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A Study on How Iron Deficiency Anemia Affects Hba1c Levels in those without Diabetes and those who are Pre-Diabetic

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ABSTRACT

- ***Background:***

Iron deficiency anemia (IDA) is the most prevalent nutritional anemia globally, affecting approximately 30% of the population. It has significant health implications, particularly in developing countries. Hemoglobin A1c (HbA1c) is a critical marker for assessing glycemic control and is used in diagnosing diabetes.

- ***Objective:***

This study aims to evaluate the influence of iron deficiency anemia on HbA1c levels in non-diabetic and pre-diabetic individuals, assessing changes in HbA1c levels pre and post-correction of iron deficiency.

- ***Methods:***

A prospective interventional study was conducted over 18 months at Katuri Medical College and Hospital. The study included 200 participants (100 with IDA and 100 controls). Various hematological parameters, including HbA1c, were measured before and after iron supplementation.

- ***Results:***

The study found that participants with IDA had significantly lower HbA1c levels before treatment (4.60 ± 0.31%) compared to post-correction levels (5.80 ± 0.31%). The results indicated a statistically significant increase in HbA1c levels following the correction of iron deficiency, aligning closely with control group levels.

- ***Conclusion:***

The findings suggest that iron deficiency anemia significantly affects HbA1c levels, which can lead to misdiagnosis of diabetes. It is crucial to consider iron status when interpreting HbA1c results in clinical practice, particularly in populations with a high prevalence of anemia. Further research is recommended to explore the relationship between IDA and glycemic control in larger cohorts.

ISSN No:-2456-2165 https://doi.org/10.5281/zenodo.14769398

TABLE OF CONTENTS

ISSN No:-2456-2165 https://doi.org/10.5281/zenodo.14769398

LIST OF TABLES

ISSN No:-2456-2165 https://doi.org/10.5281/zenodo.14769398

LIST OF FIGURES

ISSN No:-2456-2165 https://doi.org/10.5281/zenodo.14769398

CHAPTER ONE
INTRODUCTION

Iron deficiency anemia is the commonest form of nutritional anemia worldwide. WHO (World Health Organization) reported that, globally there are 2.1 billion cases of iron deficiency anemia, which is approximately 30% of the world population.

Anemia is a late indicator of iron deficiency. It is estimated that iron deficiency is 2.5 times more common than anemia.In developing countries the estimated prevalence of anemia was in children below 5 years 39%, in children between 5 to 14 years 48%, in women 15-59 years 42%, in men 15-59 years 30% and in adults more than 60 years of age group 45%. These figures show the significant impact of anemia on economic and health consequences for middle and low income countries.

Anemia and iron deficiency lead to significant productivity losses in adults. Iron deficiency in pregnant women is associated with increased maternal mortality, preterm labour, low birth weight and increased infant mortality. Iron deficiency in children leads to defective cognitive and motor development and increases susceptibility to infections.

Anemia is the major public health problem in India. According to National Family Health Survey (NFHS), 70% of children aged 6-59 months, 55% of females aged 15-49 years and 24% of males aged 15-49 years were suffering from anemia. NFHS-3 data showed that the prevalence of anemia was higher in rural areas. But there is a paucity of data about the epidemiology of anemia in rural population.

Hemoglobin A1c (HbA1c) or glycated hemoglobin is the predominant fraction of hemoglobin A. It is used as the gold standard method for assessing the glycemic control.

It reflects the glycemic status of the individual over the past 3 months. It is formed by glycation of NH2-terminal valine of the hemoglobin β chain.

According to the guidelines of American Diabetic Association, the target HbA1c in all diabetic patients is below 7%, to prevent the development of secondary micro- vascular complications. Similar to plasma glucose, HbA1c level is related to the prevalent retinopathy.

In 2009, an International Expert Committee recommended the HbA1c level of more than 6.5% as a cut-off point to diagnose diabetes. The test should be repeated to confirm the diagnosis. Repeat testing is not required if there are classical clinical symptoms and the plasma glucose levels more than 200 mg/dl.

The Committee also recommended, considering the diabetes preventive measures in individuals with HbA1c level between 6.0 to 6.5%, as they are at a higher risk. Initial studies suggested a relationship between HbA1c levels and iron deficiency anemia. They tried to explain that on the basis of structural modifications and alterations in HbA1c levels in old and new red blood cells. Few studies reported no differences in the HbA1c levels of anemic patients compared to healthy controls.

Few studies stated that higher HbA1c levels were seen in iron deficiency anemia patients and it decreased significantly after treatment. The results of various studies on relationship between HbA1c and iron deficiency anemia were conflicting. Only fewer studies have been conducted in Indian population on this topic.

The present study is aimed to study the levels of HbA1c in iron deficiency anemia patients and the changes in HbA1c level after the correction of iron deficiency anemia.

➢ *Aims & Objectives*

To asses the hba1c levels in people with iron deficiency anaemia pre and post correction and help in recognizing pre-diabetic cases early .

- To study the levels of HbA1c in iron deficiency anemia patients
- To study the changes in HbA1c level with the correction of iron deficiency anemia.

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CHAPTER TWO
REVIEW OF LITERATURE

Type 2 diabetes mellitus is still somewhat common worldwide[1]. Type 2 diabetes lowers quality of life, adds a substantial cost to public health, and causes individuals severe pain and financial hardship. As the result of a non-enzymatic interaction between hemoglobin and glucose, the measurement of glycated forms of hemoglobin represents the average plasma glucose over the preceding two or three months. [2].

Hemoglobin A1c, or HbA1c, is a key marker for screening, diagnosing, and monitoring high blood sugar levels. The oral glucose tolerance test (OGTT) is the preferred diagnostic method. [3], Hemoglobin A1c levels are easier to measure, quicker, and less influenced by physiological and pharmaceutical factors.[4–7].

HbA1c values indicate the progression of micro-vascular lesions in people with diabetes. [8]. Therefore, extensive research has been conducted on HbA1c in diabetes mellitus..[9]. In the year 2009, the American Diabetes Association, or ADA, advised that a HbA1c level of 6.5% should be considered as a diagnostic criterion for diabetes [4], especially when it is consistently elevated [10,11]. Physicians have contended that several circumstances, including as the detection technique, other existing health conditions, and patient medicines, might have a substantial impact on HbA1c readings. They say that HbA1c does not consistently indicate plasma glucose levels. [2,12,13].

The American Diabetes Association (ADA) recommended that the diagnosis of diabetes should only rely on glucose criteria for illnesses characterized by aberrant red cell turnover, such as anemia's caused by hemolysis and iron shortage. [4]Nevertheless, the use of HbA1c as the only measurement in individuals with diabetes mellitus is a topic of debate. Research has shown that iron deficiency anemia (IDA) might result in inaccurately elevated HbA1c levels, but the exact mechanism behind this is still unknown [14]. Thus, this research intends to examine the potential factors that contribute to the correlation between elevated HbA1c levels and IDA.

A. Iron Deficiency Anemia (IDA) and Glycated Hemoglobin A1c (HbA1c)

In adults, the main forms of hemoglobin are HbA (95–98%), HbA2 (2–3%), and HbF (1%). [15]. Various forms of HbA, like HbA0, HbA1a1, HbA1a2, HbA1b, and HbA1c, can be differentiated through electrophoresis.

HbA1c makes up 70-90% of HbA1 and is its glycosylated form, created through glycosylation before the amino acid proline. [7]. This process reflects blood glucose levels. HbA1c is affected by hemoglobin and glucose levels, but factors like race, age, nutrition, medication, and illnesses also play a significant role. [15].

Iron deficiency, caused by insufficient intake, use, or excess loss, affects hemoglobin production, leading to iron deficiency anemia (IDA). This condition is diagnosed by assessing ferritin, serum iron, and transferrin saturation levels. IDA accounts for 33% of anemia cases globally, particularly affecting poor nations. Symptoms include fatigue, tachycardia, pica, and cognitive issues. Babies and children can suffer neurological and cognitive effects. China's healthcare system focuses on timely detection and management of IDA, especially in children, adolescents, and women of reproductive age.

B. Latest Resarch Discoveries

Research investigations in epidemiology and clinical medicine have shown that iron deficiency anemia (IDA) leads to an elevation in HbA1c levels, regardless of the levels of glucose in the blood plasma [7,14,21].

Kim et al. [7] analyzed data from the US National Health and Nutrition Survey (1999- 2006), which included 6,666 women and 3,869 men. They discovered that women with iron deficiency but no anemia (n=1,150) saw a modest rise in their HbA1c levels (from less than 5.5% to equal to or more than 5.5%), regardless of their fasting glucose levels. Attard et al. [5] In their analysis of Chinese health data, researchers found that men with iron deficiency or anemia had a higher prediabetes risk when using just HbA1c compared to HbA1c and fasting glucose. However, studies differ on this topic. A meta- analysis discovered no impact of iron status on HbA1c levels. [22].

Additionally, a research shown that there was no correlation between hemoglobin levels and HbA1c levels in non-diabetic elderly individuals[23] and Dutch children with type 1 diabetes [24]. In their study, Christy et al. [25] A comparison was made among patients with diabetes, iron deficiency anemia (IDA), and a control group. The study revealed no correlation between ferritin or

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hemoglobin levels in any group. Past studies have indicated that both iron insufficiency and IDA can influence HbA1c levels independently.[26].

Attard et al. [5] The study evaluated HbA1c and fasting blood glucose for diagnosing diabetes impacted by iron deficiency and iron deficiency anemia. Both conditions altered HbA1c levels inaccurately, affecting diagnoses. Iron-deficient women had lower diabetes detection with HbA1c (RR ratio=0.52; 95% CI, 0.29–0.95%) while men with iron deficiency anemia had higher detection rates using HbA1c and fasting blood glucose (RR ratio=2.38; 95% CI, 2.0–4.72%). Researchers concluded that iron deficiency caused missed cases, and iron deficiency anemia led to misdiagnoses. [5]. The study authors attribute these results to factors like red blood cell renewal, modified hemoglobin structure, and glycation rate. [27].

Anemia, characterized by reduced red blood cell lifespan and lower hemoglobin levels, significantly affects HbA1c generation. Iron deficiency is associated with elevated HbA1c levels, independent of plasma glucose and anemia severity. [29]. Research has shown a significant decrease in HbA1c levels in individuals without diabetes who had well-regulated plasma glucose levels after the use of iron supplementation [30,31]. Nevertheless, another research has shown that there is no connection between indicators of iron storage (ferritin) and an elevation in HbA1c levels [25].

- *Potential Factors Contributing to Elevated HbA1c Levels in Iron Deficiency Anemia:*

The production rate of HbA1c remains consistent at a specific plasma glucose level. [26], and the pace at which red blood cells are replaced impacts the amount of HbA1c [28]. Elevated glycation may happen in red blood cells remaining in plasma for a long time. [26, 30, 32, 33].

Wu et al. [34] Ion-exchange resin microparticles measured glycated hemoglobin in aplastic anemia patients, showing decreased levels post-transfusion within 3-14 days due to immature erythrocytes. Glycated hemoglobin may indicate erythrocyte production in this condition, but longer studies are needed to assess HbA1c levels accurately after transfusion's effects on red blood cells. [34]. Previous studies suggest that plasma glucose, glycated albumin, and fructosamine may be more reliable than HbA1c in reflecting changes in the lifespan of red blood cells. [35]. In some cases, individuals with unmanaged diabetes and genetic spherocytosis may have a plasma glucose level of 379 mg/dL, despite having a relatively low Hba1c level of 6.9%.

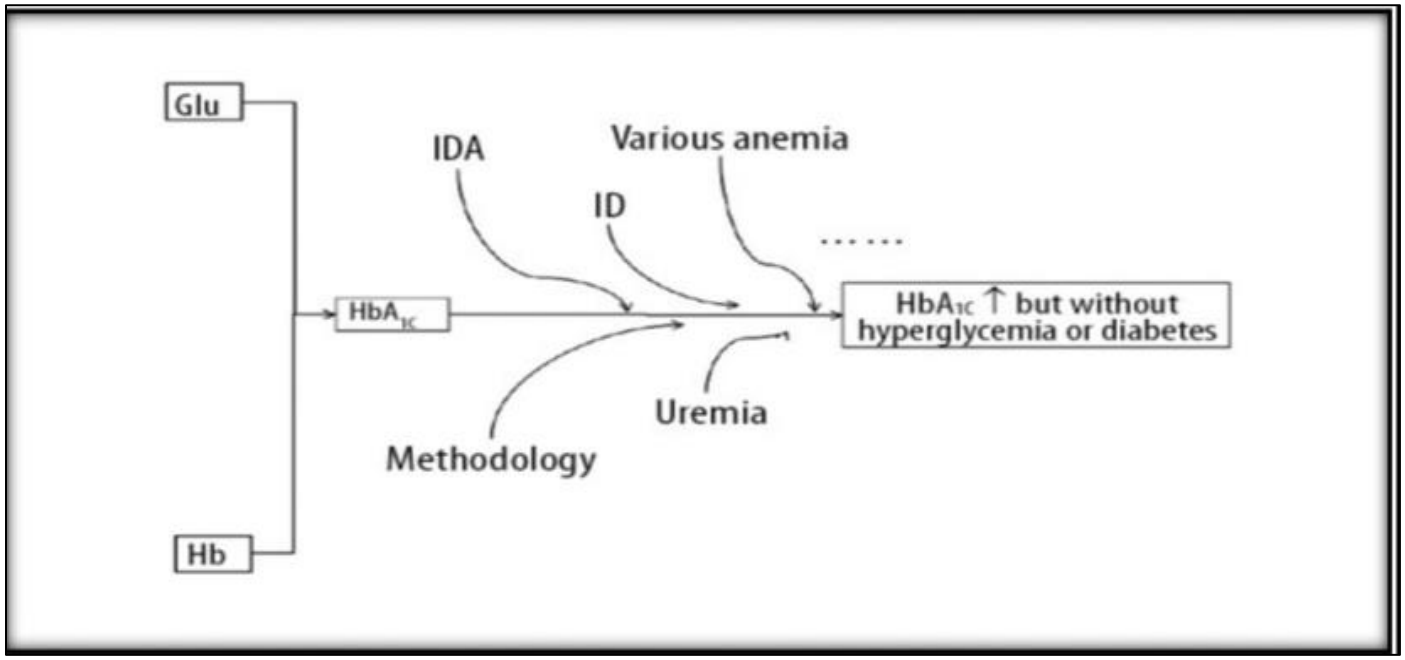

Fig 1: Ongoing Study Investigates the Correlation between Elevated Levels of Glycated Hemoglobin A1c (HbA1c) and Iron Deficiency Anemia (IDA).

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Sehrawat et al. [12] The study evaluated HbA1c's diagnostic accuracy in diabetics with liver cirrhosis and varying anemia levels. HbA1c was less reliable overall. It performed better in those with normal or mild anemia (sensitivity 82.4%) compared to moderate or severe anemia (sensitivity 72.2%). Specificity was higher in the former group (96%) than the latter (86.9%), with AUCs of 0.9 and 0.8, respectively. No significant difference in AUC between the groups (P>0.05) was found. Anemia severity influenced HbA1c levels. [12].

In the study conducted by **Xia et al.**[36], A patient with paroxysmal nocturnal hemoglobinuria showed decreased HbA1c levels from over 90 mmol/mol to 51 mmol/mol, within the normal range of 48-59 mmol/mol. This, along with high LDH and low haptoglobin levels, suggests compensated hemolytic anemia. The findings indicate poorly controlled diabetes, evident from elevated random glucose (11.0 mmol/L), glycated albumin (1.5%), and fructosamine (0.34 mmol/L) levels. [36]. The most probable reason of these observations was the shortened lifetime of red blood cells owing to hemolysis. **Silva et al.** [37] A negative association was found between HbA1c levels and hemoglobin, hematocrit, mean corpuscular volume (MCV), and ferritin (r=–0.557, r=–0.539, r=–0.488, r=–0.499) (P<0.001). [26].

Research on individuals without diabetes with hemolytic anemia has shown that hemolysis shortens red blood cell lifespan, leading to destruction and decreased HbA1c levels. [38].

Rajagopal et al. [39] a positive correlation was observed between anemia severity and increasing HbA1c levels.

In 2018, **Nakatani et al.** [40] A case study documented the regulation of blood sugar levels in a 41-year-old male with dehydrated hereditary stomatocytosis. The patient had a mutation in the PIEZO1 gene, tied to diabetes mellitus and cirrhosis due to hemochromatosis. HbA1c estimated from continuous glucose monitoring assessed glycemic control, while glycated albumin levels were also analyzed. [40]. The patient's reliability of HbA1c and glycated albumin was compromised by concomitant diseases, like pernicious anemia, resulting from vitamin B12 deficiency and marked by secondary hemolysis. [7]. Elevated levels of urea result in the production of carbamylated hemoglobin [41,42], a process analogous to glycation. The isoelectric point of carbamylated hemoglobin is similar to that of HbA1c [42]. Urea and glucose compete for binding to the N-terminal proline of hemoglobin b chain, reducing HbA1c concentration. [43]. Hematological parameters, like red blood cell count, renewal rate, and lifespan, influence HbA1c measurement. Although some labs report HbA1c as a percentage, using absolute values (mmol/mol) is recommended to minimize hemodilution effects. [44]. It is recommended to do hematological tests early to improve the accuracy of interpreting HbA1c levels. [26].

- *Heterogeneity of Hemoglobin*

Previous research has identified the repercussions of HbA1c heterogeneity based on discrepancies between measured and predicted values [45]. It has been demonstrated that hemoglobin heterogeneity influences HbA1c independently of plasma glucose. An epidemiological investigation conducted by Lacy et al. (46) revealed that individuals diagnosed with sickle cell anemia exhibited reduced concentrations of HbA1c.

It was discovered that the charge of the hemoglobin molecule was altered in the most prevalent hemoglobin variants, or mutant forms of hemoglobin, including HbS, HbC, HbD, and HbE [22]. Variants of hemoglobin cause inconsistencies in the outcomes of the various HbA1c measurement techniques. An instance of this is the substantial elevation in HbA1c that occurs when charge separation techniques, such as ion-exchange HPLC, are implemented [47].

In their research, Otabe et al. [45] investigated the clinical significance of the discrepancy between enzymatic and HPLC-determined HbA1c values in patients with diabetes. The results of an investigation involving 1,421 outpatients who received treatment and follow-up for diabetes indicated that HPLC measurements of HbA1c were substantially higher than enzymatic assay measurements. It is advisable, as suggested by the authors, to interpret HbA1c levels in light of the potential existence of hemoglobin variants [45]. Furthermore, modifications to the structure of hemoglobin glycation variants may cause alterations in the kinetics of reaction with glucose and restrict the number of NH2 groups that are capable of reacting with glucose [48]. Research has indicated that the glycation rates of HbC, HbE, and HbF vary, with HbA exhibiting the slowest rate [48]. Higgins

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et al. [49] underscored the substantial impact of glycated hemoglobin heterogeneity on HbA1c levels. They attributed this to methodological considerations that influence chromatography and immunoassays, leading to fluctuations in glycation rate and diminished erythrocyte survival (e.g., erythrocytes containing HbS have a 14– 28 day survival time). Nevertheless, prior research has demonstrated that heterozygous heterogeneity does not invariably lead to hemolytic anemia and has no discernible impact on glycosylation [50].

In the non-diabetic population, Cavagnolli et al. [22] discovered no significant difference in HbA1c levels between HbS carriers and non-carriers, suggesting that the glycation rates of HbA and HbS were comparable. Although there are instruments designed to detect hemoglobin variants [51], there are still certain constraints that clinicians should be aware of and account for, including the possibility of hemoglobin variants [45]. There is speculation regarding the potential influence of hemoglobin variants on the glycation process, which could lead to varying outcomes when attempting to measure HbA1c.

The World Health Organization (WHO) noted in 2011 that HbA1c measurements were impacted globally by hemoglobin heterogeneity [52]. While the incidence of hemoglobin variants is comparatively low, it is prudent to exercise prudence when interpreting clinical observations of HbA1c levels (Figure 2).

