytic anemia and may be at risk of complications such as splenomegaly and gallstones.<sup>58</sup>

***Other Variants and Compound Heterozygous States:*** In addition to the above-mentioned hemoglobinopathies, there are numerous other variants of hemoglobin that can lead to clinical disorders when present in certain combinations or when compounded with other genetic mutations. Examples include hemoglobin D, hemoglobin Lepore, and various combinations of hemoglobinopathies such as sickle beta thalassemia.<sup>59</sup>

These are the main types of hemoglobinopathies, each with its own distinct genetic basis, clinical features, and management considerations. Proper diagnosis, genetic counselling, and management strategies are essential for individuals affected by these disorders to optimize their health and quality of life.<sup>60</sup>

### **Pregnancy in Women with Hemoglobinopathies:**

Pregnancy in women with hemoglobinopathies indeed poses significant challenges and increased risks for both the mother and the fetus, largely due to the interplay between the physiological changes inherent to pregnancy and the underlying complexities of these conditions.<sup>61</sup>

***Increased Blood Volume:*** Pregnancy results in an expansion of blood volume to support the growing fetus. However, in women with hemoglobinopathies, such as sickle cell disease or thalassemia, this increased blood volume can exacerbate underlying anemia. Anemia, a common complication of hemoglobinopathies, can lead to reduced oxygen delivery to tissues, exacerbating symptoms such as fatigue and weakness in the mother.<sup>62</sup>

***Increased Oxygen Demand:*** The increased oxygen demand during pregnancy, primarily to support fetal growth and development, further strains the compromised oxygen-carrying capacity of hemoglobin in women with hemoglobinopathies. This can exacerbate symptoms of anemia and increase the risk of complications such as vaso-occlusive crises in sickle cell disease, where oxygen deprivation triggers painful episodes of tissue ischemia and infarction.<sup>63</sup>

***Altered Immune Function:*** Pregnancy is associated with alterations in immune function, aimed at protecting the developing fetus while maintaining maternal health. However, these changes can have implications for women with hemoglobinopathies, potentially increasing susceptibility to infections and complications such as acute chest syndrome in sickle cell disease. Additionally, the altered immune response may influence the severity and frequency of vaso-occlusive crises or other complications associated with hemoglobinopathies.<sup>64,65</sup>

***Effects on Fetal Development:*** The challenges faced by the mother with a hemoglobinopathy also impact fetal development. Reduced oxygen delivery due to maternal anemia can compromise fetal oxygenation and nutrient supply, potentially leading to *intrauterine growth restriction, preterm birth, and low birth weight*. Furthermore, maternal complications such as pre-eclampsia or thromboembolic events can directly affect fetal health and increase the risk of adverse pregnancy outcomes.<sup>66,67</sup>

In summary, the physiological changes of pregnancy exacerbate the complexities of managing hemoglobinopathies, posing increased risks for both the mother and the fetus. Understanding the interplay between hemoglobinopathies and the physiological changes of pregnancy is crucial for optimizing maternal and fetal outcomes. Close monitoring, comprehensive prenatal care, and multidisciplinary management are essential to mitigate the risks associated with hemoglobinopathies during pregnancy.<sup>68</sup>

### **Diagnosis of Hemoglobinopathies:**

The diagnosis of hemoglobinopathies during pregnancy typically involves a combination of prenatal screening and genetic evaluation.<sup>69,70</sup>

***Prenatal Screening:*** During early prenatal care visits, women may undergo routine blood tests to screen for hemoglobinopathies and to assess the risk of transmitting the condition to offspring.<sup>22</sup> These tests may include complete blood count (CBC) to assess hemoglobin levels and red blood cell indices, as well as hemoglobin electrophoresis or high-performance liquid chromatography (HPLC) to identify abnormal hemoglobin variants.<sup>71</sup>

**Genetic Counselling:** If screening tests indicate the presence of a hemoglobinopathy, genetic counselling is often recommended. Genetic counsellors can provide the information about the condition, assess the risk of passing it to offspring, and discuss available testing options like Chorionic Villus Sampling (CVS) or Amniocentesis.<sup>72</sup>

**Confirmation Testing:** Confirmation of a hemoglobinopathy diagnosis may involve additional laboratory tests, such as DNA analysis or molecular genetic testing, to identify specific mutations or gene deletions associated with the disorder. This helps confirm the diagnosis and provides more detailed information about the type and severity of the condition.<sup>73</sup>

### **Management Strategies:**

Management of hemoglobinopathies in pregnancy often requires a multidisciplinary approach involving obstetricians, hematologists, genetic counsellors, and other specialists.<sup>33</sup> This team collaborates to optimize maternal health, minimize complications, and ensure the best possible outcomes for both mother and fetus. Here are key management strategies:

**Regular Antenatal Care:** Early and regular antenatal care is essential for pregnant individuals with hemoglobinopathies. This includes monitoring maternal hemoglobin levels, hematocrit, and other blood parameters to detect anemia and monitor for the complications and assess the need for interventions.<sup>74</sup>

**Supplemental Iron and Folic Acid:** Iron and folic acid supplementation is important to prevent and treat anemia during pregnancy. Pregnant individuals with hemoglobinopathies may require higher doses of iron supplementation due to increased demands and potential iron deficiency. However, iron overload to be avoided in thalassemia major.<sup>75</sup>

***Genetic Counselling and Prenatal Testing:*** Genetic counselling should be offered to couples at risk of having a child with a hemoglobinopathy. Prenatal testing, such as chorionic villus sampling (CVS) or amniocentesis, may be recommended to diagnose fetal hemoglobinopathies and guide management decisions.<sup>76</sup>

***Maternal Monitoring:*** Throughout pregnancy, women with hemoglobinopathies require close monitoring of maternal health, including regular assessment of hemoglobin levels, red blood cell indices, and markers of disease severity. This monitoring helps detect complications such as anemia, vaso-occlusive crises, or pregnancy-related complications like pre-eclampsia.<sup>68</sup>

***Close Monitoring of Fetal Well-being:*** Regular fetal monitoring, including ultrasound examinations to assess fetal growth and development, as well as tests such as Doppler ultrasound to evaluate blood flow in the umbilical artery and fetal non-stress tests, help in assessing fetal growth and well-being and detect any signs of fetal distress or complications related to hemoglobinopathies.<sup>34,77</sup>

### **Complications and Management:**

***Maternal Risks and Management:*** Pregnant women with hemoglobinopathies are at increased risk of complications such as vaso-occlusive crises, thrombosis, and pre-eclampsia. Prompt recognition and management of these complications are essential to prevent adverse outcomes for both the mother and the fetus.<sup>52</sup>

***Anemia:*** Pregnant women with hemoglobinopathies often experience exacerbated anemia due to increased blood volume and higher iron demands. Regular monitoring of hemoglobin levels and hematocrit is crucial. Iron supplementation should be carefully managed, especially in thalassemia patients who may suffer from iron overload.<sup>78</sup>

***Vaso-occlusive & Pain Crises:*** Women with SCD are prone to vaso-occlusive crises, which can be triggered or worsened by pregnancy. Pain management strategies need to be safe for both mother and fetus, often requiring a multidisciplinary approach including obstetricians, hematologists, and pain specialists.<sup>79</sup> *Hydroxyurea*, a medication that increases fetal hemoglobin production, may be considered in pregnant individuals with sickle cell disease to reduce the frequency of vaso-occlusive crises and complications. However, the safety of hydroxyurea during pregnancy is still being studied, and its use should be carefully monitored.<sup>80</sup>

***Infections:*** Hemoglobinopathy patients are at higher risk for infections, which can be more severe during pregnancy. Prophylactic antibiotics, vaccination, and vigilant monitoring for signs of infection are essential.<sup>81</sup>

***Pregnancy-Induced Hypertension:*** There is an increased risk of hypertensive disorders, including preeclampsia, particularly in SCD. Regular blood pressure monitoring and early intervention are necessary.<sup>82</sup>

***Thrombosis:*** Pregnant women with hemoglobinopathies have a higher risk of thromboembolic events. Prophylactic anticoagulation may be considered, especially in those with a history of thrombotic events or in the postpartum period.<sup>83</sup>

#### **Fetal Risks and Management :**

***Growth Restriction:*** Fetus of mothers with hemoglobinopathies are at risk of intrauterine growth restriction (IUGR) due to placental insufficiency. Regular ultrasound monitoring for fetal growth and well-being is crucial.<sup>84</sup>

***Preterm Birth:*** There is an increased risk of preterm labour and delivery. Close monitoring and timely intervention can help manage preterm labour effectively.<sup>85</sup>

***Fetal Anemia:*** In cases of thalassemia, there is a risk of the fetus inheriting severe anemia, particularly in pregnancies where both parents are carriers. Fetal anemia can be monitored using Doppler ultrasound of the middle cerebral artery.<sup>36</sup>

***Genetic Transmission:*** Genetic counselling is important for parents who are carriers of hemoglobinopathies. Prenatal testing, such as chorionic villus sampling or amniocentesis, can diagnose hemoglobinopathies in the fetus, allowing for informed decision-making.<sup>86</sup>

### **Delivery and Postpartum Care:**

***Delivery:*** Delivery should be planned at a tertiary care centre equipped to handle high-risk pregnancies, taking into account maternal health status, fetal well-being, and the presence of any complications related to hemoglobinopathies. The timing and mode of delivery should be individualized based on maternal and fetal conditions.<sup>87</sup> Close communication between obstetricians, hematologists, and other healthcare providers is essential to ensure a safe delivery for both the mother and the baby.<sup>88</sup>

***Postpartum Care:*** The postpartum period poses a high risk for complications such as infections, thromboembolism, and worsening anemia. Close monitoring and appropriate interventions are critical during this time.<sup>89</sup>

***Breastfeeding:*** Mothers with hemoglobinopathies can generally breastfeed, but they should be supported with adequate nutritional and medical care to ensure their health and the well-being of their baby.<sup>90</sup>

A prospective study on the outcome of pregnancies in patients with hemoglobinopathies showed caesarean section as predominant obstetric outcome with sickle cell disease patients requiring more blood transfusions compared to beta

thalassemia trait. Perinatal outcomes were notably poorer among sickle cell disease cases compared to beta thalassemia and the study recorded 1.67% stillbirth and 3.33% spontaneous abortions.<sup>91</sup> A multicentre retrospective study assessed maternal and neonatal outcomes in pregnant women with a hemoglobinopathy trait. Women with the hemoglobinopathy trait experienced increased rates of adverse maternal outcomes such as abortion, gestational diabetes mellitus, urinary tract infections, intrahepatic cholestasis, abnormal placentation, and postpartum anemia with no significant difference in neonatal outcomes.<sup>61</sup> Another observational study assessed the impact of hemoglobinopathies on fetomaternal health and concluded that Hemoglobinopathies significantly impact pregnancy outcomes, increasing maternal morbidities such as pre-eclampsia and preterm labour, as well as neonatal morbidities like low birth weight and neonatal mortality.<sup>92</sup>

Overall, managing hemoglobinopathies during pregnancy requires a comprehensive and individualized approach, with close collaboration between obstetricians, hematologists, genetic counsellors, and other members of the healthcare team. The goal is to optimize maternal and fetal outcomes while minimizing the risks associated with the underlying hemoglobinopathy.<sup>93</sup>

**Public Health Measures:**

*Education and Awareness:* Public health initiatives aimed at raising awareness about hemoglobinopathies, promoting genetic counselling, and providing support to affected individuals and families are crucial for improving outcomes and reducing the burden of disease.<sup>94</sup>

**Screening for Hemoglobinopathies:** Screening programs for genetic disorders, such as Sickle Cell Disease (SCD), beta-thalassemia, and alpha-thalassemia, are essential

public health initiatives aimed at reducing the burden of these diseases.<sup>95</sup> In need for an updated guidelines, *Bain et al.*<sup>96</sup> provided comprehensive recommendations for the effective screening and diagnosis of major haemoglobinopathies to ensure accurate and timely identification and management of these conditions.

**Timing and Modalities of Screening:**

**Population-based Screening:** In areas with high disease prevalence or carrier rates, screening may be offered to children of school age to identify at-risk individuals early on. This approach aims to empower individuals to make informed choices about their future reproductive partners.<sup>97</sup>

A large population based study performed to determine the prevalence of hemoglobinopathies, focusing on beta thalassemia trait (BTT) and sickle cell trait (SCT), found a higher incidence of mild to moderate anemia in individuals with BTT and SCT and BTT emerged as the most prevalent hemoglobinopathy, emphasizing specific caste groups at higher risk for both BTT and SCT.<sup>98</sup> Another population based prevalence study showed high incidence of various hemoglobinopathies mainly beta thalassemia, HbE, HbS trait, HbS disease, HbD.<sup>99</sup> Another comparative study of hemoglobinopathies in tribal populations highlighted the significance of screening young adolescents, particularly in areas with a high incidence of hemoglobinopathies.<sup>100</sup>

A Study screened 35,413 rural individuals for hemoglobinopathies and found a notably higher prevalence of  $\beta$ -thalassemia trait compared, providing crucial data for the design and implementation of screening programs in that area.<sup>101</sup>

A 10-year study at a tertiary care hospital, screened more than one lakh population, 12.17% of prevalence of hemoglobinopathies. The most common

abnormalities identified were  $\beta$ -thalassemia trait (4.60%), HbE trait (3.02%),  $\beta$ -thalassemia major/intermedia (1.66%), and E $\beta$  thalassemia (1.16%). Other variants, including HbE disease and sickle-cell disease, were also observed.<sup>8</sup>

***Premarital/Pre-conception Screening:*** In regions where medical termination of affected pregnancies is not permitted or acceptable, screening may be offered prior to conception, often during the premarital stage. This allows couples to assess their risk of carrying genetic disorders before making reproductive decisions.<sup>76</sup>

A study program conducted as a premarital screening program for hemoglobinopathies, identified high-risk couples and successfully reduced the affected birth rate by 21.1%.<sup>104</sup> In a single-centre study, conducted among reproductive-aged women, particularly prospective mothers, revealed hemoglobinopathies prevalence rate of 4.61%.<sup>102</sup>

Another study conducted at a tertiary care hospital, emphasized the significance of screening for hemoglobinopathies, particularly beta thalassemia and hemoglobin E heterozygous with 29.3% of screened subjects tested positive and underscored the urgent need for routine premarital screening programs to detect and prevent high-risk marriages.<sup>34</sup>

***Early Gestation Screening:*** In many countries, antenatal screening is offered to pregnant women during early gestation, allowing parents to make informed decisions about their reproductive options based on the screening results.<sup>103</sup> A review study conducted by *Chakravorty et al.*<sup>104</sup>, offered insights into the current status, methods, ethical concerns, and outcomes of antenatal screening for SCD and thalassemia worldwide, emphasizing the need for expanded screening programs, particularly in regions with high prevalence such as sub-Saharan Africa and India.

**Newborn Screening:** Newborn screening programs identify infants born with genetic disorders, such as SCD and thalassemia, to offer early treatment and prevent early mortality. This complements antenatal screening efforts by providing timely intervention and support for affected individuals.<sup>105</sup>

**Antenatal Screening for Hemoglobinopathies:**

Antenatal screening for hemoglobinopathies is a critical component of prenatal care, aimed at identifying individuals or couples who carry genetic mutations associated with clinically significant hemoglobin disorders, such as thalassemia and sickle cell disease. These screening programs are typically integrated into routine prenatal care and offer genetic testing to pregnant women and their partners to assess their carrier status for hemoglobinopathies, providing the individuals at high risk of having affected babies with information and options regarding their reproductive decisions, including prenatal genetic diagnosis and the option of termination of pregnancy.

An observational study among 467 pregnant women found 14.99% prevalence of hemoglobinopathies with HbE heterozygous (9.85%) and HbE homozygous (2.57%), followed by Beta Thalassemia Trait (1.93%), double heterozygous (0.43%), and Hb-D Iran (0.21%)<sup>106</sup>. Another study evaluated a 12.26% prevalence of hemoglobinopathies with sickle cell trait (7.45%),  $\beta$ -thalassemia trait (2.89%), hemoglobin E trait (0.24%), and sickle cell disease (1.68).<sup>107</sup>

Another study among pregnant women revealed 21.4% prevalence of hemoglobinopathies, including Thalassemia carrier (17.5%), Thalassemia major (2.1%), Sickle cell disease (1.6%), HbD (1.09%), and HbE heterozygous (0.54%). The study emphasized the importance of early antenatal screening for

hemoglobinopathies and advocated for routine compulsory screening due to the high prevalence of these conditions.<sup>108</sup>

A study was conducted to identify couples at risk of severe hemoglobinopathies in their children through antenatal screening and diagnosis and found that 3.38% had  $\beta$ -thalassemia trait, 1.5% had sickle cell trait, and 0.86% had other hemoglobin variants. High-risk couples received counselling, and 78.5% chose prenatal diagnosis, resulting in the termination of three pregnancies with fetus having homozygous  $\beta$ -thalassemia. The study underscored challenges including late antenatal registration, lack of cooperation from husbands, and refusal of prenatal diagnosis, highlighting the critical need for enhanced awareness and education to improve program outcomes.<sup>71</sup>

The findings by a community-based pilot study, underscored 26.6% hemoglobinopathy prevalence, particularly Hb E (15.42%), with majority among the Rajbanshi community, emphasizing need for broad community-based initiatives for carrier detection and awareness.<sup>109</sup>

Screening methods may include laboratory tests like hemoglobin electrophoresis or high performance liquid chromatography to detect abnormal hemoglobin variants or mutations in the globin genes.<sup>69,110</sup>

A study was conducted for antenatal screening of hemoglobinopathies by using NESTROFT, a cost-effective mass screening tool, although confirmation using HPLC or hemoglobin electrophoresis is recommended.<sup>111</sup>

Another study assessed the role of high-performance liquid chromatography (HPLC) in detecting haemoglobinopathies among antenatal women at a tertiary care hospital, concluded that utilizing HPLC during pregnancy, coupled with partner

screening, provides prospective parents with the opportunity to assess fetal risk for haemoglobinopathy.<sup>112</sup>

A study compared the diagnostic efficacy of electrophoresis with high-performance liquid chromatography (HPLC) for hemoglobinopathies and concluded that HPLC, with its simplicity, superior resolution, accurate quantification, and automation, is ideal for routine clinical diagnosis of hemoglobin disorders.<sup>113</sup>

### **Risk of Inheriting Major Hemoglobinopathy:**

If a couple carries a clinically significant hemoglobinopathy trait, such as thalassemia or sickle cell trait, there is a one in four chance with each pregnancy that their child will inherit a major hemoglobinopathy. This means that with each pregnancy, there is a 25% chance that the child will inherit two copies of the mutated gene, resulting in a severe form of the hemoglobin disorder, such as thalassemia major or sickle cell disease.<sup>114</sup>

Implementing a carrier screening program for clinically significant hemoglobinopathies is a crucial public health intervention aimed at reducing the incidence and burden of these genetic disorders within society. Carrier screening involves identifying individuals who carry a single copy of a mutated gene associated with a specific disorder, such as thalassemia or sickle cell disease. Couples identified as carriers of a clinically significant hemoglobinopathy trait are at risk of having children with a major hemoglobinopathy, such as thalassemia major or sickle cell disease.<sup>97</sup>

**Key Components of a Carrier Screening Program:**

***Education and Awareness:*** An essential first step in implementing a carrier screening program is raising awareness and educating the public, healthcare providers, and at-risk communities about the significance of hemoglobinopathies, inheritance patterns, and the benefits of carrier screening.<sup>115</sup>

***Access to Screening Services:*** Accessible and affordable screening services should be available to individuals of reproductive age, particularly within communities with a high prevalence of hemoglobinopathies. Screening may be offered through primary care clinics, community health centres or specialized genetic counselling centres.<sup>13</sup>

***Genetic Counselling:*** Genetic counselling is integral to carrier screening programs, providing individuals and couples with information about their carrier status, the risk of having an affected child, reproductive options, and available genetic evaluation procedures.<sup>116</sup>

***Testing Methods:*** Screening tests for hemoglobinopathies may include laboratory techniques such as hemoglobin electrophoresis, high-performance liquid chromatography (HPLC), or molecular genetic testing. These tests can identify carriers of abnormal hemoglobin variants or mutations in the globin genes.<sup>52</sup>

A study emphasized the utility of molecular diagnostic techniques in hemoglobin disorders, focusing on their role in genetic counselling and enabling prenatal testing to prevent severe outcomes in offspring as Traditional protein-based methods like electrophoresis and chromatography, commonly used for diagnosing thalassemia and hemoglobinopathies, may fail to detect serious conditions such as  $\alpha$ -thalassemia in the presence of  $\beta$ -thalassemia or deletional forms of  $\beta$ -thalassemia.<sup>117</sup>

**Benefits of a Carrier Screening Program:**

***Prevention of Major Hemoglobinopathies:*** By identifying carrier couples and providing them with information about their reproductive risks, carrier screening programs can help prevent the birth of children with severe hemoglobinopathies, thus reducing the overall disease burden within the population.<sup>97</sup>

A prospective study conducted among tribal populations for antenatal screening for Hemoglobinopathies, found 7% with hemoglobinopathy traits or diseases, including Thalassemia and Sickle Cell Anemia and 13% of screened husbands tested positive. Amniocentesis report revealed 31.58% normal fetuses, 47.37% trait fetuses, and 21.05% affected fetuses, leading to medical termination of pregnancy as necessary, highlighting the importance of antenatal screening and diagnosis in preventing haemoglobinopathies.<sup>118</sup>

***Improved Family Planning:*** Carrier screening empowers individuals and couples to make informed decisions about family planning based on their carrier status and the risk of having an affected child. This may include options such as prenatal diagnosis, preimplantation genetic diagnosis, or adoption.<sup>119</sup>

***Early Intervention and Support:*** Early identification of carrier status allows for timely interventions, such as genetic counselling, prenatal diagnosis, and specialized medical care for affected individuals and families. Additionally, carrier screening programs can provide access to support services and resources for individuals and families affected by hemoglobinopathies.<sup>115</sup>

***Reduced Healthcare Costs:*** By preventing the birth of children with major hemoglobinopathies, carrier screening programs can potentially reduce the economic

burden associated with lifelong medical care, including hospitalizations, blood transfusions, iron chelation therapy, and complications management.<sup>120</sup>

In conclusion, implementing a carrier screening program for clinically significant hemoglobinopathies is a proactive and effective approach to reduce the incidence and burden of these genetic disorders within society. By providing individuals and couples with information about their carrier status and reproductive risks, carrier screening programs empower individuals to make informed decisions about family planning and facilitate early intervention and support for affected individuals and families. Collaboration between healthcare providers, policymakers and advocacy groups is essential for the successful implementation and sustainability of carrier screening programs.

## **MATERIALS AND METHODS**

The present study was conducted among the pregnant women attending KLE's Dr. Prabhakar Kore Hospital and Medical Research Centre.

**STUDY DESIGN:** Descriptive Observational Study

**STUDY POPULATION:** Pregnant women with Period of Gestation  $\leq$  20 weeks, meeting selection criteria, attending antenatal OPD at KLE's Dr. Prabhakar Kore Hospital and Medical Research Centre

**STUDY PERIOD:** 12 months (April 2023 to March 2024)

**SELECTION CRITERIA:**

**INCLUSION CRITERIA:**

1. The Antenatal women with period of gestation  $\leq$  20 weeks (calculated by reliable LMP/ scan) attending OPD at KLE's Prabhakar Kore Hospital and Medical Research Centre, through convenient sampling
2. Women willing to provide informed consent for the study

**EXCLUSION CRITERIA:**

1. Women who had recent blood transfusion (within 1 month)

**ETHICAL CLEARANCE:** The study was approved by JNMC Institutional Ethics Committee, Jawaharlal Nehru Medical College, Belagavi. (vide. 22/9/2022 Ref No. MDC/JNMCIEC/68)

**CTRI REGISTRATION:** The study trial was also registered with Central Trial Registry of India. (vide.10/1/2023 Reg No. CTRI/2023/01/048851)

**SAMPLE SIZE**: The minimum sample required to carry out the present study was calculated using the following formula

Formula used for sample size calculation is

$$n = \frac{p(100-p)Z^2}{E^2}$$

Where n is the sample size required

p is the percentage occurrence of condition (proportion or prevalence)

E is the percentage maximum error required = 5%

Z is the value corresponding to level of confidence required=1.96

Prevalence of hemoglobinopathies was observed to be 14.99% as assessed from (*Shah N, Khonglah Y, Raphael V et al. Antenatal Screening for Hemoglobinopathies with HPLC. Rec Adv Path Lab Med 2018; 4(3): 1-8.*)<sup>1</sup>

Considering the similar result in the current study, at 95% confidence level and 5% of maximum error, the sample size is given by,

$$n = 14.99 \times (100 - 14.99) \times 1.96^2 / 5^2$$

$$n = 195.814 \approx 196$$

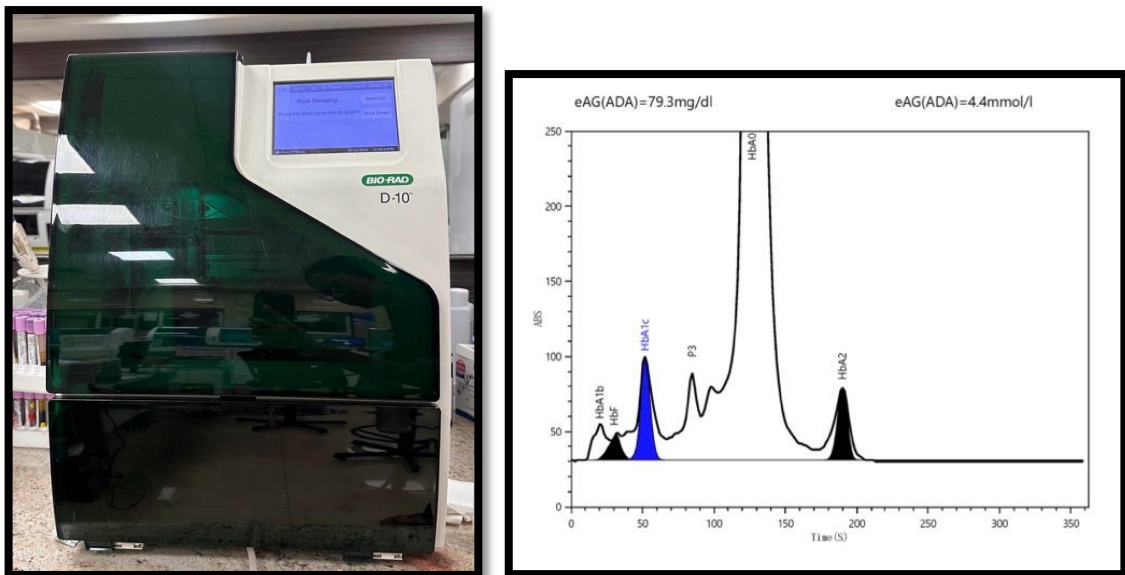
Hence, minimum sample size required is 196. As sample size increases, accuracy of result also increases.

**DATA COLLECTION:**

- After obtaining approval from ethics committee and CTRI registration, antenatal women attending outpatient department were screened using convenient sampling technique.
- After fulfilling the selection criteria, eligible participants were explained about the study and informed consent was taken.
- A detailed history, clinical findings and relevant investigations were noted as per pre-approved study related proforma. (Annexure 2)

**PROCEDURE:**

- **History:** Detailed history including *obstetric history, past history, family history, socio-economic history* and *personal history* was taken from the enrolled pregnant women.
- **Examination:** Participants were subjected to *general examination, systemic examination* and *obstetric examination*.
- **Investigations:** 4 ml of venous blood was collected from antecubital vein in two Disodium salt of ethylene diamine tetra-acetic acid (EDTA) coated vials from the participants. The blood was then subjected to *complete hemogram, peripheral blood smear and electrophoresis by HPLC Bio rad variant*.



Hemoglobinopathy W/O HBA1C by HPLC, EDTA Blood			
Hb electrophoresis by HPLC, EDTA BLOOD			
Hb A	96.50	%	94.3 - 98.5
HB A2	3.40	%	1.8 - 3.5
Hb F	0.10	%	0.0-2.0
Hb C	Not Detected	%	
Hb D	Not Detected	%	0
Hb E	Not Detected	%	0.0
Hb S	Not Detected	%	0

**Fig1. HPLC equipment, Chromatogram and Report**

- Females having abnormal blood indices and abnormal picture of chromatogram, were given professional advice and the *husband's blood* sample was taken for HPLC after counselling the couple.
- After these investigations, the couples discovered at risk, were counselled about the possibility of child with hemoglobinopathy and were offered the option of prenatal genetic evaluation through Amniocentesis.
- The couple were then advised to take an informed decision based on the amniocentesis report.