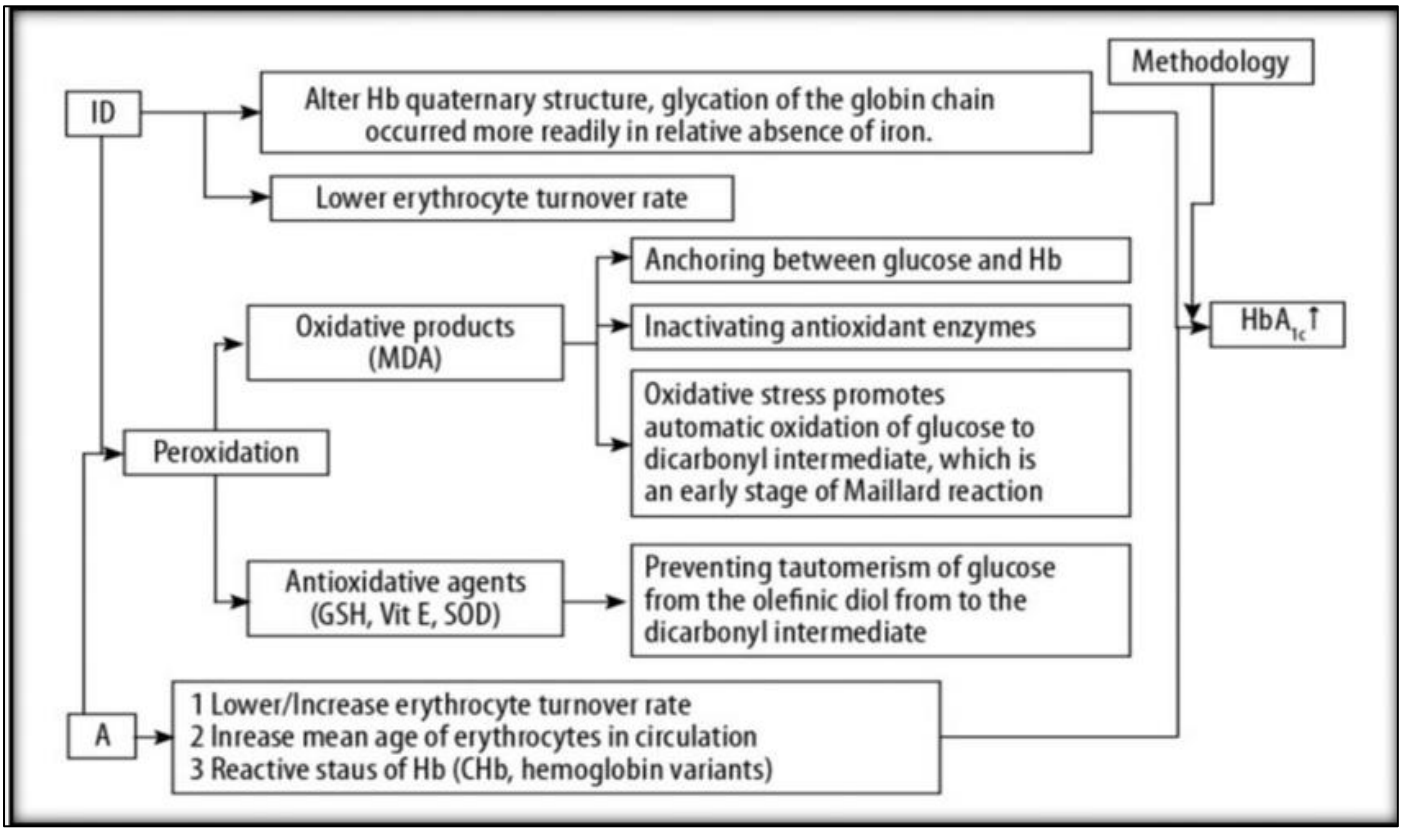

Fig 2: The Potential Factors Contributing to the Correlation between Elevated Levels of Glycated Hemoglobin A1c, also Known as HbA1c and Anemia Caused by Iron Deficiency (IDA).

➢ *Enhanced Peroxidation Caused by IDA*

Iron shortage induces terminal proline glycation via altering the hemoglobin structure [53], and simultaneously reducing the pace at which erythrocytes are replaced [21,27]. Iron deficiency may worsen diabetes progression due to increased glycation from peroxidation, a process where oxygen radicals harm cells by inducing reactive species. These radicals engage in reactions within cells and play a role in fighting infections and affecting immune responses, potentially leading to reactions with other molecules when present excessively. [60]. Iron deficiency can lower enzyme function, reducing antioxidant capacity. [54].

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Iron deficiency may trigger peroxidation and expedite glycation. Zaka-Ur-Rab et al. [55] A study found higher levels of oxidative stress biomarkers, notably malondialdehyde (MDA), in 67 children with iron deficiency anemia (IDA) compared to 31 control subjects. Antioxidant enzymes like superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx) were significantly lower in the IDA group.

In a study involving 35 children with iron deficiency anemia (IDA), iron supplementation significantly increased antioxidant levels (SOD, CAT, GPx) while decreasing MDA levels ($P<0.001$). Fructosamine correlated with MDA ($r=0.6$, $P<0.01$), adjusted for glucose influence. Ferrous sulfate tablets reduced MDA and fructosamine. Lipid peroxides influenced glycation, supported by introducing iron. In other research, exposure of erythrocytes to glucose combined with H2O2, TBH, and vitamin E showed variable glycated hemoglobin levels. MDA accelerated glycation by impacting antioxidant enzymes. Glycation linked to glucose and hemoglobin by MDA. Antioxidants like glutathione inhibited this process. Substituting glutathione with DTE/DTT demonstrated the inverse relationship between glutathione and glycated hemoglobin. Iron deficiency affects HbA1c levels through oxidative stress pathways.

C. Previous Studies:

JF Silva et al[59] a study investigated the effect of iron deficiency anemia (IDA) on HbA1c levels in non-diabetic individuals using two common measurement methods. The research involved 122 participants split evenly between those with and without IDA. Results showed significant differences in HbA1c levels between the two groups ($p < 0.001$). Strong negative correlations were found between various blood parameters and HbA1c levels. Moderate to severe anemia led to higher HbA1c values, with the impact of IDA depending on its severity. Mild anemia is unlikely to significantly affect HbA1c results, supporting its use in diabetes diagnosis.

In their research, **Sinha et al**[60] examined the impact of iron deficiency anemia on HbA1c levels and evaluated whether treating iron deficiency anemia had an influence on HbA1c levels. This research included a total of fifty individuals who were diagnosed with iron deficiency anemia. Hemoglobin A1c (HbA1c) and its absolute levels were assessed at the beginning of the study and again after 2 months of therapy. These measurements were then compared to the values seen in the control group. The average initial HbA1c level in patients with anemia (4.6%) was considerably lower compared to the control group (5.5%, $p<0.05$). There was a notable rise in the patients' absolute HbA1c levels two months following therapy (0.29 g/dL vs. 0.73 g/dL, $p<0.01$). The baseline values of patients and controls differed significantly, with patients having a mean value of 0.29 g/dL and controls having a mean value of 0.74 g/dL ($p<0.01$). Contrary to the findings of other research, our study revealed that both HbA1c levels and absolute HbA1c levels rose as a result of iron deficiency anemia therapy. This might be ascribed to a lack of adequate nutrition and/or certain unidentified factors. Additional research is necessary.

L. Rajagopal et al. [39] conducted a study at SRM Medical College Hospital to examine how iron deficiency anemia (IDA) affects HbA1C levels in non-diabetic individuals. Examined HbA1C fluctuations among mild, moderate, and severe anemia cases. 150 non-diabetic participants were involved, half with IDA. Noted medical history and conducted various tests including HbA1C. Patients with IDA had a higher mean HbA1C level compared to non-anemic individuals, with statistical significance. HbA1C levels increased with worsening anemia. Significantly different results were observed among varying degrees of anemia.

SV Madhu et al[61] analyzed the impact of iron deficiency anemia (IDA) on HbA1c levels before and after iron treatment in similar age and sex-matched groups (IDA - n = 62, healthy controls - n = 60). HbA1c levels were assessed by HPLC, and hemogram by a hematology analyzer. Serum ferritin levels were measured using ELISA per ICSH norms. IDA individuals exhibited higher HbA1c levels than healthy controls (5.51 ± 0.696 vs. $4.85 \pm 0.461\%$, $p < 0.001$). Iron treatment significantly reduced HbA1c in IDA individuals (5.51 ± 0.696 vs. 5.044 ± 0.603; $p < 0.001$). Post-treatment, 70% of pre- diabetic subjects shifted to NGT. Care is needed in interpreting HbA1c results in IDA to avoid misdiagnosing diabetes based solely on HbA1c.

S Çetinkaya Altuntaş et al[62] a study examined HbA1c levels and iron therapy effects in relation to IDA in 263 non-diabetic individuals. The group with IDA had lower HbA1c levels (5.4%) compared to healthy controls (5.9%). HbA1c decreased with increased anemia severity and increased after iron treatment. Hemoglobin and other parameters also improved with iron therapy. IDA correlated with lower HbA1c levels which improved with iron treatment, highlighting the importance of considering IDA's impact on HbA1c.

In a study done by **S Kalairajan et al**[63], the study examined how iron deficiency anemia affected HbA1c levels in 120

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individuals. Initial HbA1c levels were compared between anemic patients (4.62%) and controls (5.45%, P<0.001) before and after treatment. There was a notable rise in the patients' HbA1c values three months following therapy (5.82%, P<0.001). A strong link was seen between the levels of haemoglobin and HbA1c (correlation coefficient = 0.26, p < 0.01) in the study group prior to correction. Our study revealed a contrasting finding to earlier research, as it shown that the treatment of iron deficient anaemia led to an increase in HbA1c levels. This phenomenon may be attributed to factors such as inadequate nutrition, variances in racial and cultural backgrounds, and maybe other unidentified causes. Additional research is necessary.

In a study conducted by **Solomon et al**[64], a study at Black Lion Specialized Teaching Hospital in Addis Ababa, Ethiopia, examined how iron deficiency anemia (IDA) affects HbA1c levels in diabetic patients. Blood samples from 174 patients were analyzed for various parameters using specialized equipment. Results showed that HbA1c levels were significantly lower in diabetic individuals with IDA compared to those without IDA. This suggests caution in relying solely on HbA1c for monitoring these patients, underscoring the importance of comprehensive assessments before treatment decisions are made.

PB Renz et al[65] undertook a research to examine the impact of iron supplementation on HbA1c levels in pregnant women who do not have diabetes, both with and without anemia. Invitations were sent to pregnant women receiving prenatal care who did not have gestational diabetes (GDM) or a history of diabetes mellitus (DM) and had undergone an oral glucose tolerance test (OGTT) during the third trimester of pregnancy. Comprehensive clinical and laboratory studies were conducted, which included the use of a standardized questionnaire, oral glucose tolerance test (OGTT), complete blood count, and measurement of HbA1c levels. The research involved a cohort of 231 pregnant women who did not have diabetes mellitus (DM) or gestational diabetes mellitus (GDM). The women were categorized into four groups based on their iron and anaemia status: Group 1 (N = 86) consisted of women with no iron supplements and no anaemia, Group 2 (N = 29) consisted of women with no iron supplementation but with anaemia, Group 3 (N = 87) consisted of women with iron supplementation but no anemia, and Group 4 (N = 29) consisted of women with both iron supplementation and anaemia. There was a statistically significant difference in HbA1c values between pregnant women in Groups 1 and 4, although this difference was not clinically important. The HbA1c values in Group 1 were 5.1 ± 0.4% (32 ± 4.4 mmol/mol), whereas in Group 4 they were 4.8 ± 0.3% (29 ± 3.3 mmol/mol). The difference was significant with a p-value of less than 0.01. The HbA1c readings in pregnant women in Groups 1, 2, and 3 were comparable, regardless of the presence of anemia [5.1 ± 0.4% (32 ± 4.4 mmol/mol); 5.0 ± 0.4% (31 ± 4.4 mmol/mol); and 5.0 ± 0.4% (31 ± 4.4 mmol/mol); p > 0.05; respectively]. Iron supplementation during pregnancy does not have any effect on HbA1c levels and does not have any clinical significance in the final interpretation of data when there is no anemia or just moderate anemia present. Caution is still necessary when interpreting HbA1c findings in pregnant women who are using iron treatment and have moderate or severe anaemia.

In order to get a better understanding of the relationship between HbA1c concentrations and IDA, we did a comprehensive retrospective analysis. This study aimed to address the conflicting findings about the impact of IDA on HbA1c concentrations, as reported by LV **Rao et al**[66]. HbA1c concentrations test results were obtained from the years 2015 to 2019. We assessed a total of 12,000 individuals diagnosed with iron deficiency anemia (IDA) and 21,000 patients who did not have IDA. Patients were categorized as having iron deficiency anemia (IDA) if their tests showed blood iron, ferritin, or transferrin iron saturation levels below the age-specific limits, and transferrin iron- binding capacity or transferrin concentrations beyond the age-specific ranges. The Kruskal-Wallis statistical analysis approach was used to determine whether the two samples adhere to the same distribution and if there is statistical significance. Among females, the median HbA1c concentration was 5.7% for patients categorized with IDA and 5.4% for normal samples (P < 0.001). Among men, the median HbA1c value was 6.0% in patients categorized with IDA and 5.6% in normal samples (P < 0.001). people diagnosed with iron deficiency anemia (IDA) may have higher levels of HbA1c compared to people without IDA. Before making treatment recommendations based on HbA1c concentrations, clinicians should take into account the IDA status of the patient.

E Nasli-Esfahani et al[67] a study examined iron deficiency anemia treatment effects on HbA1c levels in type 2 diabetes patients. Ninety T2DM individuals with IDA were randomly assigned to treatment or placebo groups in a single-blind study. The treatment group received 200 mg of oral iron daily for 3 months, while the placebo group received a placebo. Results showed the treatment group had significant improvement in various blood markers and HbA1c levels compared to the control group. Considering iron status is crucial when interpreting HbA1c levels in T2DM. Iron replacement therapy may lower HbA1c in individuals with both anemia and T2DM.

In a research done by **K Sumathi et al**[68], the objective was to determine if there is a positive link between iron deficiency anemia status and HbA1c levels in individuals with diabetes. The study included 100 patients with iron-deficiency anemia and diabetes who had well-managed plasma glucose levels. The comparison group consisted of 100 diabetic persons who did not have

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anemia. The research was conducted at the facilities of Sree Balaji Medical College & Hospital. HbA1c levels were quantified using ion exchange chromatography, ferritin was tested using the particle enhanced turbidometric immunoassay technique, and fasting plasma glucose was determined using the GOD - POD method. The researchers discovered that patients with iron deficiency and diabetes had higher levels of HbA1c (7.3±0.9) compared to the normal levels (5.4±0.6) seen in the control group. The research demonstrated a direct relationship between iron deficiency anemia and elevated HbA1c values in individuals with managed diabetes. Therefore, the need of assessing the iron deficiency anemia status in diabetes patients has been shown to be crucial in determining their subsequent rapid diabetic treatment.

In their research, **Aydın et al**[69] explored iron status impact on HbA1c in 146 DM patients with IDA. They received 270 mg/day ferrous sulphate for 3 months. Hb, MCV, and ferritin increased significantly. Initial and 3-months HbA1c showed a notable drop. FPG levels remained stable. Negative correlation found between Hb and HbA1c. IDA linked to high HbA1c, improved by iron treatment. Consider IDA guidelines for HbA1c- based decisions.

Huda Ayad Hameed [70] researchers studied the link between Iron Deficiency Anemia and HbA1c levels in 120 diabetic patients at Salah al-Din Hospital from Sep 2021 to Aug 2022. 60 patients had IDA, and 60 didn't. They gathered data using a questionnaire, analyzed blood samples for HbA1c levels and hematological parameters using SPSS v21.. The Pearson's correlation coefficient and the one-sample t-test were calculated. The data was given in terms of the mean plus the standard deviation (SD). A P-value below 0.05 was deemed statistically significant. In the IDA group, the average levels of hemoglobin (Hgb), Red Blood Cell count (RBC), HBA1C, hematocrit (HCT), mean cell volume (MCV), mean cell haemoglobin concentration (MCHC), and mean cell hemoglobin (MCH) were lower compared to the non-IDA diabetic individuals. The HbA1c (%) values in the IDA group (6.78 + 7.0) were significantly lower compared to non-IDA diabetic patients (7.04 + 0.46), as shown by a statistically significant p-value. The HbA1c levels in diabetic individuals with IDA are much lower compared to diabetic patients without IDA. Consequently, it is recommended that relying just on HBA1C screening in these individuals may be misleading, so physicians and clinicians should take this into account prior to making any treatment choices. It is recommended to conduct a comprehensive evaluation with a significant sample size utilizing advanced laboratory techniques.

In a study done by **Lavanya Rajagopal et al**[71], the study at SRM Medical College analyzed the impact of iron deficiency anemia on HbA1C levels in those with controlled diabetes and those without, encompassing 150 participants with controlled diabetes and 150 without, investigating how the severity of anemia influenced outcomes. Tests were conducted for HbA1C, full Hemogram, iron profile, and FPG. A comprehensive medical history was documented. The average HbA1C levels in diabetics with and without iron deficiency anemia (IDA) were 8.81±0.13 and 5.79±0.01, respectively ($P<0.05$). In non- diabetics with and without IDA, the mean HbA1C levels were 6.84±0.07 and 5.12±0.04, respectively ($P<0.05$). The statistical analysis revealed a significant difference ($p< 0.05$) in the severity of anemia between diabetics and non-diabetics, categorized as no, mild, moderate, and severe. The groups with severe anemia had the highest mean HbA1C%. IDA erroneously increases the HbA1C level regardless of the blood glucose levels, in individuals with both well-managed diabetes and those without diabetes. Therefore, it is important to detect and rectify iron deficiency anemia (IDA) before making any changes to the treatment plan for diabetes based on HbA1C levels. Conducting simultaneous assessment for anemia is crucial in order to accurately interpret the glycemic condition in the Indian population, where iron deficiency anemia is common.

In their study, **Sreedev Narayanan et al**[72] examined the impact of total hemoglobin level on HbA1c value in patients with type 2 diabetes mellitus. They aimed to determine if a decrease in total hemoglobin level affects the HbA1c level, potentially leading to an inaccurate assessment of the patient's glycemic status. A total of 100 patients diagnosed with Type 2 Diabetes Mellitus were examined to determine their Fasting Blood Sugar (FBS), Total hemoglobin level, and HbA1c level. The purpose was to test if a decrease in total hemoglobin level had any impact on the HbA1c value. Findings: The study revealed a strong correlation between HbA1c levels and fasting blood sugar (FBS) levels in individuals with Type 2 Diabetes Mellitus. However, no association was discovered between total hemoglobin levels and HbA1c values. The fasting blood sugar level is a key determinant of the HbA1c measurement. The hemoglobin level does not seem to have an impact on the HbA1c, suggesting that hemoglobin is not a major determinant of HbA1c. Findings: This research established a notable correlation alone between fasting blood sugar (FBS) and glycated hemoglobin (HbA1c) levels. There was no observed relationship between the total Hemoglobin level and HbA1c. However, patients with mild to severe anemia still showed a high HbA1c value, which solely correlated with their blood sugar status.

Nalini Jeyaprakash et al[73] A study examined HbA1c levels in iron deficient anemia patients and post-treatment changes. 120 participants with iron insufficiency were involved. Measurements of blood count, anemia profile including HbA1c were taken pre and post-treatment. Results showed lower HbA1c levels in anemic patients compared to controls. Post-

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therapy, HbA1c levels significantly increased in the anemic group. Conclusion: Iron deficient anemia correlates with HbA1c levels. Patients show low HbA1c values which rise post-iron therapy. Consider iron deficiency in interpreting HbA1c for diabetes diagnosis.