<u>HBB GENE SEQUENCING FOR MUTATION ANALYSIS</u>	
Details	Remarks
Sample Type	Amniotic fluid
Quality of Sample	Adequate
Gestational Age	Not provided
Clinical Indication	Parents are heterozygous for c.92+5G>C in <i>HBB</i> gene.
Test Requested	Beta Globinopathy

**RESULTS**

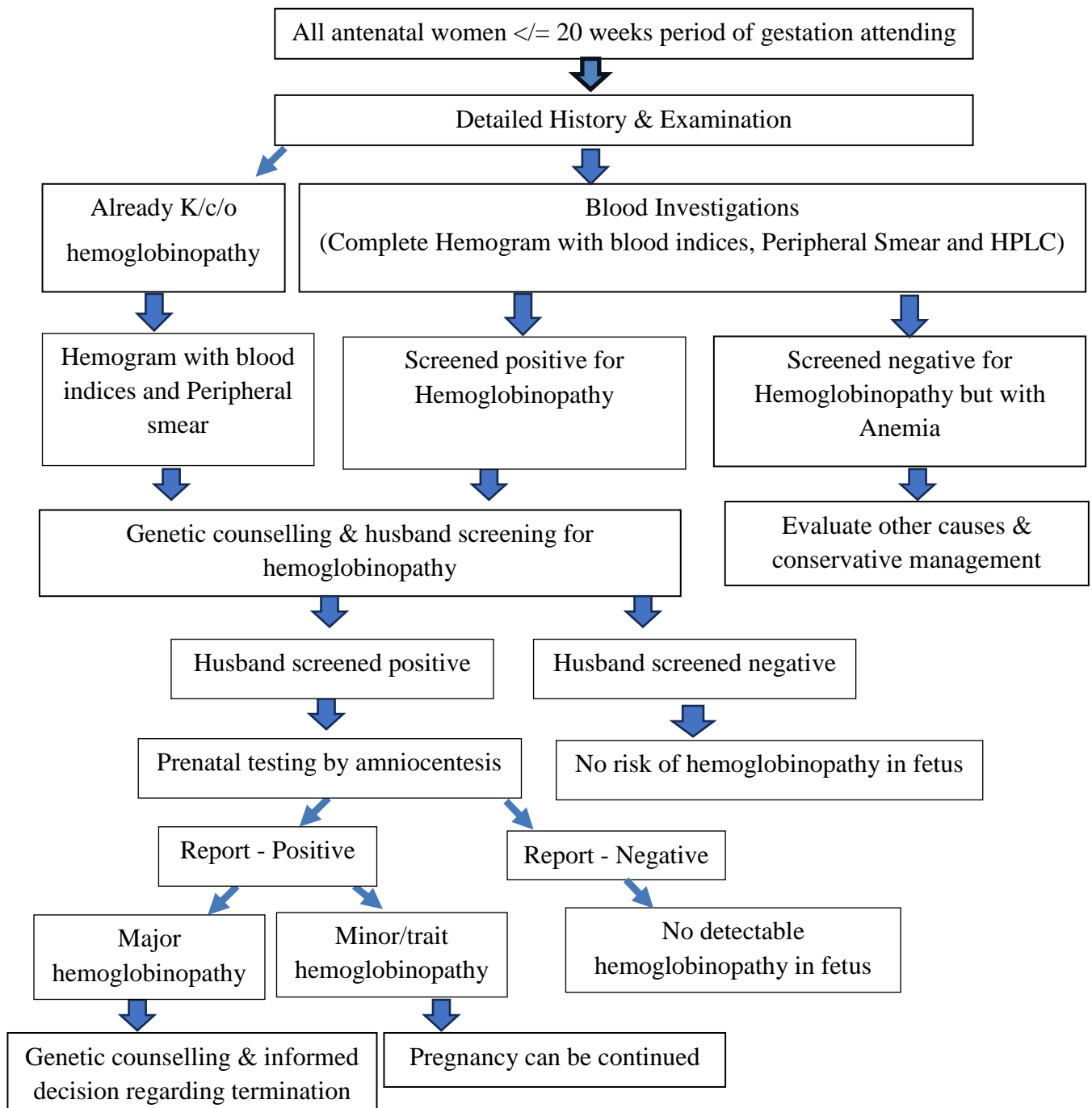
Variant Detected	Genotype	Allele Status	Clinical Significance
Common nomenclature: IVS-1-5(G>C) HGVS nomenclature: c.92+5G>C	$\beta^*/\beta$	Heterozygous	Thalassemia minor

**INTERPRETATION**

- A heterozygous variant c.92+5G>C in intron 1 of the *HBB* gene, 5 base pairs downstream to the 5' splice site of exon 1 is detected in the **Fetus sample**. The identified mutation greatly reduces the efficiency of the 5' splicing site resulting in decrease in synthesis of the  $\beta$  Globin chain. According to HbVar database this variant is classified as  $\beta^*$ , and the disease indication is likely to be **Thalassemia Minor**. The frequency of this variant among the affected population is found to be 46.55% in Maharashtrian and 30.5% in Punjabi population.

Fig 2. Amniocentesis Report

**METHODOLOGY**



**DATA PROCESSING AND ANALYSIS / STATISTICAL ANALYSIS:**

The data was entered into Microsoft Excel Sheet and was analyzed using SPSS software version 2. As data failed to follow the normal distribution as assessed using Shapiro Wilk test, Inferential statistics were performed using Non parametric test. Continuous variables were represented using mean  $\pm$  SD/median (minimum, maximum) and analyzed by Mann-Whitney test. Categorical variables were represented using frequency (percentage). Categorical data were analyzed using Chi-square test. To compare mean/distribution between two groups two sample Mann-Whitney test were used. p-value less than or equal to 0.05 indicated statistical significance.

## **RESULTS**

The present observational study was conducted at KLE'S Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi during the period of April 2023 to March 2024.

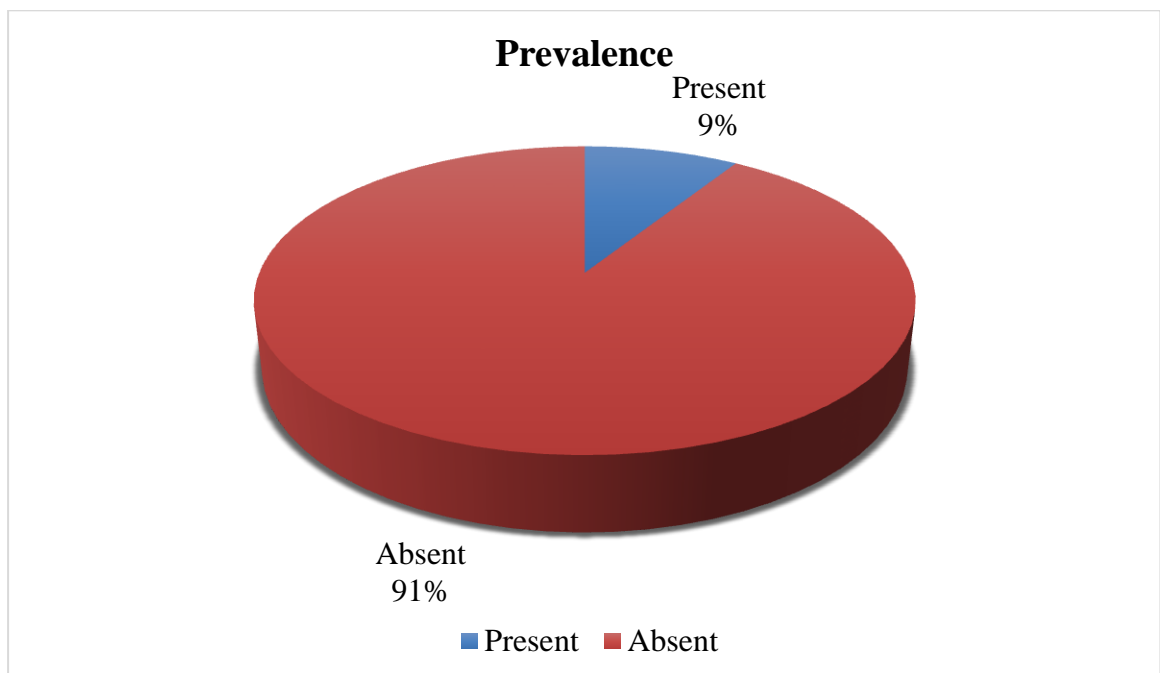
Out of total number of antenatal patients attending OPD, 629 antenatal women were screened, through convenient sampling technique. Of these, 426 women were eligible and a total of 389 women have given consent and were enrolled into the study.

After the analysis of the data using SPSS software version 21 and the final results and observations were interpreted as follows.

**Table 1: Prevalence of hemoglobinopathies**

Hemoglobinopathies	Number of participants	Percentage
Present	35	8.99
Absent	354	91.01
Total	389	100

The above table illustrates that the prevalence of hemoglobinopathies is noted to be 8.99% and the normal participants comprise 91.01%. This significant prevalence mandates the need for antenatal screening of hemoglobinopathies.

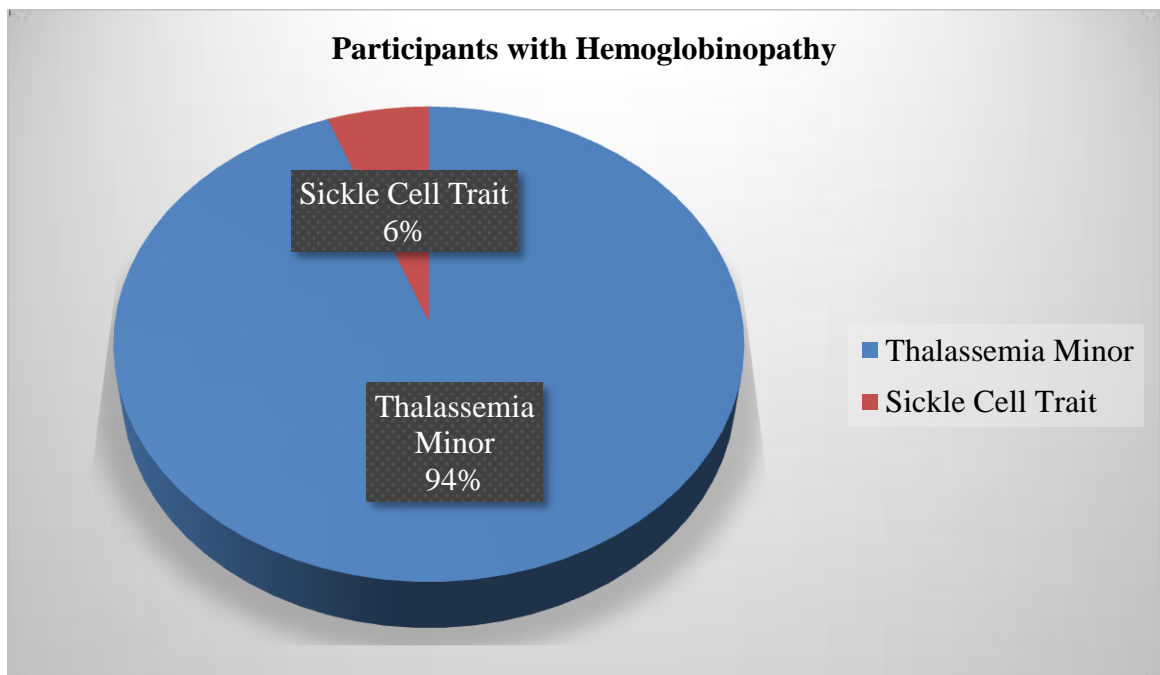
**Figure 3: Prevalence of hemoglobinopathies**

**Table 2: Distribution of participants according to type of hemoglobinopathies**

Type of Hemoglobinopathy	Number of participants With hemoglobinopathy	Percentage
Thalassemia Minor	33	94.3 %
Sickle cell Trait	2	5.7 %
Total	35	100 %

Among the 35 cases of Hemoglobinopathy, two types of hemoglobinopathy were observed of which beta thalassemia comprised 94.3 % and sickle cell trait comprised 5.7%. Beta thalassemia is the most common type of hemoglobinopathy observed in the study.

**Figure 4: Distribution of participants according to type of hemoglobinopathies**

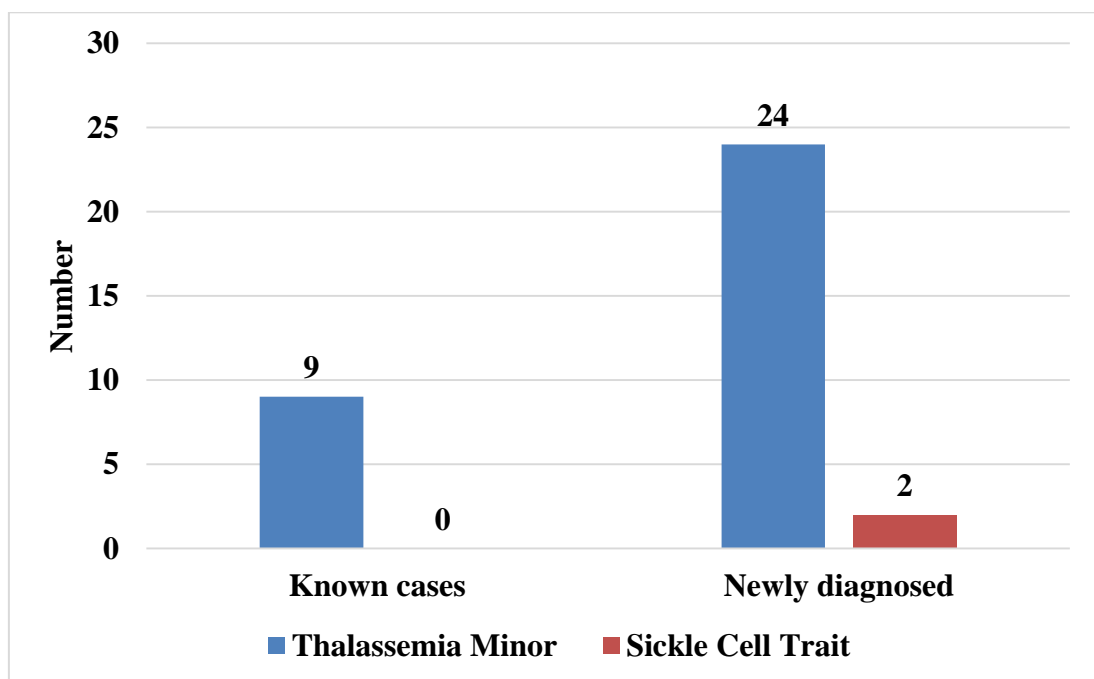


**Table 3: Distribution of participants – Newly diagnosed vs Already known cases**

Type of Hemoglobinopathy	Known cases	Newly diagnosed	Total
Thalassemia Minor	9	24	33
Sickle Cell Trait	-	2	2
Total	9 (25.7%)	26 (74.2%)	35 (100%)

Of the 35 cases of Hemoglobinopathy, 25.7% were already known cases and 74.25% were newly diagnosed cases. This finding illustrates the need for antenatal screening as majority of the individuals were unaware of their condition.

**Figure 5: Distribution of participants - Newly diagnosed vs Already known cases**

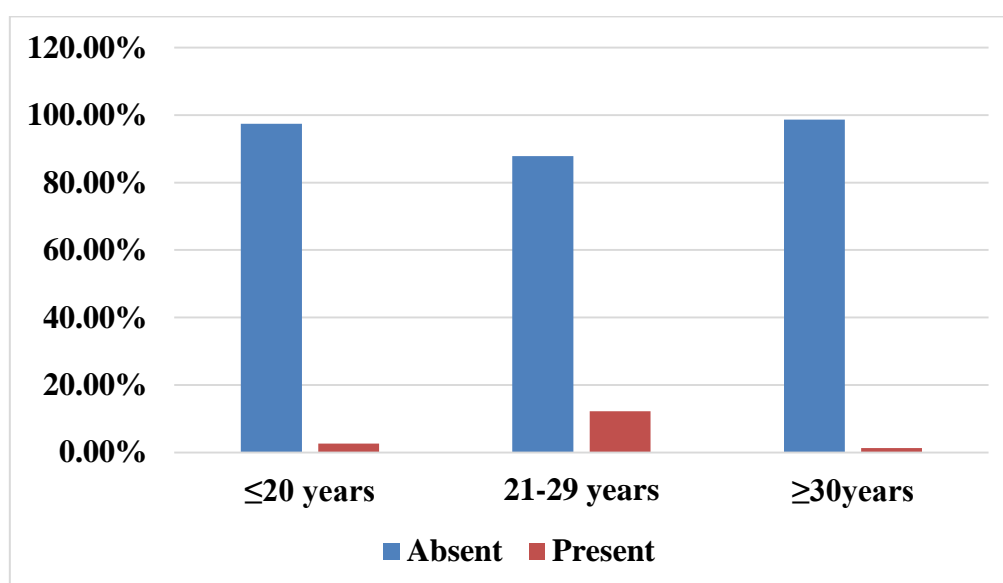


**Table 4: Age group wise distribution of participants according to prevalence of hemoglobinopathies**

			Hemoglobinopathies		Total
			Absent	Present	
Age	≤20 years	N	38	1	39
		%	10.73%	2.85%	10%
	21-29 years	N	238	33	271
		%	67.2%	94.2%	69.6%
	≥30years	N	78	1	79
		%	22%	2.85%	20.3%
Total		N	354	35	389

Out of 389 participants, majority of the participants (69.6%) were seen in the 21-29 years age group, in both the participants with hemoglobinopathy (94.2%) and without hemoglobinopathy (67.2%) as it is the reproductive age group.

**Figure 6: Age group wise distribution of participants according to prevalence of hemoglobinopathies**

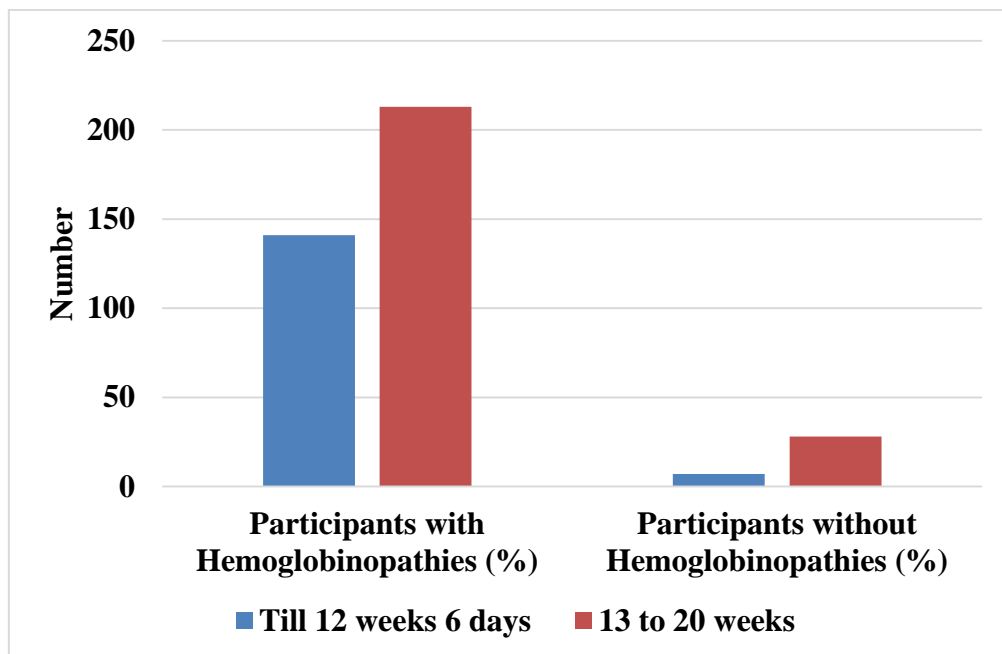


**Table 5: Distribution of participants according to Gestational age at enrollment**

Gestational Age ( weeks, days)	Participants without Hemoglobinopathies (%)	Participants with Hemoglobinopathies (%)
Till 12 weeks 6 days	141 (39.8%)	7 (20%)
13 to 20 weeks	213 (60.1%)	28 (80%)
Total	354 (100%)	35 (100%)

Among the 354 participants without hemoglobinopathy, majority of the participants (60.1%) were enrolled at 13-20 weeks gestational age and among the participants with hemoglobinopathy, majority of the participants (80%) were enrolled at 13-20 weeks of gestational age.

**Figure 7: Distribution of participants according to Gestational age at enrollment**

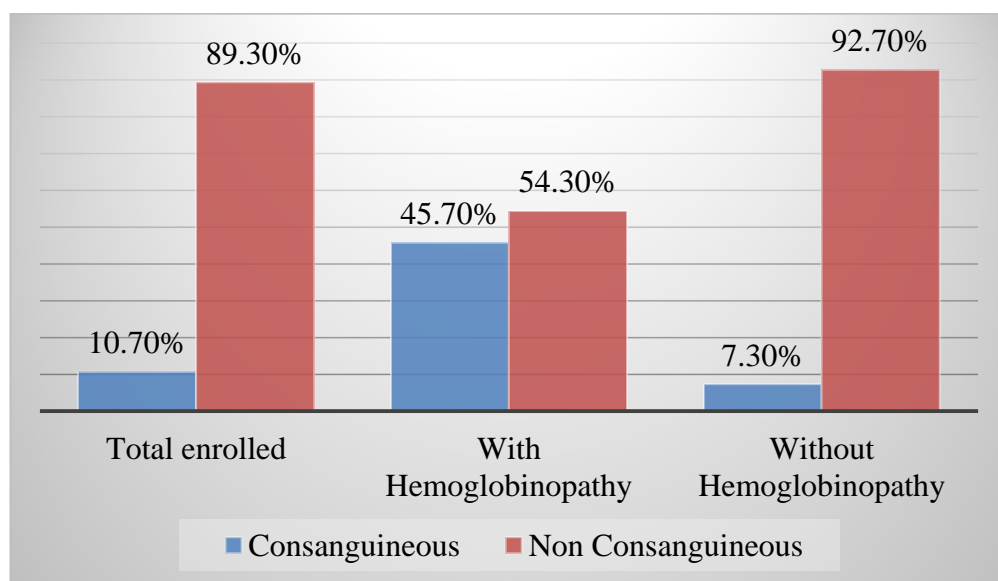


**Table 6: Association between degree of consanguinity and hemoglobinopathy**

Participants	Marriage		
	Consanguineous (%)	Non Consanguineous (%)	Total (%)
Total enrolled	42 (10.7%)	347 (89.3%)	389 (100%)
with Hemoglobinopathy	16 (45.7%)	19 (54.3%)	35 (100%)
without Hemoglobinopathy	26 (7.3%)	328 (92.7%)	354 (100%)
P value – 0.001*			

Table 6 and Fig 9: In the present study, there is a total of 10.7% prevalence of consanguinity in the study population of which the prevalence is 45.7% among the participants with hemoglobinopathy compared to 7.3% among the participants without hemoglobinopathy. There is statistically significant association between the degree of consanguinity and prevalence of hemoglobinopathies.

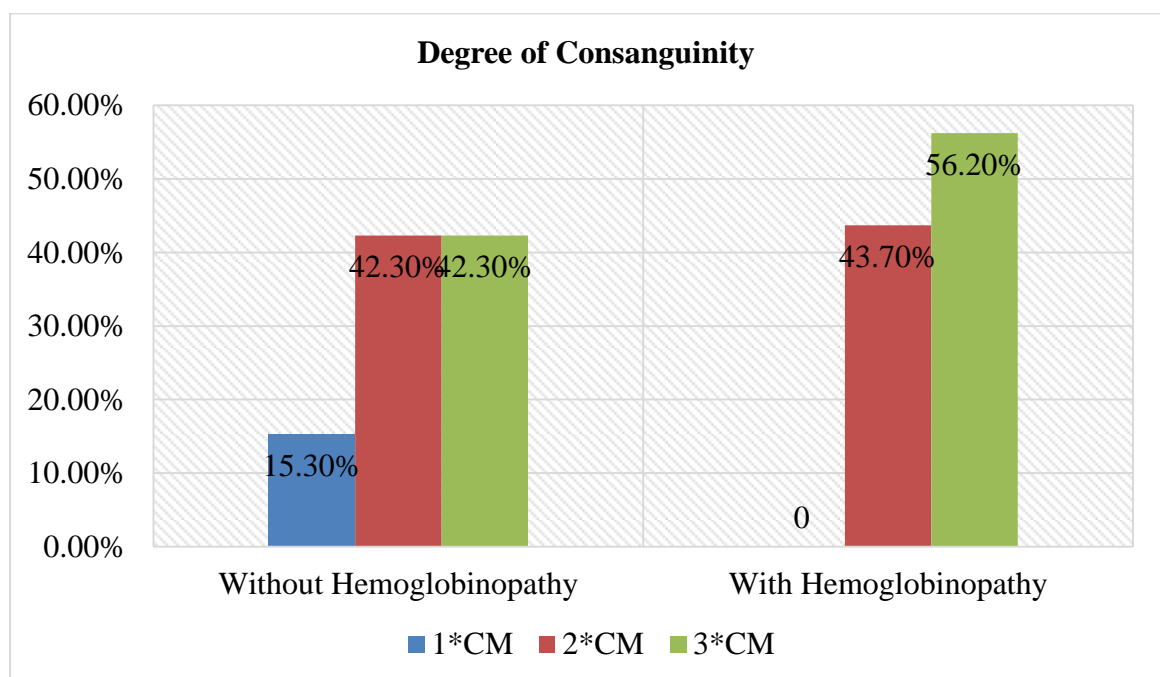
**Fig 8. Distribution of participants based on consanguinity**



**Table 7. Distribution of participants based on degree of consanguinity**

Hemoglobinopathy		Degree of Consanguinity			
		1*CM	2*CM	3*CM	Total
Absent	N	4	11	11	26
	%	15.3%	42.3%	42.3%	100%
Present	N	0	7	9	16
	%	0.0%	43.7%	56.2%	100%
Total	N	4	18	20	42
	%	9.5%	42.8%	47.6%	100%

In this study, majority of the participants with hemoglobinopathy had second (43.7%) and third degree (56.2%) consanguineous marriage.

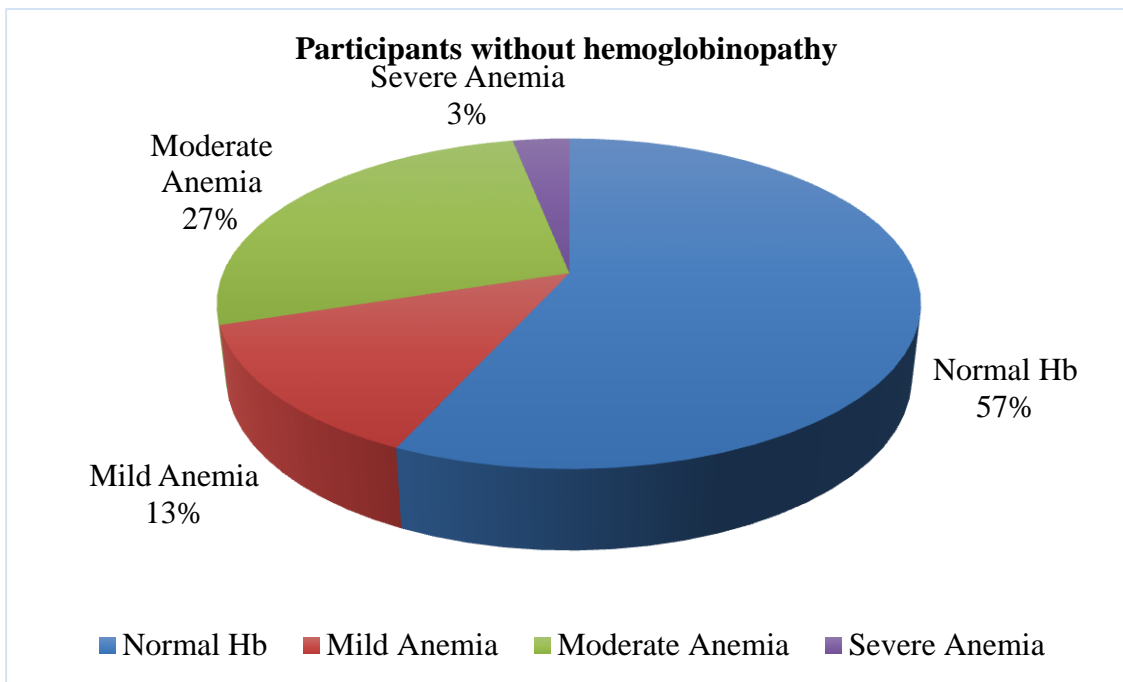
**Figure 9: Distribution based on degree of consanguinity and hemoglobinopathy**

**Table 8: Distribution of participants according to proportion of anemia**

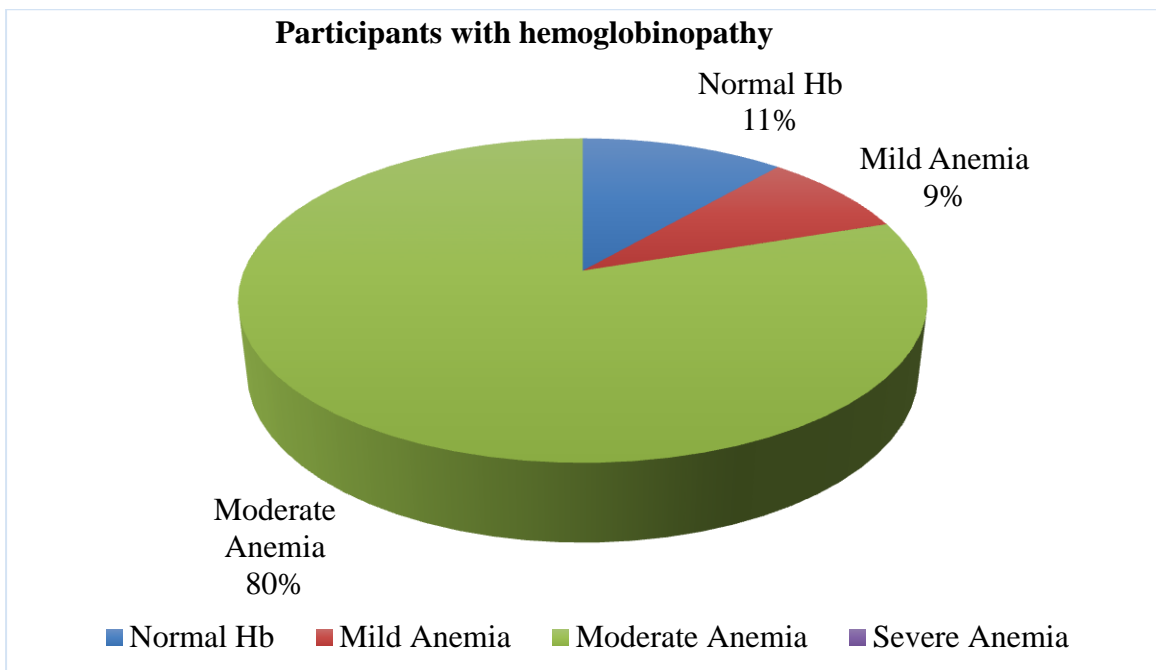
Hemoglobin range	Hemoglobinopathy - Number of Participants (%)		
	Absent (%)	Present (%)	Total
Normal Hb ( $\geq 11$ g/dl)	201 (56.7%)	4 (11.4%)	205 (52.69%)
Mild Anemia (10-10.9 g/dl)	47 (13.2%)	3 (8.5%)	50 (12.85%)
Moderate Anemia (7-9.9 g/dl)	95 (26.8%)	28 (80%)	123 (31.61%)
Severe Anemia ( $< 7$ g/dl)	11 (3.1%)	0 (0.0%)	11 (2.82%)
Total	354 (100%)	35 (100%)	389 (100%)

Among the participants without hemoglobinopathy, majority (56.7%) had normal hemoglobin. Among the participants with hemoglobinopathy, 11.4% had normal hemoglobin, 8.5% had mild anemia, 80% had moderate anemia and none with severe anemia, indicating that moderate anemia is the most prevalent anemia among the participants with hemoglobinopathy.