In a research done by **MD Qureshi et al**[74], Objective: Examine iron deficiency anemia's link with HbA1c levels in 117 diabetes cases, excluding certain conditions. Patients categorized by fasting plasma glucose: 126mg/dl threshold split into well-managed (100-126 mg/dL) and well-controlled (<100 mg/dL) sugar groups. Factors like Hemoglobin, serum ferritin, Hba1c measured health. Participants averaged 56.97±7.29 years; 38.5% female, 38.5% male (1:1.6 ratio). Mean FPG 103.3±7.6, Hba1c 6.42±0.70%. Female Hb 11.5±2.7, male Hb 10.9±3.03 g/dl. 66 cases had iron deficiency anemia, 54 had FPG >100 mg/dl. Odds ratio for HbA1c >6.5% in iron- deficient individuals: 3.90 (p=0.001). Conclusion: Iron deficiency can raise HbA1c levels; doctors should assess iron status before prescribing diabetes meds based on HbA1c.

The objective of **Vijaya Durairaj, K et al** [75] the study examined HbA1c levels in iron deficient anemia patients before and after treatment. 120 individuals with iron insufficiency were involved. Initial and post-treatment blood count, anemia profile (including serum ferritin, HbA1c) were measured and compared to controls. Iron deficient anemia patients had lower HbA1c levels (4.619 ± 0.308%) than controls (5.446 ± 0.281%). After treatment, HbA1c in anemic patients rose significantly (5.816 ± 0.323%). The study linked iron deficiency anemia with HbA1c changes. Clinicians should consider anemia when interpreting HbA1c for diabetes diagnosis.

J Intra et al[76] performed a retrospective case-control study on iron deficiency's impact on HbA1c in non-pregnant individuals above age 12. Data was collected from 2625 individuals at an Italian hospital from 1990 to 2016. 109 had iron deficiency anemia, showing higher HbA1c levels than those without anemia. Multiple linear regression revealed a negative correlation between hemoglobin and HbA1c levels. Adjusting HbA1c readings based on hemoglobin may be necessary for accurate pre-diabetes and diabetes monitoring in individuals with iron deficiency anemia.

Mohamed et al[77] A case-control study conducted from February to September 2019 at Zagazig University Hospitals investigated how iron deficiency anemia affects HbA1c levels in non-diabetic individuals. The research included 90 patients in three groups – healthy individuals, those with iron deficiency anemia, and those with different types of anemia. The study involved in-depth assessments of medical history, physical exams, and biochemical marker analysis, notably HbA1c. Results showed a significant difference in HbA1c levels pre and post-treatment in the iron deficiency group, but no notable changes in FBG or PPG levels. The research indicated a link between HbA1c and iron deficiency anemia, impacting non-diabetic individuals' HbA1c levels.

V. Porwal et al[78] conducted research on how iron deficiency anemia impacts HbA1c levels and whether treating it affects HbA1c. Studied fifty-six non-diabetic adults with iron deficiency anemia at R.D Gardi Medical College and CRG Hospital in Ujjain (Group A - Microcytic hypochromic). Additionally, 56 healthy individuals were chosen as controls (Normocytic normochromic). A thorough clinical history was collected, and biochemical testing, including HbA1c, was conducted. SPSS 26 was used to conduct the statistical analysis with the assistance of suitable statistical tools. Iron-deficient anemic nondiabetic individuals have elevated average HbA1c levels in comparison to non-Anemic non-diabetic patients. The average HbA1c values for the control group were 5.44 ± 0.3. The average HbA1c values for the case group were 5.80 ± 0.42. The disparity is statistically significant, with a p-value less than 0.001. Thus, we may deduce that iron deficiency anemia has a direct impact on HbA1c levels in individuals without diabetes, and it should be carefully evaluated in all patients with iron deficiency anemia.

In a research done by **C Gharde et al** [79], the objective was to ascertain whether individuals with iron deficient anemia but without diabetes would see an increase in HbA1c values. This cross-sectional research was undertaken in the outpatient departments (OPDs) and wards of a tertiary care center in Central India. Outcome: Among the 112 individuals who took part in the study, 56 (50%) did not have anemia, 42 (37.5%) had severe anemia, and 14 (12.5%) had mild anemia. The parameters that exhibited significant differences include the mean serum iron value (p-value <0.0000001), mean serum ferritin value (p-value <0.0000001), and mean serum TIBC value (p-value <0.0000001). The average HbA1c value for the group with anemia was 6.04 ± 0.74%, whereas for the group without anemia, it was 4.91 ± 0.65%. The difference between the two groups is statistically significant. Out of the total subjects, 21.4% have HbA1c levels equal to or less than 5.5%, 71.5% have HbA1c levels between 5.6 and 6.5%, and just one person has HbA1c values more than 6.5%. Among the 42 participants with severe anaemia, 16.7% had a HbA1c level of 5.5% or below, 40.5% have a level between 5.6% and 6.5%, and 42.8% have a level between 6.6% and 7.5%. Iron deficiency anemia is a separate component that influences the HbA1C level in persons without diabetes. It is important to interpret this carefully in all patients with anemia caused by iron deficiency.

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TI Kaltsum et al[80] a study investigated the link between HbA1c and iron deficiency anemia in 115 individuals with type 2 diabetes. Results showed a significant correlation ($p = 0.003$). Participants were mainly over 45 years old, with most having diabetes for over a decade, obesity class I, and hypertension as the most common comorbidity. It is important to monitor type 2 diabetes patients closely for iron deficiency anemia, especially in those with inadequate glycemic control.

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CHAPTER THREE
MATERIALS & METHODS

- STUDY DESIGN: PROSPECTIVE INTERVENTIONAL STUDY
- DURATION OF STUDY: 18 MONTHS (December 2022 to November 2023)
- PLACE OF STUDY: General medicine department at Katuri Medical College and Hospital in Guntur has outpatient services and a ward.
- STUDY POPULATION: Patients meeting the criteria at the general medicine department of Katuri Medical College were included. Informed written consent will be obtained.
- SAMPLE SIZE: 200 (Cases: 100 ; Control : 100)

A. Inclusion Criteria

- Age between 18 to 60 years
- Patients with Impaired fasting glucose, Impaired glucose tolerance.

B. Exclusion Criteria

- Patients with diabetes
- Patients with chronic renal failure / liver disease
- Patients with hemolytic anaemia
- Pregnancy
- known case of Malignancy
- Chronic alcoholism

C. Method of Data Collection

Interventional research involving 100 diabetes participants (aged over 18) collected data on various parameters, including HbA1c, peripheral smear, hemoglobin, MCV, MCH, MCHC, serum ferritin, and plasma glucose. Similar data were gathered from non- anemic controls matched for sex and plasma glucose levels. HbA1c levels were analyzed using HPLC with Bio-Rad D-10 analyzer consistently. Hemoglobin, MCV, MCH, MCHC were estimated using Beckman Coulter LH780. Serum ferritin was estimated with Roche/Hitachi Cobas e411 and plasma glucose with Roche Hitachi P800/917.

D. Statistical Analysis

Data Entry was done using Microsoft excel 2013 and analysis done using SPSS V 16. Qualitative data was expressed in frequencies and percentages and Quantitative data in mean and standard deviation. Parametric tests include Unpaired t test for intergroup comparison was used. Non parametric test include Chi-square test was used for qualitative data. Bar diagrams and pie chart were used to represent the data. p value of <0.05 was considered statistically significant.

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CHAPTER FOUR
RESULTS

Table 1: Distribution of the Study Population based on Age

Age in years	Study group		Control group		Total	
	N	%	N	%	N	%
21 – 30	23	23%	14	14%	37	18.5%
31 – 40	26	26%	36	36%	62	31%
41 – 50	21	21%	30	30%	51	25.5%
51 – 60	30	30%	20	20%	50	25%
Total	100	100%	100	100%	200	100%
Mean age	41.37 ± 11.30		40.86 ± 9.30			
Chi square test= 13.53, p=0.13, Not statistically significant						

The research revealed age distribution: 18.5% aged 21-30, 31% aged 31-40, 25.5% aged 41-50, and 25% aged 51-60. Average age was 41.37 ± 11.30 years, with no significant age difference among groups, indicating age equivalence.

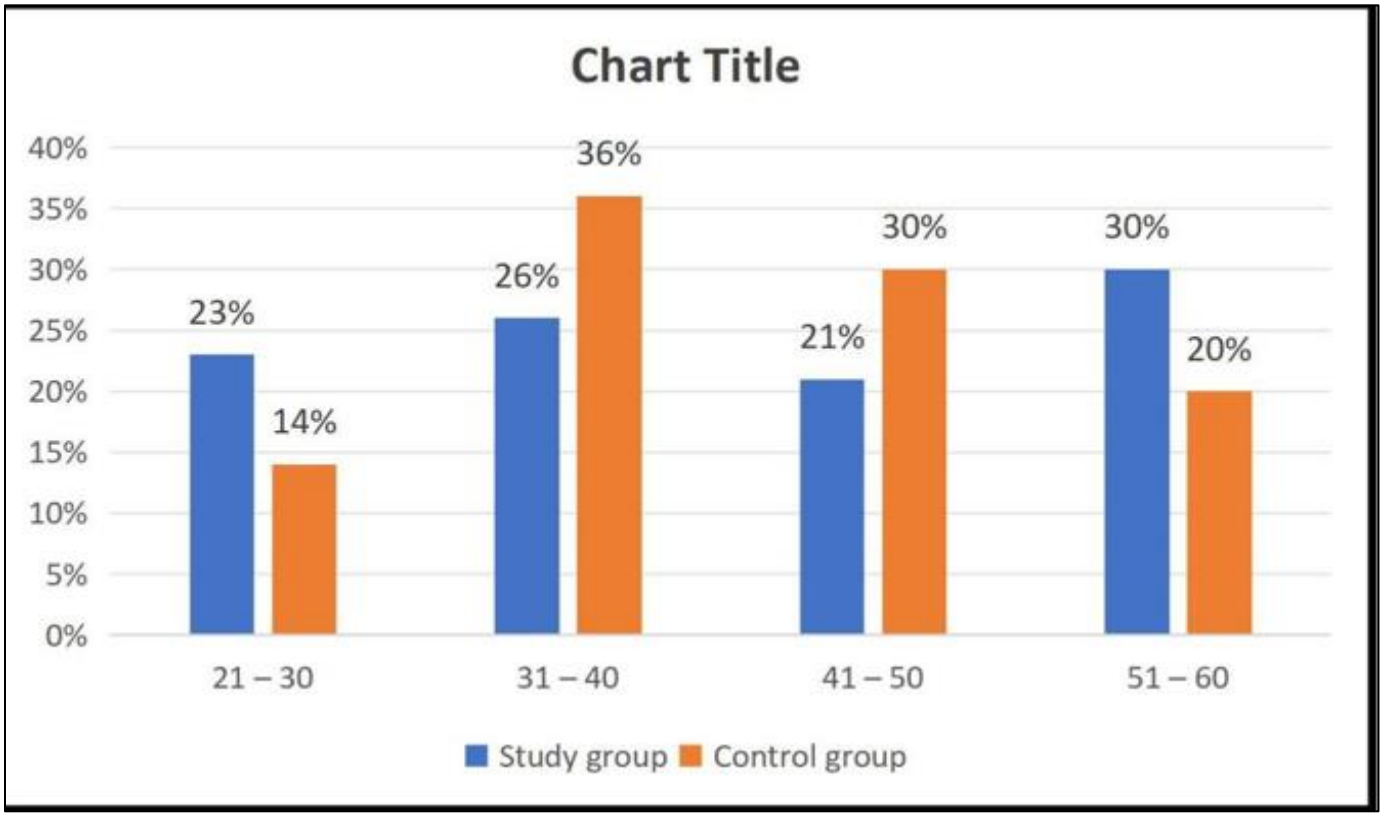

Fig 3: Distribution of the Study Population based on Age

Table 2: Distribution of the Study Population Based on Gender

Gender	Study group		Control group		Total	
	N	%	N	%	N	%
Male	35	35%	29	29%	64	32%
Female	65	65%	71	71%	136	68%
Total	100	100%	100	100%	200	100%
Chi square test= 0.82, p=0.36, Not statistically significant						

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In this study, 32% of participants were male and 68% were female. Gender distribution was similar across groups, indicating gender equivalence.

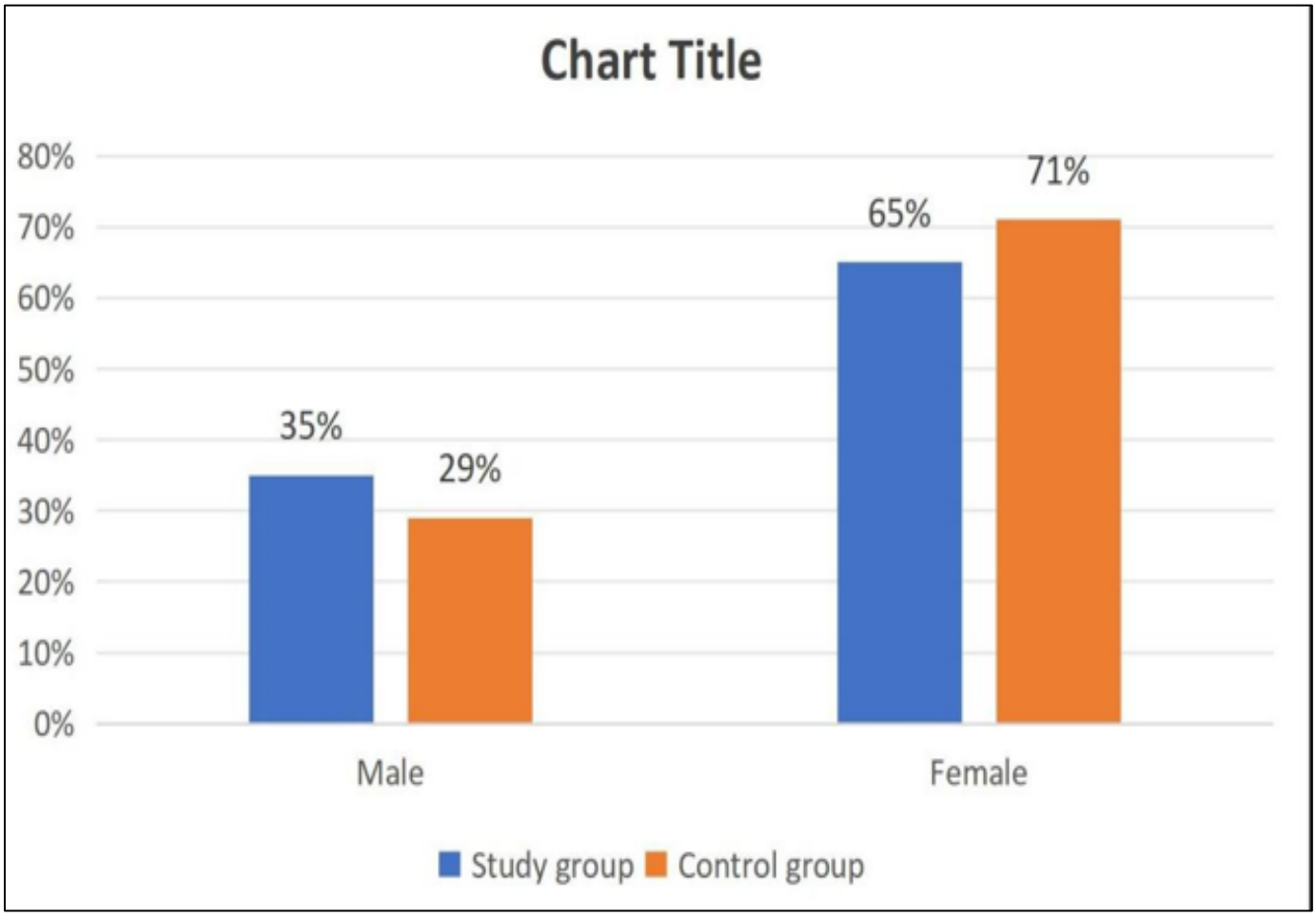

Fig 4: Distribution of the Study Population based on Gender

Table 3: Distribution of the Study Population based on FBS

FBS mg/dl	Study group		Control group		Total	
	N	%	N	%	N	%
<80	8	8%	8	8%	16	8%
81 – 90	59	59%	56	56%	115	57.5%
91 – 110	33	33%	36	36%	69	34.5%
Total	100	100%	100	100%	200	100%
Mean ± SD	88.31 ± 5.27		88.52 ± 5.33			
Chi square test= 0.78, p=0.94, Not statistically significant						

In this study, 8% had FBS levels below 80 mg/dl, 57.5% between 81-90 mg/dl, and 34.5% between 91-110 mg/dl. The average FBS level was 88.31 ± 5.27 mg/dl. Statistical analysis revealed no significant difference in average FBS levels among the groups, indicating comparability.

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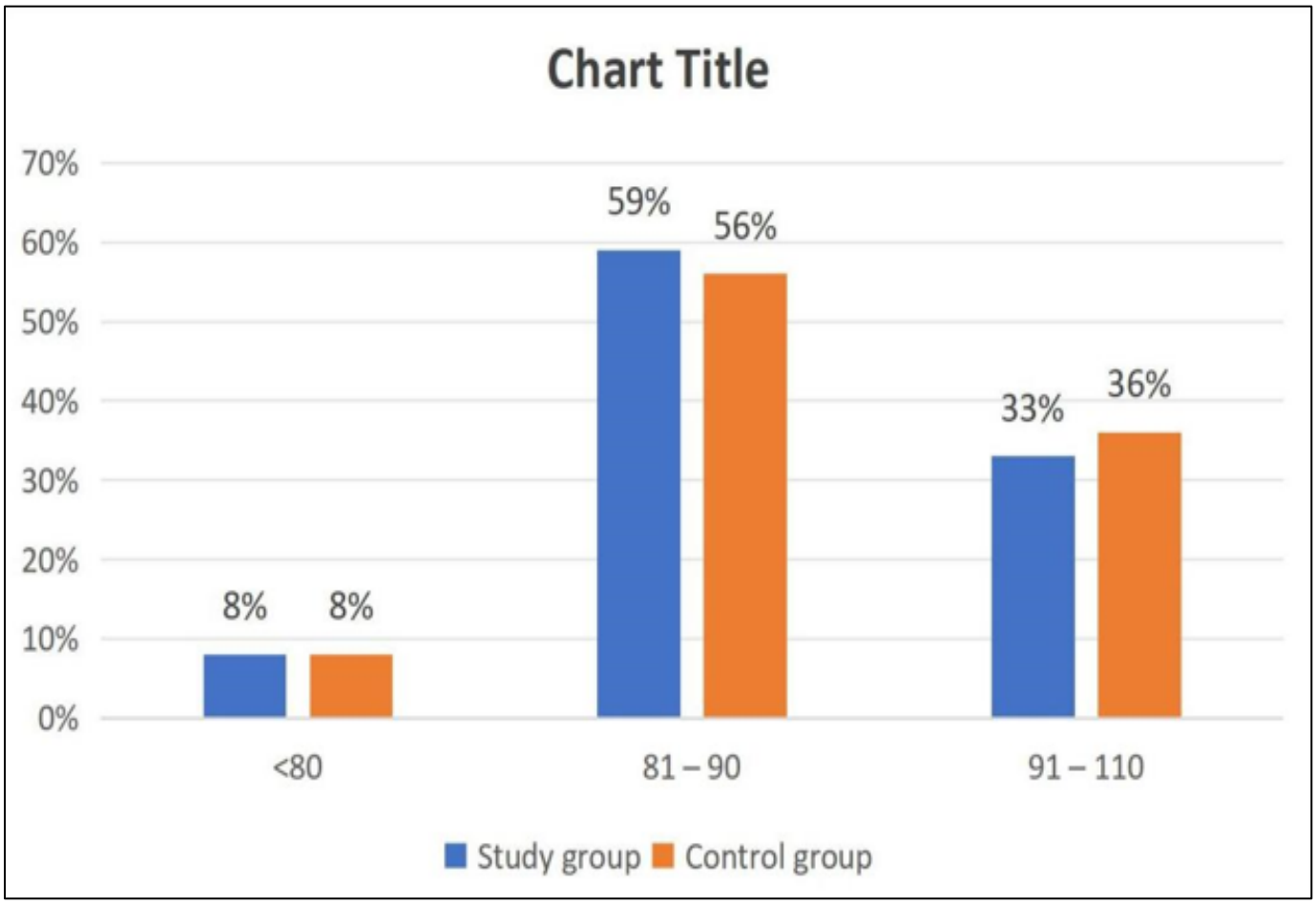

Fig 5: Distribution of the Study Population based on FBS

Table 4: Distribution of the Study Population based on PPBS

PPBS mg/dl	Study group		Control group		Total	
	N	%	N	%	N	%
<110	47	47%	40	40%	87	43.5%
111 – 120	19	19%	25	25%	44	22%
121 – 130	27	27%	26	26%	53	26.5%
131 – 140	7	7%	9	9%	16	8%
Total	100	100%	100	100%	200	100%
Mean ± SD	112.24 ± 13.68		114.58 ± 12.54			
Chi square test= 1.65, p=0.65, Not Statistically significant						

The current research found that 43.5% of the participants had a postprandial blood sugar (PPBS) level more than 110mg/dl. Additionally, 22% had a PPBS level between 111-120mg/dl, 26.5% had a PPBS level between 121-130mg/dl, and 8% had a PPBS level between 131-140mg/dl. The average PPBS level recorded in the study group was 112.24 ± 13.68 mg/dl. There was no statistically significant difference seen across the groups, indicating that they are equivalent.