**Figure 10a: Distribution of participants according to proportion of anemia among subjects without hemoglobinopathies**



**Figure 10b: Distribution of participants according to proportion of anemia among subjects with hemoglobinopathies**

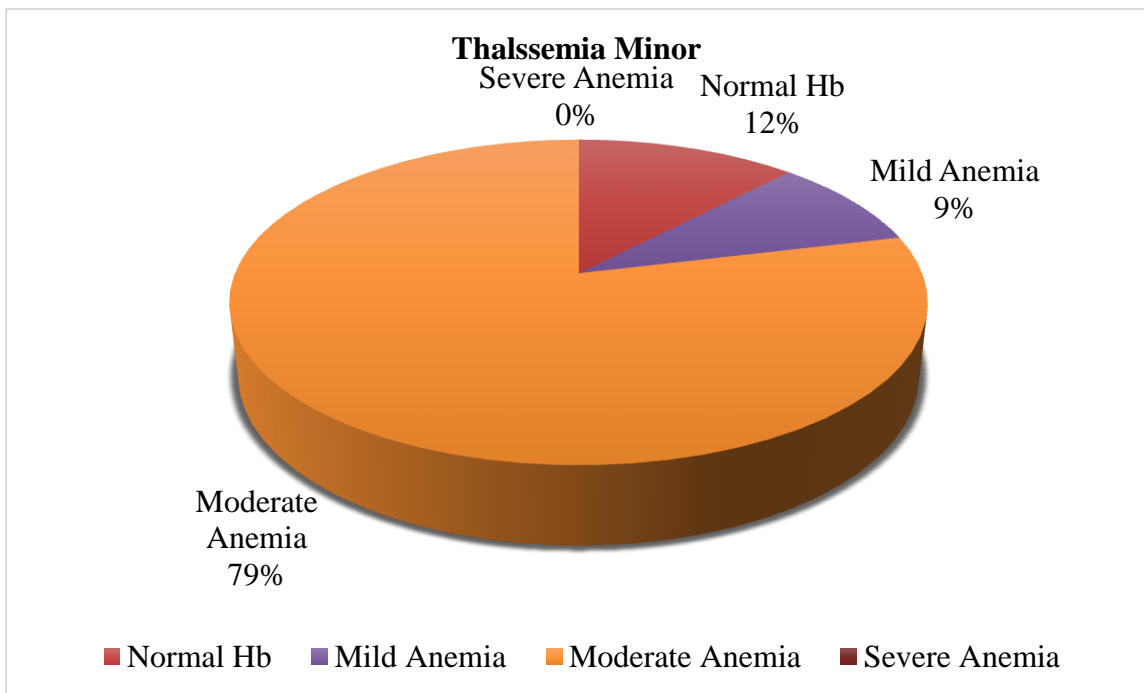


**Table 9: Distribution of participants according to proportion of anemia among various Hemoglobinopathies**

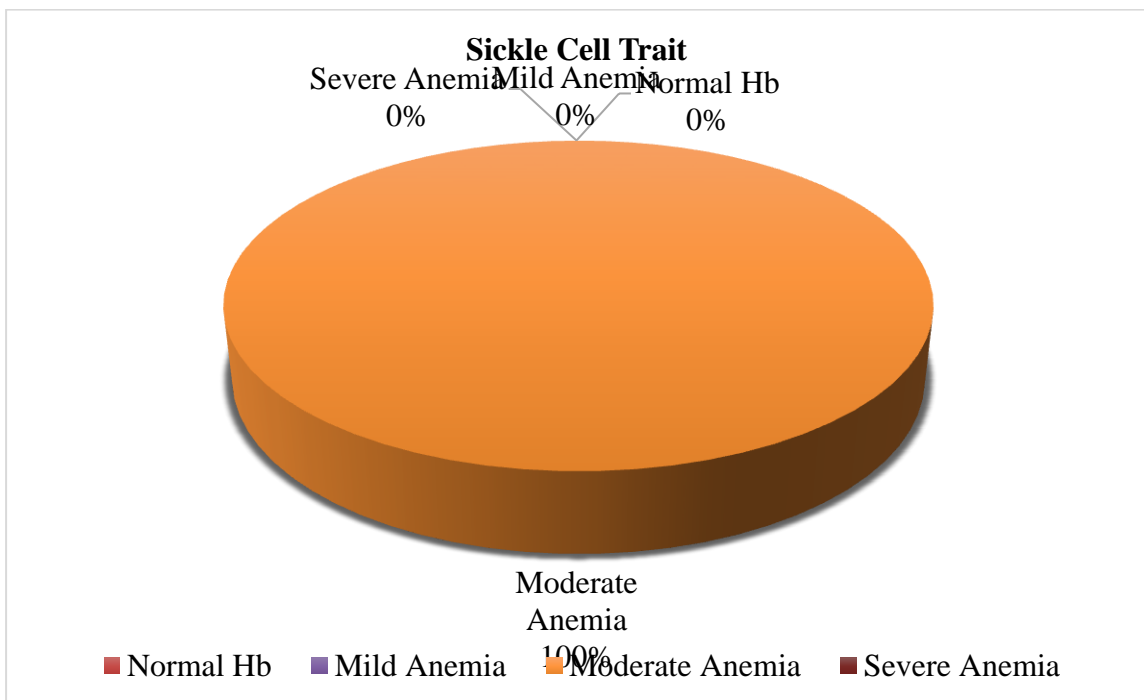
	Thalassemia Minor (HPLC) N (%)	Sickle Cell trait (HPLC) N (%)	Total
Normal Hb ( $\geq 11$ g/dl)	4 (12.12 %)	0	4 (11.4 %)
Mild Anemia (10-10.9 g/dl)	3(9.09%)	0	3(8.57 %)
Moderate Anemia (7-9.9 g/dl)	26(78.78 %)	2 (100%)	28 (80%)
Severe Anemia ( $<7$ g/dl)	0	0	0
Total	33(100%)	2 (100%)	35(100%)

Out of total 35 subjects with hemoglobinopathies, 33 had thalassemia minor and 2 had Sickle Cell trait (HPLC). 78.7% participants with thalassemia minor and 100% participants with sickle cell trait had moderate anemia and none of the participants with hemoglobinopathies had severe anemia.

**Figure 11a: Distribution of participants according to proportion of anemia among Thalassemia Minor**



**Figure 11b: Distribution of participants according to proportion of anemia among Sickle cell trait**

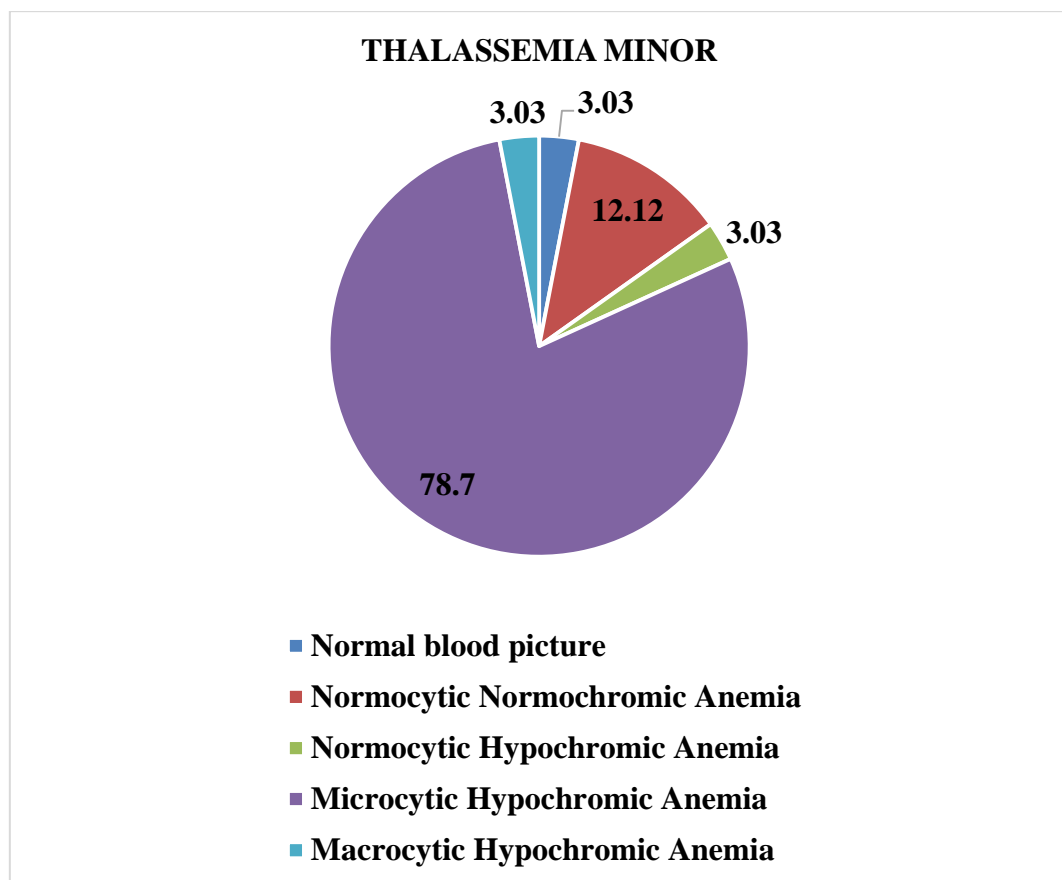
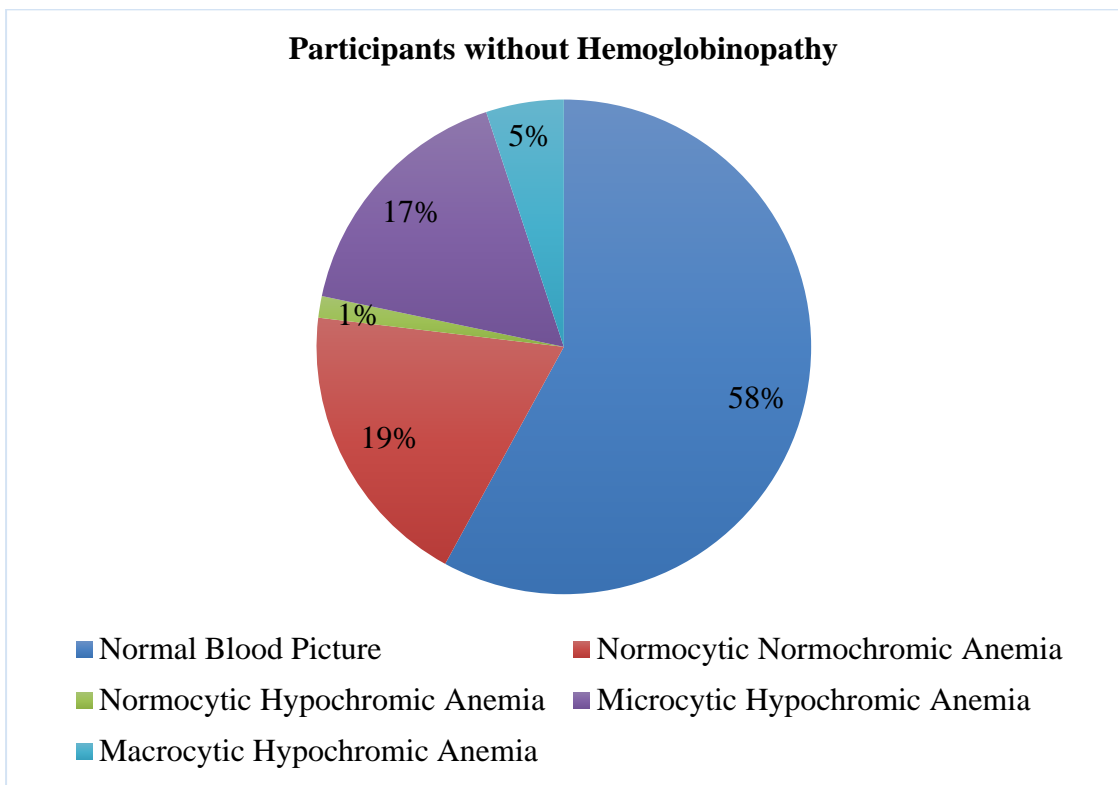


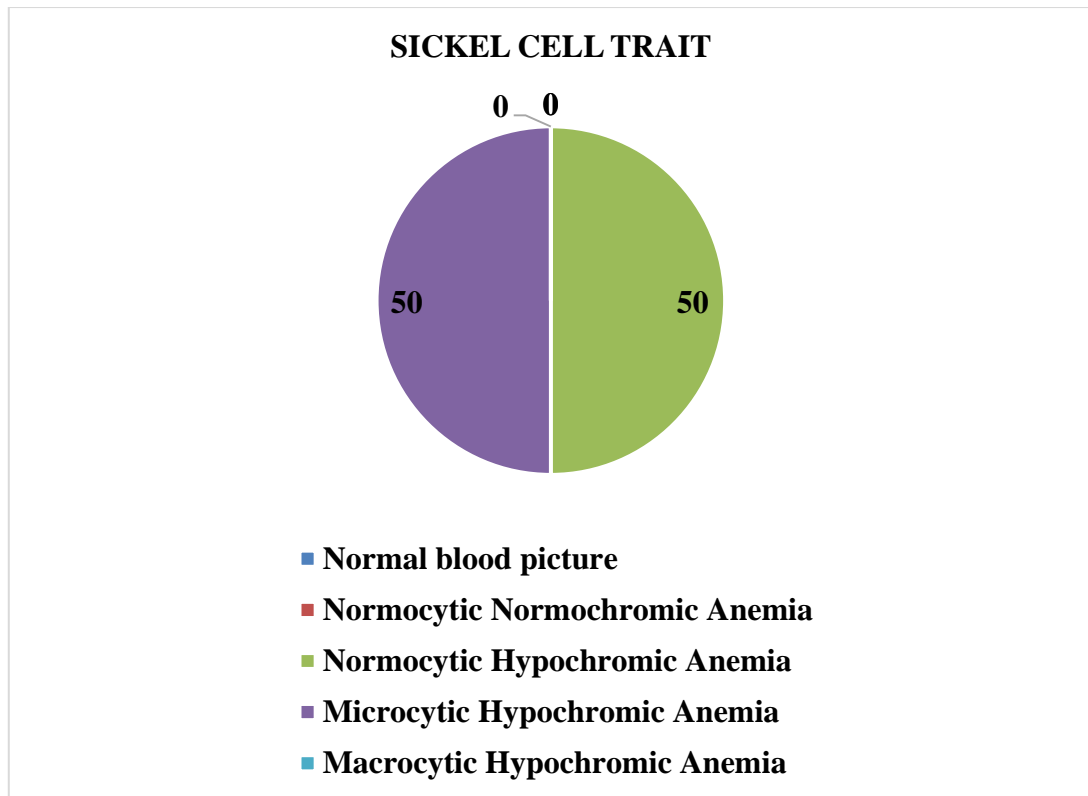
**Table 10: Distribution of participants based on Peripheral Smear**

Peripheral Smear	Normal N (%)	Hemoglobinopathies	
		Thalassemia Minor N (%)	Sickle Cell trait N (%)
Normal blood picture	205 (57.9%)	1 (3.03%)	0
Normocytic Normochromic Anemia	67 (18.9%)	4 (12.12%)	0
Normocytic Hypochromic Anemia	5 (1.41%)	1 (3.03%)	1 (50%)
Microcytic Hypochromic Anemia	59(16.6%)	26 (78.7%)	1 (50%)
Macrocytic Hypochromic Anemia	18(5.08%)	1 (3.03 %)	0
Total	354 (100%)	33 (100%)	2 (100%)

On peripheral smear, normal blood picture was seen among 57.9% of normal participants and 1 participant with thalassemia minor (3.03%). Normocytic Normochromic Anemia was seen among 18.9% of normal participants and 12.12% of participants with thalassemia minor. Normocytic Hypochromic Anemia was seen among 1.41% of normal participants, 3.03% of participants with thalassemia minor and among 50% with sickle cell trait. Microcytic hypochromic anemia was noted in 78.7% of the participants with thalassemia minor and 50% of the participants with sickle cell trait. In this study, Microcytic Hypochromic Anemia was the most common finding among Hemoglobinopathies.

Figure 12: Distribution of participants based on Peripheral Smear





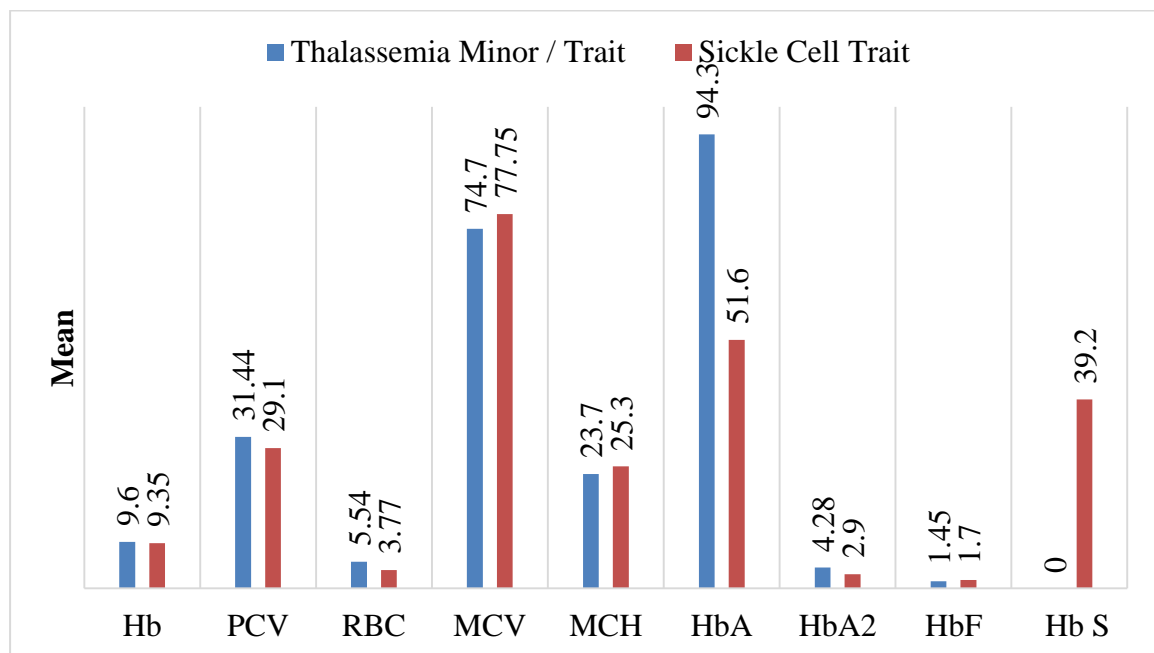
**Table 11: Distribution of participants according to Hematological Profile in Hemoglobinopathies**

Hemoglobinopathy (HPLC Report)	No of subjects (%)	Hb	PCV	RBC	MCV	MCH	HbA	HbA2	HbF	HbS
		Mean $\pm$ SD								
Thalassemia Minor / Trait	24	9.6 $\pm$ 1.67	31.44 $\pm$ 8.38	5.54 $\pm$ 5.31	74.7 $\pm$ 10.98	23.7 $\pm$ 3.79	94.31 $\pm$ 0.63	4.28 $\pm$ 0.81	1.45 $\pm$ 0.67	0
Sickle Cell Trait	2	9.35 $\pm$ 0.64	29.1 $\pm$ 0.85	3.77 $\pm$ 0.47	77.75 $\pm$ 7.57	25.3 $\pm$ 0.99	51.6 $\pm$ 0.38	2.9 $\pm$ 0.44	1.7 $\pm$ 0.42	39.2 $\pm$ 0.34

Among the subjects with thalassemia minor, mean Hemoglobin (Hb) was found to be  $9.6 \pm 1.67$  g/dL. Mean PCV was found to be  $31.44 \pm 8.38$ . Mean RBC count was found to be  $5.54 \pm 5.31 \times 10^6/\mu\text{L}$ . Mean MCV was found to be  $74.7 \pm 10.98$ . Mean MCH was found to be  $23.7 \pm 3.79$ , Mean HbA was found to be  $94.31 \pm 0.63$ , Mean HbA2 was found to be  $4.28 \pm 0.81$ , mean HbF was found to be  $1.45 \pm 0.67$  and Mean HbS was found to be 0 indicating that there was decrease in Hb, PCV, MCV, MCH, HbA and mild increase in HbA2 in the participants with thalassemia minor.

Among the subjects with sickle cell trait, mean Hemoglobin (Hb) was found to be  $9.35 \pm 0.64$ . Mean PCV was found to be  $29.1 \pm 0.85$ . Mean RBC count was found to be  $3.77 \pm 0.47$ . Mean MCV was found to be  $77.75 \pm 7.57$ , and Mean MCH was found to be  $25.3 \pm 0.99$ , Mean HbA was found to be  $51.6 \pm 0.38$ , Mean HbA2 was found to be  $2.9 \pm 0.44$ , mean HbF was found to be  $1.7 \pm 0.42$  and Mean HbS was found to be  $39.2 \pm 0.34$  indicating that there was decrease in Hb, PCV, MCV, MCH, HbA and significant increase in HbS.

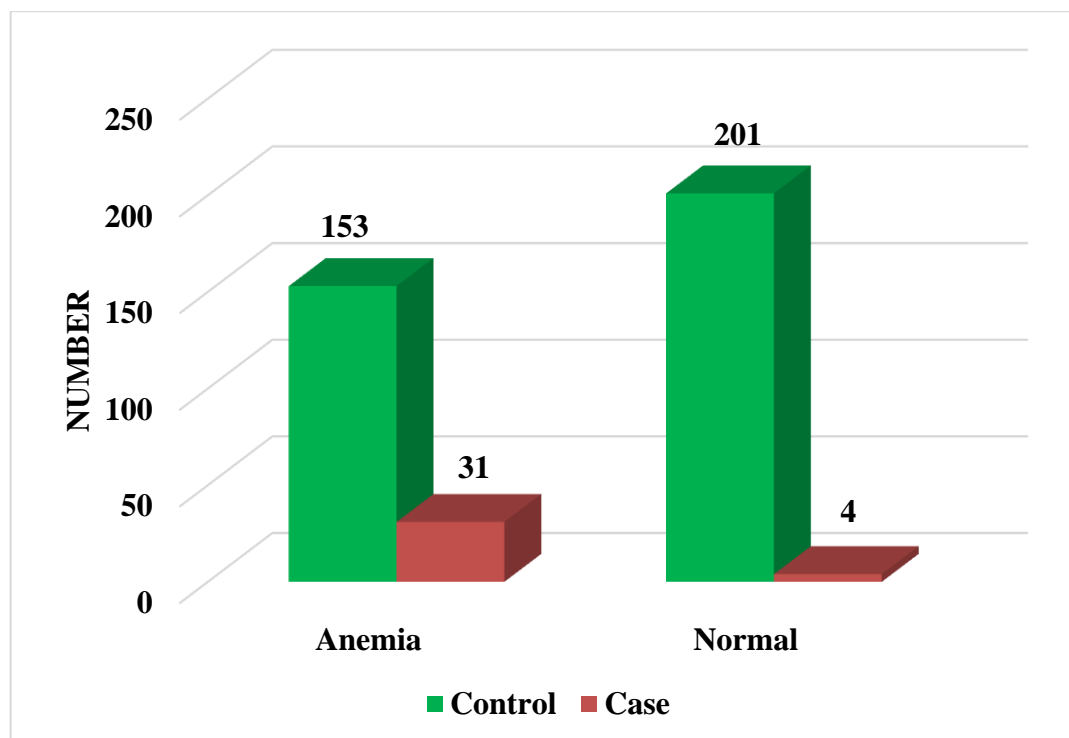
**Figure 13. Distribution of participants according to Hematological Profile in Hemoglobinopathies**



**Table 12: Association between Anemia and Hemoglobinopathy**

Variable	Subcategory	Hemoglobinopathy			p-value
		Absent	Present	Total	
Anemia/ Normal Hb	Anemia	153 (39.33%)	31 (7.96%)	184 (47.3%)	<0.001* <sup>C</sup>
	Normal	201 (51.67%)	4 (1.02%)	205 (52.7%)	
Total		354 (91%)	35 (9%)	389 (100%)	

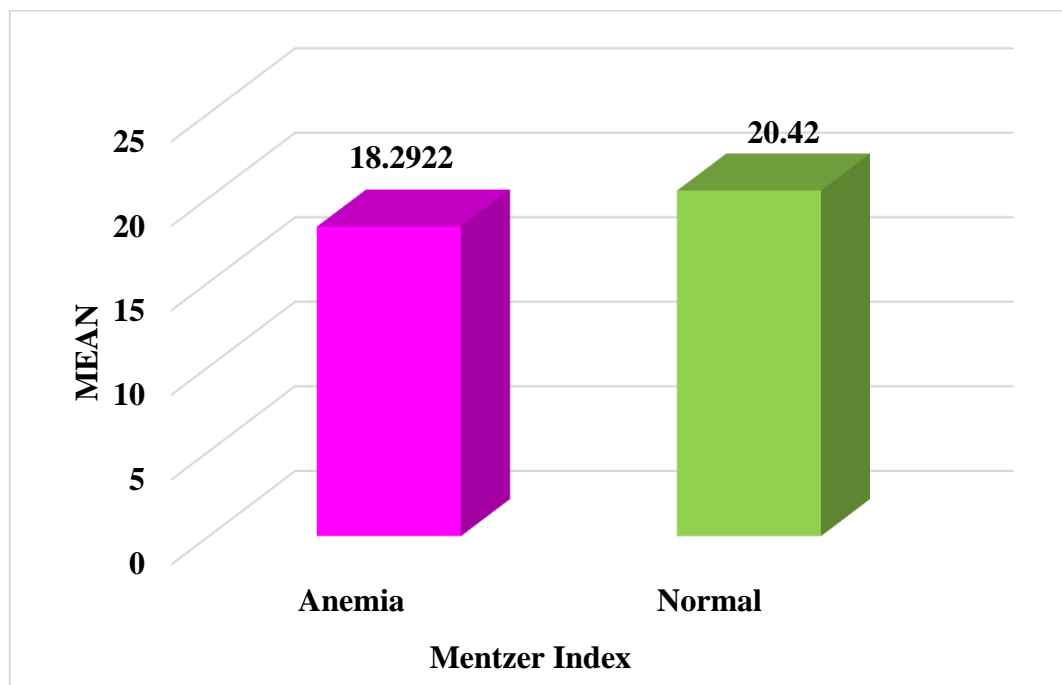
Significant association was observed between Anemia and Hemoglobinopathy. Significantly more number of anemic subjects had hemoglobinopathies.

**Figure14: Association between Anemia and Hemoglobinopathy**

**Table 13: Association between Anemia and Mentzer index**

		N	Mean	Standard Deviation (SD)	Standard Error Mean (SE)
Mentzer Index	Anemia	184	18.2922	6.32320	0.4661
	Normal	205	20.420	3.3106	0.231
P value - 0.001*					

Significant association was noted between anemia and Mentzer index. Mentzer index was found to be significantly lower anemic subjects.

**Figure 15: Association between anemia and Mentzer index**

**Table 14: Association between Hemoglobinopathy and Mentzer index**

	Hemoglobinopathy	N	Mean	Standard Deviation	Standard Error Mean
Mentzer Index	Absent	354	19.97	4.914	0.261
	Present	35	13.72	2.553	0.4316
P value - 0.001*					

Significant association was seen between Hemoglobinopathy and Mentzer index. Mentzer index was found to be significantly lower among the participants with hemoglobinopathy.

**Figure 16: Association between Hemoglobinopathy and Mentzer index**

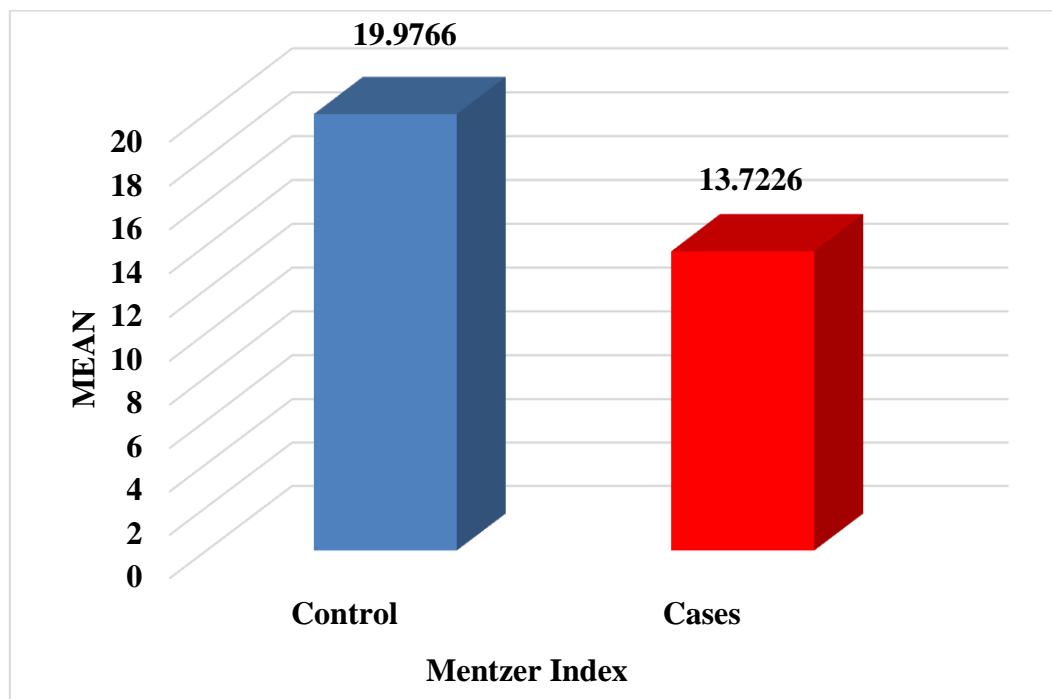
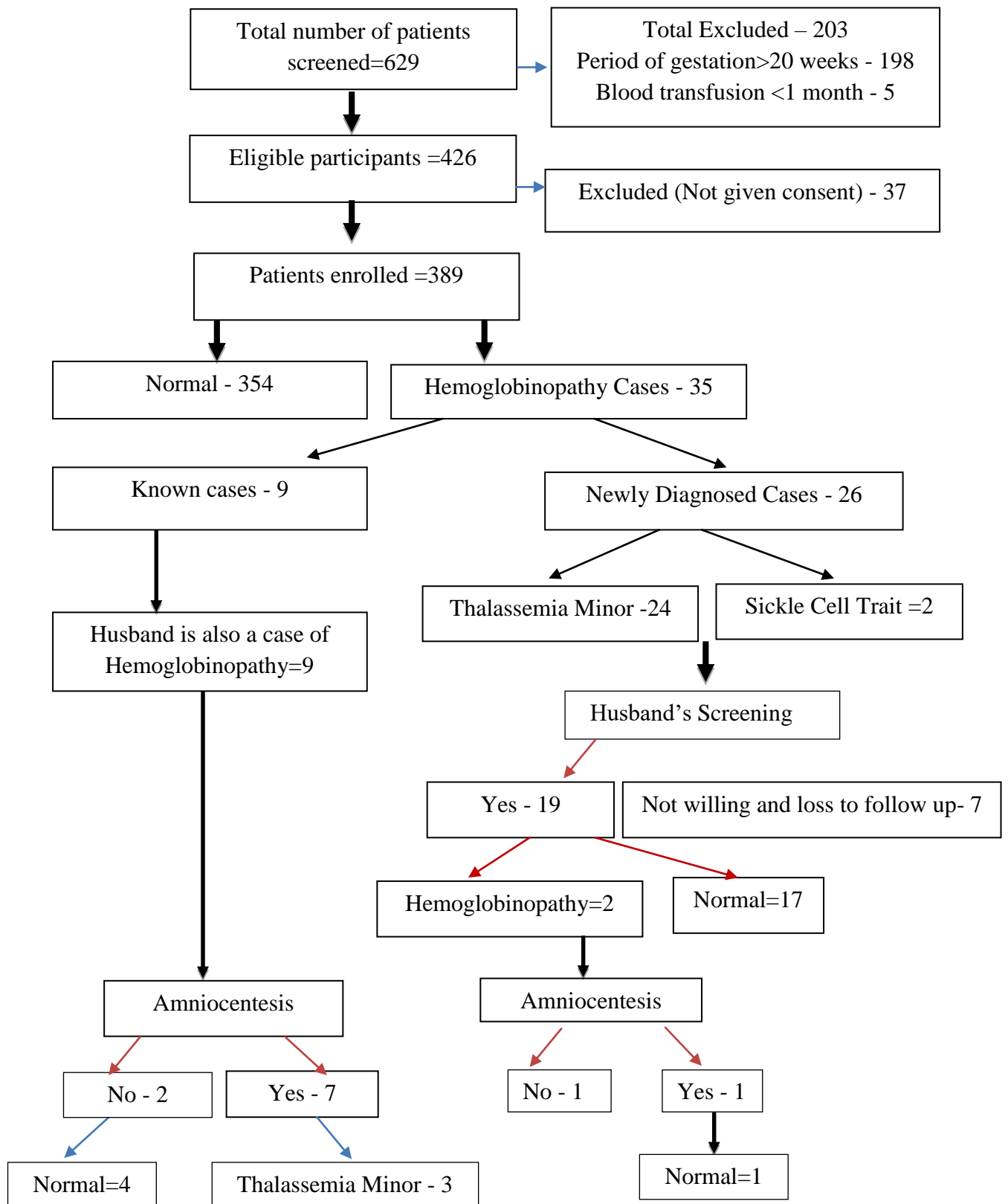


Figure 17: STROBE diagram



## **DISCUSSION**

Hemoglobinopathies are common inherited genetic disorders resulting from deficiencies in the synthesis of hemoglobin (Hb), either quantitatively or qualitatively. As per the World Health Organization, about 5% of the human population is affected by hemoglobinopathies, and between 300,000 and 400,000 newborns are born each year with severe hemoglobinopathies. Hemoglobinopathies were once only found in the Middle East and certain regions of Asia and Africa, but the massive international migration and intercultural and intercommunal marriages have led to their widespread prevalence today.<sup>121,122</sup>

### ***Prevalence of Hemoglobinopathies:***

In the present study, hemoglobinopathies were seen among 35 cases out of 389 owing to the prevalence of 8.99% and this prevalence aligns with previous research indicating a significant burden of these genetic disorders in certain populations. In a study by Shah et al.<sup>106</sup> (2018), 467 antenatal mothers had a prevalence of 14.99% prevalence of hemoglobinopathy on HPLC. A study by Priyadarshini et al,<sup>92</sup> 2022, reported the prevalence of hemoglobinopathies in the same range (8-10%). In a study by Shanthi et al,<sup>118</sup>(2023) the prevalence of hemoglobinopathies was found to be 7%. On comparison of similar studies conducted in India, Sharma et al.<sup>108</sup> had very high prevalence compared to our study whereas Sawaimul et al<sup>122</sup> and Singh et al<sup>123</sup>, have reported comparatively less prevalence rates of hemoglobinopathies among antenatal populations compared to our study.