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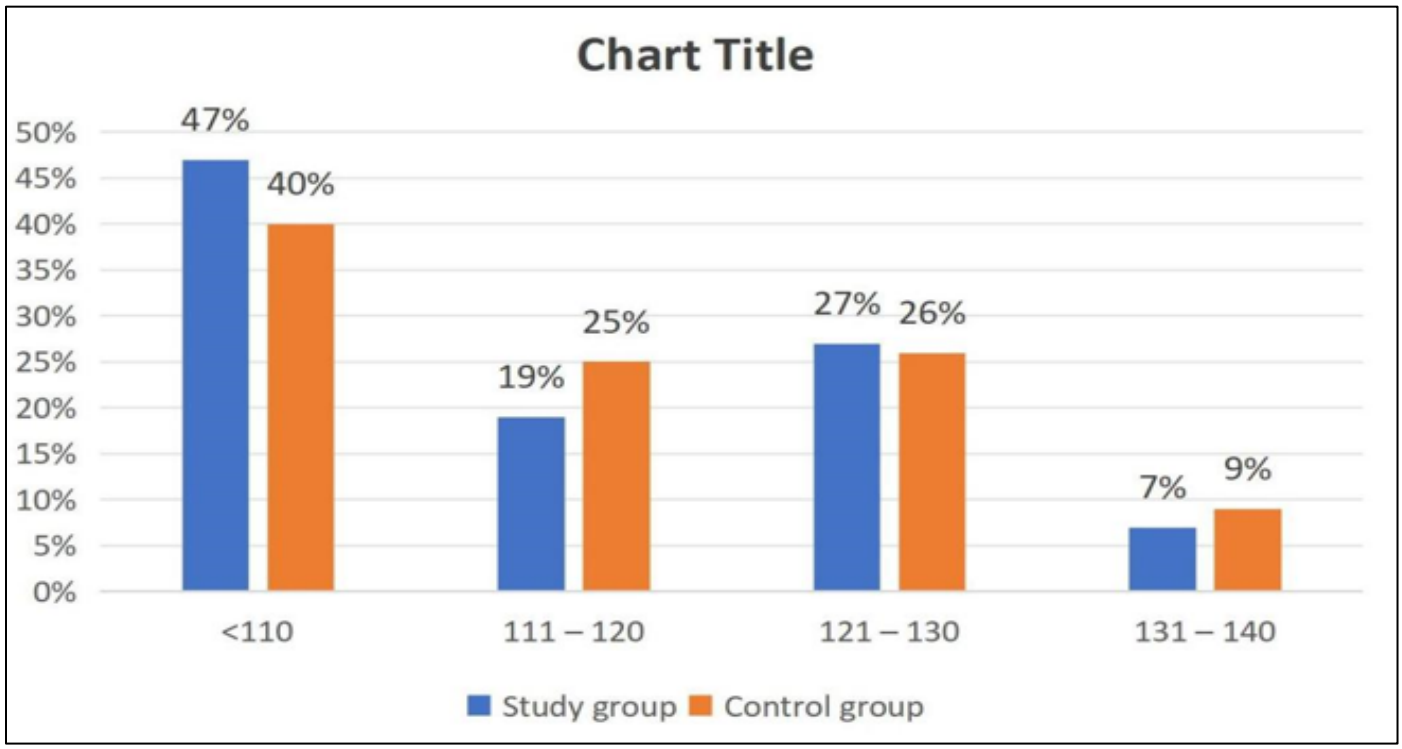

Fig 6: Distribution of The Study Population based on PPBS

Table 5: Distribution of the Study Population based on HB levels – Study Group Pre Correction

Hb g/dl	Frequency	Percentage
≤8	86	86%
8.1 – 8.9	14	14%
Total	100	100%
Mean ± SD	6.87 ± 0.96	

In the study, 86% of participants had hemoglobin levels of 8g/dl or lower, with 14% ranging from 8.1 to 8.9 g/dl. The average hemoglobin level recorded was 6.87 ± 0.96 g/dl.

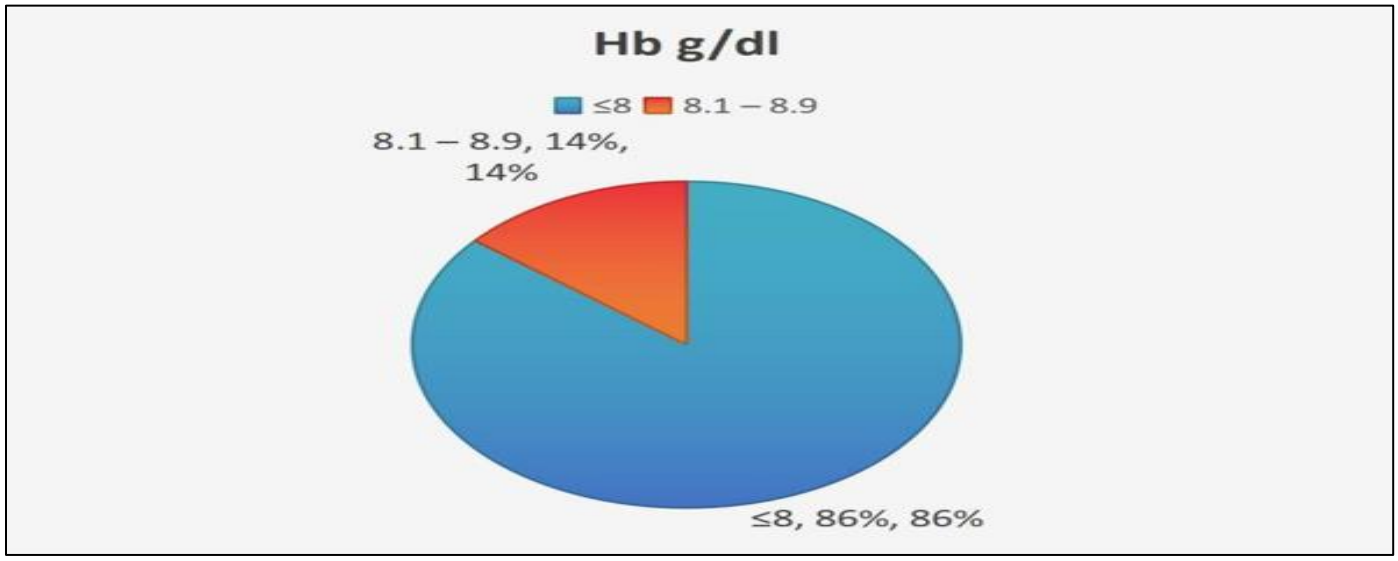

Fig 7: Distribution of the Study Population Based on HB Levels – Study Group Pre Correction

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Table 6: Distribution of the Study Population based on HB Levels – Study Group Post Correction

Hb g/dl	Frequency	Percentage
12 – 12.9	67	67%
>13	33	33%
Total	100	100%
Mean ± SD	12.64 ± 0.42	

After adjustment, 67% of participants had hemoglobin levels between 12-12.9 g/dl, while 33% had levels exceeding 13 g/dl. The average hemoglobin level was 12.64 ± 0.42 g/dl.

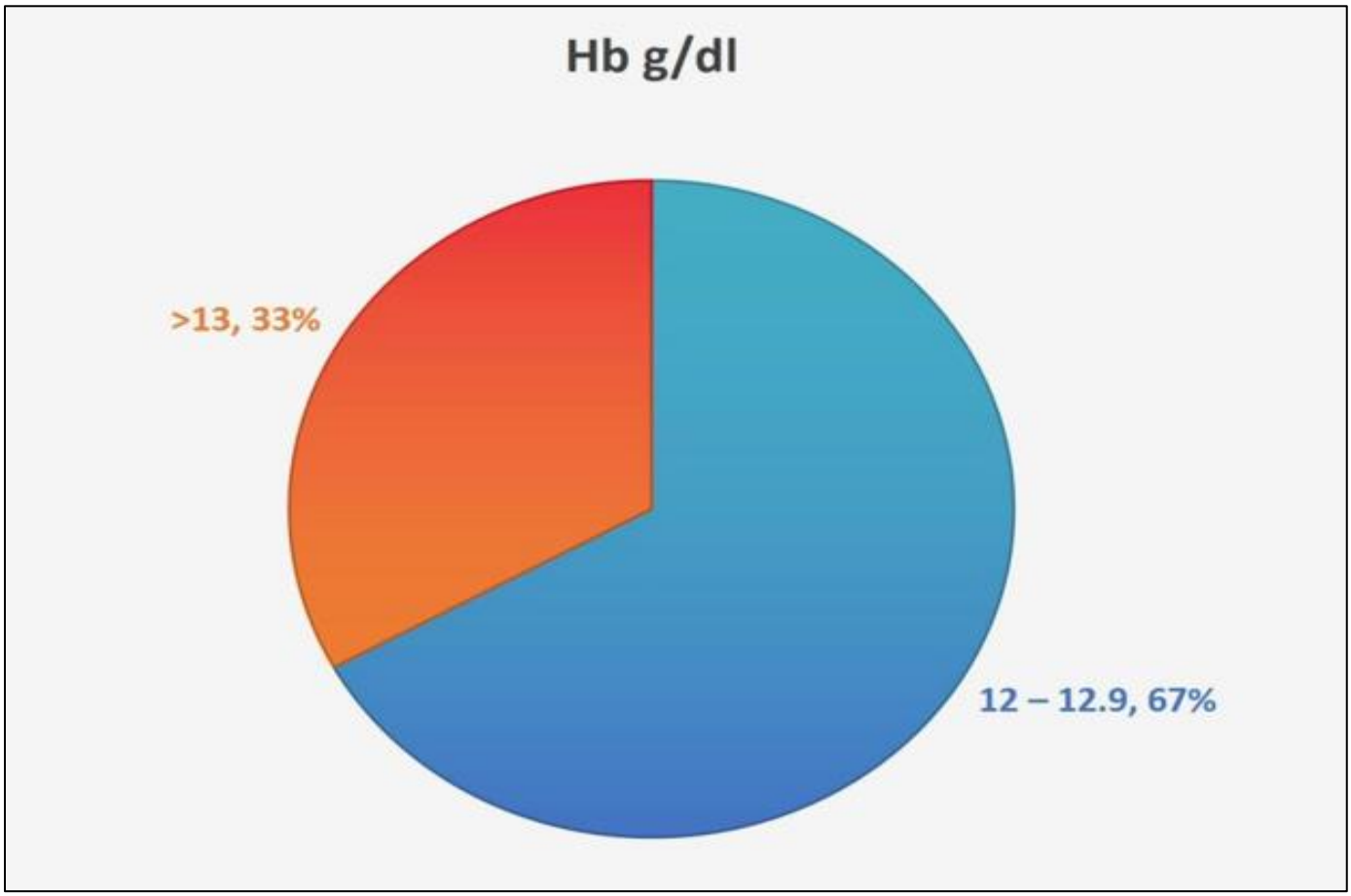

Fig 8: Distribution of the Study Population based on HB levels – Study Group Post Correction

Table 7: Distribution of the Study Population based on HB Levels – Control Group

Hb g/dl	Frequency	Percentage
12.5 – 13.0	18	18%
13.1 – 14	82	82%
Total	100	100%
Mean ± SD	**13.39 ± 0.33**	

In the control group, 18% had hemoglobin levels between 12.5 - 13.0 g/dl, while another 18% had levels between 13.1 - 14 g/dl. The average hemoglobin level was 13.39 ± 0.33 g/dl.

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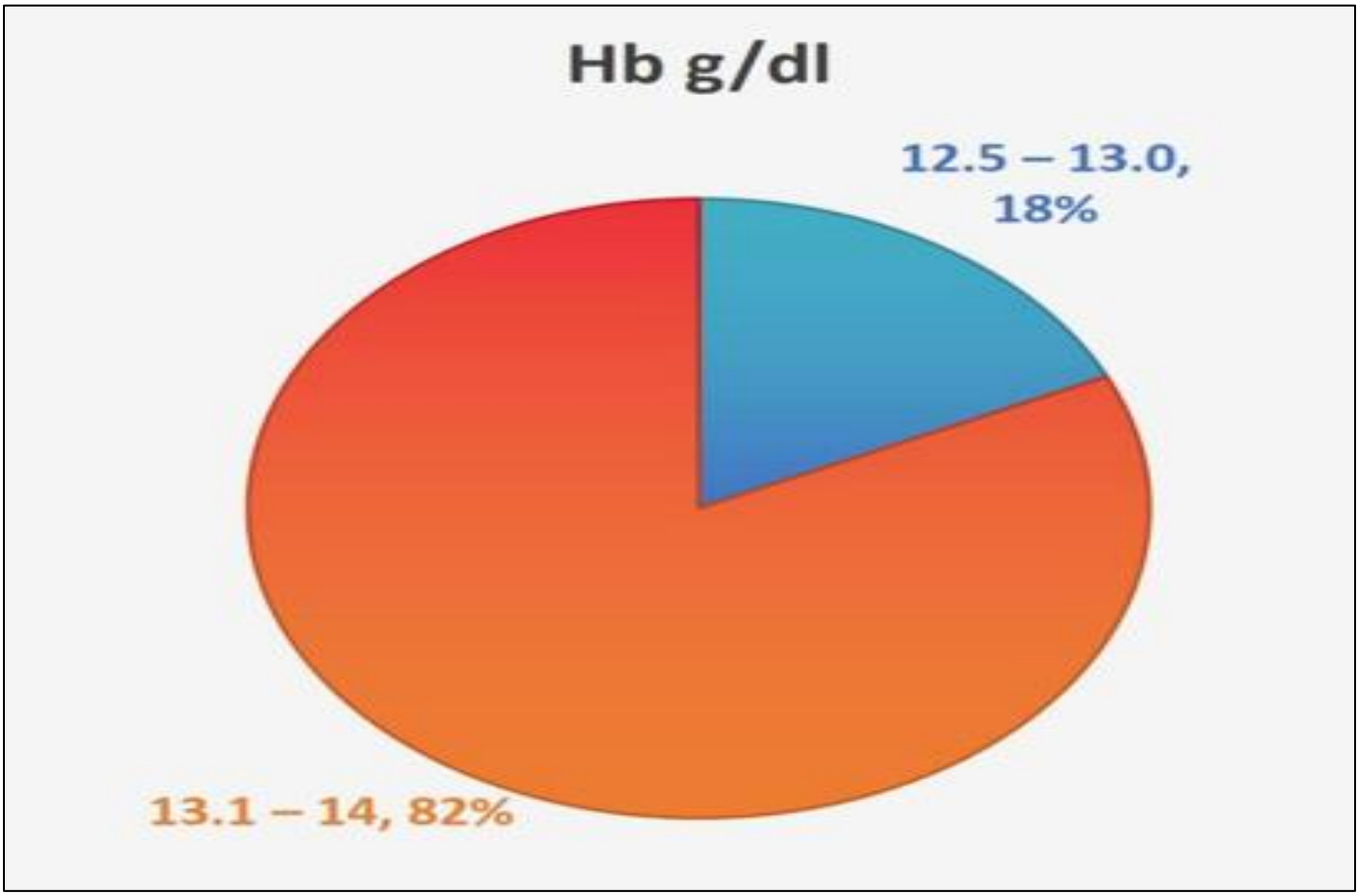

Fig 9: Distribution of the Study Population based on HB levels – Control Group

Table 8: Distribution of Study Population Based on MCV

MCV (fl)	Study Group Pre Correction		Study Group Post Correction		Control Group	
	N	%	N	%	N	%
<80	100	100%	0	0%	0	0%
80 – 90	0%	0%	35	35%	45	45%
91 – 100	0%	0%	65	65%	55	55%
Total	100	100%	100	100%	200	100%
Mean ± SD	64.36 ± 6.71		91.52 ± 2.81		91.38 ± 2.81	
p=0.0001*, Statistically significant						

Regarding MCV, all patients in the research group had MCV values below 80. After adjustment, none of the cases had MCV values below 80. 35% of the cases had MCV values between 80-90fl, while 65% of the cases had MCV values between 91-100.

The average MCV before correction was found to be 64.36 ± 6.71, but after adjustment it was 91.52 ± 2.81. Among the control group, the average MCV was 91.38 ± 2.81. It was noted that there was a substantial rise in the MCV after correction, and the value was almost identical to that of the control group.

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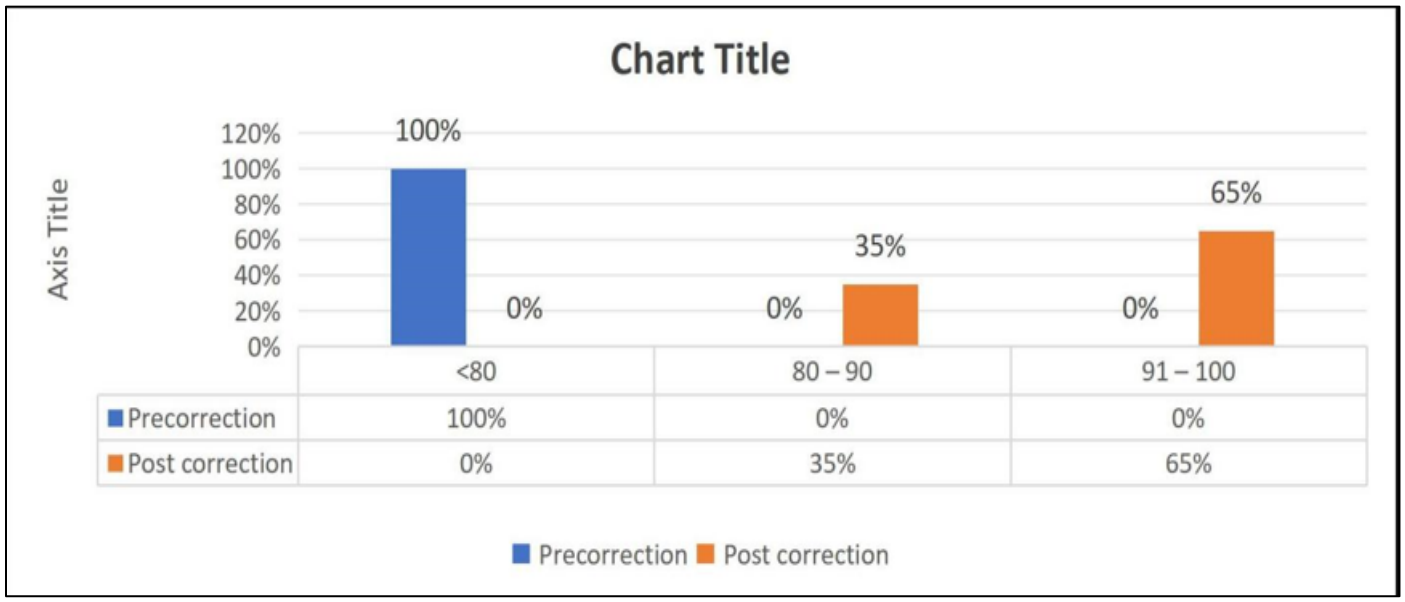

Fig 10: Distribution of Study Population based on MCV

Table 9: Distribution of the Study Population based on MCH

MCH (pg/cell)	Study group pre correction		Study group post correction		Control group	
	N	%	N	%	N	%
<26	100	100%	0	0%	0	0%
26 – 28	0	0%	36	36%	35	35%
>28	0	0%	64	64%	65	65%
Total	100	100%	100	100%	100	100%
Mean ± SD	19.52 ± 2.98		28.38 ± 0.84		28.40 ± 0.83	
Chi square test= 300.03, p=0.0001*, Statistically significant						

In this study, the initial mean corpuscular hemoglobin (MCH) was below 26 pg/cell. After correction, 36% had MCH values between 26-28 and 65% above 28. The pre-correction mean MCH was 19.52 ± 2.98 pg/cell, rising post-correction to 28.38 ± 0.84 pg/cell. This increase was statistically significant, aligning closely with the control group's MCH values.

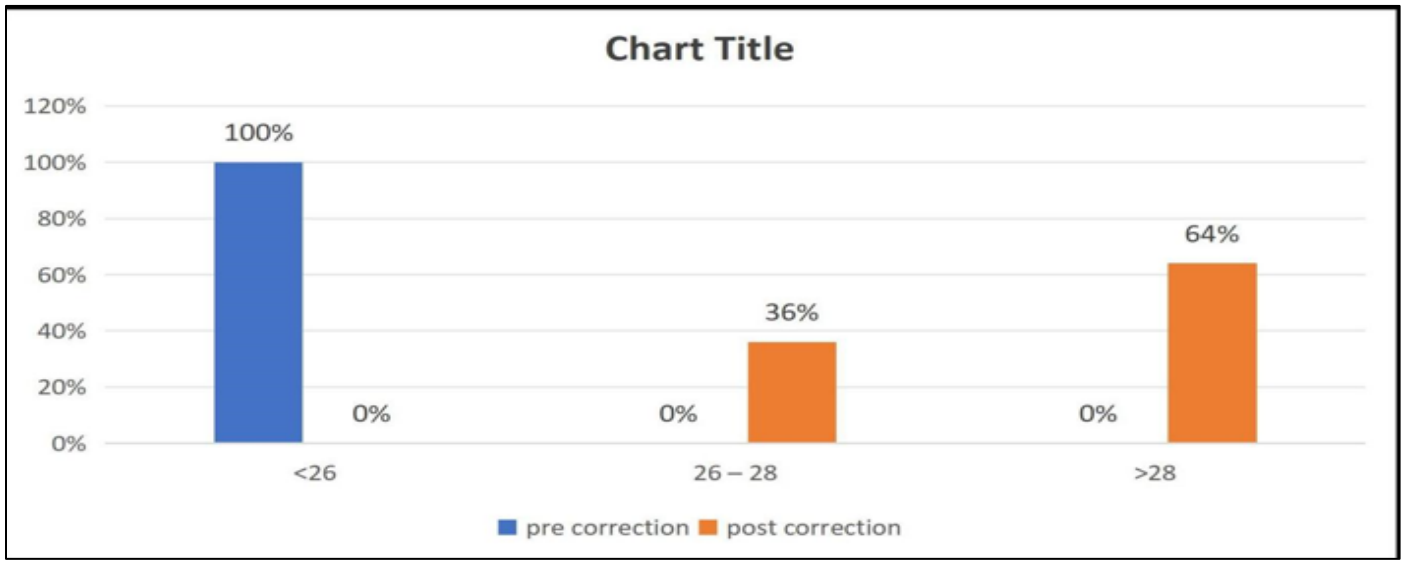

Fig 11: Distribution of the Study Population based on MCH

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Table 10: Distribution of the Study Population based on Serum Iron

Serum Iron (µgm/dl)	Study group pre correction		Study group post correction		Control group	
	N	%	N	%	N	%
<30	100	100%	0	0%	0	0%
31 – 60	0	0%	0	0%	0	0%
61 – 90	0	0%	2	2%	2	2%
91 – 120	0	0%	51	51%	48	48%
>120	0	0%	47	47%	52	52%
Total	100	100%	100	100%	200	100%
Mean ± SD	21.51 ± 4.62		118.04 ± 11.96		117.77 ± 12.74	
Chi square test= 299.19, p=0.0001*, Statistically significant						

Before correction, all study group participants had serum iron levels < 30 mcg/dL. Post- correction, 51% had levels between 91-120, 47% above 120. Average pre-correction level: 21.51 ± 4.62 mcg/dL; post-correction soared significantly to 118.04 ± 11.96 mcg/dL. MCH also rose significantly post-correction, nearly matching the control group level.

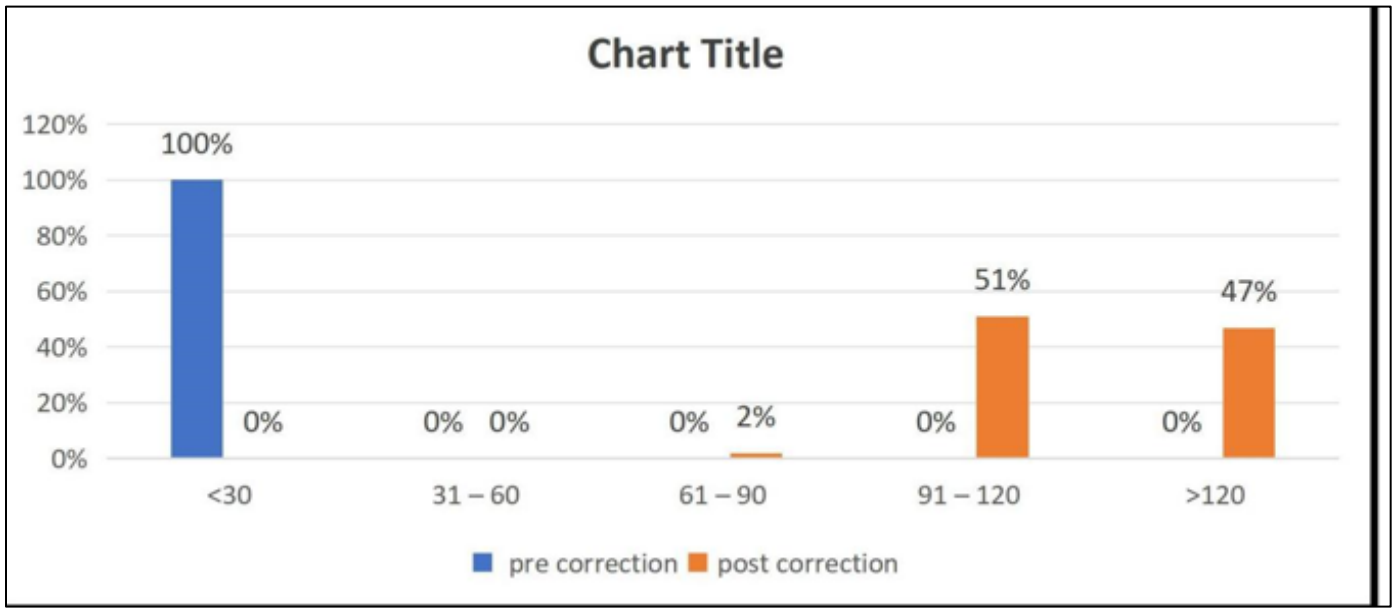

Fig 12: Distribution of the Study Population based on Serum Iron

Table 11: Distribution of the Study Population based on Ferritin Levels

Serum Ferritin (g/L)	Study group pre correction	Study group post correction	Control group
Mean ± SD	6.89 ± 1.42	238.48 ± 25.25	231.52 ± 29.80
F value= 3406.41, p=0.0001*, Statistically significant			

In this study, the average level of serum ferritin was measured during the pre-correction phase and found to be 6.89 ± 1.42g/L. During the post-correction phase, the average level increased to 238.48 ± 25.25g/L. Serum ferritin levels notably improved post-correction, resembling those of the control group.

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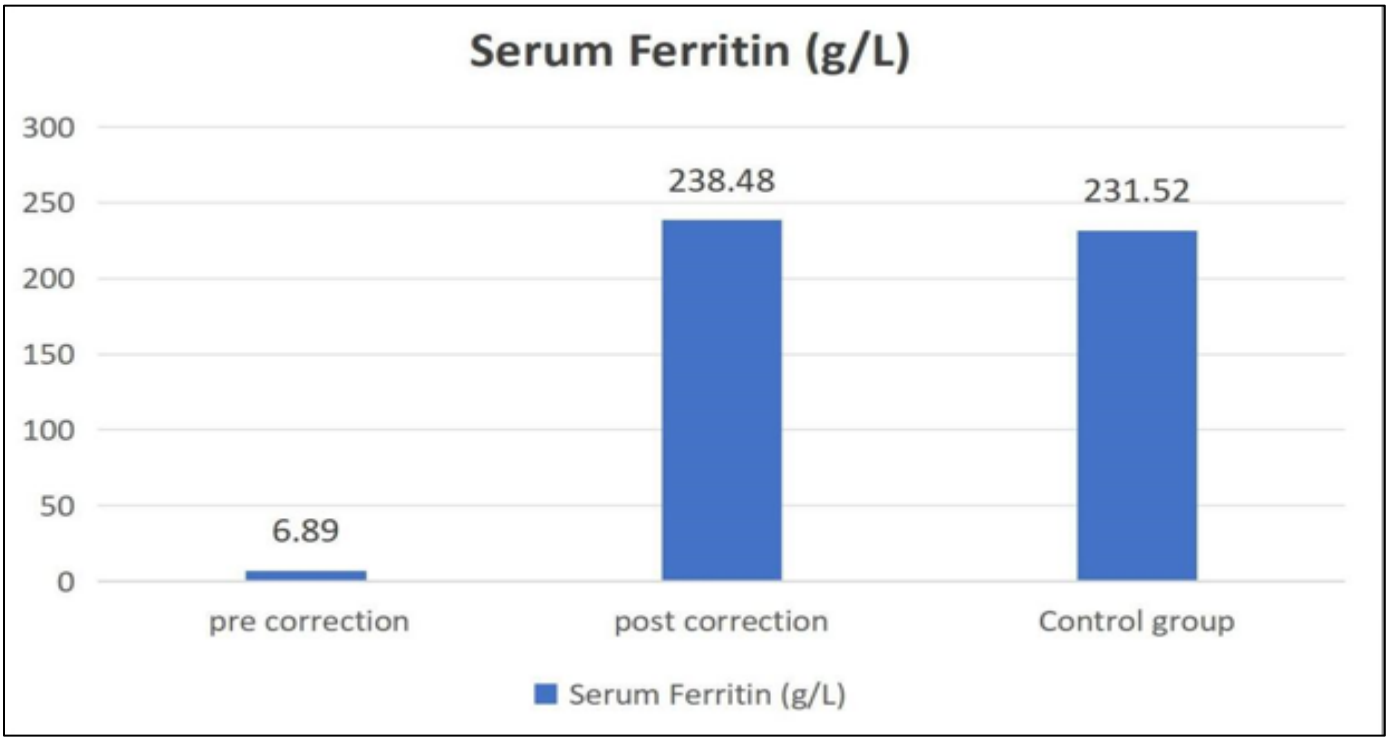

Fig 13: Distribution of the Study Population based on Ferritin Levels

Table 12: Distribution of the Study Population based on HbA1c Levels

HbA1c (%)	Study group pre correction		Study group post correction		Control group	
	N	%	N	%	N	%
4.5 – 5	88	88%	0	0%	74	74%
5.1 – 5.5	12	12%	28	28%	26	26%
5.6 – 6.0	0	0%	47	47%	0	0%
6.1 – 6.5	0	0%	25	25%	0	0%
Total	100	100%	100	100%	200	100%
Mean ± SD	4.60 ± 0.31		5.80 ± 0.31		5.01 ± 0.16	
Chi square test= 233.72, p=0.0001*, Statistically significant						

In this study, it was found that 88% of the cases had HbA1C levels between 4.5-5 during the pre-correction period. Among the remaining cases, 12% had HbA1C levels between 5.1-5.5. During the post-correction period, 28% of the cases had HbA1C levels between 5.1-5.5, 47% had levels between 5.6-6.0, and 25% had levels between 6.1-6.5. During the ore correction period, the average HbA1C level was found to be 4.60 ± 0.31. In the post correction period, it increased to 5.80 ± 0.31. It was observed that the HbA1C level was significantly higher in the post correction period compared to the pre correction period, and it was nearly the same as the level observed in the control group.

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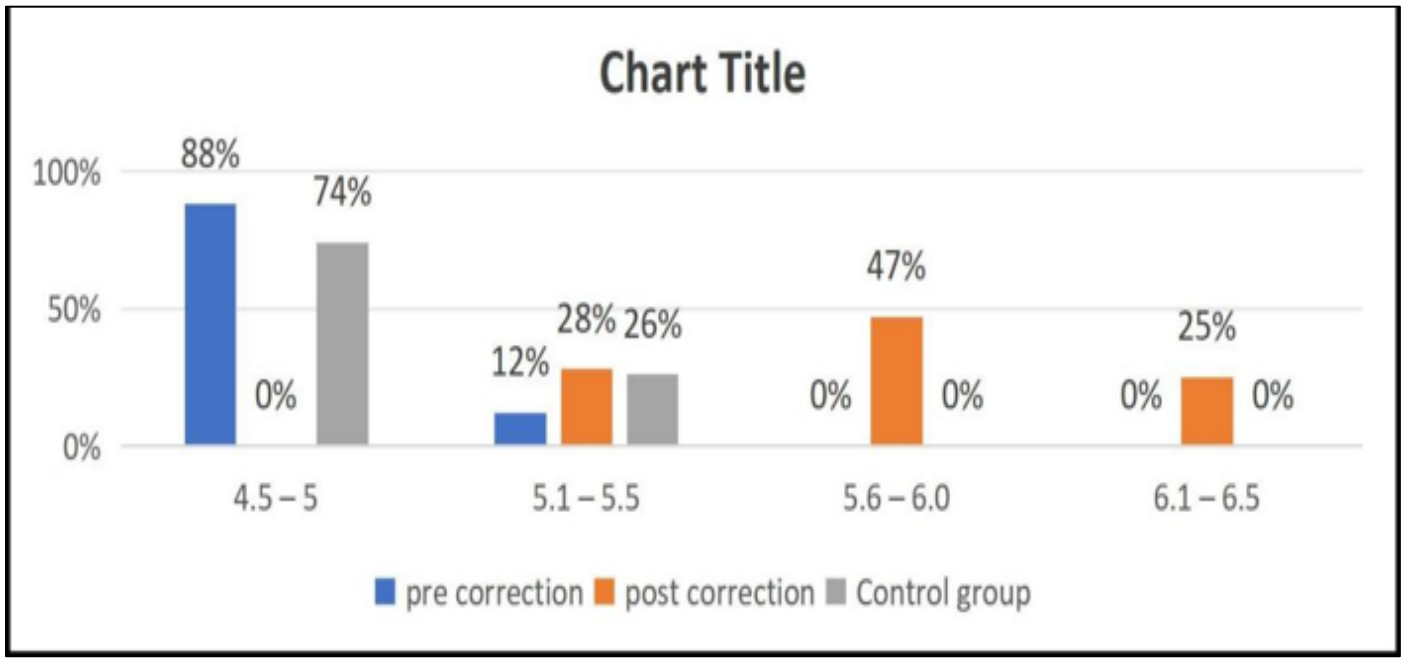

Fig 14: Distribution of the Study Population based on HbA1c Levels

Table 13: Mean Difference in Pre and Post Correction for Hb and HbA1c Values

	Pre	Post	Mean difference	P value
Hb levels	6.78 ± 1.13	12.64 ± 0.42	5.86	0.0001*
HbA1c	4.60 ± 0.31	5.80 ± 0.31	1.19	0.0001*

During the pre-correction period, the Hb level was 6.78 ± 1.13 g/dl. In the post- correction phase, it increased significantly to 12.64 ± 0.42 g/dl.

During the pre-correction phase, the average HbA1C level was 4.60 ± 0.31. In the post- correction period, the average HbA1C level increased to 5.80 ± 0.31. It was found that the mean HbA1C level was substantially higher during the post-correction period compared to the level seen during the pre-correction period.

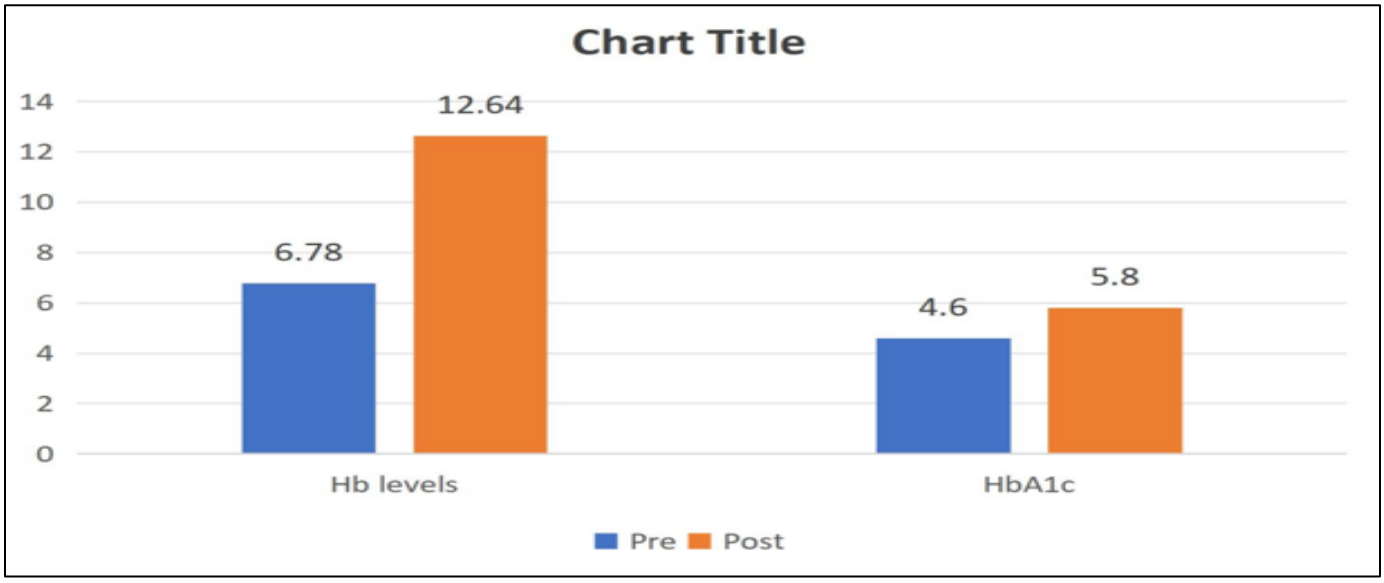

Fig 15: Mean Difference in Pre and Post Correction for Hb and HbA1c Values

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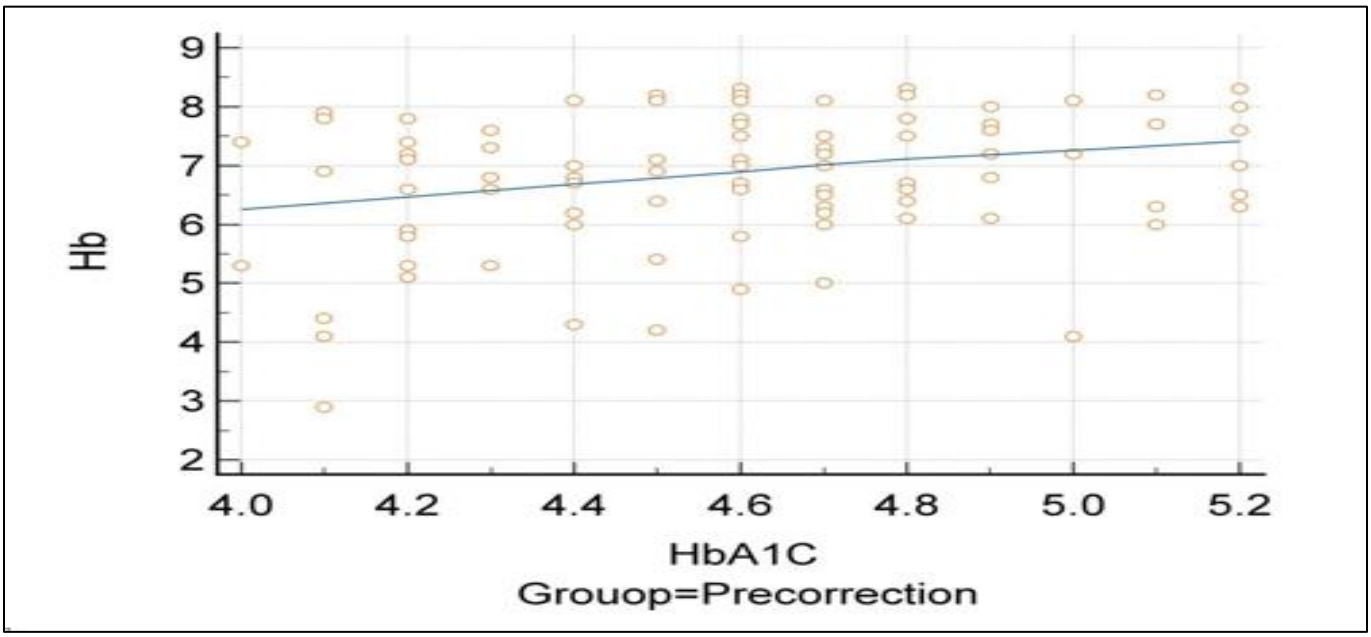

Table 14: Correlation between HbA1c and Hb levels before correction

Sample size	**100**
Correlation coefficient r	0.3131
Significance level	P=0.0015
95% Confidence interval for r	0.1243 to 0.4800

The study found a significant association between Hb and HbA1C levels in the pre- correction phase.