Out of these 35 cases, 9 (25.7%) were known cases of hemoglobinopathy and 26 (74.3%) were newly diagnosed cases. This emphasizes the need for screening antenatal patients as the majority of the carriers are unaware of their condition and

future prospects. The prevalence of hemoglobinopathies in India as estimated by Christianson et al.<sup>124</sup> was 1.2/1000 live births. According to this, each year, 32,400 are born with a significant hemoglobin abnormality. These findings underscore the importance of routine screening for hemoglobinopathies in prenatal care settings for early diagnosis, prevention and treatment.

<b>Study trial</b>	<b>Study area</b>	<b>Prevalence</b>
<b>Priyadarshini et al<sup>92</sup></b>	Cuttack, East Orissa	9.2%
<b>Shah et al<sup>106</sup></b>	Shillong, Meghalaya	14.99%
<b>Balgir et al<sup>107</sup></b>	Jabalpur, Madhya Pradesh	12.26%
<b>Sharma et al<sup>108</sup></b>	Amritsar, Punjab	21.4%
<b>Shanthi et al<sup>118</sup></b>	Tribal hilly areas, Tamilnadu	7%
<b>Sawaimul et al<sup>122</sup></b>	Pune, Maharashtra	4%
<b>Singh et al<sup>123</sup></b>	Rishikesh, Uttarakhand	2.8%
<b>Mukhopadhyay et al<sup>125</sup></b>	Kolkata, West Bengal	12.55%
<b>Ahuja et al<sup>126</sup></b>	Ahmedabad, Gujarat	9.75%

**Age:**

In our study, the majority of subjects (69.6%) were between 21-29 years, while 20.3% were  $\geq 30$  years, and 10% were  $\leq 20$  years. This age distribution reflects a typical reproductive age group. The age range of 21–29 years old included the majority of the participants affected with hemoglobinopathies. This highlights the need for targeted screening in this reproductive age group to identify and manage hemoglobinopathies early, reducing the risk of complications during pregnancy. A

study by Bhukhanvala et al. (2019)<sup>71</sup> found a similar clustering of hemoglobinopathies among women in the 21-29 age group.

***Gestational Age:***

Gestational age distribution indicated that 38% of subjects were within 12 weeks and 7 days, and 62% were between 13 to 20 weeks. Among those with hemoglobinopathies, a significant number (80%) had gestational ages between 13 to 20 weeks, compared to only 20% till 12 weeks 7 days. The association between gestational age and hemoglobinopathy prevalence highlights the importance of timing in antenatal screening, as emphasized by Gupta et al. (2019).<sup>127</sup> In our study, the majority had the booked visit in the late first trimester and early second trimester but the early visit is recommended and it facilitates early detection and counselling on pregnancy and potential complications and ensures timely intervention with a multi-disciplinary approach.

***Consanguinity:***

Distribution of subjects based on consanguinity indicated that 10.8% of subjects were found to have h/o consanguineous marriage. Among the cases of hemoglobinopathy, 45.7% of subjects have history of consanguinity, of which 46.7% of subjects have history of second-degree consanguineous marriage and 56.3% of subjects have history of third-degree consanguineous marriage. The subjects with consanguinity had a higher prevalence of previous children with hemoglobinopathies.

This finding underscores the significance of consanguinity and its positive correlation with hemoglobinopathies in the offspring.

***Anemia:***

Distribution of subjects based on hemoglobin range indicated that 52.6% of subjects had normal hemoglobin levels (Hb>11) whereas 47.4% of subjects were found to be anemic (Hb<11). This substantial proportion of anemia highlights a critical public health concern, especially in pregnant women where anemia can lead to adverse maternal and fetal outcomes.

Among the cases of Hemoglobinopathy, 11.4% of subjects had Hemoglobinopathy without Anemia and 88.5% had Hemoglobinopathy with Anemia. Thus, hemoglobinopathies were more prevalent among those with anemia, with 8.5% cases in mild anemia (10 to 10.9g/dL) & 80% in moderate anemia (7-9.9g/dL). No cases of hemoglobinopathies were found in subjects with severe anemia (<7 g/dL) when categorized according to NFHS -5 criteria. These findings emphasize the necessity of addressing moderate anemia in pregnant women from a wider perspective as a significant proportion may have underlying hemoglobinopathies.

Hemoglobinopathy and anemia were shown to be significantly correlated ( $p<0.001$ ). A higher proportion of anemic subjects had hemoglobinopathies compared to those with normal hemoglobin levels. This strong association between anemia status and hemoglobinopathy prevalence underscores the clinical relevance of integrating hemoglobinopathy screening into routine anemia assessments during antenatal care. This finding is consistent with studies by Verma et al. (2016)<sup>128</sup> and Siddiqui et al. (2020)<sup>129</sup>, which emphasize the utility of comprehensive anemia screening protocols for detecting underlying hemoglobinopathies. According to a study by Priyadarshini et al.,<sup>92</sup> of the antenatal women with hemoglobinopathy, 09.09% were not anemic and 90.91% were. The fact that hemoglobinopathies themselves can cause anemia suggests that the high incidence of anemia may be

caused by their combined effects. As such, routine screening for hemoglobinopathy should be conducted during pregnancy when anemia is being investigated. The high prevalence of anemia in pregnancy in our nation may be partially attributed to hemoglobinopathy. The majority of these disorders (carriers) usually remain asymptomatic and require diagnostic testing using appropriate technology (HPLC or automated capillary zone electrophoresis).

***Hematological profile:***

Hemoglobinopathies can significantly affect the hematological parameters of the subjects. PCV has typically reduced due to their reduced size (microcytosis). RBCs are elevated despite a reduced PCV, as the RBCs are smaller (microcytic) and more numerous. MCV (Mean Corpuscular Volume), MCH (Mean Corpuscular Hemoglobin), and MCHC (Mean Corpuscular Hemoglobin Concentration) are significantly reduced due to microcytosis and hypochromia.<sup>130</sup> According to the current investigation, hemoglobinopathy cases with coexisting anemia had lower MCV and MCH levels. The findings are consistent with research conducted on antenatal mothers by Chakrabarti et al,<sup>131</sup> which showed that individuals with coexisting iron deficiency and  $\beta$ -thalassemia trait had considerably lower hemoglobin, MCV, and MCH levels than those with beta-thalassemia only.

Subjects with thalassemia minor had mean hemoglobin levels of  $9.6 \pm 1.67$  g/dL, lower than the normal range, indicating mild to moderate anemia. Their RBC count was elevated, consistent with the compensatory mechanism in thalassemia. In contrast, sickle cell trait subjects had slightly lower hemoglobin levels ( $9.35 \pm 0.64$  g/dL) and RBC counts, with a significant presence of HbS ( $39.2 \pm 0.34$ ), characteristic of sickle cell disease. These profiles are critical for the differential diagnosis and management of hemoglobinopathies.

***Peripheral Smear:***

Peripheral smear analysis revealed that the majority of controls (57.9%) had a normal blood picture. In contrast, those with thalassemia minor predominantly showed microcytic hypochromic anemia (78.7%), and sickle cell trait subjects had a mix of normocytic hypochromic and microcytic hypochromic anemia. These findings align with the typical hematological presentations of thalassemia and sickle cell disorders, providing a useful diagnostic tool for clinicians.

***HPLC Analysis:***

Mean HbA, HbA<sub>2</sub>, and HbF values among the cases of hemoglobinopathy were found to be  $91.01 \pm 11.61$ ,  $4.20 \pm 0.84$ , and  $1.45 \pm 0.63$  respectively. HbA was found to be lesser than the normal average value of 95-97% whereas Hb A<sub>2</sub> was found to be a little higher than the normal range i.e. 0-3. HbF is in the normal range category i.e. 0-2%.

In order to diagnose different hemoglobin problems, a thorough and meticulous peripheral blood smear examination (PBS) and full blood count are crucial. When the HPLC is completely normal in  $\alpha$ -thalassemia trait but the HbA<sub>2</sub> level is either low or normal, careful correlation of the microcytosis, which cannot be explained by BTT or iron deficiency, can lead to the suspicion of  $\alpha$ -thalassemia condition, which can be validated by DNA analysis. The sickling test and HPLC can both confirm the diagnosis of sickle cell disease, as the PBS in these cases indicates sickle cells. The blood picture in various hemoglobinopathies may not be specific, and high-performance liquid chromatography (HPLC) is a highly useful diagnostic tool for identifying and measuring a number of normal and abnormal hemoglobins.<sup>132</sup>

***Mentzer Index:***

The inability to distinguish between beta thalassemia trait and iron deficiency anemia based solely on blood picture, along with the high cost of procedures in low resource settings, has resulted in the development of blood count indices. The Mentzer Index is computed as follows: MCV (fl) /RBC count (millions per microliter); a number less than 13 suggests beta cell thalassemia, whereas a result greater than 13 suggests IDA as the most likely diagnosis. Out of 35 subjects with hemoglobinopathies, 26 were found to have a Mentzer index below 13 indicating the likelihood of beta thalassemia. In the present study, a significant association ( $p < 0.001$ ) was seen between Hemoglobinopathy and Mentzer index. These findings are consistent with Shagufta Tabassum et al.<sup>133</sup>

***Types of Hemoglobinopathies:***

Out of 35 subjects with hemoglobinopathies, 94.29% had thalassemia minor, and 5.71% had sickle cell trait. Among these, most cases of anemia were moderate (78.78% in thalassemia minor and 100% in sickle cell trait). The predominance of Thalassemia Minor among subjects with hemoglobinopathies is in line with regional genetic profiles and previous studies conducted in India Colah et al.,<sup>15</sup> 2014. The hematological profiles observed in this study, including microcytic hypochromic anemia and characteristic HPLC patterns, are consistent with the expected manifestations of Thalassemia Minor and Sickle Cell Trait, as reported by Verma et al. (2016)<sup>128</sup> and Mukherjee et al. (2018).<sup>134</sup>

Contrary to the outcomes of the present investigation, Shanthi et al.<sup>118</sup> found out that the Thalassemia trait was seen in 40.8% of cases, the Sickle cell trait was seen in 27.6 % of cases, and Sickle Cell Anemia was seen in 2% of cases. There were no

cases of sickle cell trait or disease in research by Shah et al.<sup>106</sup> Additionally, there were no Hb S cases from Tripura or Arunachal Pradesh reported by Sengupta B et al.<sup>100</sup> This suggests that thalassemia minor is the most common hemoglobinopathy in this cohort, often leading to moderate anemia. The presence of sickle cell trait, though less frequent, also contributes to moderate anemia, underscoring the need for targeted interventions for these conditions.

***Acceptance for Screening and Prenatal Diagnosis:***

In this study, all the husbands of 9 already known cases were also known cases of hemoglobinopathy but they were screened after their prior children got affected with hemoglobinopathy. Among 26 newly diagnosed cases, 19 husbands (73%) accepted to undergo screening, of which 2 (10.5%) were diagnosed with hemoglobinopathy and 17 (89.4%) were diagnosed normal. This underscores that the husbands of carriers exhibited reluctance to undergo screening even after the genetic counselling, due to general unawareness about the disease and lack of education as documented by Colah et al.<sup>15</sup>

Of the total 11(31.4%) at risk couple, 2 couple (18.2%) from newly diagnosed and 9 couple (81.8%) from already known cases, 8 (72.7%) underwent genetic evaluation through amniocentesis of which 5(62.5% ) were normal and 3 (37.5%) were cases of hemoglobinopathy(thalassemia minor).

In this study, the couples who were at risk rejected to undergo genetic evaluation, despite receiving appropriate counselling. The level of acceptance for screening and genetic evaluation is anticipated to be still lower in villages and remote areas of the country. Hence, it is imperative for both governmental and non-

governmental organizations to educate and disseminate information regarding the disease in order to establish a proficient screening and control initiative.

The findings of this study underscore the importance of implementing comprehensive antenatal screening protocols for hemoglobinopathies in antenatal women. Screening strategies that integrate family history, consanguinity, anemia status and hematological profiling can facilitate early detection and appropriate management of these genetic disorders, as advocated by guidelines from the Indian Council of Medical Research (ICMR, 2018). By contextualizing these findings within the existing literature on antenatal screening for hemoglobinopathies, this discussion emphasizes the importance and need screening strategies and highlights opportunities for reducing the burden of hemoglobinopathies in the country.

***Strengths:***

- This study was performed in North Karnataka, a non-endemic area of Hemoglobinopathy and the results reported high prevalence. It emphasizes that hemoglobinopathies are no more an endemic disorder, owing to global migration and hence the need for universal screening of hemoglobinopathies in all the antenatal women, to reduce the burden of hemoglobinopathies in our country.
- Although the objective of the study was to find the prevalence in early antenatal period, husband's screening and identification of couples at risk and genetic evaluation and counselling was done, on ethical grounds, with consideration to provide couples with the option of termination in cases of major hemoglobinopathy, as India has largest number of children with thalassemia major in the world.

***Limitations:***

- Convenient sampling is followed in this study.
- The prevalence rate of hemoglobinopathies with the current study population (pregnant women less than or equal to 20 weeks period of gestation) is 9%. It would have been still high, had it been taken inclusion criteria as all the pregnant women irrespective of gestational age. Nevertheless, along with the prevalence, genetic counselling and informed decision by the couple were in consideration and hence, early antenatal screening was done in this study.
- Despite all the advantages of antenatal screening through HPLC, conducting a universal screening would not be cost effective. However, at 9% prevalence rate of hemoglobinopathies in the current study with few hemoglobinopathies showing normal blood indices, routine hemogram would not suffice and it is advisable to universally screen all the pregnant women for hemoglobinopathies with electrophoresis.

***Recommendations:***

- Most of the countries have adopted either early antenatal screening program (Sweden, Italy) or neonatal screening program (Netherlands, Belgium, Spain, France) or a combined antenatal and neonatal screening program (UK) or a pre-conceptual screening program (Greece, Turkey, Cyprus).
- In recent decades, the globalization of migration has contributed to generate multiethnic societies and India, being the most populous country, with multiple races, religions and ethnicities, needs to implement a hemoglobinopathy screening protocol for pregnant women for overall good maternal and fetal health outcomes.

- This can be achieved with collaborative efforts between healthcare providers, policymakers by raising awareness about the importance of antenatal screening for hemoglobinopathies through community health programs and ensuring the availability of diagnostic resources and trained healthcare providers in antenatal clinics to facilitate early detection and management of these conditions.

## **CONCLUSION**

The study underscores a significantly high prevalence of hemoglobinopathies among the pregnant women, even in a non-endemic area, highlighting the need for universal screening of antenatal women and public health interventions. By adopting a comprehensive screening and management strategies for hemoglobinopathies, we can reduce the burden of Hemoglobinopathies in our country and improve maternal and fetal health outcomes, ultimately contributing to healthier pregnancies and stronger communities.

## SUMMARY

A descriptive Observational Study was conducted among antenatal women attending KLE's Dr. Prabhakar Kore Hospital and Medical Research Centre, during the study period of twelve months i.e. April 2023 to March 2024.

The study investigated the prevalence of hemoglobinopathies among 389 pregnant women, attending OPD at KAHER's Prabhakar Kore Hospital, with a period of gestation less than or equal to 20 weeks (calculated by reliable LMP/ scan)

Key findings from the study are summarized as follows:

- The Hemoglobinopathies were present in 35 out of 389 participants leading to prevalence of 9%.
- Out of those 35 participants with hemoglobinopathies, 94.29% had thalassemia minor, and 5.71% had sickle cell trait.
- 74.2% of hemoglobinopathy cases are newly diagnosed and 25.7% are already known cases.
- The Majority of participants in the study (69.6%) were aged 21-29 years, also the majority of participants with Hemoglobinopathies with 97.1%.
- 45.7% of Hemoglobinopathy subjects have h/o consanguinity and majority of them had history of previous children with hemoglobinopathies.
- Of the total participants, 52.6% had normal hemoglobin levels ( $\geq 11$  g/dL), while 47.4% were anemic ( $< 11$  g/dL).
- There was a strong correlation between hemoglobinopathies and anemia ( $p < 0.001$ ), with 88.57% of hemoglobinopathy cases presenting with anemia, especially moderate anemia corresponding to 80%.

- Majority of the participants with thalassemia minor (78.7%) and all the sickle cell trait (100%) had moderate anemia.
- Participants with Thalassemia minor showed predominantly microcytic hypochromic anemia (78.7%), whereas Sickle cell trait had a mix of normocytic hypochromic and microcytic hypochromic anemia.
- Thalassemia minor participants had lower mean hemoglobin levels ( $9.6 \pm 1.67$  g/dL) and elevated RBC counts and Sickle cell trait participants had lower mean hemoglobin levels ( $9.35 \pm 0.64$  g/dL) and significant HbS presence ( $39.2 \pm 0.34$ ).
- Mentzer index was significantly lower in hemoglobinopathy cases ( $p=0.001$ ), aiding in differential diagnosis.
- All the husbands of already known cases of hemoglobinopathy(9) are also cases of hemoglobinopathy.
- The husbands of newly diagnosed participants with hemoglobinopathy, who opted for screening, constitute 74.2% (19 of 26).
- The couples at risk in this study constitute 11, of which 72.7% underwent genetic evaluation with amniocentesis.
- The fetus detected with hemoglobinopathy on amniocentesis constituted 3 (37.5%) and were Thalassemia Minor.

The study's findings reinforce the importance of routine screening for anemia and hemoglobinopathies in pregnant women, particularly during early pregnancy. Early detection and management of these conditions can prevent adverse maternal and fetal outcomes.

**BIBLIOGRAPHY**

1. Benson CS, Shah A, Frise MC, Frise CJ. Iron deficiency anaemia in pregnancy: A contemporary review. *Obstet Med* 2021; 14: 67-76.
2. Ministry of Health and Family Welfare. Guidelines for control of iron deficiency anaemia. Jul 2021. Available from: <http://www.nhm.gov.in/images/pdf/programmes/child-health/guidelines/Control-of-Iron-DeficiencyAnaemia.Pdf>.
3. World Health Organization. Anaemia. Geneva: World Health Organization, 2021. Available from: <https://www.who.int/newsroom/factsheets/detail/anaemia>
4. Kuppusamy P, Prusty RK, Khan SA. Assessing the prevalence and predictors of anemia among pregnant women in India: findings from the India National Family Health Survey 2019-2021. *Curr Med Res Opin* 2024; 40: 51-58.
5. Kohne E. Hemoglobinopathies: clinical manifestations, diagnosis, and treatment. *Dtsch Arztebl Int* 2011; 108: 532-40.
6. Weatherall DJ. Hemoglobinopathies worldwide: present and future. *Curr Mol Med* 2008; 8: 592-599.
7. National Health Mission. Guidelines on Hemoglobinopathies in India. [Internet]. India: National Health Mission; [cited 2024 Jun 11]. Available from: [https://nhm.gov.in/images/pdf/infocus/NHM\\_Guidelines\\_on\\_Hemoglobinopathies\\_in\\_India.pdf](https://nhm.gov.in/images/pdf/infocus/NHM_Guidelines_on_Hemoglobinopathies_in_India.pdf)
8. Mondal SK, Mandal S. Prevalence of thalassemia and hemoglobinopathy in eastern India: A 10-year high-performance liquid chromatography study of 119,336 cases. *Asian J Transfus Sci* 2016; 10: 105-10.

9. Ali S, Mumtaz S, Shakir HA, et al. Current status of beta-thalassemia and its treatment strategies. *Mol Genet Genomic Med* 2021; 9: e1788.
10. Bajwa H, Basit H. Thalassemia. [Updated 2023 Aug 8]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK545151/>
11. Hossain MS, Raheem E, Sultana TA, et al. Thalassemias in South Asia: clinical lessons learnt from Bangladesh. *Orphanet J Rare Dis* 2017; 12: 93.
12. Goh LPW, Chong ETJ, Lee PC. Prevalence of Alpha ( $\alpha$ )-Thalassemia in Southeast Asia (2010-2020): A Meta-Analysis Involving 83,674 Subjects. *Int J Environ Res Public Health* 2020; 17: 7354.
13. Aggarwal P, Bhat D. Genetic counseling in sickle cell disease: Insights from the Indian tribal population. *J Community Genet* 2023; 14: 345-353.
14. Ilesanmi OO. Pathological basis of symptoms and crises in sickle cell disorder: implications for counseling and psychotherapy. *Hematol Rep* 2010; 2: e2.
15. Colah RB, Mukherjee MB, Martin S, Ghosh K. Sickle cell disease in tribal populations in India. *Indian J Med Res* 2015; 141: 509-15.
16. Van Vliet ME, Kerkhoffs JH, Harteveld CL, Houwink EJJ. Hemoglobinopathy prevention in primary care: a reflection of under detection and difficulties with accessibility of medical care, a quantitative study. *Eur J Hum Genet* 2022; 30: 790-794.
17. Biswas B, Naskar NN, Basu K, et al. An Epidemiological Study of the Quality of Life of Children With Beta-Thalassemia Major ( $\beta$ -TM) and Its Correlates in Kolkata, West Bengal, India. *Cureus* 2023; 15: e36888.

18. Karnon J, Zeuner D, Brown J, et al. Lifetime treatment costs of beta-thalassaemia major. *Clin Lab Haematol* 1999; 21:377-385.
19. Dharmarajan S, Pawar A, Bhide P, Kar A. Undiagnosed haemoglobinopathies among pregnant women attending antenatal care clinics in Pune, India. *J Community Genet* 2021; 12: 337-344.
20. Harteveld CL, Achour A, Arkesteijn SJG, et al. The hemoglobinopathies, molecular disease mechanisms and diagnostics. *Int J Lab Hematol* 2022;4:28-36.
21. Colizzi M, Lasalvia A, Ruggeri M. Prevention and early intervention in youth mental health: is it time for a multidisciplinary and trans-diagnostic model for care? *Int J Ment Health Syst* 2020;14:23.
22. Mandrile G, Barella S, Giambona A, Gigante A, Grosso M, Perrotta S, et al. First and Second Level Haemoglobinopathies Diagnosis: Best Practices of the Italian Society of Thalassemia and Haemoglobinopathies (SITE). *J Clin Med* 2022 Sep 15;11(18):5426.
23. Stephen G, Mgongo M, Hussein Hashim T, Katanga J, Stray-Pedersen B, Msuya SE. Anaemia in Pregnancy: Prevalence, Risk Factors, and Adverse Perinatal Outcomes in Northern Tanzania. *Anemia* 2018 May 2;2018:1846280.
24. Georgieff MK. Iron deficiency in pregnancy. *Am J Obstet Gynecol* 2020 Oct;223(4):516-524.
25. Means RT. Iron Deficiency and Iron Deficiency Anemia: Implications and Impact in Pregnancy, Fetal Development, and Early Childhood Parameters. *Nutrients* 2020 Feb 11;12(2):447.

26. Oguizu AD, Chigbundu SJ. Assessment of anaemia and dietary intake of pregnant women in Ikwuano local government area Abia State, Nigeria. *J Hum Nutr Food Sci* 2016;4(2):1085.
27. Bansal R, Bedi M, Kaur J, Kaur K, Shergill HK, Khaira HK, et al. Prevalence and factors associated with anemia among pregnant women attending antenatal clinic. *Adesh Univ J Med Sci Res* 2020;2(1):42-8.
28. Sinha NK, Chattopadhyay JC, Das PK, Maiti S, Maiti K. Prevalence of anemia and its possible attributing factors in psychologically healthy women of reproductive ages in Midnapore (Jangalmahal-area), India. *Indian J Community Health* 2013;25:226–32.
29. Suryanarayana R, Chandrappa M, Santhuram AN, Prathima S, Sheela SR. Prospective study on prevalence of anemia of pregnant women and its outcome: A community based study. *JFamily MedPrim Care* 2017 Oct-Dec;6(4):739-743.
30. Shi H, Chen L, Wang Y, Sun M, Guo Y, Ma S, Wang X, Jiang H, Wang X, Lu J, Ge L, Dong S, Zhuang Y, Zhao Y, Wei Y, Ma X, Qiao J. Severity of Anemia During Pregnancy and Adverse Maternal and Fetal Outcomes. *JAMA Netw Open* 2022 Feb 1;5(2)
31. Tandon R, Jain A, Malhotra P. Management of Iron Deficiency Anemia in Pregnancy in India. *Indian J Hematol Blood Transfus* 2018 Apr;34(2):204-215.
32. Osungbade KO, Oladunjoye AO. Preventive treatments of iron deficiency anaemia in pregnancy: a review of their effectiveness and implications for health system strengthening. *J Pregnancy* 2012;2012:454601.

33. Rappaport VJ, Velazquez M, Williams K. Hemoglobinopathies in pregnancy. *Obstet Gynecol Clin North Am* 2004;31(2):287-vi.
34. Jain D, Atmapoojya P, Colah R, Lodha P. Sickle Cell Disease and Pregnancy. *Mediterr J Hematol Infect Dis* 2019 Jul 1;11(1)
35. Figueira CO, Surita FG, Fertrin K, Nobrega GM, Costa ML. Main Complications during Pregnancy and Recommendations for Adequate Antenatal Care in Sickle Cell Disease: A Literature Review. *Rev Bras Ginecol Obstet* 2022 Jun;44(6):593-601.
36. Petrakos G, Andriopoulos P, Tsironi M. Pregnancy in women with thalassemia: challenges and solutions. *Int J Womens Health* 2016 Sep 8;8:441-51.
37. Saleh-Gohari N, Mohammadi-Anaie M. Co-inheritance of sickle cell trait and thalassemia mutations in South central iran. *Iran J Public Health* 2012;41(10):81-6.
38. Marengo-Rowe AJ. The thalassemias and related disorders. *Proc (Bayl Univ Med Cent)* 2007 Jan;20(1):27-31.
39. Forget BG, Bunn HF. Classification of the disorders of hemoglobin. *Cold Spring Harb Perspect Med* 2013 Feb 1;3(2)
40. Farid Y, Bowman NS, Lecat P. Biochemistry, Hemoglobin Synthesis. In: *StatPearls*. Treasure Island (FL): StatPearls Publishing; May 1, 2023.
41. Ahmed MH, Ghatge MS, Safo MK. Hemoglobin: Structure, Function and Allostery. *Subcell Biochem* 2020; 94: 345-382.

42. Safo MK, Ahmed MH, Ghatge MS, Boyiri T. Hemoglobin-ligand binding: understanding Hb function and allostery on atomic level. *Biochim Biophys Acta* 2011; 1814: 797-809.
43. Benner A, Patel AK, Singh K, Dua A. Physiology, Bohr Effect. In: *StatPearls*. Treasure Island (FL): StatPearls Publishing; 2023 Aug 8.
44. Patel S, Jose A, Mohiuddin SS. Physiology, Oxygen Transport and Carbon Dioxide Dissociation Curve. In: *StatPearls* [Internet]. Treasure Island (FL): StatPearls Publishing; 2024. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK539815>.
45. Malte H, Lykkeboe G, Wang T. The magnitude of the Bohr effect profoundly influences the shape and position of the blood oxygen equilibrium curve. *Comp Biochem Physiol A Mol Integr Physiol* 2021; 254: 110880.
46. Vandegriff KD, Benazzi L, Ripamonti M, et al. Carbon dioxide binding to human hemoglobin cross-linked between the alpha chains. *J Biol Chem* 1991; 266:2697-2700.
47. Thom CS, Dickson CF, Gell DA, Weiss MJ. Hemoglobin variants: biochemical properties and clinical correlates. *Cold Spring Harb Perspect Med* 2013; 3: a011858.
48. Inusa BPD, Hsu LL, Kohli N, et al. Sick Cell Disease-Genetics, Pathophysiology, Clinical Presentation and Treatment. *Int J Neonatal Screen* 2019;5:20.
49. Mettananda S, Gibbons RJ, Higgs DR.  $\alpha$ -Globin as a molecular target in the treatment of  $\beta$ -thalassemia. *Blood* 2015; 125: 3694-701.