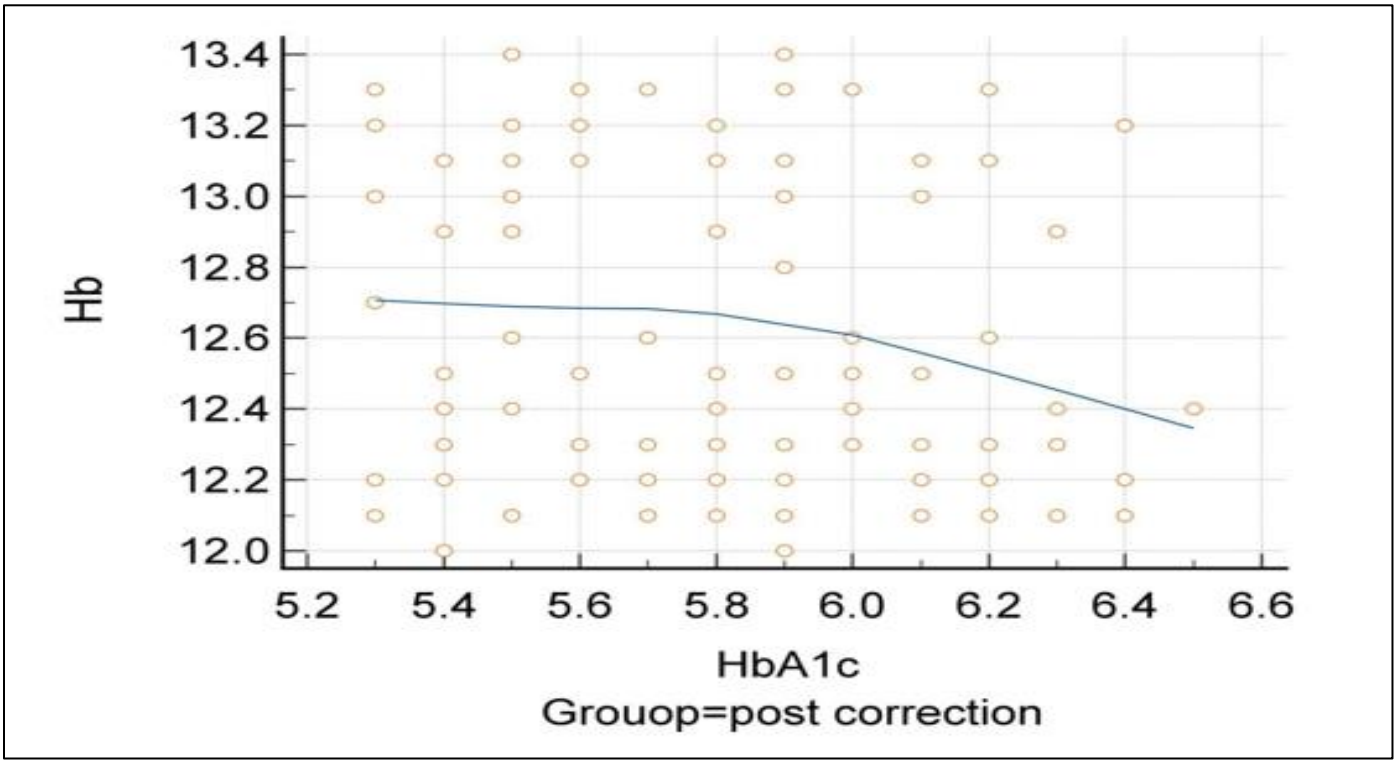

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Table 15: Correlation between HbA1c and Hb Levels Post Correction

Sample Size	**100**
Correlation coefficient r	-0.1723
Significance level	P=0.0865
95% Confidence interval for r	-0.3566 to 0.02498

The current study found a negative correlation between Hb and HbA1C levels. After correction, there was an increase in HbA1C levels, but this observation was not statistically significant. However, it was observed that as Hb levels increased, HbA1C levels also increased.

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CHAPTER FIVE
DISCUSSION

Iron deficiency anemia is the predominant kind of anemia. HbA1c is a form of hemoglobin that has been attached to sugar molecules, and it may be used to evaluate the blood sugar control of a diabetic patient during the last three months. In addition to blood sugar, HbA1c levels may be influenced by other illnesses such as hemolytic anemia's, hemoglobinopathies, acute and chronic blood loss, pregnancy, and uremia.

The research will examine HbA1c levels in Iron Deficiency Anemia patients and the impact on these levels post-treatment. Study objectives include exploring HbA1c changes in this context.

- To analyse HbA1c levels in Iron Deficiency Anaemia patients.
- To investigate how HbA1c levels change after correcting Iron Deficiency Anaemia.

A. Socio Demographic characteristics:

In the present study, 18.5% of the participants were aged between 21-30 years, 31% were aged between 31-40 years, 25.5% were aged between 41-50 years, 25%. The participants' ages ranged from 51 to 60 years, with a mean age of 41.37 ± 11.30 years. There was no significant age difference among the groups, making them comparable in this regard.

In the study by Sinha N et al., the mean age was about 30.3 years, which was 10 years younger than the mean age in our study.

In the study done by Intra J et al.,[82] the mean age of participants during the tests was approximately 43 years, closely aligned with the present study's observed average age. In a study by Jeyaprekash N et al.,[83], the mean age of participants was around 34 years, a decade younger than in the present study.

In the present study 32% of the participants were male and 68% were female. There was no statistically significant difference across the groups in terms of gender distribution thus they stand comparable in terms of gender.

In the study done by Sinha N et al.,[81]68% of the participants were female and 32% of the cases were male and this gender distribution was on close consonance with the present study findings.

In the study done by Intra J et al.,[82] Additionally, a notable majority of females were observed among the subjects with diabetes, aligning with the current study's findings.

In the study done by Jeyaprakash N et al.,[83] they have stated that there was a stron female preponderance in the study and this was observed across majority of the studies which might be because of increased prevalence of IDA among women.

B. Hb Levels:

In this study, 86% of subjects had Hb levels ≤8g/dl, while 14% had Hb levels between 8.1-8.9 g/dl. The overall mean Hb was 6.87 ± 0.96 g/dl.

In the study done by **Kumar C et al.,**[84] they found that the mean HbA1C levels were lower in the cases after 2 months of treatment, but this study did not observe a significant difference, although a difference was noted, it was not statistically significant. In the study done by **Sinha N et al.,**[81] severe anaemia was seen among 76% of the cases, 24% were having moderate anaemia and no cases showed mild anaemia, aligning closely with the current study's findings.

In the study **Mohammad et al.,**[85] they found that 8.3% had mild anaemia, 66.7% moderate, and 25% severe.

In the present study, post correction 67% of the subjects had Hb levels 12-12.9g/dl, 33% of the cases had it Hb >13 g/dl. The overall mean Hb observed in the present study was 12.64 ± 0.42 g/dl.

In the control group, 18% of the subjects had Hb between 12.5-13.0, 18% of the subjects had Hb between 13.1-14g/dl and the

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mean Hb level was observed to be 13.39 ± 0.33 g/dl.

Table : HB Values Pre and Post Correction in Different Studies

Studies	Pre correction (g/dl)	Post correction (g/dl)
Present study	6.87 ± 0.96 g/dl.	12.64 ± 0.42
Sinha N et al.,[81]	6.2±2.1	12.5±1.0
Mohammed et al.,[85]	10.41±1.11	14.09±1.33
Jeyaprekash N et al.,[83]	6.71 ±1.0	12.6±0.446

C. Changes in Blood Parameters:

All patients in the study initially had MCV values below 80, which changed after adjustments. None exceeded 80. 35% had MCV between 80-90fl, while 65% fell between 91-100.

The initial MCV was 64.36 ± 6.71, increasing significantly to 91.52 ± 2.81 post- adjustment, similar to the control group's mean MCV of 91.38 ± 2.81.

The study revealed that initially, the mean corpuscular hemoglobin (MCH) in the study group was below 26 pg/cell, but after correction, 36% showed MCH values between 26- 28, and 65% had values above 28. The MCH mean increased significantly from 19.52 ± 2.98 to 28.38 ± 0.84 pg/cell post-correction, aligning closely with the control group's levels.

In **Mohamed et al.'s** study,[85], it was found that individuals with anemia had lower average morphological characteristics in their red blood cells (RBC). These parameters improved post-therapy, consistent with our current findings.

In the research conducted by **Manisha G et al.**[86], a statistically significant difference was observed and reported in the average values of red blood cells (RBC) and hemoglobin. (Hb), mean corpuscular volume (MCV), and mean corpuscular hemoglobin (MCH) between the patients and controls. This finding aligns with the observations made in the current investigation.

Before correction, participants' serum iron levels were below 30 mcg/dl. After correction, 51% showed levels between 91-120 and 47% had levels above 120. Pre-correction, the average serum iron level was 21.51 ± 4.62 mcg/dl, significantly increasing post-correction to 118.04 ± 11.96 mcg/dl. There was a notable rise in MCH post-correction, nearing the control group’s level.

The study conducted by **Intra et al.** [82] found significant variations in the average levels of hematological and biochemical parameters differ between individuals with iron deficiency anemia (IDA) and the control group.Specifically, subjects with IDA had lower levels of hemoglobin, hematocrit, red blood cell count, mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC). The ferritin levels were similar to those in the control group, aligning with the current study's observations.

During the study, serum ferritin levels increased significantly from 6.89 ± 1.42g/L in the pre-correction phase to 238.48 ± 25.25g/L in the post-correction phase, aligning closely with the control group's levels.

Table : Ferritin Values Pre and Post Correction in Different Studies

Studies	Pre correction (g/L)	Post correction (g/L)
Present study	6.89 ± 1.42	238.48 ± 25.25
Sinha N et al.,[81]	7.0±3.1	232.8 ± 76.7
Mohammed et al.,[85]	26.17 ± 4.36	270.4±23.6
Jeyaprekash N et al.,[83]	6.871 ±1.5	237.239 ±25.267

The current study found a significant difference in serum iron, TIBC, and Transferrin saturation between healthy control and cases with iron deficiency anemia, both before and after correction. This finding aligns with the observations made in a previous study by **Mahmood et al.,**[85].

In the research conducted by **Kumar C et al.,**[84], The average initial iron levels in the blood were significantly lower in

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patients than the control group, consistent with the current investigation's observations. Similarly, patients had notably lower initial serum ferritin levels compared to controls, in line with the study's findings.

In the study conducted by **Sinha et al.,**[81], The study revealed lower initial serum ferritin levels in patients than in the control group. Additionally, anemic patients showed increased hemoglobin and serum ferritin levels after 2 months of iron treatment. These findings are consistent with the results of the present study and the observations made in the study conducted by **Mohamed et al.,**[85].

D. Blood Sugar Levels:

In the present study 8% of the participants had FBS <80 mg/dl, 57.5% of the subjects had FBS between 81-90 mg/dl, 34.5% of the subjects had FBS between 91-110 mg/dl and the mean FBS was 88.31 ± 5.27 mg/dl in the study group, showing no statistically significant difference between the groups, making them comparable.

The study found that 43.5% had PPBS >110mg/dl, 22% had it between 111-120, 26.5% between 121-130, and 8% between 131-140; the mean PPBS in the group was 112.24 ± 13.68 mg/dl. No significant statistical difference was found among the groups, indicating their comparability.

In the study done by **Mohamed et al.,**[85] majority did not show variation sin FBS and PPBS with changes in anaemia.

In the study done by **Jeyaprakash N et al.,**[83] they reported no statistically significant difference in mean FBS and PPBS between the cases and controls, aligning with this study's findings.

E. Modifications in HbA1C after Correction:

Glycated hemoglobin (HbA1c) is the preferred criterion for assessing the extent of long- term glycemic control in individuals with diabetes mellitus (DM)[87].

It provides an indication of the patient's blood sugar levels throughout the last 3 months. HbA1c is often used as a screening tool for diabetes mellitus. Moreover, a HbA1c level of ≥6.5% (48mmol/mol) is advised as the threshold for diagnosing DM.

HbA1c is a kind of hemoglobin that has a glucose molecule connected to the end of one or both of its HbA beta chains, specifically to the terminal NH2 group (valine residue). Red blood cells allow glucose to pass easily and have similar glucose levels to plasma, making HbA1c a precise indicator of glycemic control over months based on their lifespan. HbA1c assesses glycemic changes through serial readings.

This helps evaluate whether patients are meeting their HbA1c objectives and may also be used as a diagnostic tool for diabetes mellitus [89]. Various variables may influence or disrupt HbA1c findings, depending on the measurement methods used[87].

Before correction, 88% of cases had HbA1C levels between 4.5-5, with 12% between 5.1-5.5. After, levels were 25% between 6.1-6.5, 47% between 5.6-6.0, and 28% between 5.1-5.5.

During the ore correction period, the average HbA1C level was 4.60 ± 0.31, increasing to 5.80 ± 0.31 post correction. The post correction levels significantly surpassed the pre- correction values and closely matched those of the control group.

Table : Hba1c Values Pre and Post Correction in Different Studies in Comparision to Present Study

Studies	**Pre correction**	**Post correction**
Present study	4.60 ± 0.31	5.80 ± 0.31
Sinha N et al.,**[81]**	4.60 ± 0.6	5.90 ± 0.6
Jeyaprakash N et al.,**[83]**	4.619 ±0.308	5.816 ±0.323

HbA1C levels were initially low in the study group but increased after correction to match the control group, aligning with study observations by Sinha N et al.,[81] Madhu et al.,[90] and Jeyaprekash N et al.,[83]

F. Impact of Hb on HbA1C:

Protein glycation commonly occurs. Hemoglobin reacts non-enzymatically with glucose at the beta chain's N-terminus to form

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a Schiff base, then 1-deoxyfructose via an Amadori rearrangement. Elevated blood glucose causes glucose attachment to red blood cell hemoglobin.As hyperglycemia persists in the bloodstream, there is an increased binding of glucose to hemoglobin in the red blood cells, resulting in elevated levels of glycated hemoglobin. Once a hemoglobin molecule undergoes glycation, it permanently retains that modification. Glycated hemoglobin accumulating in the red blood cell indicates the average glucose level the cell has been exposed to during its lifespan. Glycated hemoglobin measurement evaluates the efficacy of treatment by monitoring the long-term management of glucose levels in the blood. A1c is a calculated average of blood glucose levels across the lifespan of red blood cells, which is around 117 days for men and 106 days for women. Thus, glucose levels that are closer to the test day have a much greater impact on the A1c level compared to values that are farther away from the test date.

In this research, the level of Hb was measured to be 6.78 ± 1.13 g/dl during the pre- correction period. However, during the post-correction phase, the level dramatically increased to 12.64 ± 0.42 g/dl, indicating a substantial improvement compared to the pre-correction period.

During the pre-correction phase, the HbA1C level was 4.60 ± 0.31. In the post- correction period, the HbA1C level increased to 5.80 ± 0.31. It was noted that the mean HbA1C level was considerably higher during the post-correction period compared to the pre-correction period.

The current investigation found a significant correlation between the levels of hemoglobin (Hb) and glycated hemoglobin (HbA1C) during the pre-correction period. A negative correlation was observed between the levels of Hb and HbA1C. After correction, there was an increase in HbA1C levels, but this observation was not statistically significant. However, it was observed that as the levels of Hb increased, the level of HbA1C also increased.

In the research conducted by Mohamad et al.,[85], it was noticed that there was a notable disparity in the levels of HbA1C between the groups before and after treatment. This finding aligns with the results of the current investigation.

The research done by Intra et al. [82] severely anemic individuals had notably higher HbA1C levels compared to non-anemic individuals, but this contradicted the current investigation's results.

The research conducted by Solomon et al.[91] a link was discovered between HbA1C levels and hematological parameters in Iron Deficiency Anemia (IDA) individuals via Pearson correlation analysis. The analysis did not show any significant correlation between HbA1C and RBC, MCV, MCH, or MCHC, consistent with this study's findings.

A research conducted by Silva et al. [92] examined the impact of iron deficiency anemia (IDA) on HbA1c levels in 122 patients. The study definitively concluded that IDA has an effect on HbA1c outcomes, and this effect is influenced by the severity of anemia. The detected changes are statistically significant, but they lack clinical relevance when considering total variability. Similarly, in the current investigation, no statistically significant difference was identified, which aligns with the results of the present study.

In Bharadwaj et al.'s study, anaemic patients had higher HbA1C levels than the control group. Post-treatment, HbA1C levels reduced from 6.6 to 5.74, opposing present study results.

Contrary studies conflict with the current research by Sinha et al.,[81], in this study, they found that the non-anemic group had significantly lower average HbA1C levels compared to anemic individuals from lower socioeconomic backgrounds.The study conducted by Intra J et al. [82] Iron therapy increased HbA1c levels in anaemic individuals, indicating nutritional deficiencies contribute significantly to iron deficiency development, aligning with this study's observations.

According to the source, the specific cause of the patho-physiology of diabetes is diverse. However, in a research conducted by Grossman et al., it was said that the precise mechanism responsible for the anemia in relation to HbA1C levels was not determined. They said that the severity of iron deficiency impacts the lifetime of red blood cells, resulting in a reduced quantity of erythrocytes. This leads to an older population of red blood cells that remain in touch with plasma glucose for a longer duration, generating inaccurately elevated HbA1c values. In the studies conducted by Selvaraj N et al.,[94] and Zaka- Ur-Rab Z et al.,[95], Iron deficiency disrupts antioxidant defense, increasing enzyme activity and lipid peroxidation.In the research conducted by Del Rio D et al.,[96], Individuals with iron deficiency anemia (IDA) showed significantly higher malondialdehyde levels than the control group, indicating increased oxidative stress.

The research conducted by Selvaraj N et al.,[94] and Zaka- Ur-Rab Z et al.,[95] have shown that iron supplementation has

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been found to reduce levels of oxidative stress indicators, namely decreasing MDA levels, in youngsters. Decreased MDA levels lead to a reduction in the glycation of hemoglobin, hence minimizing the impact on HbA1c readings.

In Mitchell et al.'s In the study, researchers found no statistically significant difference in HbA1C levels before and after treatment, which aligns with our current findings.

The study conducted by Van Heyningen et al,[98], no significant difference in HbA1C levels was found between the anaemic and control groups. The researchers suggested that any observed variations were likely caused by changes in the methodology employed to quantify HbA1C.