50. Tesio N, Bauer DE. Molecular Basis and Genetic Modifiers of Thalassemia. *Hematol Oncol Clin North Am* 2023; 37: 273-299.
51. Makkawi M, Alasmari S, Hawan AA, et al. Hemoglobinopathies: An update on the prevalence trends in Southern Saudi Arabia. *Saudi Med J* 2021;42:784-789.
52. Arishi WA, Alhadrami HA, Zourob M. Techniques for the Detection of Sickle Cell Disease: A Review. *Micromachines (Basel)* 2021; 12: 519.
53. Marengo-Rowe AJ, McCracken AW, Flanagan P. Complete suppression of haemoglobin A synthesis in haemoglobin D Los Angeles—beta thalassaemia. *J Clin Path* 1968; 21: 508–510.
54. Wasi P. Haemoglobinopathies including thalassaemia. Part 1: Tropical Asia. *Clin Haematol* 1981; 10: 707–729.
55. Vijian D, Wan Ab Rahman WS, Ponnuraj KT, et al. Molecular Detection of Alpha Thalassemia: A Review of Prevalent Techniques. *Medeni Med J* 2021; 36: 257-269.
56. Cao A, Galanello R. Beta-thalassemia. *Genet Med* 2010; 12: 61-76.
57. Piel FB, Howes RE, Patil AP, et al. The distribution of haemoglobin C and its prevalence in newborns in Africa. *Sci Rep* 2013; 3: 1671.
58. Fucharoen S, Weatherall DJ. The hemoglobin E thalassemias. *Cold Spring Harb Perspect Med* 2012; 2: a011734.
59. Harteveld CL, Achour A, Arkesteijn SJG, et al. The hemoglobinopathies, molecular disease mechanisms and diagnostics. *Int J Lab Hematol* 2022; 44 Suppl 1: 28-36.

60. Ferreira TD, Freire AS, Silveira-Lacerda Ede P, García-Zapata MT. A model of genetic guidance for hemoglobinopathy patients and laboratory diagnosis of family members as educational and preventive measures. *Rev Bras Hematol Hemoter* 2012; 34: 339-44.
61. Kasperek J, Burkhardt T, Hoesli I, Amstad Bencaiova G. Pregnancy outcomes in women with a hemoglobinopathy trait: a multicenter, retrospective study. *Arch Gynecol Obstet* 2021; 304: 1197-1203.
62. Sorrentino F, Maffei L, Caprari P, et al. Pregnancy in Thalassemia and Sickle Cell Disease: The Experience of an Italian Thalassemia Center. *Front Mol Biosci* 2020; 7: 16.
63. Fisher A, Nemeth E. Iron homeostasis during pregnancy. *Am J Clin Nutr* 2017; 106 (Suppl): 1567S–74S.
64. Bakhsh E, Alkhalidi M, Shaban M. Exploring the Link between Maternal Hematological Disorders during Pregnancy and Neurological Development in Newborns: Mixed Cohort Study. *Life (Basel)* 2023; 13: 2014.
65. Abu-Raya B, Michalski C, Sadarangani M, Lavoie PM. Maternal Immunological Adaptation During Normal Pregnancy. *Front Immunol* 2020; 11: 575197.
66. Abu-Ouf NM, Jan MM. The impact of maternal iron deficiency and iron deficiency anemia on child's health. *Saudi Med J* 2015; 36: 146-9.
67. Society for Maternal-Fetal Medicine; Sinkey RG, Ogunbile FJ, Kanter J, et al. Society for Maternal-Fetal Medicine Consult Series #68: Sickle cell disease in pregnancy. *Am J Obstet Gynecol* 2024; 230: B17-B40.

68. Shegekar T, Pajai S. A Comprehensive Review of Pregnancy in Sickle Cell Disease. *Cureus* 2023; 15: e41165.
69. Thomas P, Oni L, Alli M, et al. Antenatal screening for haemoglobinopathies in primary care: a whole system participatory action research project. *Br J Gen Pract* 2005; 55: 424-8.
70. Cao A, Galanello R, Rosatelli MC. Prenatal diagnosis and screening of the haemoglobinopathies. *Baillieres Clin Haematol* 1998; 11: 215-238.
71. Bhukhanvala DS, Sorathiya SM, Sawant P, et al. Antenatal screening for identification of couples for prenatal diagnosis of severe hemoglobinopathies in Surat, South Gujarat. *J Obstet Gynaecol India* 2013; 63: 123-7.
72. Petrou M. Genetic counselling. In: Angastiniotis M, Eleftheriou A, Galanello R, et al., editors. *Prevention of Thalassaemias and Other Haemoglobin Disorders: Volume 1: Principles* [Internet]. 2nd ed. Nicosia (Cyprus): Thalassaemia International Federation; 2013. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK190461>.
73. Trent RJ. Diagnosis of the haemoglobinopathies. *Clin Biochem Rev* 2006; 27: 27-38.
74. Smith LA, Young BC. Antenatal optimization of maternal anemia leads to decreased risks of maternal morbidity. *Curr Obstet Gynecol Rep* 2023; 3: 1-7.
75. Aroke D, Tchouakam DN, Kadia BM, Choukem SP. Iron supplementation in pregnant sicklers: an opinion. *BMC Pregnancy Childbirth* 2018; 18: 256.

76. Malik SD, Al-Shafai M, Abdallah AM. The special features of prenatal and preimplantation genetic counseling in Arab countries. *Genes (Basel)* 2022; 13: 167.
77. David AL, Spencer RN. Clinical assessment of fetal well-being and fetal safety indicators. *J Clin Pharmacol* 2022; 62 Suppl 1: S67-S78.
78. Wang M, Zhang X, Zhao Y, et al. Prevalence of iron-deficiency anemia in pregnant women with various thalassemia genotypes: Thoughts on iron supplementation in pregnant women with thalassemia genes. *Front Nutr* 2022; 9: 1005951.
79. Oteng-Ntim E, Shangaris P. Evidence-based management of pregnant women with sickle cell disease in high-income countries. *Hematology Am Soc Hematol Educ Program* 2022; 2022: 408-413.
80. McGann PT, Ware RE. Hydroxyurea therapy for sickle cell anemia. *Expert Opin Drug Saf* 2015; 14: 1749-58.
81. Leung T, Lao T. Thalassemia in pregnancy. *Best Pract Res Clin Obstet Gynaecol* 2012; 26: 37–51.
82. Fox R, Kitt J, Leeson P, et al. Preeclampsia: Risk Factors, Diagnosis, Management, and the Cardiovascular Impact on the Offspring. *J Clin Med* 2019; 8: 1625.
83. Maughan BC, Marin M, Han J, et al. Venous thromboembolism during pregnancy and the postpartum period: risk factors, diagnostic testing, and treatment. *Obstet Gynecol Surv* 2022; 77: 433-444.

84. Sharma D, Shastri S, Sharma P. Intrauterine growth restriction: antenatal and postnatal aspects. *Clin Med Insights Pediatr* 2016; 10: 67-83.
85. Requejo J, Merialdi M, Althabe F, et al. Born too soon: care during pregnancy and childbirth to reduce preterm deliveries and improve health outcomes of the preterm baby. *Reprod Health* 2013; 10 (Suppl 1): S4.
86. Stevens B. Impact of emerging technologies in prenatal genetic counseling. *Cold Spring Harb Perspect Med* 2020; 10: a036517.
87. Barnes SG, Sutliff B, Wendel MP, Magann EF. Maternal transport, what do we know: a narrative review. *Int J Womens Health* 2024; 16: 877-889.
88. Rajauria S, Atreja CB, Mujalda A, et al. The effect of sickle cell hemoglobinopathy on pregnancy, labor, puerperium, and fetal outcome: a retrospective cohort study from a single centre. *Cureus* 2023; 15: e34318.
89. Tepper NK, Boulet SL, Whiteman MK, et al. Postpartum venous thromboembolism: incidence and risk factors. *Obstet Gynecol* 2014; 123:987-996.
90. Carretero-Krug A, Montero-Bravo A, et al. Nutritional status of breastfeeding mothers and impact of diet and dietary supplementation: a narrative review. *Nutrients* 2024; 16: 301.
91. Chauhan A, Prasad M. Outcome of pregnancy with hemoglobinopathy in a tertiary care center. *J Obstet Gynaecol India* 2018; 68: 394-9.
92. Priyadarsini B, Mohapatra K, Naik M, Behuria S. Antenatal screening for hemoglobinopathies with HPLC and their fetomaternal outcome. *Int J Health Sci* 2022; 6 (S9): 2958–68.

93. Bedrick BS, Kohn TP, Pecker LH, et al. Fertility preservation for pediatric patients with hemoglobinopathies: multidisciplinary counseling needed to optimize outcomes. *Front Endocrinol (Lausanne)* 2022; 13: 985525.
94. Twum S, Fosu K, Felder RA, et al. Bridging the gaps in newborn screening programmes: challenges and opportunities to detect hemoglobinopathies in Africa. *Afr J Lab Med* 2023; 12: 2225.
95. Tegha G, Topazian HM, Kamthunzi P, et al. Prospective newborn screening for sickle cell disease and other inherited blood disorders in Central Malawi. *Int J Public Health* 2021; 66: 629338.
96. Bain BJ, Daniel Y, Henthorn J, et al. Significant haemoglobinopathies: a guideline for screening and diagnosis: a British Society for Haematology guideline. *Br J Haematol* 2023; 201: 1047-65.
97. Cousens NE, Gaff CL, Metcalfe SA, Delatycki MB. Carrier screening for beta-thalassaemia: a review of international practice. *Eur J Hum Genet* 2010; 18: 1077-83.
98. Patel AG, Shah AP, Sorathiya SM, Gupte SC. Hemoglobinopathies in South Gujarat population and incidence of anemia in them. *Indian J Hum Genet* 2012; 18: 294-8.
99. Baruah MK, Saikia M, Baruah A. Pattern of hemoglobinopathies and thalassemiias in upper Assam region of North Eastern India: high performance liquid chromatography studies in 9000 patients. *Indian J Pathol Microbiol* 2014; 57: 236-43.

100. Sengupta B, De M, Dasgupta I, Poddar S. Comparative study of haemoglobinopathies in tribal populations of Arunachal Pradesh and Tripura (North East India). *Int J Hum Genet* 2002; 2: 169-72.
101. Dolai TK, Dutta S, Bhattacharyya M, Ghosh MK. Prevalence of hemoglobinopathies in rural Bengal, India. *Hemoglobin* 2012; 36: 57-63.
102. Sur D, Chakravorty R. Prevalence of hemoglobinopathies and thalassemia carriers in women of reproductive age group: a single center study in West Bengal. *J Hematol* 2016; 5: 99-102.
103. Aksoy S. Antenatal screening and its possible meaning from the unborn baby's perspective. *BMC Med Ethics* 2001; 2: E3.
104. Chakravorty S, Dick MC. Antenatal screening for haemoglobinopathies: current status, barriers, and ethics. *Br J Haematol* 2019; 187: 431-440.
105. Thaker P, Colah RB, Patel J, et al. Newborn screening for sickle cell disease among tribal populations in Gujarat and Madhya Pradesh, India: evaluation and outcome over 6 years. *Front Med (Lausanne)* 2022; 8: 731884.
106. Shah N, Khonglah Y, Raphael V, et al. Antenatal screening for hemoglobinopathies with HPLC. *Lab Med* 2018; 4: 1-8.
107. Balgir RS. Prevalence of hemolytic anemia and hemoglobinopathies among pregnant women attending a tertiary hospital in central India. *Thalassemia Reports* 2015; 5: 16-20.
108. Sharma A, Uppal N, Kukreja S, et al. Screening of thalassemia in pregnant females visiting a tertiary hospital in Amritsar. *Int J Clin Biochem Res* 2020; 7: 226-31.

109. Ghosh P, Dasgupta A, Paul B, et al. A cross-sectional study on prevalence and determinants of anemia among women of reproductive age in a rural community of West Bengal. *J Family Med Prim Care* 2020; 9: 5547-5553.
110. Ai S, Cliffe C, Kidson-Gerber G. Antenatal haemoglobinopathy screening: experiences of a large Australian centre. *Obstet Med* 2021; 14: 89-94.
111. Gosavi M, Chavan R, Bellad MB. NESTROFT—A cost-effective mass screening tool for the detection of  $\beta$ -thalassemia carrier status in anemic pregnant women. *J Lab Physicians* 2021; 13: 368-73.
112. Sawaimul KD, Iqbal MB, Sawaimul VD, et al. Study to identify the role of high-performance liquid chromatography in detecting haemoglobinopathies in antenatal patients. *Indian J Pathol Oncol* 2018; 5: 6-11.
113. Ou CN, Rognerud CL. Diagnosis of hemoglobinopathies: electrophoresis vs. HPLC. *Clin Chim Acta* 2001; 313: 187-194.
114. Wendt AS, Brintrup J, et al. Thalassemia and hemoglobinopathy prevalence in a community-based sample in Sylhet, Bangladesh. *Orphanet J Rare Dis* 2023; 18: 192.
115. Ontario Health (Quality). Carrier screening programs for cystic fibrosis, fragile X syndrome, hemoglobinopathies and thalassemia, and spinal muscular atrophy: a health technology assessment. *Ont Health Technol Assess Ser* 2023; 23: 1-398.
116. Edwards S, Laing N. Genetic counselling needs for reproductive genetic carrier screening: a scoping review. *J Pers Med* 2022; 12: 1699.

117. Sabath DE. Molecular diagnosis of thalassemias and hemoglobinopathies: an ACLPS critical review. *Am J Clin Pathol* 2017; 148: 6-15.
118. Shanthi S, Beula D, Rajendran A, et al. Antenatal screening for haemoglobinopathies among the tribal population in Tamil Nadu, India. *HemaSphere* 2023; 7: 5.
119. Bajaj K, Gross SJ. Carrier screening: past, present, and future. *J Clin Med* 2014; 3: 1033–42.
120. Alshamsi S, Hamidi S, Narci HO. Healthcare resource utilization and direct costs of transfusion-dependent thalassemia patients in Dubai, United Arab Emirates: a retrospective cost-of-illness study. *BMC Health Serv Res* 2022; 22: 304.
121. Weatherall DJ, Clegg JB. Inherited haemoglobin disorders: an increasing global health problem. *Bull World Health Organ* 2001; 79: 704–712.
122. Sawaimul KD, Iqbal MB, Sawaimul VD, Kambale T, Hanmante R. Study to identify the role of high-performance liquid chromatography in detecting haemoglobinopathies in antenatal patients. *Indian J Pathol Oncol*. 2018 Jan;5(1):6-11.
123. Singh N, Chowdhury N, Bahadur A, Ahuja S, Arathi K, Jeladharan R, Mirza AA, Gupta AK, Chandra H, Rao S. Thalassemia and hemoglobinopathy screening in women attending antenatal clinic at a tertiary care center in Uttarakhand, India: a re-look at the laboratory parameters mandating high-performance liquid chromatography workup. *Cureus*. 2023 Jun;15(6).

124. Christianson A, Howson C, Modell B. March of Dimes global report on birth defects: the hidden toll of dying and disabled children. The March of Dimes Birth Defects Foundation, 2006. Available from: March of Dimes.
125. Mukhopadhyay A, Bhadra M, Bose K. Anthropometric assessment of nutritional status of adolescents of Kolkata, West Bengal. *J Hum Ecol* 2005; 18: 251-256.
126. Ahuja T, Bhatnagar N, Shah M, Shah S. Screening of antenatal patients for anaemia and haemoglobinopathies. *Hematol Transfus Int* 2023; 11: 67-71.
127. Gupta S, Naert M, Lam-Rachlin J, et al. Outcomes in patients with early-onset fetal growth restriction without fetal or genetic anomalies. *J Matern Fetal Neonatal Med* 2019; 32: 2662-2666.
128. Verma P, Ghilidiyal A, Krishan A, Singh S. Prevalence of hemoglobinopathies in different regions and castes of Uttar Pradesh, India: a hospital-based study. *Asian J Med Sci* 2015; 6: 21-25.
129. Siddiqui SH, Ishtiaq R, Sajid F, Sajid R. Quality of life in patients with thalassemia major in a developing country. *J Coll Physicians Surg Pak* 2014; 24: 477-480.
130. Chaudhry HS, Kasarla MR. Microcytic hypochromic anemia. [Updated 2023 Aug 14]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan. Available from: StatPearls.
131. Chakrabarti A, Bhattacharya D, Deb S, Chakraborty M. Differential thermal stability and oxidative vulnerability of the hemoglobin variants, HbA2 and HbE. *PLoS One* 2013; 8: e82081.

132. Khan A, Rehman AU. Laboratory evaluation of beta thalassemia. [Updated 2023 Aug 28]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan.
133. Tabassum S, Khakwani M, Fayyaz A, Taj N. Role of Mentzer index for differentiating iron deficiency anemia and beta thalassemia trait in pregnant women. *Pak J Med Sci* 2022; 38: 878-882.
134. Mukherjee MB, Nadkarni AH, Gorakshakar AC, Ghosh K, Mohanty D, Colah RB. Clinical, hematologic, and molecular variability of sickle cell- $\beta$  thalassemia in western India. *Indian J Hum Genet* 2010; 16: 154-158.

---

## **ANNEXURE I – INFORMED CONSENT FORM**

### **“Antenatal Screening for Hemoglobinopathies in a Tertiary Care Centre**

#### **A One Year Descriptive Observational Study”**

Principal investigator:

REG.NO: BJ0121008

Post Graduate,

Department of OBG,

J.N. Medical College, KAHER, Belagavi.

Guide:

Dr.

Professor,

Department of OBG,

J.N. Medical College, KAHER, Belagavi.

**Purpose of the Study:** Hemoglobinopathies are group of diseases characterized by abnormalities both quantitative and qualitative in the production of hemoglobin. In India, major concerned hemoglobinopathic disorders are Sickle cell anemia and  $\beta$ -thalassemia. If a couple carry a clinically significant hemoglobinopathy trait, there is 1 in 4 chance with each pregnancy that their children will inherit a major hemoglobinopathy.

The most effective approach to reduce the burden of society is to reduce the incidence by implementation of a carrier screening programme.

Detection of carrier status using complete hemogram and Hb Electrophoresis during pregnancy along with couple screening provides prospective parents with the option of testing their foetus for hemoglobinopathy.

**Explanation of Procedure:** Once you agree to participate in the study, you will be enrolled and detailed history including obstetric history, past history, family history, socio-economic history and personal history will be taken and you will be subjected to general examination, systemic examination and obstetric examination. 4 ml of venous blood will be taken from antecubital vein in two Disodium salt of ethylene

diamine tetra-acetic acid (EDTA) coated vials and will be subjected to complete hemogram, peripheral blood smear and electrophoresis by HPLC.

**Voluntary Participation:** Your participation in this research is voluntary. It is your choice whether to participate or not. Your decision whether to participate in the study or not will not change the present or future health care services offered to you and will not affect your relationship with Dr.Prabhakar Kore Charitable Hospital and Research Centre, J.N. Medical College, KAHER, Belagavi. If you choose not to participate in this study, you will still be offered good treatment at our hospital and you will continue to receive the routine pregnancy care at our hospital. If you decide to participate, you are free to withdraw at any time.

**Possible benefits from participating in the study:** You will be screened for hemoglobinopathies and if reported positive, further screening of your husband and genetic counselling and evaluation will be advised, there by the status of fetus for hemoglobinopathy will be known and you can take an informed decision.

**Possible risks from participating in the study:** There are no risks involved in participating in this study

**Privacy and confidentiality:** Your confidentiality will be respected. No information that discloses your identity will be released. All the information taken through interviews with you will be kept safely and no person other than authorized local key investigators will be able to trace the information to your name or your address.

**Financial incentives:** You will not receive any payment for participating in this study.

**Authorization for publication of aggregated data:** Results obtained after processing of the aggregated data will be published for scientific purposes and or presented to scientific groups. However, your identity will never be revealed.

**Whom to contact with questions about the study:** We have given you information about the study called “Antenatal Screening for Hemoglobinopathies in a Tertiary Care Centre - A One Year Descriptive Observational Study”. We have discussed about the study and you understand that you do not have to agree to be in the study or may decide later not to be part of the study. This will not affect your or your baby’s care in any way.

If you have any questions, please call: Reg No: BJ0121008.

If you have questions about your rights as a study participant, please contact Dr.Harsha Hegde, Chairperson, JNMC, IEC and Scientist D, ICMR, National Institute of Traditional Medicine, Belagavi-9480422500

**CONSENT STATEMENT**

I have read this consent form or it has been read to me in presence of a witness in my vernacular. I was given opportunity to ask questions and they were answered to my satisfaction. By signing this document, I declare that I have consented to participate in this study.

Signature or Thumbprint of Person Providing Consent

Date

Signature or Thumbprint of Witness/parents

Date

Signature of Person Obtaining Consent

Date

Person requesting consent, please check applicable boxes:

- Consent obtained (for adult respondent)
- Assent (for minor respondents)
- Consent from authorized person of minor respondent

**ANNEXURE II – PROFORMA****SCREENING FORM**

**“Antenatal Screening for Hemoglobinopathies in a Tertiary Care Centre – A One Year Descriptive Observational Study”.**

Screening Number :

Date of Screening (dd-mm-yyyy) :

OP / IP Number:

First Name :

Middle Name :

Last Name :

Age (years) :

Address : H.no. -

Street -

Taluka -

District -

Phone Number :

**Eligibility:**

YES – 1 NO – 2

A. Period of Gestation  $\leq$  20 weeks

a. LMP Known

b. Dating Scan

<input type="text"/>
<input type="text"/>
<input type="text"/>

Last menstrual period (dd-mm-yyyy):

Expected date of delivery (dd-mm-yyyy):

USG EDD (dd-mm-yyyy):

Period of gestation (weeks/ days )

According to LMP / CEDD :

B. Known Case of Hemoglobinopathy

C. No History of Recent Blood Transfusion ( within 1 month )

Is she eligible?

If eligible,

Does the woman assent to participate?

Enrolled into the study

YES – 1 NO – 2

Date of Enrollment:       
(dd-mm-yyyy)

Enrollment Number :

## DATA COLLECTION INSTRUMENT

## “Antenatal Screening for Hemoglobinopathies in a Tertiary Care Centre – A One Year Descriptive Observational Study”.

Screening Number : Enrollment Number : **Obstetric history:**Married Life (years) : Consanguinity :  (YES - 1, NO - 2)

If yes,

Degree of consanguinity : 

Obstetric score :

Gravida  Para  Live  Abortion **Menstrual history:**Menarche (age in years): 

YES – 1, NO – 2

Regular Past menstrual cycles: Last menstrual period (dd-mm-yyyy):   Expected date of delivery (dd-mm-yyyy):   USG EDD (dd-mm-yyyy):   

Period of gestation (weeks/ days)

According to LMP / C.EDD :  **Past History : YES – 1 , NO – 2**a. Known case of Diabetes mellitus :If yes, Duration (in years) : Treatment received  
  
b. Known case of Hypertension :If yes, Duration (in years) : Treatment received :



**Personal History :**

YES – 1 , NO – 2

- a. Diet
  - Vegetarian
  - Mixed
- b. Normal appetite
- c. Adequate sleep
- d. Normal Bowel & Bladder habits

**General Physical Examination :**

Height (in centimetres)

Weight (in kilogram)

BMI

YES – 1 , NO – 2

Pallor

Icterus

Pedal Oedema

Blood pressure (mmHg)  /

Pulse rate (beats per minute)

**Systemic Examination :**

Cardiovascular : \_\_\_\_\_

Respiratory : \_\_\_\_\_

Per Abdomen : \_\_\_\_\_

FHR

**Investigations-**

Date (dd-mm-yyyy) :

Blood Group:

Haemoglobin (g/dl) :   
(cyanmethemoglobin method)

Packed cell volume (%)

RBC :

MCV :

MCH :

MCHC :

Peripheral Smear :

**HPLC :**

Hb A

Hb A2

Hb F

Other Hb

Report

**Provisional diagnosis:**

Investigator's Signature :

Patient's Signature

# **ANNEXURE III**

# **MASTER CHART**

Antenatal Screening of Hemoglobinopathies in a Tertiary Care Centre - One Year Descriptive Observational Study

Antenatal Screening of Hemoglobinopathies in a Tertiary Care Centre - One Year Descriptive Observational Study																									
Screening No.	Enrollment No.	Age	Obst. Score	Gestational Age	Degree of Consanguinity	H/o Blood Transfusion	K/c/o Hemoglobinopathy			Blood Group	HB	PCV	RBC	MCV	MCH	MCHC	Mentzer Index	Peripheral Smear	HbA	HbA2	HbF	Other Hb	Report	Husband's HPLC	Amniocentesis
							Patient	Husband	Prior Children																
1	1	22	Primigravida	11 weeks 3 days	NCM	No	No	No	No	O positive	11.8	35.4	3.79	93.40	31.13	33.33	24.64	Normal Blood Picture	96.9	2.3	0.8	0	Normal Hb Variant		
4	2	31	G3P1L1A1	19 weeks 2 days	NCM	No	No	No	No	O Positive	7.9	23.7	2.64	89.77	29.92	30.20	34.00	Microcytic Hypochromic Anemia	95.9	3.3	0.8	0	Normal Hb Pattern		
5	3	32	G3A2	12 weeks 1 day	NCM	No	No	No	No	O Positive	12	36	3.7	97.30	32.43	33.20	26.30	Normal Blood Picture	96.9	2.3	0.8	0	Normal Hb Variant		
8	4	31	G2P1L1	8 weeks 2 days	3*CM	No	No	No	No	A Positive	13.6	40.8	4.35	93.79	31.26	32.10	21.56	Normal Blood Picture	96.7	2.5	0.8	0	Normal Hb Variant		
12	5	26	Primigravida	7 weeks 4 days	NCM	No	No	No	No	O Positive	9.9	31.9	4.93	63.90	20.50	30.80	12.96	Microcytic Hypochromic Anemia	94.8	4.4	0.8	0	Thalassemia Trait	Normal	
14	6	34	G3P2L1	18 weeks 2 days	NCM	No	No	No	No	B Positive	13.2	39.6	4.44	89.19	29.73	33.20	20.09	Normal Blood Picture	96.8	2.4	0.8	0	Normal Hb Variant		
18	7	21	Primigravida	13 weeks 3 days	NCM	No	No	No	No	O Positive	6.5	19.5	3.59	54.32	18.11	28.20	15.13	Microcytic Hypochromic Anemia	97.4	1.8	0.8	0	Normal Hb Variant		
19	8	26	Primigravida	14 weeks 2 days	NCM	No	No	No	No	O Positive	10.3	30.9	3.53	87.54	29.18	33.00	24.80	Normal Blood Picture	96	2.6	1.4	0	Normal Hb Variant		
22	9	27	G2P1L1	11 weeks 1 day	NCM	No	No	No	No	A Positive	12.9	38.7	3.91	98.98	32.99	33.40	25.31	Normal Blood Picture	96.3	2.9	0.8	0	Normal Hb Variant		
25	10	32	G4P1L1A2	10 weeks 3 days	NCM	No	No	No	No	AB Positive	11.3	33.9	4.31	78.65	26.22	32.00	18.25	Normocytic Normochromic Anemia	95.5	2.7	1.8	0	Normal Hb Variant		
28	11	24	G2A1	8 weeks 1 day	NCM	No	No	No	No	B Positive	11.8	35.4	3.71	95.42	31.81	33.60	25.72	Normal Blood Picture	96.4	2.8	0.8	0	Normal Hb Variant		
29	12	26	Primigravida	weeks 4 da	NCM	No	No	No	No	O Positive	8.4	27.6	4.98	64.5	19.6	30.4	12.95	Microcytic Hypochromic Anemia	95.1	3.8	1.1	0	Thalassemia Minor	Not done	
31	13	20	Primigravida	15 weeks 4 days	NCM	No	No	No	No	B Positive	10.9	32.7	3.98	82.16	27.39	32.60	20.64	Normocytic Normochromic Anemia	96.2	2.9	0.9	0	Normal Hb Variant		
33	14	30	G3P1L1A1	18 weeks 1 day	NCM	No	No	No	No	A Positive	10.9	32.7	3.84	85.16	28.39	31.60	22.18	Normal Blood Picture	96.8	2.4	0.8	0	Normal Hb Variant		
35	15	25	G2A1	19 weeks 4 days	NCM	No	No	No	No	B Positive	11.8	35.4	3.88	91.24	30.41	32.40	23.51	Normal Blood Picture	96.2	2.4	1.4	0	Normal Hb Variant		
37	16	24	Primigravida	15 weeks 3 days	NCM	No	No	No	No	B Positive	12.5	37.5	4.19	89.50	29.83	32.20	21.36	Normal Blood Picture	96.8	2.4	0.8	0	Normal Hb Variant		

Antenatal Screening of Hemoglobinopathies in a Tertiary Care Centre - One Year Descriptive Observational Study