G. Implications Drawn in Comparison to Literature:

Anemia caused by a lack of iron is the prevailing kind of anemia in India. In various studies conducted by Brooks et al.,[99] Sluiter et al.,[100], and Mitchel et al.,[97], research shows a link between iron deficiency anemia (IDA) and HbA1C levels, explaining it through pathophysiology and RBC age influence.

In subsequent investigations conducted by Heyningen et al. [98] and Hansen et al. [101], it was concluded that there was no discernible disparity in HbA1C levels between the experimental group and the control group. These findings support the current research results.

In Rai et al.'s[102] study, various methods were tested to measure HbA1C. They found that patients with iron deficiency anemia (IDA) had high HbA1C levels, which decreased after iron therapy.

In their study, Christy et al.[103] discovered that anaemia can artificially elevate HbA1C levels, emphasizing the importance of meticulous treatment adjustments for diabetic patients.This finding is supported by Mohamed et al.,[85], Individuals with fasting plasma glucose levels of 100-126 mg/dl showed notably elevated HbA1C levels, as per the observation made by [name].They concluded that anaemia may amplify the representation of glycemic status in these individuals. However, our current study cannot definitively establish this finding due to the fact that most of the participants included in the study had fasting blood sugar (FBS) and postprandial blood sugar (PPBS) levels that were close to normal. As a result, the HbA1C levels were not significantly elevated enough to demonstrate statistically significant differences. This is consistent with the findings of previous studies conducted by Kim C et al.,[104]. Sinha et $_{al.}$ (81)

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CHAPTER SIX
CONCLUSION

The conclusion suggests conducting larger studies with more participants to assess HbA1C changes in individuals with Anaemia and Diabetes. Considering the impact of iron deficiency in monitoring Prediabetes and Diabetes individuals is crucial. Therefore, measuring iron, Hb, and HbA1C in diabetic patients simultaneously helps understand their glycemic status accurately and improves the interpretation of their hyper-glycemic condition.

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ANNEXURES

INFORMED CONSENT ACCEPTANCE SHEET

Patient I.D. No.

Title of the Project: "A STUDY ON HOW IRON DEFICIENCY ANEMIA AFFECTS HBA1C LEVELS IN THOSE WITHOUT DIABETES AND THOSE WHO ARE PRE-DIABETIC".

Principal Investigator Name: Dr.M.NAGA PRADEEP

Contact No:

Information Sheet: I have read the information sheet carefully/ explained in the language I understand, and I fully understand the contents. I confirm that I have had an opportunity to askquestions. I have been explained in detail about the nature and purpose of the study and its potential risks/benefits, and the expected duration of the study and other related details of the study. I understand that my participation is voluntary and i am free to withdraw at any time without giving any reason, my medical care and legal rights.

I agree to participate in the study.

.....
(signature / left) Date:

Place:
Name of the participant:.....
Son/ Daughter/ Spouse of:.....
Complete postal address: .....

This certifies that the above consent has been taken in my presence.

.....
(Signature of principal investigator)

Date: .....
Place: .....
1) witness- I..... 2) Witness - II .....

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PROFORMA

NAME:

DOA:

AGE:

DOD:

IP NO.:
PRESENTING COMPLAINTS: DETAILED HISTORY: GENERAL EXAMINATION: VITALS:

TEMP:

BP:

PULSE:

RR:

SYSTEMIC EXAMINATION: CVS:

CNS:

RS:

P/A:

INVESTIGATIONS
COMPLETE HEMOGRAM
Hb (g/dL)
TC (cells/cu.mm) DC
RBC (millions/cu.mm) Platelets (Lakhs/cu.mm) PCV

ANAEMIA PROFILE
Peripheral smear Serum Iron Serum ferritin TIBC
Transferrin saturation
Vit B 12 and folic acid level

HBA1C

URINE ANALYSIS
Albumin Sugar Deposits
BLOOD SUGAR (MG/DL)—FBS/PPBS/GTT LIVER FUNCTION TEST
RENAL FUNCTION TEST
BLOOD UREA (mg/dl) CREATININE (mg/dl)
Serum Electrolytes ECG in all leads Chest x ray PA view
USG Abdomen & pelvis
DURING FOLLOW UP AT THIRD MONTH
History Examination
Complete hemogram and anaemia profile HbA1c levels

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MASTER CHART

STUDY GROUP PRE CORRECTION														STUDY GROUP POST CORRECTION								CONTROL GROUP											
SNO	AGE_S	SEX_S	FBS_S	PPBS_S	Hb_S1	MCV_S1	MCH_S1	MCHC_S1	SIRON_S1	TIBC_S1	ISATN_S1	FERRITIN_S1	HbA1C_S1	Hb_S2	MCV_S2	MCH_S2	MCHC_S2	SIRON_S2	TIBC_S2	ISATN_S2	FERRITIN_S2	HbA1C_S2	AGE_c	SEX_c	FBS_c	PPBS_c	Hb_c	MCV_c	MCH_c	MCHC_c	SIRON_c	TIBC_c	ISATN_c	FERRITIN_c	HbA1C_c
1	25	F	87	128	8.1	68.9	23.7	29.2	22.6	397	5.7	5.5	5	12.6	92.3	27.8	33.6	108.78	312	34.86	241.5	5.5	36	F	78	90	13.4	90.4	27.9	33.2	87.8	306	28.69	185.4	5.1
2	34	F	85	93	6.3	62.9	20.4	27.8	13.27	475	2.79	3.9	5.2	12.4	90.5	26.9	34.1	120.43	328	36.71	254.3	5.8	56	M	86	97	13.8	95.6	29.2	33.1	99.76	312	31.97	306.5	5.2
3	49	F	98	132	2.9	50.5	14.8	29.3	24.5	463	5.3	6.2	4.1	12.1	93.4	30.4	33.1	116.31	310	37.51	236.6	5.3	31	M	91	126	13.1	94.3	28.7	34.6	104.82	321	32.65	284.3	5.1
4	57	M	92	97	7.4	68.1	22.4	26.6	9.35	506	1.85	8.2	4	13.1	90.6	29.3	33.6	104.82	317	33.06	279.8	6.1	36	F	84	106	13.8	88.7	27.3	33.8	96.3	317	30.37	190.7	5.0
5	56	F	84	98	6.6	72.7	25.1	27.2	17.8	484	3.67	6.8	4.7	12.3	89.5	26.9	33.4	97.65	322	30.32	282.4	6	51	F	85	98	14	96.6	29.8	33.5	133.42	315	42.35	244.6	5.0
6	58	F	88	102	8.2	72.5	20.3	28	27.6	445	6.2	9.6	5.1	12.8	95.4	28.8	32.8	89.65	313	28.64	195.3	5.9	25	F	86	107	12.8	91.2	28.3	33.9	120.34	303	39.71	232.8	4.9
7	36	M	95	103	4.9	66.9	17.9	26.7	18.9	412	4.59	7.2	4.6	13	96	29.4	33.2	120.12	328	36.62	254.6	5.5	34	M	97	115	13	91.4	28.1	34.1	132.12	320	41.28	280.3	5.0
8	43	F	87	97	8	76	24.1	28.9	27	382	7.1	8.7	4.9	12.9	93.6	29.8	34.2	96.33	305	31.58	210.8	5.8	39	M	96	134	13.3	93.1	28.6	33.1	135.62	311	43.6	255.4	5.1
9	53	F	83	99	7.6	78.1	23.8	28.5	28.2	398	7.08	6.3	5.2	12.3	92.2	29.1	32.6	102.26	310	32.98	251.7	6.1	33	F	84	130	13.4	94.2	28.9	33.5	122.73	309	39.71	226.7	5.0
10	37	F	88	122	6.6	59.9	16.5	27.5	21.66	406	5.33	5.6	4.2	12.3	91.2	28.2	33.1	124.78	306	40.77	235.5	5.7	42	F	90	98	13.8	87.9	27.8	33.8	90.6	319	28.4	188.5	5.0
11	58	F	93	112	8.2	72	20.1	28	25.94	480	5.41	4.9	5.1	13.3	87.9	27.8	33.6	99.45	323	30.78	267.8	5.9	36	F	92	112	13	91.2	28.2	33.9	104.52	312	33.5	214.6	5.0
12	39	F	90	95	6.8	72.6	23	29.7	29.05	459	6.32	8	4.9	12.2	94.2	28.9	33.5	106.34	319	33.33	212.3	6.2	32	M	88	127	13.2	92.2	29.1	32.9	124.55	306	40.7	229.8	4.9
13	24	F	92	118	7.5	58.6	16.8	28.6	27.93	460	6.07	6.2	4.8	12.5	93.1	28.6	33.1	124.6	303	41.12	204.5	5.6	28	M	93	109	13.5	93.6	29.8	32.7	131.42	312	42.12	277.4	5.1
14	35	F	85	134	7	55.1	15.8	27.6	26.06	562	4.64	7.1	5.2	12.3	91.4	28.1	34.1	108.65	314	34.6	237.6	5.8	42	F	87	99	12.9	96	29.4	34.2	125.36	305	41.1	207.5	5.0
15	31	M	95	124	4.4	49	13.2	26.8	28.66	468	6.12	9.71	4.1	13.2	91.2	28.3	33.9	116.24	310	37.49	266.2	5.5	57	F	82	135	13.1	95.4	28.8	33.1	122.72	308	39.84	196.4	5.0
16	28	M	97	130	7.7	79	22.6	28.7	23.66	398	5.94	8.6	5.1	13	96.6	29.8	33.5	109.81	303	36.24	282.3	5.9	48	F	87	96	12.8	89.5	26.9	32.8	112.6	323	34.86	186.3	5.0
17	32	F	86	128	8.3	64.8	19.2	29.7	26.12	487	5.36	6.3	5.2	12.9	95.3	27.3	33.8	97.25	312	31.16	238.6	6.3	45	M	96	106	13.4	90.6	29.3	33.4	124.65	328	38	212.5	5.0
18	57	F	85	130	8.3	59.3	18	29.3	17.93	417	4.3	9.6	4.8	13.1	96.1	28.7	33.9	108.44	317	34.2	254.6	5.4	31	F	88	120	13.9	93.4	30.4	33.6	104.88	317	33.08	202.6	5.0
19	36	F	84	95	6.6	52.8	14.1	26.7	16.97	502	3.38	3.3	4.3	12.3	86.4	29.2	33.1	114.56	321	35.68	224.5	6.1	36	F	84	108	13.3	90.5	26.9	33.1	96.52	330	29.24	177.1	5.1
20	36	M	90	97	7.3	58.3	17.9	30.7	20.98	502	4.18	4.6	4.7	13.3	91.7	27.9	33.2	122.6	312	39.29	277.4	6	32	M	92	105	13.4	92.3	27.8	34.5	97.65	328	29.77	176.5	5.0
21	35	M	86	96	4.1	55.2	14.4	26.2	18.4	538	3.42	7.8	5	13.1	87.6	28.7	32.8	132.72	306	43.37	254.4	5.6	51	F	98	126	13	88.5	27.5	33.6	99.38	320	31.05	183.4	5.0
22	50	F	78	90	5.8	56.8	20.1	26.8	15.32	450	3.12	3.48	4.2	12.2	91.8	27.6	33.7	115.65	313	36.94	198.5	5.9	54	F	85	117	13.2	93.2	28.1	33.9	105.2	314	33.5	198.3	5.0
23	35	F	98	118	6.7	58	16	27.9	26.52	428	6.2	5.5	4.6	12.1	86.5	29.3	33.2	131.45	316	41.59	237.5	6.2	36	M	78	112	13.6	94.5	29.2	33.5	118.76	312	38.06	233.4	5.0
24	35	F	92	105	7	73.6	21.4	29	14.49	438	3.31	4.3	4.7	12.4	96.1	31.6	34.5	122.33	325	37.64	202.6	5.5	49	F	90	126	12.9	94.6	28.4	32.9	124.6	304	40.98	215.6	5.0
25	56	F	87	98	8.1	66.2	19.8	29.9	22.8	388	5.88	9.5	4.4	12.9	95.3	30.5	34.3	130.46	331	39.41	196.7	5.4	56	F	94	114	13.2	93.8	28.6	34.8	106.76	317	33.67	184.67	5.1
26	51	M	88	97	6.6	62.4	18	28.8	18.42	422	4.36	7.8	4.8	13.2	94.2	28.5	33.9	96.75	310	31.2	267.5	5.8	28	F	90	123	13.4	95.5	29.3	33.3	99.97	308	32.45	232.8	5.0
27	57	F	96	128	7.8	62.6	20	27.9	17.8	435	4.09	8.4	4.1	12.5	88.6	27.8	33.6	121.37	321	37.8	244.3	5.6	25	M	85	96	13.8	88.7	27.8	32.9	122.44	314	38.99	244.7	5.0
28	37	M	81	109	6.2	60	17	28.4	22	402	5.47	9.2	4.4	13.1	90.2	28.2	34.1	134.76	315	42.78	272.1	5.9	33	F	87	124	13.2	89.9	27.6	34.2	98.64	322	30.63	177.6	5.0
29	41	F	91	108	5.1	59.2	15.7	26.5	21.8	432	5.05	7.8	4.2	12.1	91.3	29.2	32.9	133.14	307	43.36	208.5	6.4	51	M	96	132	13.9	91.4	28.7	33.9	130.12	316	41.17	250.6	5.1
30	48	M	84	96	6.3	62	18.1	27.5	23	397	5.79	6.41	4.7	13.3	89.2	27.9	33.6	109.54	318	34.44	245.6	5.9	47	M	89	94	13.3	88.6	27.9	33.2	121.22	305	39.74	223.5	5.1
31	46	F	97	126	6	65	20.2	29.2	14.38	497	2.89	7.6	5.1	12.2	95.4	29.5	34.3	123.46	309	39.95	262.3	5.3	29	F	78	108	13.6	90.5	28.5	33.5	99.78	312	31.98	206.4	5.0
32	28	F	92	97	7.2	70	22.3	28	26.7	453	5.89	8.5	4.9	12.4	90.5	28.5	32.8	114.33	321	35.61	233.5	6.3	42	F	77	102	13.1	95.7	29.5	32.7	127.34	310	41.07	228.6	5.1
33	45	F	86	98	5.3	57	15.5	27.1	24.5	388	6.31	5.9	4.3	12.2	88.6	27.9	33.5	106.72	315	33.87	212.5	6.4	34	M	90	127	13.5	89.2	27.9	34.3	115.64	303	38.16	235.6	5.0
34	26	F	88	91	8.2	66.6	20.1	29.8	18.5	476	3.89	6.9	4.5	13.1	91.4	28.7	33.2	120.64	322	37.46	272.2	5.5	46	M	92	105	13.8	91.2	29.2	33.7	107.82	313	34.44	252.3	5.0
35	38	F	92	116	7.7	73.6	23.5	28	28.6	445	6.43	7.2	4.9	12.7	89.9	27.6	33.9	116.56	306	38.09	242.1	5.3	55	F	88	118	13	90	28.2	32.9	120.46	308	39.11	216.5	5.0
36	29	M	90	124	8.2	62	17.7	28.3	22.3	437	5.11	6.2	4.5	13.2	88.7	27.8	34.2	132.54	310	42.75	260.5	5.6	56	F	86	115	13.2	88.6	27.8	34.1	115.74	315	36.74	198.2	5.0
37	57	F	77	96	6.5	62.2	18.2	29.2	20.44	459	4.45	3.9	4.7	12.2	95.5	29.3	32.9	127.45	302	42.2	227.6	5.4	42	M	92	124	13.9	94.2	28.5	33.6	123.65	322	38.4	215.6	5.0
38	51	M	78	89	7.9	60.3	19.2	29.8	16.68	449	3.71	5.8	4.1	13.1	93.8	28.6	33.3	99.76	317	31.47	256.7	5.9	40	F	97	114	13.5	95.3	30.5	33.9	131.54	312	42.16	220.4	4.9
39	38	F	89	97	7.7	59.6	19.4	28.5	19.2	465	4.13	7.4	4.6	12.5	94.6	28.4	34.8	103.24	323	31.96	212.4	5.4	44	M	84	135	13.4	96.1	31.6	34.3	130.22	314	41.47	234.5	5.0
40	57	F	96	130	7.6	65	17.7	27.2	22.2	452	4.91	6.8	4.9	12.6	94.5	29.2	32.9	97.48	301	32.38	207.6	5.5	36	M	91	119	13.8	86.5	29.3	34.5	120.35	306	39.33	267.3	5.0
41	26	F	87	97	4.3	56.7	17.3	29.5	16.3	529	3.08	9.8	4.4	12.1	93.3	28.1	33.5	130.22	324	40.19	243.4	5.8	45	F	81	108	13.3	91.8	27.6	33.2	97.48	324	30.08	281.4	5.1
42	42	M	85	114	7.1	72	21.3	27.6	22.8	417	5.46	7.1	4.6	13.1	89.5	27.5	33.9	115.66	306	37.79	262.3	6.2	37	M	96	120	13.7	87.6	28.7	33.7	103.24	301	34.29	276.6	5.1