38	17	28	G2A1	9 weeks 6 days	1*CM	No	No	No	No	O Positive	9.8	29.4	3.92	75.00	25.00	30.10	19.13	Microcytic Hypochromic	96.6	2	1.4	0	Normal Hb Variant		
40	18	24	Primigravida	11 weeks 2 days	NCM	No	No	No	No	O Positive	10.6	31.8	4.5	70.67	23.56	30.60	15.70	Microcytic Hypochromic	97.2	2	0.8	0	Normal Hb Variant		
41	19	23	Primigravida	9 weeks 5 days	NCM	No	No	No	No	AB Positive	8.3	24.9	4.69	53.09	17.70	30.40	11.32	Microcytic Hypochromic	97.7	1.5	0.8	0	Normal Hb Variant		
44	20	26	G2P1L1	8 weeks 6 days	NCM	No	No	No	No	B Positive	9.8	29.4	4.46	65.92	21.97	31.20	14.78	Microcytic Hypochromic	97.1	2.1	0.8	0	Normal Hb Variant		
46	21	26	Primigravida	9 weeks 4 days	NCM	No	No	No	No	A Positive	9.5	28.5	4.97	64.00	30.80	33.30	12.88	Normocytic Normochromic	94.6	2.9	2.5	0	Thalassemia Minor	Not done	
48	22	32	G2P1L1	19 weeks 3 days	NCM	No	No	No	No	O Positive	12.2	36.6	4.6	79.57	26.52	30.10	17.30	Microcytic Hypochromic	96.8	2.4	0.8	0	Normal Hb Variant		
49	23	31	G2P1L1	18 weeks 3 days	NCM	No	No	No	No	B Positive	13.6	40.8	4.97	82.09	27.36	34.40	16.52	Normal Blood Picture	96.5	2.7	0.8	0	Normal Hb Variant		
52	24	23	Primigravida	13 weeks 6 days	2*CM	No	No	No	No	O Positive	13.1	39.3	4.7	83.62	27.87	29.90	17.79	Normocytic Normochromic	96.2	2.6	1.2	0	Normal Hb Variant		
55	25	21	Primigravida	11 weeks 6 days	NCM	No	No	No	No	B Positive	12.3	36.9	4.6	80.22	26.74	30.40	17.44	Normocytic Normochromic	96.5	2.5	1	0	Normal Hb Pattern		
57	26	25	Primigravida	8 weeks 2 days	NCM	No	No	No	No	A Positive	12.1	36.3	4.19	86.63	28.88	35.20	20.68	Normocytic Normochromic	96.8	2.6	0.6	0	Normal Hb Variant		
58	27	26	G2P1L1	8 weeks 6 days	NCM	No	No	No	No	A Positive	8.6	25.8	4.69	55.01	18.34	30.20	11.73	Microcytic Hypochromic	96.3	2.9	0.8	0	Normal Hb Variant		
60	28	23	Primigravida	weeks 4 da	3*CM	No	No	No	No	B Positive	9.8	31.7	4.89	64.8	20	30.9	13.25	Microcytic Hypochromic Anemia	97.2	2.5	0.3	0	Normal Hb Variant		
62	29	25	G2A1	10 weeks 4 days	NCM	No	No	No	No	A Positive	12.4	37.2	4.6	80.87	26.96	31.20	17.58	Normocytic Normochromic Anemia	96.9	2.3	0.8	0	Normal Hb Variant		
63	30	24	G2P1L1	11 weeks 4 days	NCM	No	No	No	No	A Positive	12.6	37.8	4.6	82.17	27.39	29.40	17.86	Normocytic Normochromic Anemia	96.6	2.7	0.7	0	Normal Hb Pattern		
66	31	27	G3P2L1	10 weeks 4 days	NCM	No	No	No	No	A Positive	8.1	24.3	4.69	51.81	17.27	30.40	11.05	Macrocytic Hypochromic Anemia	97.2	2.8	0	0	Normal Hb Variant		
68	32	30	G2A1	9 weeks 5 days	NCM	No	No	No	No	A Positive	8.3	24.9	4.55	54.73	18.24	29.10	12.03	Macrocytic Hypochromic Anemia	97.3	2.1	0.6	0	Normal Hb Variant		
69	33	29	G3P1L1A1	8 weeks 6 days	NCM	No	No	No	No	B Positive	12.2	36.6	4.19	87.35	29.12	29.90	20.85	Macrocytic Hypochromic Anemia	95.8	2.6	1.6	0	Normal Hb Variant		
71	34	27	G2P1L1	7 weeks 2 days	NCM	No	No	No	No	B Positive	12.6	36.6	4.5	81.33	28.00	29.60	18.07	Normocytic Normochromic Anemia	96.8	2.6	0.6	0	Normal Hb Variant		
73	35	28	G2A1	16 weeks 4 days	2*CM	No	No	No	No	B Positive	9.7	29.1	3.79	76.78	25.59	33.60	20.26	Normocytic Normochromic Anemia	96.3	2.9	0.8	0	Normal Hb Variant		
74	36	34	G3A2	17 weeks 6 days	NCM	No	No	No	No	B Positive	9.4	28.2	4.18	67.46	22.49	34.10	16.14	Microcytic Hypochromic Anemia	96.4	2.7	0.9	0	Normal Hb Variant		

Antenatal Screening of Hemoglobinopathies in a Tertiary Care Centre - One Year Descriptive Observational Study

76	37	21	Primigravida	15 weeks 4 days	NCM	No	No	No	No	O positive	9.2	28.2	4.98	63.45	21.30	30.20	12.74	Microcytic Hypochromic Anemia	92.5	5.8	1.7	0	Thalassemia Minor	Not done
78	38	26	G2P1L1	12 weeks 5 days	NCM	No	No	No	No	O Positive	11.6	34.8	3.79	91.82	30.61	33.33	24.23	Normal Blood Picture	94.9	2.7	2.4	0	Normal Hb Variant	
79	39	27	G3P1L1A1	19 weeks 2 days	NCM	No	No	No	No	O Positive	7.5	22.5	2.64	85.23	28.41	30.20	32.28	Macrocytic Hypochromic Anemia	96.4	2.9	0.7	0	Normal Hb Variant	
82	40	30	G3P2L1	17 weeks 5 days	NCM	No	No	No	No	O Positive	12.2	36.6	3.7	98.92	32.97	33.20	26.73	Normal Blood Picture	96.9	2.7	0.4	0	Normal Hb Variant	
85	41	34	G2P1L1	14 weeks 4 days	NCM	No	No	No	No	O Positive	12.8	38.4	4.35	88.28	29.43	32.10	20.29	Normal Blood Picture	96.9	2.3	0.8	0	Normal Hb Variant	
88	42	29	G3P1L1A1	12 weeks 3 days	NCM	No	No	No	No	O Positive	9.2	31.9	4.8	66.50	20.50	30.80	13.85	Normocytic Normochromic Anemia	96.6	2.7	0.7	0	Normal Hb Variant	
89	43	24	Primigravida	weeks 3 da	NCM	No	No	No	No	O Positive	9.7	33.9	5.18	65.4	18.7	28.7	12.63	Microcytic Hypochromic Anemia	94.1	4.1	1.8	0	Thalassemia Minor	Normal
91	44	32	G3P1L1A1	10 weeks 4 days	2*CM	No	No	No	No	O Positive	6.4	19.2	3.59	53.48	17.83	28.20	14.90	Microcytic Hypochromic Anemia	96.3	2.9	0.8	0	Normal Hb Variant	
92	45	32	Primigravida	12 weeks 3 days	NCM	No	No	No	No	O Positive	10.1	30.3	3.53	85.84	28.61	33.00	24.32	Normocytic Normochromic Anemia	96.4	2.7	0.9	0	Normal Hb Variant	
94	46	35	G4P1L1A2	18 weeks 2 days	NCM	No	No	No	No	O Positive	11.6	34.8	3.91	89.00	29.67	33.40	22.76	Normal Blood Picture	96.4	2.8	0.8	0	Normal Hb Variant	
95	47	28	G2P1L1	14 weeks 3 days	NCM	No	No	No	No	O Positive	11.1	33.3	4.31	77.26	25.75	32.00	17.93	Normocytic Normochromic Anemia	96	3.1	0.9	0	Normal Hb Pattern	
97	48	26	G2A1	17 weeks 3 days	NCM	No	No	No	No	O Positive	11.5	34.5	3.71	92.99	31.00	33.60	25.07	Normal Blood Picture	96.8	2.4	0.8	0	Normal Hb Variant	
98	49	22	Primigravida	18 weeks 4 days	NCM	No	No	No	No	O Positive	12.1	36.3	4.19	86.63	28.88	32.90	20.68	Normal Blood Picture	97.4	1.8	0.8	0	Normal Hb Variant	
100	50	20	Primigravida	13 weeks 2 days	3*CM	No	No	No	No	B Positive	14.4	45.6	4.92	63.80	30.20	32.40	12.97	Normocytic Normochromic	94.2	2.4	3.4	0	Thalassemia Minor	Normal
102	51	22	Primigravida	11 weeks 3 days	NCM	No	No	No	No	O Positive	10.3	30.9	3.98	77.64	25.88	32.60	19.51	Microcytic Hypochromic Anemia	96.9	3.1	0	0	Normal Hb Variant	
103	52	20	Primigravida	10 weeks 4 days	NCM	No	No	No	No	O Positive	10.2	45	3.84	117.19	26.56	31.60	30.52	Macrocytic Hypochromic Anemia	96.9	3.1	0	0	Normal Hb Variant	
104	53	29	G2P1L1	9 weeks 6 days	NCM	No	No	No	No	O Positive	11.4	34.2	3.88	88.14	29.38	32.40	22.72	Normocytic Normochromic Anemia	97	3	0	0	Normal Hb Variant	
105	54	20	Primigravida	11 weeks 3 days	NCM	No	No	No	No	O Positive	12.2	36.6	4.19	87.35	29.12	32.20	20.85	Normal Blood Picture	97.2	2.8	0	0	Normal Hb Variant	
107	55	19	Primigravida	12 weeks 2 days	NCM	No	No	No	No	O Positive	9.6	28.8	3.92	73.47	24.49	30.10	18.74	Microcytic Hypochromic Anemia	97.3	2.1	0.6	0	Normal Hb Variant	
109	56	24	G2P1L1	14 weeks 4 days	NCM	No	No	No	No	O Positive	10.5	31.5	4.5	70.00	23.33	30.60	15.56	Microcytic Hypochromic Anemia	97.1	2.5	0.4	0	Normal Hb Variant	

Antenatal Screening of Hemoglobinopathies in a Tertiary Care Centre - One Year Descriptive Observational Study

112	57	31	G3P1L1A1	15 weeks 2 days	NCM	No	No	No	No	O Positive	8.1	24.3	4.69	51.81	17.27	30.40	11.05	Microcytic Hypochromic Anemia	96.9	2.5	0.6	0	Normal Hb Variant		
113	58	32	G4P1L1A2	17 weeks 3 days	NCM	No	No	No	No	O Positive	9.7	29.1	4.46	65.25	21.75	31.20	14.63	Normocytic Normochromic Anemia	96.4	2.9	0.4	0	Normal Hb Variant		
114	59	30	G2P1L1	15 weeks 5 days	NCM	No	No	No	No	O Positive	9.4	28.5	3.08	92.50	30.80	33.30	30.03	Normocytic Normochromic Anemia	96.3	2.9	0.8	0	Normal Hb Variant		
118	60	33	G3P2L2	11 weeks 6 days	2*CM	No	No	No	No	O Positive	12.1	36.3	4.6	78.91	26.30	30.10	17.16	Normal Blood Picture	96.8	2.6	0.6	0	Normal Hb Variant		
120	61	35	G3P2L1	9 weeks 2 days	NCM	No	No	No	No	O Positive	13.5	40.5	4.97	81.49	27.16	34.40	16.40	Normal Blood Picture	96.3	2.9	0.8	0	Normal Hb Variant		
122	62	30	G2P1L1	8 weeks 4 days	NCM	No	No	No	No	O Positive	12.9	38.7	4.7	82.34	27.45	29.90	17.52	Normal Blood Picture	96.4	2.7	0.9	0	Normal Hb Variant		
125	63	25	Primigravid a	10 weeks 4 days	NCM	No	No	No	No	O Positive	11.7	35.1	4.6	76.30	25.43	30.40	16.59	Normal Blood Picture	96.4	2.8	0.8	0	Normal Hb Variant		
127	64	20	Primigravid a	11 weeks 1 day	NCM	No	No	No	No	O Positive	11.8	35.4	4.19	84.49	28.16	35.20	20.16	Normal Blood Picture	96	3.1	0.9	0	Normal Hb Pattern		
130	65	30	G3P1L1A1	9 weeks 6 days	NCM	No	No	No	No	O Positive	8.4	25.2	4.69	53.73	17.91	30.20	11.46	Microcytic Hypochromic Anemia	94.9	2.7	2.4	0	Normal Hb Variant		
135	66	26	G3P2L1D1	13 weeks 3 days	2*CM	No	Thalasse mia minor	Thalassemi a Minor	PI-Thal Major / P2- Thal Minor	O Positive	9.2	31.7	4.3	73.70	21.30	28.90	17.14	Microcytic Hypochromic Anemia				0	K/C/O	K/C/O	Thalasse mia Minor
137	67	29	G2P1L1	19 weeks 4 days	NCM	No	No	No	No	O Positive	11.5	34.5	3.79	91.03	30.34	33.33	24.02	Normal Blood Picture	96.9	2.3	0.8	0	Normal Hb Variant		
140	68	45	Primigravid a	18 weeks 5 days	NCM	No	No	No	No	O Positive	7.3	21.9	2.64	82.95	27.65	30.20	31.42	Microcytic Hypochromic Anemia	96.6	2.7	0.7	0	Normal Hb Variant		
142	69	33	G3P2L2	13 weeks 4 days	3*CM	No	No	No	No	O Positive	12.3	36.9	3.7	99.73	33.24	33.20	26.95	Normal Blood Picture	96.8	2.8	0.4	0	Normal Hb Variant		
145	70	26	G2P1L1	12 weeks 2 days	NCM	No	No	No	No	O Negative	12.7	41.1	4.56	90.10	28.70	31.90	19.76	Normal Blood Picture	96.8	2.4	0.8	0	Normal Hb Variant		
149	71	25	G2A1	17 weeks 3 days	NCM	No	No	No	No	B Positive	12.1	40.4	4.81	84.10	26.50	31.50	17.48	Normal Blood Picture	96.4	2.9	0.7	0	Normal Hb Variant		
151	72	23	Primigravid a	16 weeks 4 days	NCM	No	No	No	No	O Positive	12.8	38.2	4.55	83.90	27.90	32.10	18.44	Normal Blood Picture	96.9	2.7	0.4	0	Normal Hb Variant		
153	73	25	G2P1L1	10 weeks 5 days	NCM	No	No	No	No	B Positive	8.3	32.5	4.62	70.50	19.20	27.20	15.26	Microcytic Hypochromic Anemia	96.9	2.3	0.8	0	Normal Hb Variant		
155	74	33	G3P2L1	12 weeks 3 days	NCM	No	No	No	No	A Positive	10.1	38.4	3.67	104.40	31.10	29.80	28.45	Macrocytic Hypochromic Anemia	96.6	2.7	0.7	0	Normal Hb Variant		
156	75	25	G2P1L1	15 weeks 3 days	2*CM	No	No	No	No	A Negative	9.5	40.3	3.72	108.40	32.40	29.80	29.14	Macrocytic Hypochromic Anemia	96.8	2.8	0.4	0	Normal Hb Variant		
162	76	28	G3A2	17 weeks 2 days	NCM	No	No	No	No	B Positive	12.3	36.9	4.6	80.22	26.74	29.50	17.44	Normal Blood Picture	96.8	2.4	0.8	0	Normal Hb Variant		

Antenatal Screening of Hemoglobinopathies in a Tertiary Care Centre - One Year Descriptive Observational Study

164	77	26	G2P1L1	11 weeks 4 days	NCM	No	No	No	No	A Positive	12.5	37.5	4.6	81.52	27.17	31.20	17.72	Normal Blood Picture	97.4	1.8	0.8	0	Normal Hb Variant		
166	78	22	Primigravida	14 weeks 4 days	NCM	No	No	No	No	A Positive	12.4	37.2	4.6	80.87	26.96	29.40	17.58	Normal Blood Picture	96.8	2.6	0.6	0	Normal Hb Variant		
167	79	22	Primigravida	8 weeks 2 days	NCM	No	No	No	No	B Positive	8.3	24.9	4.69	53.09	17.70	30.40	11.32	Microcytic Hypochromic Anemia	96.3	2.9	0.8	0	Normal Hb Variant		
168	80	20	Primigravida	9 weeks 5 days	NCM	No	No	No	No	O positive	8.8	26.4	4.55	58.02	19.34	29.10	12.75	Microcytic Hypochromic Anemia	96.4	2.7	0.9	0	Normal Hb Variant		
169	81	29	G2P1L1	10 weeks 6 days	NCM	No	No	No	No	O positive	12.5	37.5	4.19	89.50	29.83	29.90	21.36	Normal Blood Picture	96.4	2.8	0.8	0	Normal Hb Variant		
170	82	20	Primigravida	11 weeks 2 days	NCM	No	No	No	No	B Negative	12.2	36.6	4.5	81.33	27.11	29.60	18.07	Normocytic Normochromic Anemia	96	3.1	0.9	0	Normal Hb Pattern		
171	83	19	Primigravida	18 weeks 3 days	NCM	No	No	No	No	O positive	9.4	28.2	3.79	74.41	24.80	33.60	19.63	Normocytic Normochromic Anemia	96.8	2.4	0.8	0	Normal Hb Variant		
172	84	24	G2P1L1	16 weeks 5 days	NCM	No	No	No	No	AB Positive	9.1	27.3	4.18	65.31	21.77	34.10	15.62	Microcytic Hypochromic Anemia	95.1	4.1	0.8	0	Thalassemia Minor	Not done	
174	85	25	G2A1	15 weeks 2 days	NCM	No	No	No	No	A Positive	9.8	28.2	4.65	76.80	21.30	30.20	16.52	Microcytic Hypochromic Anemia	96.8	2.6	0.6	0	Normal Hb Variant		
175	86	28	G2P1L1	12 weeks 4 days	NCM	No	No	No	No	O Positive	14.1	42.3	4.35	97.24	32.41	32.10	22.35	Normal Blood Picture	96.3	2.9	0.8	0	Normal Hb Variant		
177	87	28	G3P1L1A1	10 weeks 2 days	NCM	No	No	No	No	O Positive	9.9	31.9	4.8	66.50	20.50	30.80	13.85	Microcytic Hypochromic Anemia	96.4	2.7	0.9	0	Normal Hb Variant		
178	88	24	G2A1	13 weeks 5 days	NCM	No	No	No	No	O Positive	13.1	39.3	4.44	88.51	29.50	33.20	19.94	Normal Blood Picture	96.4	2.8	0.8	0	Normal Hb Variant		
179	89	18	Primigravida	11 weeks	NCM	No	No	No	No	O Positive	6.6	19.8	3.59	55.15	18.38	28.20	15.36	Microcytic Hypochromic Anemia	96	3.1	0.9	0	Normal Hb Pattern		
181	90	23	Primigravida	13 weeks 1 day	NCM	No	No	No	No	O Positive	11.3	33.5	4.32	89.90	34.40	38.20	20.81	Normal Blood Picture	96	3.1	0.9	0	Normal Hb Pattern		
182	91	27	G3P1L1A1	15 weeks 2 days	NCM	No	No	No	No	O Positive	10.1	30.3	3.53	85.84	28.61	33.00	24.32	Microcytic Hypochromic Anemia	96.8	2.4	0.8	0	Normal Hb Variant		
183	92	20	Primigravida	13 weeks 3 days	3*CM	No	No	No	No	O Positive	12.5	37.5	3.91	95.91	31.97	33.40	24.53	Normal Blood Picture	97.4	1.8	0.8	0	Normal Hb Variant		
184	93	29	G2P1L1	16 weeks 3 days	NCM	No	No	No	No	O Positive	11.4	34.2	4.31	79.35	26.45	32.00	18.41	Normal Blood Picture	96.8	2.6	0.6	0	Normal Hb Variant		
185	94	33	G3P1L1A1	17 weeks 1 day	NCM	No	No	No	No	O Positive	11.7	35.1	3.71	94.61	31.54	33.60	25.50	Normal Blood Picture	96.3	2.9	0.8	0	Normal Hb Variant		
188	95	21	Primigravida	12 weeks	NCM	No	No	No	No	O Positive	12.3	36.9	4.19	88.07	29.36	32.90	21.02	Normal Blood Picture	96.4	2.7	0.9	0	Normal Hb Variant		
190	96	30	G2P1L1	14 weeks 3 days	NCM	No	No	No	No	O Positive	10.4	31.2	3.98	78.39	26.13	32.60	19.70	Normocytic Normochromic Anemia	96.4	2.8	0.8	0	Normal Hb Variant		

Antenatal Screening of Hemoglobinopathies in a Tertiary Care Centre - One Year Descriptive Observational Study

192	97	23	Primigravida	19 weeks 2 days	NCM	No	No	No	No	O Positive	9.1	32.1	4.99	64.20	21.30	27.80	12.87	Microcytic Hypochromic Anemia	95.4	3.9	0.7	0	Thalassemia Minor	Normal	
195	98	33	G2P1L1	11 weeks 1 day	NCM	No	No	No	No	O Positive	11.7	35.1	3.88	90.46	30.15	32.40	23.32	Normocytic Normochromic Anemia	96.8	2.4	0.8	0	Normal Hb Variant		
197	99	27	G2A1	10 weeks 4 days	NCM	No	No	No	No	O Positive	12.7	38.1	4.19	90.93	30.31	32.20	21.70	Normal Blood Picture	97.4	1.8	0.8	0	Normal Hb Variant		
199	100	22	Primigravida	9 weeks 3 days	NCM	No	No	No	No	O Positive	9.7	36	3.92	91.84	24.74	30.10	23.43	Macrocytic Hypochromic Anemia	96.8	2.6	0.6	0	Normal Hb Variant		
200	101	26	G2P1L1	10 weeks 4 days	2*CM	No	Thalassemia minor	Thalassemia Minor	Thalassemia Major	O Positive	9.8	37.4	4.96	64.70	24.00	32.00	13.04	Microcytic Hypochromic Anemia				0	K/C/O	K/C/O	Normal
202	102	24	G2P1L1	7 weeks 1 day	NCM	No	No	No	No	O Positive	10.5	31.5	4.5	70.00	23.33	30.60	15.56	Normocytic Normochromic Anemia	96.4	2.7	0.9	0	Normal Hb Variant		
203	103	27	G2P1L1	11 weeks 5 days	NCM	No	No	No	No	O Positive	8.4	25.2	4.69	53.73	17.91	30.40	11.46	Microcytic Hypochromic Anemia	97.2	2.8	0	0	Normal Hb Variant		
205	104	25	Primigravida	16 weeks 2 days	NCM	No	No	No	No	B Positive	10.6	33.5	3.5	95.70	30.90	32.30	27.34	Microcytic Hypochromic Anemia	96.2	3.1	0.7	0	Normal Hb Variant		
207	105	21	Primigravida	10 weeks 2 days	NCM	No	No	No	No	B Positive	12.4	36.6	4.5	81.33	27.56	29.60	18.07	Normal Blood Picture	96.3	3.1	0.6	0	Normal Hb Variant		
209	106	31	G2P1L1	14 weeks 5 days	NCM	No	No	No	No	O Positive	11.7	38	4.08	93.20	29.10	31.20	22.84	Normal Blood Picture	96.3	3.1	0.6	0	Normal Hb Variant		
211	107	27	G2A1	17 weeks 3 days	NCM	No	No	No	No	O Positive	12.4	36.6	4.5	81.33	27.56	29.60	18.07	Normal Blood Picture	96.4	2.9	0.4	0	Normal Hb Variant		
215	108	26	G2P1L1	16 weeks 4 days	2*CM	No	No	No	No	O Positive	10.8	31.3	3.51	89.30	30.80	34.50	25.44	Normocytic Normochromic Anemia	96.9	3.1	0	0	Normal Hb Variant		
216	109	36	Primigravida	12 weeks 5 days	NCM	No	No	No	No	O Negative	12.8	38	4.2	90.50	30.30	33.50	21.55	Normal Blood Picture	96.9	3.1	0	0	Normal Hb Variant		
219	110	25	Primigravida	10 weeks 4 days	NCM	No	No	No	No	A Positive	8.9	28.5	4.99	64.8	26	31.4	12.99	Microcytic Hypochromic Anemia	51.6	2.9	1.7	S - 3%	Sickle Cell Trait	Normal	
221	111	21	Primigravida	9 weeks 3 days	NCM	No	No	No	No	O Positive	12.6	40	4.33	92.40	29.20	31.60	21.34	Normal Blood Picture	97.2	2.8	0	0	Normal Hb Variant		
225	112	24	G2P1L1	12 weeks 2 days	NCM	No	No	No	No	B Positive	7.6	29	3.52	82.40	21.70	26.30	23.41	Microcytic Hypochromic Anemia	97.3	2.1	0.6	0	Normal Hb Variant		
227	113	23	Primigravida	14 weeks 5 days	NCM	No	No	No	No	A Positive	10.6	37.4	4.24	88.10	25.10	28.50	20.78	Normocytic Normochromic Anemia	97.1	2.5	0.4	0	Normal Hb Variant		
230	114	33	G2P1L1	11 weeks 1 day	NCM	No	No	No	No	A Negative	10.2	34.4	3.84	89.40	26.40	29.60	23.28	Microcytic Hypochromic Anemia	96.9	2.5	0.6	0	Normal Hb Variant		
232	115	28	G2A1	10 weeks	NCM	No	No	No	No	B Positive	11.6	35.6	3.92	90.80	29.70	32.70	23.16	Normal Blood Picture	96.4	2.9	0.4	0	Normal Hb Variant		
235	116	22	Primigravida	15 weeks 2 days	NCM	No	No	No	No	B Positive	10.8	36	3.77	95.50	28.70	30.10	25.33	Normocytic Normochromic Anemia	96.3	2.9	0.8	0	Normal Hb Variant		

Antenatal Screening of Hemoglobinopathies in a Tertiary Care Centre - One Year Descriptive Observational Study

238	117	25	Primigravida	13 weeks 1 day	3*CM	No	No	No	No	O Positive	9.8	29.4	4.46	65.92	21.97	31.20	14.78	Normocytic Normochromic Anemia	96.8	2.6	0.6	0	Normal Hb Variant		
241	118	34	G2P1L1	15 weeks 4 days	NCM	No	No	No	No	B Positive	9.5	28.5	3.08	92.50	30.80	33.30	30.03	Normocytic Normochromic Anemia	96.3	2.9	0.8	0	Normal Hb Variant		
244	119	26	G2P1L1	11 weeks 2 days	NCM	No	No	No	No	A Positive	12.2	36.6	4.6	79.57	26.52	30.10	17.30	Normal Blood Picture	96.4	2.7	0.9	0	Normal Hb Variant		
246	120	29	G2P1L1	13 weeks	NCM	No	No	No	No	A Negative	13.6	40.8	4.97	82.09	27.36	34.40	16.52	Normal Blood Picture	96.4	2.8	0.8	0	Normal Hb Variant		
247	121	24	Primigravida	18 weeks 3 days	NCM	No	No	No	No	B Positive	13.1	39.3	4.7	83.62	27.87	29.90	17.79	Normal Blood Picture	96	3.1	0.9	0	Normal Hb Pattern		
250	122	27	G2P1L1	12 weeks 3 days	3*CM	No	Thalassaemia minor	Thalassaemia Minor	Thalassaemia Major	B Positive	9.9	38.1	3.89	79.00	24.00	27.30	20.31	Microcytic Hypochromic Anemia				0	K/C/O	K/C/O	Normal
252	123	42	G4P1L1A2	14 weeks 3 days	NCM	No	No	No	No	A Positive	10.4	36.1	3.8	88.20	26.10	28.70	23.21	Microcytic Hypochromic Anemia	97	2.6	0.7	0	Normal Hb Variant		
255	124	19	Primigravida	11 weeks 4 days	NCM	No	No	No	No	A Positive	9.1	27.3	4.69	58.21	19.40	30.20	12.41	Microcytic Hypochromic Anemia	96	3.1	0.9	0	Normal Hb Variant		
258	125	20	Primigravida	9 weeks 2 days	NCM	No	No	No	No	B Positive	11.7	40.4	4.18	96.80	28.10	29.00	23.16	Normal Blood Picture	96.4	2.9	0.7	0	Normal Hb Variant		
257	126	26	G2P1L1	18 weeks 3 days	NCM	No	No	No	No	AB Positive	9.6	34.4	4.33	79.50	22.20	27.90	18.36	Microcytic Hypochromic Anemia	96.8	2.6	0.6	0	Normal Hb Variant		
259	127	34	G2A1	15 weeks 5 days	2*CM	No	No	No	No	B Positive	12.3	36.9	4.6	80.22	26.74	30.40	17.44	Microcytic Hypochromic Anemia	96.3	2.9	0.8	0	Normal Hb Variant		
262	128	25	Primigravida	16 weeks 4 days	NCM	No	No	No	No	O Positive	12.1	36.3	4.19	86.63	28.88	35.20	20.68	Normocytic Normochromic Anemia	96.4	2.7	0.9	0	Normal Hb Variant		
265	129	28	Primigravida	19 weeks	NCM	No	No	No	No	B Positive	8.6	25.8	4.69	55.01	18.34	30.20	11.73	Microcytic Hypochromic Anemia	96.4	2.8	0.8	0	Normal Hb Variant		
270	130	25	G2P1L1	18 weeks 3 days	NCM	No	No	No	No	A Positive	12.7	38.1	4.6	82.83	27.61	29.50	18.01	Normal Blood Picture	96	3.1	0.9	0	Normal Hb Pattern		
272	131	20	Primigravida	13 weeks 3 days	NCM	No	No	No	No	A Negative	12.4	37.2	4.6	80.87	26.96	31.20	17.58	Normal Blood Picture	96.3	2.9	0.8	0	Normal Hb Variant		
273	132	23	Primigravida	14 weeks 4 days	NCM	No	No	No	No	A Positive	6.3	20.7	3.16	80.20	24.50	26.90	25.38	Microcytic Hypochromic Anemia	97.2	2.5	0.3	0	Normal Hb Variant		
277	133	23	Primigravida	16 weeks 1 day	NCM	No	No	No	No	O positive	8.9	30.2	4.98	64.30	24.23	26.31	12.91	Microcytic Hypochromic Anemia	94	4.8	1.2	0	Thalassaemia Minor	Normal	
281	134	29	G2A1	12 weeks 3 days	NCM	No	No	No	No	B Positive	12.6	37.8	4.6	82.17	27.39	29.40	17.86	Normal Blood Picture	96.4	2.7	0.9	0	Normal Hb Variant		
283	135	18	Primigravida	14 weeks 1 day	NCM	No	No	No	No	O Positive	8.1	24.3	4.69	51.81	17.27	30.40	11.05	Microcytic Hypochromic Anemia	96.4	2.8	0.8	0	Normal Hb Variant		
285	136	20	Primigravida	17 weeks 3 days	NCM	No	No	No	No	B Positive	8.3	24.9	4.55	54.73	18.24	29.10	12.03	Microcytic Hypochromic Anemia	96	3.1	0.9	0	Normal Hb Variant		

Antenatal Screening of Hemoglobinopathies in a Tertiary Care Centre - One Year Descriptive Observational Study

287	137	24	G2A1	18 weeks 3 days	NCM	No	No	No	No	A Positive	12.2	36.6	4.19	87.35	29.12	29.90	20.85	Normal Blood Picture	97	2.4	0.8	0	Normal Hb Variant		
291	138	25	Primigravida	8 weeks 4 days	NCM	No	No	No	No	A Positive	12.6	37.7	4.24	88.90	29.70	33.40	20.97	Normal Blood Picture	96.8	2.8	0.4	0	Normal Hb Variant		
294	139	20	Primigravida	10 weeks 5 days	NCM	No	No	No	No	B Positive	8.9	27	4.34	58.80	19.34	33.40	13.55	Microcytic Hypochromic Anemia	97	2.7	0.8	0	Normal Hb Variant		
295	140	33	G2P1L1	12 weeks 2 days	NCM	No	No	No	No	B Positive	12.8	41.1	4.64	88.60	27.60	31.10	19.09	Normal Blood Picture	97	2.4	0.8	0	Normal Hb Variant		
297	141	31	G3P1L1A1	10 weeks 3 days	NCM	No	No	No	No	O Positive	9.7	29.1	3.79	76.78	25.59	33.60	20.26	Normocytic Normochromic Anemia	96.5	2.9	0.8	0	Normal Hb Variant		
300	142	31	G2P1L1	9 weeks	NCM	No	No	No	No	B Positive	9.4	28.2	4.18	67.46	22.49	34.10	16.14	Microcytic Hypochromic Anemia	96	3.1	0.9	0	Normal Hb Variant		
303	143	26	Primigravida	13 weeks 3 days	2*CM	No	No	No	No	A Positive	9.6	30.4	4.98	64.20	24.56	36.23	12.89	Microcytic Hypochromic Anemia	94.1	5.2	0.7	0	Thalassemia Minor	Thalassemia N	Loss to follow up
305	144	22	Primigravida	17 weeks 4 days	NCM	No	No	No	No	O Positive	8.6	25.8	4.69	55.01	18.34	30.20	11.73	Microcytic Hypochromic Anemia	97.4	1.8	0.8	0	Normal Hb Variant		
307	145	19	Primigravida	12 weeks 5 days	NCM	No	No	No	No	B Positive	12.7	38.1	4.6	82.83	27.61	29.50	18.01	Normal Blood Picture	97.5	1.7	0.8	0	Normal Hb Variant		
310	146	25	G2P1L1	11 weeks 3 days	NCM	No	No	No	No	O Positive	14.1	42.6	4.73	90.10	29.70	33.00	19.05	Normal Blood Picture	96.1	3.1	0.8	0	Normal Hb Variant		
312	147	28	G2P1L1	10 weeks	NCM	No	No	No	No	AB Positive	12.4	39.5	3.99	99.00	31.10	31.40	24.81	Normal Blood Picture	96.8	2.4	0.8	0	Normal Hb Variant		
316	148	22	Primigravida	12 weeks 1 day	NCM	No	No	No	No	O Positive	8.1	24.3	4.69	51.81	17.27	30.40	11.05	Microcytic Hypochromic Anemia	96.7	2.7	0.6	0	Normal Hb Variant		
319	149	25	G2P1L1	14 weeks 2 days	1*CM	No	No	No	No	B Positive	8.3	24.9	4.55	54.73	18.24	29.10	12.03	Microcytic Hypochromic Anemia	97	2.3	0.8	0	Normal Hb Variant		
321	150	23	G2P1L1	16 weeks 3 days	NCM	No	No	No	No	A Positive	8.6	25.8	3.81	88.60	28.70	31.20	23.25	Microcytic Hypochromic Anemia	96.8	2.6	0.6	0	Normal Hb Variant		
325	151	26	G2P1L1	16 weeks 2 days	1*CM	No	Yes	No		O Positive	8.6	25.8	3.81	88.60	28.70	31.20	23.25	Microcytic Hypochromic Anemia	96.8	2.6	0.6	0	Normal Hb Variant		
327	152	23	Primigravida	15 weeks 4 days	1*CM	No	No	No	No	AB positive	13.1	39.3	4.44	88.51	29.50	33.20	19.94	Normal Blood Picture	97.8	1.4	0.8	0	Normal Hb Variant		
329	153	22	G3P1L1A1	19 weeks 3 days	NCM	No	No	No	No	O Positive	6.6	19.8	3.59	55.15	18.38	28.20	15.36	Microcytic Hypochromic Anemia	97.1	2.1	0.8	0	Normal Hb Variant		
332	154	27	G2P1L1	19 weeks 5 days	NCM	No	No	No	No	B Positive	10.4	31.2	3.53	88.39	29.46	33.00	25.04	Normocytic Normochromic Anemia	96.8	2.4	0.8	0	Normal Hb Variant		
333	155	26	Primigravida	14 weeks 6 days	3*CM	No	No	No	No	A Positive	8.5	25.2	4.98	64.60	29.40	34.20	12.97	Macrocytic Hypochromic Anemia	94	5.2	1.3	0	Thalassemia Minor	Normal	
335	156	21	Primigravida	9 weeks 2 days	NCM	No	No	No	No	B Positive	10.3	33.5	3.75	89.20	27.40	30.70	23.79	Normocytic Normochromic	97	2.6	0.8	0	Normal Hb Pattern		

Antenatal Screening of Hemoglobinopathies in a Tertiary Care Centre - One Year Descriptive Observational Study

336	157	34	G3P1L1A1	11 weeks 2 days	NCM	No	No	No	No	AB Positive	11.4	34.4	3.52	97.70	32.40	33.10	27.76	Normocytic Normochromic	96.8	2.4	0.8	0	Normal Hb Variant		
339	158	23	Primigravida	9 weeks 4 days	NCM	No	No	No	No	O Positive	11.2	36.3	3.98	91.30	28.00	30.70	22.94	Normal Blood Picture	97.4	1.8	0.8	0	Normal Hb Variant		
340	159	30	G2P1L1	12 weeks 2 days	NCM	No	No	No	No	B Negative	12.5	40.9	3.98	101.10	30.90	31.10	25.40	Normal Blood Picture	96.8	2.6	0.6	0	Normal Hb Variant		
344	160	29	G3P1L1A1	16 weeks 4 days	NCM	No	No	No	No	O Positive	12	39.4	4.27	92.30	28.10	30.50	21.62	Normal Blood Picture	96.3	2.9	0.8	0	Normal Hb Variant		
346	161	30	G3P2L2	14 weeks 2 days	NCM	No	No	No	No	B Positive	10.1	32.9	3.11	105.70	32.60	30.80	33.99	Normocytic Normochromic	96.4	2.7	0.9	0	Normal Hb Variant		
348	162	23	G2P1L1	12 weeks 4 days	NCM	No	No	No	No	AB Positive	11.2	37.6	3.69	101.70	30.20	29.70	27.56	Normal Blood Picture	96.4	2.8	0.8	0	Normal Hb Variant		
350	163	22	Primigravida	16 weeks 2 days	NCM	No	No	No	No	B Positive	10.5	38.3	4.53	84.60	23.10	27.30	18.68	Microcytic Hypochromic	96	3.1	0.9	0	Normal Hb Pattern		
351	164	22	G2P1L1	15 weeks 3 days	NCM	No	No	No	No	A Positive	12	39.4	4.27	92.30	28.10	30.50	21.62	Normal Blood Picture	96.4	2.8	0.8	0	Normal Hb Pattern		
355	165	20	Primigravida	18 weeks 2 days	NCM	No	No	No	No	B Positive	11.3	38.3	4.61	83.20	24.40	29.40	18.05	Normocytic Hypochromic	96	3.1	0.9	0	Normal Hb Pattern		
357	166	24	Primigravida	14 weeks 4 days	NCM	No	No	No	No	B Positive	10.7	36.1	3.66	98.60	29.20	29.60	26.94	Normocytic Normochromic	96.2	2.9	0.9	0	Normal Hb Pattern		
359	167	32	G2P1L1	12 weeks 6 days	NCM	No	No	No	No	A Negative	12.4	39.3	3.96	99.20	31.40	31.60	25.05	Normal Blood Picture	96.8	2.4	0.8	0	Normal Hb Pattern		
361	168	27	G2P1D1	18 weeks 3 days	2*CM	No	Thalassaemia minor	Thalassaemia Minor	Thalassaemia Major	B Positive	9.2	29.2	3.45	88.20	29.70	30.20	25.57	Microcytic Hypochromic				0	K/C/O	K/C/O	Thalassaemia minor
362	169	26	Primigravida	12 weeks 5 days	NCM	No	No	No	No	O Positive	11.5	34.5	3.79	91.03	30.34	33.33	24.02	Normal Blood Picture	96.8	2.4	0.8	0	Normal Hb Variant		
364	170	30	G2P1L1	9 weeks 5 days	NCM	No	No	No	No	O Positive	7.5	22.5	2.64	85.23	28.41	30.20	32.28	Microcytic Hypochromic Anemia	97.4	1.8	0.8	0	Normal Hb Variant		
365	171	27	G2A1	16 weeks 3 days	NCM	No	No	No	No	O Negative	12.3	36.9	3.7	99.73	33.24	33.20	26.95	Normal Blood Picture	96.8	2.6	0.6	0	Normal Hb Variant		
367	172	21	Primigravida	11 weeks 5 days	NCM	No	No	No	No	B Positive	13.5	40.5	4.35	93.10	31.03	32.10	21.40	Normal Blood Picture	96.3	2.9	0.8	0	Normal Hb Variant		
368	173	19	Primigravida	17 weeks 2 days	NCM	No	No	No	No	O Positive	9.8	31.9	4.8	66.50	20.50	30.80	13.85	Microcytic Hypochromic Anemia	96.4	2.7	0.9	0	Normal Hb Variant		
370	174	20	Primigravida	13 weeks 5 days	NCM	No	No	No	No	B Positive	13.1	39.3	4.44	88.51	29.50	33.20	19.94	Normal Blood Picture	96.4	2.8	0.8	0	Normal Hb Variant		
372	175	29	Primigravida	weeks 4 da	NCM	No	No	No	No	B Positive	9.8	32.3	5	64.6	19.6	30.3	12.92	Microcytic Hypochromic Anemia	94.6	3.8	1.6	0	Thalassaemia Minor	Not done	
373	176	24	G2P1L1	15 weeks 5 days	NCM	No	No	No	No	A Negative	10.4	31.2	3.53	88.39	29.46	33.00	25.04	Normocytic Normochromic Anemia	96.2	2.9	0.9	0	Normal Hb Variant		

Antenatal Screening of Hemoglobinopathies in a Tertiary Care Centre - One Year Descriptive Observational Study

374	177	26	Primigravida	16 weeks 3 days	NCM	No	No	No	No	B Positive	12.5	37.5	3.91	95.91	31.97	33.40	24.53	Normal Blood Picture	96.8	2.4	0.8	0	Normal Hb Variant		
377	178	27	G3P1L1A1	12 weeks 3 days	NCM	No	No	No	No	B Positive	11.5	34.5	4.31	80.05	26.68	32.00	18.57	Normal Blood Picture	96.8	2.2	1	0	Normal Hb Variant		
378	179	26	Primigravida	18 weeks 5 days	NCM	No	No	No	No	B Positive	11.9	35.7	3.71	96.23	32.08	33.60	25.94	Normal Blood Picture	96.8	2.4	0.8	0	Normal Hb Variant		
380	180	18	Primigravida	12 weeks 5 days	3*CM	No	No	No	No	O Positive	12.4	37.2	4.19	88.78	29.59	32.90	21.19	Normal Blood Picture	97	2	1	0	Normal Hb Variant		
381	181	24	G2P1L1	18 weeks 4 days	NCM	No	No	No	No	O Positive	11	33	3.98	82.91	27.64	32.60	20.83	Normal Blood Picture	97.2	2	0.8	0	Normal Hb Variant		
384	182	28	G3P1L1A1	12 weeks 3 days	NCM	No	No	No	No	B Positive	11.1	33.3	3.84	86.72	28.91	31.60	22.58	Normal Blood Picture	97.8	1.4	0.8	0	Normal Hb Variant		
385	183	21	Primigravida	20 weeks 4 days	NCM	No	No	No	No	A Positive	11.7	35.1	3.88	90.46	30.15	32.40	23.32	Normocytic Normochromic Anemia	97.1	2.1	0.8	0	Normal Hb Variant		
388	184	26	G2P1L1	11 weeks 3 days	NCM	No	No	No	No	A Negative	12.4	37.2	4.19	88.78	29.59	32.20	21.19	Normal Blood Picture	97	2.2	0.8	0	Normal Hb Variant		
390	185	24	G2P1L1	13 weeks 2 days	NCM	No	No	No	No	B Positive	9.7	29.1	3.92	74.23	24.74	30.10	18.94	Microcytic Hypochromic Anemia	96.4	2.8	0.8	0	Normal Hb Variant		
393	186	29	G2P1L1	17 weeks 2 days	3*CM	No	Thalassaemia minor	Thalassaemia Minor	P1 - Thalassaemia Major	B Positive	10.4	31.2	4.89	63.60	23.11	30.60	13.01	Microcytic Hypochromic Anemia					K/C/O	K/C/O	Normal
395	187	28	G3P1L0A1	17 weeks 2 days	2*CM	2 Pint PCV	Thalassaemia minor	Thalassaemia Minor	P1-Thal Major / P2-Thal Minor	A Positive	10.8	37.2	4.99	64.20	23.10	29.10	12.87	Microcytic Hypochromic Anemia				0	K/C/O	K/C/O	Thalassaemia minor
396	188	21	Primigravida	10 weeks 3 days	NCM	No	No	No	No	O Positive	8.4	25.2	4.69	53.73	17.91	30.40	11.46	Microcytic Hypochromic Anemia	96.8	2.4	0.8	0	Normal Hb Variant		
397	189	21	Primigravida	9 weeks 3 days	NCM	No	No	No	No	O Positive	9.9	29.7	4.46	66.59	22.20	31.20	14.93	Microcytic Hypochromic Anemia	97.4	1.8	0.8	0	Normal Hb Variant		
400	190	25	G2P1L1	11 weeks 5 days	2*CM	No	No	No	No	O Positive	9.4	28.5	3.08	92.50	30.80	33.30	30.03	Microcytic Hypochromic Anemia	96.8	2.6	0.6	0	Normal Hb Variant		
402	191	24	primigravida	13 weeks 3 days	NCM	No	No	No	No	O Negative	12.4	37.2	4.6	80.87	26.96	30.10	17.58	Normal Blood Picture	96.3	2.9	0.8	0	Normal Hb Variant		
404	192	32	G2P1L1	16 weeks 4 days	NCM	No	No	No	No	B Positive	13.2	39.6	4.97	79.68	26.56	34.40	16.03	Normal Blood Picture	96.4	2.7	0.9	0	Normal Hb Variant		
405	193	36	G3P1L1A1	15 weeks 3 days	NCM	No	No	No	No	O Positive	13	39	4.7	82.98	27.66	29.90	17.66	Normal Blood Picture	96.4	2.8	0.8	0	Normal Hb Variant		
407	194	25	G4P2L1	16 weeks 6 days	NCM	No	No	No	No	B Positive	12.2	36.6	4.6	79.57	26.52	30.40	17.30	Normal Blood Picture	96	3.1	0.9	0	Normal Hb Pattern		
408	195	18	Primigravida	18 weeks 3 days	NCM	No	No	No	No	A Positive	12.2	36.6	4.19	87.35	29.12	35.20	20.85	Normal Blood Picture	96.2	2.9	0.9	0	Normal Hb Variant		
410	196	20	Primigravida	12 weeks 5 days	NCM	No	No	No	No	A Negative	8.8	26.4	4.69	56.29	18.76	30.20	12.00	Microcytic Hypochromic Anemia	96.8	2.4	0.8	0	Normal Hb Variant		

Antenatal Screening of Hemoglobinopathies in a Tertiary Care Centre - One Year Descriptive Observational Study

412	197	20	G2A1	19 weeks 3 days	NCM	No	No	No	No	B Positive	12.4	37.2	4.6	80.87	26.96	29.50	17.58	Normal Blood Picture	96.8	2.2	1	0	Normal Hb Variant		
414	198	27	G3A2	13 weeks 6 days	NCM	No	No	No	No	A Positive	12.6	37.8	4.6	82.17	27.39	31.20	17.86	Normal Blood Picture	96.8	2.4	0.8	0	Normal Hb Variant		
416	199	22	Primigravida	21 weeks 3 days	NCM	No	No	No	No	B Positive	12.1	36.3	4.6	78.91	26.30	29.40	17.16	Normal Blood Picture	97	2	1	0	Normal Hb Variant		
418	200	28	G2P1L1	26 weeks 2 days	NCM	No	No	No	No	B Positive	8.3	28.1	4.96	63.30	22.20	29.50	12.76	Microcytic Hypochromic Anemia	94.7	4.1	1.2	0	Thalassemia minor	Normal	
420	201	25	G3P1L1A1	9 weeks 4 days	NCM	No	No	No	No	O Positive	8	24	4.69	51.17	17.06	30.40	10.91	Microcytic Hypochromic Anemia	97.8	1.4	0.8	0	Normal Hb Variant		
422	202	22	Primigravida	14 weeks 2 days	NCM	No	No	No	No	O Positive	8.4	25.2	4.55	55.38	18.46	29.10	12.17	Microcytic Hypochromic Anemia	97.1	2.1	0.8	0	Normal Hb Variant		
424	203	26	G3P1L1A1	11 weeks 4 days	NCM	No	No	No	No	O Negative	12.4	37.2	4.19	88.78	29.59	29.90	21.19	Normal Blood Picture	97	2.2	0.8	0	Normal Hb Variant		
426	204	31	G2P1L1	16 weeks 3 days	NCM	No	No	No	No	B Positive	12	36.6	4.5	81.33	26.67	29.60	18.07	Normal Blood Picture	96.3	2.9	0.8	0	Normal Hb Variant		
427	205	29	G3P2L2	13 weeks 4 days	NCM	No	No	No	No	O Positive	11.9	35.7	3.79	94.20	31.40	33.33	24.85	Normal Blood Picture	96.4	2.7	0.9	0	Normal Hb Variant		
428	206	27	G4P2L1	19 weeks 1 day	NCM	No	No	No	No	B Positive	7.5	22.5	2.64	85.23	28.41	30.20	32.28	Microcytic Hypochromic Anemia	96.4	2.8	0.8	0	Normal Hb Variant		
430	207	21	Primigravida	15 weeks 4 days	NCM	No	No	No	No	A Positive	12.1	36.3	3.7	98.11	32.70	33.20	26.52	Normal Blood Picture	96	3.1	0.9	0	Normal Hb Pattern		
432	208	34	G2P1L1	13 weeks 3 days	NCM	No	No	No	No	A Negative	13.5	40.5	4.35	93.10	31.03	32.10	21.40	Normal Blood Picture	96.2	2.9	0.9	0	Normal Hb Variant		
436	209	22	Primigravida	17 weeks 4 days	NCM	No	No	No	No	B Positive	9.8	31.9	4.8	66.50	20.50	30.80	13.85	Microcytic Hypochromic Anemia	96.8	2.4	0.8	0	Normal Hb Variant		
438	210	19	Primigravida	15 weeks 2 days	NCM	No	No	No	No	O Positive	13.1	39.3	4.44	88.51	29.50	33.20	19.94	Normal Blood Picture	96.8	2.2	1	0	Normal Hb Variant		
440	211	25	Primigravida	19 weeks 4 days	NCM	No	No	No	No	O Positive	11.1	36.6	4.93	63.98	24.90	30.30	12.98	Normocytic Hypochromic Anemia	94.4	5.1	0.5	0	Thalassemia Minor	Normal	
442	212	21	G2P1L1	12 weeks 5 days	3*CM	No	No	No	No	O Negative	10.2	30.6	3.53	86.69	28.90	33.00	24.56	Normal Blood Picture	96.4	2.7	0.9	0	Normal Hb Variant		
443	213	25	G3P2L2	11 weeks 4 days	NCM	No	No	No	No	B Positive	11.2	33.6	3.91	85.93	28.64	33.40	21.98	Normal Blood Picture	96.4	2.8	0.8	0	Normal Hb Variant		
445	214	27	G3P1L1A1	12 weeks 2 days	NCM	No	No	No	No	O Positive	11.4	34.2	4.31	79.35	26.45	32.00	18.41	Normocytic Normochromic Anemia	96	3.1	0.9	0	Normal Hb Pattern		
447	215	22	Primigravida	15 weeks 3 days	3*CM	No	No	No	No	B Positive	11.5	34.5	3.71	92.99	31.00	33.60	25.07	Normal Blood Picture	96.2	2.9	0.9	0	Normal Hb Variant		
449	216	29	G2P1L1	17 weeks 5 days	NCM	No	No	No	No	A Positive	12.2	36.6	4.19	87.35	29.12	32.90	20.85	Normal Blood Picture	96.8	2.4	0.8	0	Normal Hb Variant		

Antenatal Screening of Hemoglobinopathies in a Tertiary Care Centre - One Year Descriptive Observational Study

451	217	31	G3P1L1A1	12 weeks 1 day	NCM	No	No	No	No	A Negative	10.5	31.5	3.98	79.15	26.38	32.60	19.89	Normocytic Normochromic Anemia	96.8	2.2	1	0	Normal Hb Variant		
453	218	28	Primigravida	16 weeks 4 days	NCM	No	No	No	No	B Positive	10.1	30.3	3.84	78.91	26.30	31.60	20.55	Normal Blood Picture	96.2	2.9	0.9	0	Normal Hb Variant		
455	219	27	G2A1	15 weeks 3 days	NCM	No	No	No	No	A Positive	8.8	29.2	4.98	63.70	24.20	30.20	12.79	Microcytic Hypochromic Anemia	94.1	5.1	1.3	0	Thalassemia Minor	Normal	
395	220	22	Primigravida	12 weeks 2 days	NCM	No	No	No	No	O Positive	11.7	35.1	3.79	92.61	30.87	33.33	24.44	Normal Blood Picture	96.8	2.4	0.8	0	Normal Hb Variant		
396	221	24	G2P1L1	9 weeks 4 days	NCM	No	No	No	No	O Positive	7.7	23.1	2.64	87.50	29.17	30.20	33.14	Microcytic Hypochromic Anemia	97.4	1.8	0.8	0	Normal Hb Variant		
397	222	25	G2P1L1	14 weeks 4 days	2*CM	No	No	No	No	O Negative	11.2	33.6	3.7	90.81	30.27	33.20	24.54	Normal Blood Picture	96.8	2.6	0.6	0	Normal Hb Variant		
398	223	33	G2P1L1	11 weeks 4 days	NCM	No	No	No	No	B Positive	13.1	39.3	4.35	90.34	30.11	32.10	20.77	Normal Blood Picture	96.3	2.9	0.8	0	Normal Hb Variant		
399	224	27	Primigravida	12 weeks 5 days	NCM	No	No	No	No	O Positive	9.1	31.9	4.8	66.50	20.50	30.80	13.85	Normocytic Normochromic Anemia	96.4	2.7	0.9	0	Normal Hb Variant		
400	225	27	Primigravida	17 weeks 2 days	NCM	No	No	No	No	B Positive	12.8	38.4	4.44	86.49	28.83	33.20	19.48	Normal Blood Picture	96.4	2.8	0.8	0	Normal Hb Variant		
402	226	30	Primigravida	13 weeks 5 days	NCM	No	No	No	No	A Positive	6.2	18.6	3.59	51.81	17.27	28.20	14.43	Normal Blood Picture	96	3.1	0.9	0	Normal Hb Pattern		
403	227	25	G2P1L1	19 weeks 4 days	NCM	No	No	No	No	A Negative	10.1	30.3	3.53	85.84	28.61	33.00	24.32	Normocytic Normochromic Anemia	96.2	2.9	0.9	0	Normal Hb Variant		
404	228	30	Primigravida	14 weeks 5 days	NCM	No	No	No	No	B Positive	12.4	37.2	3.91	95.14	31.71	33.40	24.33	Normal Blood Picture	96.8	2.4	0.8	0	Normal Hb Variant		
405	229	22	Primigravida	19 weeks 1 day	3*CM	No	No	No	No	O Positive	9.2	29.9	4.96	62.90	25.75	32.00	12.68	Microcytic Hypochromic Anemia	94.8	4.2	1	0	Thalassemia minor	Normal	
406	230	31	Primigravida	16 weeks 4 days	3*CM	No	No	No	No	O Positive	11.4	34.2	3.71	92.18	30.73	33.60	24.85	Normal Blood Picture	96.8	2.4	0.8	0	Normal Hb Variant		
407	231	24	G2P1L1	14 weeks 4 days	NCM	No	No	No	No	O Negative	12	36	4.19	85.92	28.64	32.90	20.51	Microcytic Hypochromic	97	2	1	0	Normal Hb Variant		
409	232	33	G3P1L1A1	15 weeks 1 day	NCM	No	No	No	No	B Positive	10.6	31.8	3.98	79.90	26.63	32.60	20.08	Microcytic Hypochromic	97.2	2	0.8	0	Normal Hb Variant		
410	233	27	Primigravida	12 weeks	NCM	No	No	No	No	O Positive	10.3	30.9	3.84	80.47	26.82	31.60	20.96	Microcytic Hypochromic	97.8	1.4	0.8	0	Normal Hb Variant		
412	234	22	G2P1L1	16 weeks 4 days	NCM	No	No	No	No	B Positive	11.2	33.6	3.88	86.60	28.87	32.40	22.32	Microcytic Hypochromic	97.1	2.1	0.8	0	Normal Hb Variant		
413	235	20	Primigravida	11 weeks 1 day	NCM	No	No	No	No	A Positive	12.2	36.6	4.19	87.35	29.12	32.20	20.85	Normal Blood Picture	97	2.2	0.8	0	Normal Hb Variant		
415	236	27	G2A1	20 weeks	NCM	No	No	No	No	A Negative	9.6	28.8	3.92	73.47	24.49	30.10	18.74	Microcytic Hypochromic Anemia	97.4	1.8	0.8	0	Normal Hb Variant		

Antenatal Screening of Hemoglobinopathies in a Tertiary Care Centre - One Year Descriptive Observational Study

416	237	22	G2P1L1	11 weeks 4 days	NCM	No	No	No	No	B Positive	10.1	30.3	4.5	67.33	22.44	30.60	14.96	Normal Blood Picture	96.5	2.6	0.9	0	Normal Hb Variant		
417	238	26	G2P1L1	13 weeks 5 days	NCM	No	No	No	No	O Positive	8.3	24.9	4.69	53.09	17.70	30.40	11.32	Normal Blood Picture	96.9	2.3	0.8	0	Normal Hb Variant		
418	239	24	G2P1L1	12 weeks 1 day	NCM	No	No	No	No	O Positive	9.8	29.4	4.46	65.92	21.97	31.20	14.78	Normocytic Normochromic Anemia	96.4	2.7	0.9	0	Normal Hb Variant		
419	240	24	primigravid a	14 weeks 4 days	NCM	No	No	No	No	O Negative	9.5	28.5	3.08	92.50	30.80	33.30	30.03	Normal Blood Picture	96.4	2.8	0.8	0	Normal Hb Variant		
420	241	24	primigravid a	16 weeks 1 days	NCM	No	No	No	No	B Positive	12.2	36.6	4.6	79.57	26.52	30.10	17.30	Normal Blood Picture	96.1	3	0.9	0	Normal Hb Variant		
421	242	30	G3A2	14 weeks 3 days	NCM	No	No	No	No	O Positive	13.6	40.8	4.97	82.09	27.36	34.40	16.52	Normocytic Normochromic Anemia	96.2	2.9	0.9	0	Normal Hb Variant		
422	243	25	G3P2L2	15 weeks 1 day	NCM	No	No	No	No	B Positive	13.1	39.3	4.7	83.62	27.87	29.90	17.79	Normal Blood Picture	96.7	2.5	0.8	0	Normal Hb Variant		
424	244	26	G2P1L1	16 weeks 4 days	NCM	No	No	No	No	A Positive	12.3	36.9	4.6	80.22	26.74	30.40	17.44	Normal Blood Picture	97.2	2.2	0.6	0	Normal Hb Variant		
425	245	27	Primigravid a	12 weeks 4 days	NCM	No	No	No	No	A Negative	12.1	36.3	4.19	86.63	28.88	35.20	20.68	Normal Blood Picture	96.8	2.4	0.8	0	Normal Hb Variant		
426	246	26	Primigravid a	13 weeks 5 days	NCM	No	No	No	No	B Positive	9.8	32	4.68	68.40	21.00	30.70	14.62	Microcytic Hypochromic	94.3	4.3	1.4	0	Beta Thalassemia Trait	Normal	
427	247	24	Primigravid a	16 weeks 4 days	NCM	No	No	No	No	O Positive	12.7	38.1	4.6	82.83	27.61	29.50	18.01	Microcytic Hypochromic	97.2	2	0.8	0	Normal Hb Variant		
429	248	21	Primigravid a	14 weeks 4 days	NCM	No	No	No	No	O Positive	12.4	37.2	4.6	80.87	26.96	31.20	17.58	Microcytic Hypochromic	97.6	1.6	0.8	0	Normal Hb Variant		
430	249	26	G2P1L1	12 weeks 4 days	NCM	No	No	No	No	O Negative	12.6	37.8	4.6	82.17	27.39	29.40	17.86	Microcytic Hypochromic	97.1	2.1	0.8	0	Normal Hb Variant		
432	250	29	G3P1L1A1	16 weeks 6 days	NCM	No	No	No	No	B Positive	8.1	24.3	4.69	51.81	17.27	30.40	11.05	Normal Blood Picture	96.9	2.4	0.7	0	Normal Hb Variant		
433	251	20	G2P2L1	17 weeks	NCM	No	No	No	No	O Positive	8.3	24.9	4.55	54.73	18.24	29.10	12.03	Microcytic Hypochromic Anemia	97.2	2	0.8	0	Normal Hb Variant		
434	252	29	G3P2L2	15 weeks 6 days	NCM	No	No	No	No	B Positive	12.2	36.6	4.19	87.35	29.12	29.90	20.85	Normal Blood Picture	96.4	2.6	1	0	Normal Hb Variant		
435	253	28	Primigravid a	8 weeks 5 days	NCM	No	No	No	No	A Positive	12.6	36.6	4.5	81.33	28.00	29.60	18.07	Normal Blood Picture	96.3	2.9	0.8	0	Normal Hb Variant		
436	254	20	Primigravid a	16 weeks 3 days	NCM	No	No	No	No	A Negative	11.6	34.8	3.79	91.82	30.61	33.33	24.23	Normocytic Normochromic Anemia	96.5	2.6	0.9	0	Normal Hb Variant		
438	255	28	G3P2L1	13 weeks 4 days	NCM	No	No	No	No	B Positive	7.8	23.4	2.64	88.64	29.55	30.20	33.57	Normal Blood Picture	96.4	2.8	0.8	0	Normal Hb Variant		
439	256	28	G3P2L3	17 weeks 5 days	NCM	No	No	No	No	O Positive	12.1	36.3	3.7	98.11	32.70	33.20	26.52	Normal Blood Picture	96.1	3	0.9	0	Normal Hb Variant		

Antenatal Screening of Hemoglobinopathies in a Tertiary Care Centre - One Year Descriptive Observational Study

440	257	21	G2P1L1	18 weeks 1 days	NCM	No	No	No	No	O Positive	13.5	40.5	4.35	93.10	31.03	32.10	21.40	Normocytic Normochromic Anemia	96.2	2.9	0.9	0	Normal Hb Variant		
441	258	29	G3P2L2	20 weeks	NCM	No	No	No	No	O Negative	9.9	31.9	4.8	66.50	20.50	30.80	13.85	Normal Blood Picture	96.8	2.4	0.8	0	Normal Hb Variant		
442	259	23	Primigravida	weeks 4 da	NCM	No	No	No	No	O Positive	13.7	46.9	5.26	68.1	26	29.2	12.95	Normal Blood Picture	94.1	4.3	1.6	0	Thalassemia Minor	Normal	
443	260	23	G3P2L2	10 weeks 4 days	NCM	No	No	No	No	O Positive	6.2	18.6	3.59	51.81	17.27	28.20	14.43	Normal Blood Picture	96.8	2.4	0.8	0	Normal Hb Variant		
444	261	25	G3P2L2	15 weeks 5 days	NCM	No	No	No	No	B Positive	10.2	30.6	3.53	86.69	28.90	33.00	24.56	Microcytic Hypochromic	97.2	1.8	1	0	Normal Hb Variant		
445	262	32	G4P3L3	11 weeks 4 days	NCM	No	No	No	No	A Positive	12.3	36.9	3.91	94.37	31.46	33.40	24.14	Microcytic Hypochromic	97.2	2	0.8	0	Normal Hb Variant		
446	263	23	G2P1L1	15 weeks 3 days	NCM	No	No	No	No	A Negative	11.2	33.6	4.31	77.96	25.99	32.00	18.09	Microcytic Hypochromic	97.3	1.9	0.8	0	Normal Hb Variant		
447	264	32	G2P1L1	14 weeks 3 days	NCM	No	No	No	No	B Positive	11.8	35.4	3.71	95.42	31.81	33.60	25.72	Microcytic Hypochromic	97.1	2.1	0.8	0	Normal Hb Variant		
449	265	20	Primigravida	16 weeks 5 days	NCM	No	No	No	No	O Positive	12.1	36.3	4.19	86.63	28.88	32.90	20.68	Normal Blood Picture	96.8	2.4	0.8	0	Normal Hb Variant		
450	266	20	G3P2L2	15 weeks 1 day	NCM	No	No	No	No	O Positive	10.5	31.5	3.98	79.15	26.38	32.60	19.89	Microcytic Hypochromic Anemia	97.3	1.9	0.8	0	Normal Hb Variant		
451	267	33	G3P2L2	15 weeks 4 days	NCM	No	No	No	No	O Positive	10.2	30.6	3.84	79.69	26.56	31.60	20.75	Normal Blood Picture	96.4	2.6	1	0	Normal Hb Variant		
452	268	34	Primigravida	15 weeks 6 days	NCM	No	No	No	No	O Negative	11.1	33.3	3.88	85.82	28.61	32.40	22.12	Normal Blood Picture	96.3	2.9	0.8	0	Normal Hb Variant		
453	269	32	G2P1L1	15 weeks 4 days	NCM	No	No	No	No	B Positive	12.1	36.3	4.19	86.63	28.88	32.20	20.68	Normocytic Normochromic Anemia	96.5	2.7	0.8	0	Normal Hb Variant		
454	270	24	G2P2L1	9 weeks 2 days	NCM	No	No	No	No	O Positive	9.7	29.1	3.92	74.23	24.74	30.10	18.94	Normal Blood Picture	96.4	2.8	0.8	0	Normal Hb Variant		
456	271	30	Primigravida	13 weeks 3 days	NCM	No	No	No	No	B Positive	10.5	31.5	4.5	70.00	23.33	30.60	15.56	Normal Blood Picture	96	3.1	0.9	0	Normal Hb Variant		
457	272	21	Primigravida	weeks 3 da	3*CM	No	No	No	No	O Positive	9.1	29.2	4.93	62.5	21.5	31.2	12.68	Microcytic Hypochromic Anemia	93.8	4.8	1.4	0	Thalassemia Minor	Normal	
458	273	27	Primigravida	13 weeks 2 days	NCM	No	No	No	No	A Negative	9.7	29.1	4.46	65.25	21.75	31.20	14.63	Normal Blood Picture	96.8	2.4	0.8	0	Normal Hb Variant		
460	274	22	Primigravida	16 weeks 5 days	NCM	No	No	No	No	B Positive	9.2	28.5	3.08	92.50	30.80	33.30	30.03	Normal Blood Picture	96.5	2.6	0.9	0	Normal Hb Variant		
461	275	19	Primigravida	12 weeks 1 day	NCM	No	No	No	No	O Positive	11.9	35.7	4.6	77.61	25.87	30.10	16.87	Normal Blood Picture	96.8	2.4	0.8	0	Normal Hb Variant		
462	276	35	G3P1L1A1	18 weeks 2 days	NCM	No	No	No	No	O Positive	13.5	40.5	4.97	81.49	27.16	34.40	16.40	Microcytic Hypochromic	97.4	1.8	0.8	0	Normal Hb Variant		

Antenatal Screening of Hemoglobinopathies in a Tertiary Care Centre - One Year Descriptive Observational Study

463	277	35	G3P2L2	9 weeks 4 days	NCM	No	No	No	No	O Positive	13.9	41.7	4.7	88.72	29.57	29.90	18.88	Microcytic Hypochromic	97.1	2.1	0.8	0	Normal Hb Variant		
464	278	25	Primigravida	13 weeks 3 days	3*CM	No	No	No	No	O Negative	12.2	36.6	4.6	79.57	26.52	30.40	17.30	Microcytic Hypochromic	97.8	1.4	0.8	0	Normal Hb Variant		
465	279	22	Primigravida	16 weeks 4 days	NCM	No	No	No	No	B Positive	11.8	35.4	4.19	84.49	28.16	35.20	20.16	Microcytic Hypochromic	96.8	2.4	0.8	0	Normal Hb Variant		
467	280	30	G3P2L2	12 weeks 5 days	NCM	No	No	No	No	O Positive	8.1	24.3	4.69	51.81	17.27	30.20	11.05	Normal Blood Picture	96.8	2.4	0.8	0	Normal Hb Variant		
468	281	43	G3P2L0	15 weeks 5 days	NCM	No	No	No	No	B Positive	12.3	36.9	4.6	80.22	26.74	29.50	17.44	Microcytic Hypochromic Anemia	97.2	2	0.8	0	Normal Hb Variant		
469	282	33	G2P2L1	12 weeks 7 days	NCM	No	No	No	No	A Positive	12.1	36.3	4.6	78.91	26.30	31.20	17.16	Normal Blood Picture	96.7	2.6	0.7	0	Normal Hb Variant		
470	283	30	G2P2L1	12 weeks 6 days	NCM	No	No	No	No	A Negative	12.8	38.4	4.6	83.48	27.83	29.40	18.15	Normal Blood Picture	96.3	2.9	0.8	0	Normal Hb Variant		
472	284	21	Primigravida	16 weeks 4 days	NCM	No	No	No	No	B Positive	8.1	24.3	4.69	51.81	17.27	30.40	11.05	Normocytic Normochromic Anemia	96.3	2.7	1	0	Normal Hb Variant		
473	285	24	G2P1L1	weeks 2 da	2*CM	No	Thalassemia	Thalassemia M	Thalassemia N	A Positive	9.8	33.5	5.13	65.3	20.1	30.7	12.73	Microcytic Hypochromic Anemia	94.1	4.3	1.6	0	K/C/O	K/C/O	Loss to follow up
475	286	31	Primigravida	16 weeks 1 day	NCM	No	No	No	No	O Positive	12.2	36.6	4.19	87.35	29.12	29.90	20.85	Normal Blood Picture	96.4	2.7	0.9	0	Normal Hb Variant		
476	287	24	Primigravida	19 weeks 5 days	NCM	No	No	No	No	O Positive	12.6	36.6	4.5	81.33	28.00	29.60	18.07	Normocytic Normochromic Anemia	96.2	2.9	0.9	0	Normal Hb Variant		
477	288	25	G3P2L2	12 weeks 2 days	NCM	No	No	No	No	O Negative	11.8	35.4	3.79	93.40	31.13	33.33	24.64	Normal Blood Picture	96.8	2.4	0.8	0	Normal Hb Variant		
478	289	23	G2P1L1	13 weeks 4 days	NCM	No	No	No	No	B Positive	7.9	23.7	2.64	89.77	29.92	30.20	34.00	Normal Blood Picture	96.6	2.5	0.9	0	Normal Hb Variant		
479	290	22	G2P1L1	16 weeks 5 days	NCM	No	No	No	No	O Positive	12	36	3.7	97.30	32.43	33.20	26.30	Normal Blood Picture	96.8	2.4	0.8	0	Normal Hb Variant		
481	291	24	Primigravida	13 weeks 6 days	NCM	No	No	No	No	B Positive	13.6	40.8	4.35	93.79	31.26	32.10	21.56	Microcytic Hypochromic	97.5	1.8	0.7	0	Normal Hb Variant		
482	292	34	G2P1L1	12 weeks 4 days	NCM	No	No	No	No	A Positive	9.9	31.9	4.8	66.50	20.50	30.80	13.85	Microcytic Hypochromic	96.7	2.5	0.8	0	Normal Hb Variant		
484	293	21	G2A1	17 weeks 5 days	NCM	No	No	No	No	A Negative	13.2	39.6	4.44	89.19	29.73	33.20	20.09	Microcytic Hypochromic	97.8	1.4	0.8	0	Normal Hb Variant		
485	294	21	Primigravida	15 weeks 4 days	NCM	No	No	No	No	B Positive	6.5	19.5	3.59	54.32	18.11	28.20	15.13	Microcytic Hypochromic	97.1	2.1	0.8	0	Normal Hb Variant		
487	295	25	Primigravida	17 weeks 5 days	NCM	No	No	No	No	O Positive	10.3	30.9	3.53	87.54	29.18	33.00	24.80	Normal Blood Picture	96.8	2.4	0.8	0	Normal Hb Variant		
488	296	19	Primigravida	8 weeks 2 days	NCM	No	No	No	No	O Positive	12.9	38.7	3.91	98.98	32.99	33.40	25.31	Microcytic Hypochromic Anemia	97.3	1.9	0.8	0	Normal Hb Variant		

Antenatal Screening of Hemoglobinopathies in a Tertiary Care Centre - One Year Descriptive Observational Study

490	297	27	Primigravida	15 weeks 1 day	NCM	No	No	No	No	O Positive	11.3	33.9	4.31	78.65	26.22	32.00	18.25	Normal Blood Picture	96.6	2.6	0.8	0	Normal Hb Variant		
491	298	27	Primigravida	14 weeks 3 days	NCM	No	No	No	No	O Negative	11.8	35.4	3.71	95.42	31.81	33.60	25.72	Normal Blood Picture	96.3	2.9	0.8	0	Normal Hb Variant		
492	299	25	Primigravida	17 weeks 5 days	NCM	No	No	No	No	B Positive	12.2	36.6	4.19	87.35	29.12	32.90	20.85	Normocytic Normochromic Anemia	96.5	2.5	1	0	Normal Hb Variant		
493	300	35	G3P2L2	12 weeks 4 days	NCM	No	No	No	No	O Positive	10.9	32.7	3.98	82.16	27.39	32.60	20.64	Normal Blood Picture	96.4	2.8	0.8	0	Normal Hb Variant		
494	301	23	G4P3L1	13 weeks 5 days	NCM	No	No	No	No	B Positive	10.9	32.7	3.84	85.16	28.39	31.60	22.18	Normal Blood Picture	96	3.1	0.9	0	Normal Hb Variant		
495	302	22	G2P1L1	16 weeks 5 days	NCM	No	No	No	No	A Positive	11.8	35.4	3.88	91.24	30.41	32.40	23.51	Normocytic Normochromic Anemia	96.2	2.9	0.9	0	Normal Hb Variant		
497	303	25	G2P1L1	16 weeks 1 day	NCM	No	No	No	No	A Negative	12.5	37.5	4.19	89.50	29.83	32.20	21.36	Normal Blood Picture	96.8	2.4	0.8	0	Normal Hb Variant		
498	304	21	Primigravida	11 weeks 1 day	NCM	No	No	No	No	B Positive	9.8	29.4	3.92	75.00	25.00	30.10	19.13	Normal Blood Picture	97	2.2	0.8	0	Normal Hb Variant		
499	305	29	G2P1L1	17 weeks 5 days	NCM	No	No	No	No	O Positive	10.6	31.8	4.5	70.67	23.56	30.60	15.70	Normal Blood Picture	96.8	2.4	0.8	0	Normal Hb Variant		
500	306	30	G2P1L1	13 weeks 4 days	NCM	No	No	No	No	O Positive	8.3	24.9	4.69	53.09	17.70	30.40	11.32	Microcytic Hypochromic	97.2	1.8	1	0	Normal Hb Variant		
501	307	30	G2P1L1	17 weeks 5 days	NCM	No	No	No	No	O Positive	9.8	29.4	4.46	65.92	21.97	31.20	14.78	Microcytic Hypochromic	97.2	2	0.8	0	Normal Hb Variant		
502	308	27	G2A1	10 weeks 4 days	NCM	No	No	No	No	O Negative	9.5	28.5	3.08	92.50	30.80	33.30	30.03	Microcytic Hypochromic	97.8	1.4	0.8	0	Normal Hb Variant		
503	309	27	G5P3L2A1	12 weeks 4 days	NCM	No	No	No	No	B Positive	12.2	36.6	4.6	79.57	26.52	30.10	17.30	Microcytic Hypochromic	97.1	2.1	0.8	0	Normal Hb Variant		
505	310	24	Primigravida	weeks 4 da	NCM	No	No	No	No	B Negative	9.1	30.9	4.99	62.8	21.4	29.6	12.59	Microcytic Hypochromic Anemia	93.5	5.1	1.4	0	Thalassemia Minor	Normal	
506	311	31	G2P2L1	17 weeks 5 days	NCM	No	No	No	No	B Positive	13.1	39.3	4.7	83.62	27.87	29.90	17.79	Microcytic Hypochromic Anemia	97.4	1.8	0.8	0	Normal Hb Variant		
507	312	23	Primigravida	13 weeks 4 days	NCM	No	No	No	No	A Positive	12.3	36.9	4.6	80.22	26.74	30.40	17.44	Normal Blood Picture	96	2.6	1.4	0	Normal Hb Variant		
508	313	25	Primigravida	18 weeks 1 day	NCM	No	No	No	No	A Negative	12.1	36.3	4.19	86.63	28.88	35.20	20.68	Normal Blood Picture	96.7	2.5	0.8	0	Normal Hb Variant		
509	314	26	Primigravida	19 weeks 4 days	NCM	No	No	No	No	B Positive	8.6	25.8	4.69	55.01	18.34	30.20	11.73	Normocytic Normochromic Anemia	95.5	2.7	1.8	0	Normal Hb Variant		
511	315	23	G3P2L2	13 weeks 3 days	NCM	No	No	No	No	O Positive	12.7	38.1	4.6	82.83	27.61	29.50	18.01	Normal Blood Picture	96.4	2.8	0.8	0	Normal Hb Variant		
512	316	26	Primigravida	15 weeks 6 days	NCM	No	No	No	No	O Positive	12.4	37.2	4.6	80.87	26.96	31.20	17.58	Normal Blood Picture	96.6	2.5	0.9	0	Normal Hb Variant		

Antenatal Screening of Hemoglobinopathies in a Tertiary Care Centre - One Year Descriptive Observational Study

513	317	21	Primigravida	weeks 4 da	3*CM	No	No	No	No	B Positive	10.7	36.1	4.98	64.30	21.50	29.70	12.91	Microcytic Hypochrom	94.2	3.5	2.3	0	Thalassemia Minor	Normal
515	318	26	G2P1L1	14 weeks 3 days	NCM	No	No	No	No	O Negative	8.1	24.3	4.69	51.81	17.27	30.40	11.05	Normal Blood Picture	96.8	2.4	0.8	0	Normal Hb Variant	
517	319	24	G2P1L1	11 weeks 4 days	NCM	No	No	No	No	B Positive	8.3	24.9	4.55	54.73	18.24	29.10	12.03	Normal Blood Picture	96.8	2.2	1	0	Normal Hb Variant	
518	320	25	Primigravida	14 weeks 5 days	NCM	No	No	No	No	O Positive	12.2	36.6	4.19	87.35	29.12	29.90	20.85	Normal Blood Picture	96.8	2.4	0.8	0	Normal Hb Variant	
521	321	29	G2A1	17 weeks 2 days	NCM	No	No	No	No	B Positive	12.6	36.6	4.5	81.33	28.00	29.60	18.07	Microcytic Hypochromic	97.5	1.8	0.7	0	Normal Hb Variant	
523	322	32	G3P1L1A1	19 weeks 5 days	NCM	No	No	No	No	A Positive	10.6	30.9	4.5	82.40	28.10	30.60	18.31	Microcytic Hypochromic	97.2	2	0.8	0	Normal Hb Variant	
525	323	25	Primigravida	12 weeks 6 days	NCM	No	No	No	No	A Negative	8.3	26	4.69	80.60	27.20	30.40	17.19	Microcytic Hypochromic	97.8	1.4	0.8	0	Normal Hb Variant	
526	324	22	G2P1L1	15 weeks 3 days	NCM	No	No	No	No	B Positive	9.8	27.3	4.46	82.60	28.20	31.20	18.52	Microcytic Hypochromic	97.1	2.1	0.8	0	Normal Hb Variant	
528	325	29	G2P1L1	17 weeks 2 days	NCM	No	No	No	No	A Positive	10	27.3	4.46	82.60	28.20	31.20	18.52	Microcytic Hypochromic	96.8	2.4	0.8	0	Normal Hb Variant	
531	326	27	G2P1L1	19 weeks 5 days	NCM	No	No	No	No	B Positive	9.6	27.3	4.46	82.60	28.20	31.20	18.52	Microcytic Hypochromic	97.2	2	0.8	0	Normal Hb Variant	
532	327	25	G3P2L2	18 weeks 1 day	2*CM	No	Thalassaemia minor	Thalassaemia Minor	PT - Thalassaemia Minor	A Positive	9.6	29.4	4.98	63.40	28.40	30.20	12.73	Microcytic Hypochromic				0	K/C/O	K/C/O Normal
534	328	18	Primigravida	12 weeks 2 days	NCM	No	No	No	No	O Positive	12.9	39.7	4.44	90.20	31.20	33.20	20.32	Normal Blood Picture	96.4	2.9	0.7	0	Normal Hb Variant	
536	329	33	Primigravida	15 weeks 4 days	NCM	No	No	No	No	O Positive	6.4	22.5	3.59	78.20	29.20	28.20	21.78	Microcytic Hypochromic Anemia	95.6	2.7	1.7	0	Normal Hb Variant	
538	330	25	Primigravida	17 weeks 5 days	NCM	No	No	No	No	O Positive	10	31.1	3.53	88.30	29.10	33.00	25.01	Normal Blood Picture	96.4	2.8	0.8	0	Normal Hb Variant	
541	331	28	G2P1L1	19 weeks 4 days	NCM	No	No	No	No	O Negative	12.9	38.6	3.91	98.90	33.00	33.40	25.29	Normal Blood Picture	95.9	3.1	1	0	Normal Hb Variant	
543	332	23	Primigravida	14 weeks 3 days	NCM	No	No	No	No	B Positive	11.6	35.5	4.31	82.30	26.30	32.00	19.10	Normocytic Normochromic Anemia	96.2	2.9	0.9	0	Normal Hb Variant	
545	333	24	Primigravida	13 weeks 5 days	2*CM	No	No	No	No	O Positive	11.8	35.1	3.71	88.60	31.80	33.60	23.88	Normal Blood Picture	96.8	2.4	0.8	0	Normal Hb Variant	
547	334	24	G2P1	18 weeks 2 days	NCM	No	No	No	No	B Positive	12.5	37	4.19	88.30	29.10	32.90	21.07	Normal Blood Picture	96.5	2.1	1.4	0	Normal Hb Variant	
548	335	24	Primigravida	11 weeks 4 days	NCM	No	No	No	No	A Positive	9	33.4	3.98	84.00	27.40	32.60	21.11	Normocytic Normochromic Anemia	96.8	2.4	0.8	0	Normal Hb Variant	
551	336	25	G3P2L2	17 weeks 2 days	NCM	No	No	No	No	A Negative	11	32.7	3.84	90.20	31.10	31.60	23.49	Normal Blood Picture	96.8	1.8	1.4	0	Normal Hb Variant	

Antenatal Screening of Hemoglobinopathies in a Tertiary Care Centre - One Year Descriptive Observational Study

553	337	26	Primigravida	16 weeks 3 days	NCM	No	No	No	No	B Positive	11.8	36.4	3.88	93.90	30.40	32.40	24.20	Normal Blood Picture	96.9	2.3	0.8	0	Normal Hb Variant		
554	338	27	Primigravida	11 weeks 4 days	NCM	No	No	No	No	O Positive	13	37.6	4.19	92.80	31.20	32.20	22.15	Normal Blood Picture	97.8	1.4	0.8	0	Normal Hb Variant		
555	339	26	G2P1L1	9 weeks 4 days	NCM	No	No	No	No	O Negative	9.8	30.7	3.92	83.20	27.40	30.10	21.22	Microcytic Hypochromic	97.1	2.1	0.8	0	Normal Hb Variant		
558	340	25	G2P1L1	8 weeks 5 days	NCM	No	No	No	No	B Positive	10	30.9	4.5	82.40	28.10	30.60	18.31	Microcytic Hypochromic	96.9	2.3	0.8	0	Normal Hb Variant		
559	341	24	Primigravida	14 weeks 4 days	NCM	No	No	No	No	O Positive	8.3	26	4.69	80.60	27.20	30.40	17.19	Microcytic Hypochromic	96.8	1.8	1.4	0	Normal Hb Variant		
560	342	25	Primigravida	17 weeks 5 days	3*CM	No	No	No	No	B Positive	9.8	27.3	4.46	82.60	28.20	31.20	18.52	Microcytic Hypochromic	97.2	2	0.8	0	Normal Hb Variant		
563	343	30	Primigravida	11 weeks 4 days	NCM	No	No	No	No	A Positive	9.7	27.3	4.46	82.60	28.20	31.20	18.52	Microcytic Hypochromic	97.9	1.3	0.8	0	Normal Hb Variant		
564	344	26	G2P1L1	16 weeks 3 days	NCM	No	No	No	No	A Negative	9.5	27.3	4.46	82.60	28.20	31.20	18.52	Microcytic Hypochromic	97.1	2.1	0.8	0	Normal Hb Variant		
565	345	26	G3P1L1A1	18 weeks 2 days	3*CM	No	Thalassemia minor	Thalassemia Minor	P1 - Thalassemia Major	B Positive	9.2	29.2	4.96	63.80	27.20	29.40	12.86	Microcytic Hypochromic				0	K/C/O	K/C/O	Loss to follow up
567	346	25	Primigravida	13 weeks 4 days	NCM	No	No	No	No	O Positive	11.9	35.7	3.79	94.20	31.40	33.33	24.85	Normochromic Anemia	96.9	2.1	1	0	Normal Hb Variant		
569	347	20	Primigravida	11 weeks 1 day	NCM	No	No	No	No	O Positive	7.6	22.8	2.64	86.36	28.79	30.20	32.71	Microcytic Hypochromic Anemia	96.8	2.4	0.8	0	Normal Hb Variant		
571	348	26	G2P2L2	16 weeks 3 days	NCM	No	No	No	No	O Positive	12.1	36.3	3.7	98.11	32.70	33.20	26.52	Normal Blood Picture	97.4	1.8	0.8	0	Normal Hb Variant		
572	349	25	Primigravida	17 weeks 2 days	NCM	No	No	No	No	O Negative	13.5	40.5	4.35	93.10	31.03	32.10	21.40	Normal Blood Picture	96.9	2.3	0.8	0	Normal Hb Variant		
574	350	21	Primigravida	14 weeks 5 days	NCM	No	No	No	No	B Positive	9.8	31.9	4.8	66.50	20.50	30.80	13.85	Microcytic Hypochromic	97.9	1.4	0.7	0	Normal Hb Variant		
176	351	23	Primigravida	19 weeks 5 days	NCM	No	No	No	No	O Positive	13.4	40.2	4.44	90.54	30.18	33.20	20.39	Normal Blood Picture	97.1	2.1	0.8	0	Normal Hb Variant		
578	352	22	G2P2L2	15 weeks 4 days	NCM	No	No	No	No	B Positive	6.4	19.2	3.59	53.48	17.83	28.20	14.90	Microcytic Hypochromic Anemia	96.8	2.3	0.9	0	Normal Hb Variant		
579	353	28	G2P2L1	12 weeks 6 days	NCM	No	No	No	No	A Positive	10.2	30.6	3.53	86.69	28.90	33.00	24.56	Normochromic Anemia	97.2	1.8	1	0	Normal Hb Variant		
580	354	25	Primigravida	19 weeks 5 days	NCM	No	No	No	No	A Negative	12.8	38.4	3.91	98.21	32.74	33.40	25.12	Normal Blood Picture	97.1	2	0.9	0	Normal Hb Variant		
582	355	23	Primigravida	weeks 4 days	NCM	No	No	No	No	B Positive	11.4	38.2	4.99	63.78	31.10	30.30	12.78	Normochromic	94	3.8	2.2	0	Thalassemia Minor	Normal	
584	356	24	G2P1L1	17 weeks 5 days	NCM	No	No	No	No	O Positive	11.7	35.1	3.71	94.61	31.54	33.60	25.50	Normal Blood Picture	97.9	1.3	0.8	0	Normal Hb Variant		

Antenatal Screening of Hemoglobinopathies in a Tertiary Care Centre - One Year Descriptive Observational Study

585	357	37	G4P3L2	19 weeks 5 days	NCM	No	No	No	No	B Positive	12.1	36.3	4.19	86.63	28.88	32.90	20.68	Normal Blood Picture	96.9	2.1	1	0	Normal Hb Variant		
587	358	23	G2P1L1	9 weeks 2 days	NCM	No	No	No	No	A Negative	10.8	32.4	3.98	81.41	27.14	32.60	20.45	Microcytic Hypochromic Anemia	96.8	2.4	0.8	0	Normal Hb Variant		
588	359	21	Primigravida	17 weeks 5 days	NCM	No	No	No	No	B Positive	10.7	32.1	3.84	83.59	27.86	31.60	21.77	Microcytic Hypochromic Anemia	97.4	1.8	0.8	0	Normal Hb Variant		
590	360	22	G2A1	13 weeks 4 days	NCM	No	No	No	No	O Positive	11.7	35.1	3.88	90.46	30.15	32.40	23.32	Normal Blood Picture	96.8	2.6	0.6	0	Normal Hb Variant		
592	361	23	G3P1L1A1	18 weeks 1 day	NCM	No	No	No	No	O Positive	12.4	37.2	4.19	88.78	29.59	32.20	21.19	Normal Blood Picture	96.3	2.9	0.8	0	Normal Hb Variant		
593	362	24	G2P1L1	19 weeks 4 days	NCM	No	No	No	No	O Positive	9.7	29.1	3.92	74.23	24.74	30.10	18.94	Microcytic Hypochromic Anemia	96.4	2.7	0.9	0	Normal Hb Variant		
594	363	29	G4P3L3	13 weeks 3 days	NCM	No	No	No	No	O Negative	10.5	31.5	4.5	70.00	23.33	30.60	15.56	Microcytic Hypochromic Anemia	96.4	2.8	0.8	0	Normal Hb Variant		
596	364	26	G2A1	15 weeks 6 days	NCM	No	No	No	No	B Positive	8.2	24.6	4.69	52.45	17.48	30.40	11.18	Microcytic Hypochromic Anemia	96	3.1	0.9	0	Normal Hb Pattern		
597	365	20	Primigravida	18 weeks 2 days	NCM	No	No	No	No	O Positive	9.7	35	4.46	94.00	21.75	31.20	21.08	Macrocytic Hypochromic Anemia	96.2	2.9	0.9	0	Normal Hb Variant		
599	366	22	Primigravida	14 weeks 3 days	NCM	No	No	No	No	B Positive	9.6	28.5	3.08	92.50	30.80	33.30	30.03	Microcytic Hypochromic Anemia	96.8	2.4	0.8	0	Normal Hb Variant		
601	367	23	G3A2	11 weeks 4 days	NCM	No	No	No	No	A Positive	12.1	36.3	4.6	78.91	26.30	30.10	17.16	Normal Blood Picture	96.8	2.2	1	0	Normal Hb Variant		
602	368	21	G2A1	14 weeks 5 days	NCM	No	No	No	No	A Negative	13.2	39.6	4.97	79.68	26.56	34.40	16.03	Normal Blood Picture	96.8	2.4	0.8	0	Normal Hb Variant		
603	369	21	Primigravida	17 weeks 2 days	NCM	No	No	No	No	B Positive	9.8	29.7	4.99	64.10	24.60	30.20	12.85	Microcytic Hypochromic	51.6	2.9	1.7	39	Sickle cell trait	Not done	
604	370	22	G2P1L1	19 weeks 5 days	NCM	No	No	No	No	A Positive	12.1	36.3	4.6	78.91	26.30	30.40	17.16	Normal Blood Picture	97.2	2	0.8	0	Normal Hb Variant		
605	371	18	Primigravida	12 weeks 6 days	NCM	No	No	No	No	B Positive	12.4	37.2	4.19	88.78	29.59	35.20	21.19	Normal Blood Picture	97.8	1.4	0.8	0	Normal Hb Variant		
606	372	26	G2P1L1	15 weeks 3 days	NCM	No	No	No	No	A Negative	8.5	25.5	4.69	54.37	18.12	30.20	11.59	Microcytic Hypochromic Anemia	97.1	2.1	0.8	0	Normal Hb Variant		
608	373	29	G3P1L1A1	17 weeks 2 days	3*CM	No	No	No	No	B Positive	12.6	37.8	4.6	82.17	27.39	29.50	17.86	Normal Blood Picture	97	2.2	0.8	0	Normal Hb Variant		
610	374	23	Primigravida	19 weeks 5 days	NCM	No	No	No	No	O Positive	12.3	36.9	4.6	80.22	26.74	31.20	17.44	Normal Blood Picture	96.4	2.8	0.8	0	Normal Hb Variant		
612	375	30	G3P2L2	2 weeks 1 day	NCM	No	No	No	No	O Positive	12.4	37.2	4.6	80.87	26.96	29.40	17.58	Normal Blood Picture	96	3.1	0.9	0	Normal Hb Pattern		
614	376	22	Primigravida	weeks 4 days	NCM	No	No	No	No	B Positive	9.2	34.4	4.95	63.80	30.30	33.50	12.89	Microcytic Hypochromic Anemia	94	5.1	0.9	0	Thalassemia	Normal	