ISSN No:-2456-2165 https://doi.org/10.5281/zenodo.14769398

43	32	F	90	116	7.5	68	19.3	28.5	29.3	511	5.74	8.2	4.7	12.5	96.1	29.6	32.9	123.46	314	39.31	231.5	6.1	46	F	88	118	13.2	89.8	27.5	32.8	98.75	323	30.57	209.6	5.0
44	33	F	94	135	6.4	67	20.5	29	18.6	426	4.36	7.2	4.8	12.4	95.6	28.7	33.8	131.54	312	42.16	196.7	6	58	M	87	104	13.9	94.3	28.4	34.6	127.34	317	40.17	230.1	5.0
45	55	F	90	122	5.4	67	22.5	27.2	9.35	506	1.85	4.9	4.5	12.2	89.6	28.4	34.2	123.65	322	38.4	212.4	5.9	40	F	92	122	13.2	96.6	29.4	34.2	134.23	302	44.44	255.4	5.0
46	27	M	83	125	7.8	68	19.8	29.1	28.3	384	7.4	7.7	4.2	13.3	93.4	29.1	34.4	115.74	315	36.74	270.4	5.3	33	M	98	104	13.5	95.4	28.7	33.5	119.62	310	38.58	244.6	5.0
47	29	F	88	99	6.9	70	20.5	29.2	26.4	435	6.07	6.8	4.5	12.2	95.2	28.4	32.9	120.46	308	39.11	231.3	5.7	42	F	93	130	13	93.1	28.1	34.6	127.31	306	41.6	234.6	4.9
48	43	F	87	118	8.3	71.7	21.5	28.3	20.61	465	4.43	5.6	4.6	13.1	87.5	29.7	33.7	107.82	313	34.44	227.5	5.4	38	M	86	115	13.4	88.6	28.5	34.2	125.49	322	38.97	228.3	5.0
49	48	M	94	130	5	55	14.8	26.7	15.37	472	3.25	5.6	4.7	13	88.4	29.6	33.4	115.64	303	38.16	282.1	6.1	46	F	96	120	13.7	89.5	29.3	33.3	107.66	315	34.17	212.4	5.0
50	56	F	90	126	6	61.7	18.8	27.4	21.8	392	5.56	7.5	4.7	12.1	92.7	28.1	34.3	127.34	310	41.07	199.4	6.3	32	F	95	134	13.2	91.4	27.8	33.7	112.43	321	35.02	188.9	4.9
51	56	F	88	96	7.3	59.9	17.4	29	24.92	428	5.82	6.2	4.3	12.5	91.6	27.7	32.9	99.78	312	31.98	220.3	5.4	40	M	87	100	13.8	88.9	28.9	34.1	126.87	309	41.05	230.6	5.1
52	53	F	98	124	6.8	65	22.3	27.6	25.44	433	5.87	7.6	4.4	12.3	94.3	27.6	33.1	121.22	305	39.74	252.4	5.9	25	F	95	130	13.2	94.3	28.2	33.9	109.54	318	34.44	218.8	5.1
53	33	M	92	132	7.2	70	23.9	28.2	26.1	426	6.12	5.4	4.2	13.2	89.4	27.9	33.5	130.41	318	41	275.4	5.8	44	M	82	118	13.4	92.2	27.8	33.4	110.72	321	34.49	276.5	5.0
54	44	F	86	94	6.2	66.8	20.4	26.5	18.6	436	4.26	6.9	4.4	12	88.6	28.5	33.8	134.54	311	43.26	221.5	5.4	48	M	85	99	13.6	88.6	28.5	33.8	105.46	330	31.95	266.2	5.0
55	24	F	87	108	6.4	76.2	24.3	28.7	18.88	429	4.4	7.2	4.5	12.2	92.2	27.8	33.4	124.78	306	40.77	208.4	5.6	32	F	96	125	13.3	89.4	27.9	33.5	99.85	314	31.79	195.7	5.1
56	27	F	86	104	6.8	59.7	16.2	27.2	28.36	436	6.5	6.7	4.3	12.1	94.3	28.2	33.9	100.52	321	31.31	271.3	5.5	35	F	90	122	13.9	94.3	27.5	32.9	98.64	302	32.66	208.6	5.0
57	25	F	96	127	7.1	57.5	16.9	29.4	26.9	460	5.84	7.6	4.6	12.6	88.9	28.9	34.1	126.53	303	41.75	234.2	6.2	57	F	85	135	13.3	91.6	27.3	32.8	88.97	325	27.37	178.6	5.1
58	24	F	85	105	6.7	66.1	19.4	27.8	22.5	418	5.38	5.7	4.4	12.1	91.4	27.8	33.7	114.96	323	35.59	255.4	6.1	39	F	96	116	13.2	92.7	28.1	34.3	112.78	305	36.97	214.4	5.1
59	52	F	90	116	7.5	58.6	16.8	28.6	19.6	475	4.12	8.8	4.6	12.4	89.5	29.3	33.3	119.65	320	37.39	211.3	5.4	49	M	86	114	13.4	88.4	29.6	33.4	124.66	312	39.95	232.6	4.9
60	24	M	96	115	5.3	52	13	24.9	25.2	422	5.97	7.4	4.2	13.1	88.6	28.5	34.2	122.75	311	39.46	266.2	5.5	43	M	87	97	13.5	87.5	29.7	33.7	119.12	306	38.92	244.5	5.0
61	24	F	85	124	7.7	64.5	19.8	29.7	22.3	415	5.37	8.2	4.9	12.5	93.1	28.1	34.5	131.32	307	42.77	233.1	5.8	44	F	86	130	12.8	95.2	28.4	32.9	130.65	303	43.11	253.12	5.0
62	51	F	82	91	8.1	64.2	20	28.3	24.1	447	5.39	6.8	4.6	12.8	95.4	28.7	33.5	110.34	315	35.02	206.2	5.9	39	M	92	97	13.8	93.3	29.1	34.4	127.62	312	40.9	246.1	5.0
63	58	F	95	135	4.1	55	14.7	27	5.12	483	1.06	7.4	4.1	12.1	96.6	29.4	34.2	123.69	312	39.64	195.6	6.4	26	F	98	129	13.4	89.6	28.4	34.2	133.4	310	43.03	228.7	5.0
64	44	F	87	114	6.6	57.4	16.4	28.5	22.2	456	4.86	3	4.7	12.2	94.3	28.4	34.1	126.85	309	41.05	207.5	6.1	56	F	88	106	13.2	95.6	28.7	33.8	126.85	309	41.05	232.6	5.0
65	33	M	95	109	4.3	54.2	14.8	27.3	15.8	405	3.9	7.2	4.4	13.2	89.8	27.5	32.9	133.4	310	43.03	258.4	6.4	29	F	90	124	12.8	97	29.6	32.9	123.69	312	39.64	220.8	5.0
66	55	F	96	120	7	64.3	17.8	27.7	26.51	388	6.83	6.4	4.6	12.3	93	28.3	33.1	127.62	312	40.9	223.8	5.6	26	F	94	116	13	90.4	27.6	33.8	110.32	315	35.02	192.6	5.0
67	56	M	86	127	7.7	62.4	19.1	29.5	18.6	426	4.36	6.9	5.1	13.3	93.7	27.2	33.2	130.65	303	43.11	276.9	5.7	40	M	87	98	13.4	88.7	29.1	34.5	131.32	307	42.77	270.3	5.0
68	44	M	93	118	6.5	63	17.6	28.1	18.41	435	4.24	5.3	5.2	13	87.4	28.4	34.7	119.12	306	38.92	245.3	5.3	35	M	88	91	13.2	91.4	28.8	34.2	122.79	311	39.48	246.7	5.0
69	28	M	82	93	8.2	65	17.9	27.7	28.37	418	6.78	5.9	4.8	13.2	91.4	27.8	33.5	124.66	312	39.95	224.8	5.8	33	M	83	98	13.9	92.3	28.3	33.2	119.65	320	37.39	240.8	4.9
70	39	F	81	104	7.6	58	16.5	28.7	13.54	503	2.69	7	4.3	12.6	86.5	27.6	34.2	112.78	305	36.97	235.3	5.5	26	F	80	126	13.6	87.6	29.3	32.7	114.98	323	35.59	212.7	5.0
71	57	M	90	130	4.2	49	13.5	27.8	28.5	450	6.33	5.6	4.5	13.1	89.3	28.1	32.8	88.97	325	27.37	197.6	5.9	43	M	94	120	13.8	88.4	29.1	33.3	125.63	303	41.46	228.5	5.1
72	52	F	94	122	6.6	63	16.2	26	21.9	428	5.11	9.1	4.7	12.4	91.7	27.8	33.2	98.64	302	32.66	217.3	6.5	46	F	87	108	12.9	92.5	28.2	34.7	100.45	321	31.29	187.5	5.0
73	57	F	78	97	5.3	66.7	21.1	29.6	17.2	390	4.41	5.9	4	12.1	94.2	28.4	34.1	99.86	314	31.8	245.6	6.3	44	M	82	114	13.6	89.8	28.7	33.9	124.78	306	40.77	225.4	5.0
74	28	F	86	102	7.8	62.3	18	28.8	24.35	404	6.02	6.7	4.8	12.5	93.6	29.3	33.7	105.46	330	31.95	273.1	6	49	M	91	102	13.4	89	28.3	33.4	134.54	311	43.26	265.4	5.0
75	45	F	91	130	5.8	52.9	15.4	29.1	18.3	465	3.93	7.2	4.6	12	90.5	27.5	34.2	110.72	321	34.49	254.2	5.9	39	F	90	97	12.8	94.5	28.5	34.2	130.41	306	42.61	284.2	5.0
76	27	M	83	123	7.2	65.3	19.9	29.4	20.4	440	4.63	7.4	5	13.3	93.6	28.2	33.6	132.56	311	42.62	260.5	5.6	43	F	78	99	13.8	91.5	27.9	34.6	130.63	324	40.31	273.5	5.0
77	57	M	85	114	6.4	61.1	20.3	28.9	21.6	502	4.3	5.6	4.5	13.1	87.6	27.1	34.5	123.65	306	40.4	255.6	5.8	29	F	93	122	13	87.6	27.1	34.5	128.2	305	42.03	265.78	5.0
78	42	F	89	126	6.7	65.5	22.4	27.7	22.9	455	5.03	7.8	4.6	12.3	91.5	27.9	34.3	98.41	312	31.54	225.2	5.6	24	M	87	112	13.5	93.6	28.2	33.6	120.77	310	38.95	256.6	4.9
79	26	F	81	112	8.1	68	19.1	28.3	25.2	425	5.92	6.6	4.7	12.9	94.5	28.5	34.5	120.46	310	38.85	198.1	6.3	32	F	85	95	13.1	90.5	27.5	34.2	108.46	322	33.68	188.6	4.9
80	41	F	96	117	6.3	72	21.6	29	20.7	446	4.64	6.4	5.1	12.3	89.8	28.3	33.4	108.98	303	35.96	267.3	6.2	54	F	95	118	13.2	93.6	29.3	33.7	124.65	310	40.2	246.7	5.1
81	34	F	95	126	7.1	63.7	22.3	28.5	19.8	522	3.79	7.7	4.2	12.5	88.7	28.7	33.9	119.22	332	35.9	231.6	6	53	M	96	134	13.6	94.2	28.4	34.1	135.64	301	45.06	250.8	5.1
82	55	M	85	105	6.4	65.6	24.3	28.5	21.3	435	4.89	8.6	4.5	13.4	92.5	28.3	34.1	126.78	321	39.49	272.4	5.5	51	M	81	124	14	91.7	27.8	33.3	128.74	312	41.26	261.2	5.1
83	28	F	87	108	7	70.2	24	27.6	19.9	438	4.54	7.5	4.4	12.2	88.4	29.1	33.3	125.43	310	40.46	252.3	5.8	35	F	89	130	13.9	89.3	28.1	32.8	123.88	322	38.47	232.4	5.0
84	29	F	93	120	6.1	62.7	22.8	27.9	18.4	414	4.44	8	4.8	12.1	87.6	29.3	32.7	118.74	312	38.05	212.5	5.9	34	F	85	128	12.8	85.6	27.6	34.2	109.36	313	34.93	253.34	5.0
85	33	F	78	106	6.9	64.4	19.5	26.6	19.5	388	5.02	6.4	4.1	12.3	92.3	28.3	33.2	136.54	309	44.18	245.6	5.4	43	M	83	130	13.8	91.4	27.8	33.5	130.67	325	40.2	198.7	5.0
86	44	F	90	96	7.3	75	21.4	27.4	21.5	428	5.02	7.6	4.7	12.6	91.4	28.8	34.2	140.12	306	45.79	228.5	5.5	54	F	91	115	13.2	87.4	28.4	34.7	131.42	307	42.8	276.4	5.0
87	51	M	91	135	8.2	73.2	23.5	27.6	26.1	452	5.77	7.3	4.6	13.2	88.7	29.1	33.7	120.45	326	36.94	235.6	5.8	33	M	86	97	13.4	93.7	27.2	33.2	120.45	326	36.94	246.5	5.0
88	29	F	82	98	7.1	74.7	24.5	28.2	25.3	466	5.42	8.5	4.5	12.5	90.4	27.6	33.8	131.42	307	42.8	210.8	5.9	48	M	78	102	13.6	93	28	33.1	140.12	306	45.79	236.8	5.0
89	27	F	87	109	6	64.8	18.9	24.6	19.1	408	4.68	7	4.4	12.1	93.5	28.4	33.6	130.67	325	40.2	197.7	5.7	55	F	94	116	13.9	89.5	27.8	34.2	136.54	309	44.18	229.7	5.0

ISSN No:-2456-2165 https://doi.org/10.5281/zenodo.14769398

90	34	M	94	127	6.7	67.9	20.8	26.5	18.6	435	4.27	6.2	4.8	13.3	95.4	27.7	33.4	109.36	313	34.93	270.9	6.2	41	F	90	135	12.8	84.7	27.4	34.4	118.7	312	38.04	241.2	5.1
91	57	F	79	112	7.4	76.3	22.4	27.8	20.5	464	4.41	6.7	4.2	12.6	94.3	27.3	34.3	123.88	322	38.47	261.4	5.7	53	M	81	105	13.8	87.5	28.7	33.5	125.43	310	40.46	235.7	5.1
92	57	F	80	96	7.2	64	18.7	26.1	21.9	429	5.1	7.5	4.7	12.5	85.6	27.8	33.8	108.48	312	34.76	225.8	6.1	28	M	93	127	13.4	88.4	28.4	33.8	127.62	321	39.75	218.6	5.1
93	40	F	86	130	6.2	67.8	20.4	26.6	17.2	390	4.41	9.2	4.7	12.3	88.9	28.5	33.3	128.74	301	42.77	247.6	6.3	38	M	80	98	13.9	90.5	27.8	32.8	119.22	332	35.9	253.4	5.1
94	55	M	90	134	5.9	64.5	23.5	28.2	20.1	424	4.74	6.3	4.2	13.1	87.7	27.3	34.5	135.64	310	43.75	258.7	5.4	33	M	85	128	13.5	88.6	28.5	33.4	108.98	303	35.96	267.2	5.0
95	38	F	82	115	6.6	58.9	19.8	24.6	22.1	472	4.68	6.8	4.6	12.2	90.5	27.8	34.2	124.65	322	38.71	216.3	5.8	56	F	92	109	13	86.3	26.7	34.1	120.46	310	38.85	227.5	5.0
96	49	M	93	106	7.8	60.4	23.7	28.6	23.35	460	5.07	5.2	4.6	13.4	91.4	28.1	33.1	120.75	307	39.33	244.3	5.9	43	F	90	132	13.2	90.8	27.9	33.9	98.41	312	31.54	204.4	5.0
97	46	F	87	98	8	72.1	24.6	27.1	24.2	444	5.45	5.5	5.2	12.6	87.6	27.4	33.9	128.23	315	40.7	208.6	6	54	F	84	110	13.1	92.1	28.1	35.2	123.65	306	40.4	254.3	5.0
98	33	F	80	105	8.1	75.4	22.6	27.8	22.6	502	4.5	8.3	4.5	12.9	88.6	26.7	34.2	130.63	324	40.31	243.5	5.5	44	M	90	128	13.7	91.6	27.7	34.6	132.56	312	42.48	272.4	5.0
99	47	F	86	126	6.1	68.7	21.7	26.2	19.8	398	4.97	6.6	4.9	12.3	90.2	27.5	33.8	122.34	305	40.11	223.8	5.6	50	M	87	120	13.6	90.4	28.3	33.8	130.34	304	42.87	268.6	5.0
100	50	M	89	118	7	68.2	19.6	27.1	20.8	452	4.6	7.1	4.6	13.2	89.3	27.2	34.4	128.56	322	39.92	262.4	5.3	30	F	95	108	13.2	89.3	27.2	34.5	107.28	330	32.5	232.5	5.1

ISSN No:-2456-2165 https://doi.org/10.5281/zenodo.14769398

STUDY GROUP				CONTROL GROUP		
SNO	NAME_S	AGE_S	SEX_S	NAME_c	AGE_c	SEX_c
1	CHARITHA	25	F	SUSMITHA	36	F
2	SUSNEHA	34	F	PRASAD	56	M
3	JANAKI	49	F	SAIRAM	31	M
4	SUBBAIAH	57	M	ASHRITHA	36	F
5	PUNAMMA	56	F	VANITHA	51	F
6	PARVATHI	58	F	VYSHNAVI	25	F
7	CHANDRASEKHAR	36	M	SUNEEL	34	M
8	VANI	43	F	RAGHAVENDRA	39	M
9	SUREKHA	53	F	RADHIKA	33	F
10	SUVARTHAMMA	37	F	USHA	42	F
11	APAMMA	58	F	AKSHARA	36	F
12	UMA	39	F	SACHIN	32	M
13	ANUSHA	24	F	MANAS	28	M
14	VIRAJITHA	35	F	PRIYATHI	42	F
15	SUJITH	31	M	MAHESWARI	57	F
16	MANMOHIT	28	M	GOPIKA	48	F
17	SRAVYA	32	F	ABHIMANYU	45	M
18	KANAKAMMA	57	F	SATHVIKA	31	F
19	LAKSHMI	36	F	AKHILA	36	F
20	NARENDRA	36	M	SUDHAKAR	32	M
21	RAMCHANDRAIH	35	M	BHUVANESWARI	51	F
22	SESHAMMA	50	F	AYESHA	54	F
23	SAMYUKTHA	35	F	PANDIT	36	M
24	ANITHA	35	F	NIRMALA	49	F
25	ANJANAMMA	56	F	ROSHAMMA	56	F
26	ASHOK	51	M	SARAYU	28	F
27	SUJATHA	57	F	SANDEP	25	M
28	DINESH	37	M	ANANTHA LAKSHMI	33	F
29	SUNITHA	41	F	KISHAN	51	M
30	PRASAD	48	M	AJAY KHANA	47	M
31	ASHA	46	F	SUCHITRA	29	F
32	BINDHU	28	F	SIRISHA	42	F
33	SUDHA	45	F	PRANAV	34	M
34	ALEKHYA	26	F	ADITYA	46	M
35	RADHA	38	F	ARAVINDA	55	F
36	KRISHNA	29	M	VASUNDARA	56	F
37	SUMATHI	57	F	CHARAN	42	M
38	SUKESH	51	M	SUNDARI	40	F
39	ANANYA	38	F	BALA MURALI	44	M
40	NAGESWARAMMA	57	F	VIVEK	36	M
41	SANJANA	26	F	SARASWATHI	45	F

ISSN No:-2456-2165 https://doi.org/10.5281/zenodo.14769398

42	RAMBABU	42	M	ELIYASU	37	M
43	PRIYANKA	32	F	NIRUPAMMA	46	F
44	GAYATRI	33	F	MADHAVA KUMAR	58	M
45	KALYANI	55	F	BRAHMINI	40	F
46	RAHUL	27	M	THARUN	33	M
47	NAKSHATRA	29	F	ANUDEEPIKA	42	F
48	SAI LEELA	43	F	SAIDA	38	M
49	MAHATHI	48	M	NAGESWARI	46	F
50	SANGEETHA	56	F	GEETHANJALI	32	F
51	SHARMILA	56	F	MAHESWARAIAH	40	M
52	SHILPA	53	F	ANUHYA	25	F
53	MADHU BABU	33	M	JAGAN	44	M
54	PAVITHRA	44	F	KUMARA SWAMY	48	M
55	PREETHI	24	F	SAKSHI	32	F
56	TANYA	27	F	MANDODARI	35	F
57	SWETHA	25	F	ARSHIYA KAHANAM	57	F
58	SUPRITHA	24	F	HIMAJA	39	F
59	AMUDHA	52	F	CHANDRA MOHAN	49	M
60	SHANTHI PRIYA	24	M	VENKATESWARA RAO	43	M
61	SWAIRA	24	F	GRUHA LAKSHMI	44	F
62	JHANSI	51	F	LOHITH KUMAR	39	M
63	SUVARTHA	58	F	SUMAJA	26	F
64	SAVITHRI	44	F	CHAMUNDESWARI	56	F
65	MAHESH	33	M	PALLAVI	29	F
66	ANNAPURNA	55	F	NIHITHA	26	F
67	CHANDRAIAH	56	M	PRADEEP	40	M
68	SAI DATTA	44	M	AZAD	35	M
69	DEEPAK	28	M	SIVA KRISHNA	33	M
70	MADHURIKA	39	F	KOMALI	26	F
71	NAGA CHAITANYA	57	M	RANJITH	43	M
72	BASAVAMMA	52	F	ANANDI	46	F
73	PARVATHAMMA	57	F	KESHAV	44	M
74	SUNANDA	28	F	KRUNAL CHETAN	49	M
75	NAZRIYA	45	F	SAVITHA	39	F
76	YESU BABU	27	M	KATYAYINI	43	F
77	BIKSHALU	57	M	SMRITI	29	F
78	KARUNA	42	F	VISHNU CHARAN	24	M
79	ARAADHANA	26	F	SHAGUFTA ANJUN	32	F
80	KRISHNA BHARATHI	41	F	BALA SUNDARI	54	F
81	HEMA	34	F	SURESH KUMAR	53	M
82	SRINIVASA RAO	55	M	VARA PRASAD RAJU	51	M
83	HEMAMALINI	28	F	JYOSTNA	35	F
84	SULOCHANA	29	F	DEVIKA	34	F

ISSN No:-2456-2165 https://doi.org/10.5281/zenodo.14769398

85	**SHREYA**	**33**	**F**	**VIJAY GOVINDA**	**43**	**M**
86	**MADHAVI**	**44**	**F**	**GEETHAMBIKA**	**54**	**F**
87	**MANIKYAM**	**51**	**M**	**NISHANTH**	**33**	**M**
88	**RASHMIKA**	**29**	**F**	**PRAKSAH RAO**	**48**	**M**
89	**SIVATMIKA**	**27**	**F**	**VARA LAKSHMI**	**55**	**F**
90	**NARAYANA**	**34**	**M**	**TULASAMMA**	**41**	**F**
91	**NAGAMMA**	**57**	**F**	**TULASI RAM**	**53**	**M**
92	**NILAMBHARI**	**57**	**F**	**ISHAAN**	**28**	**M**
93	**MEENAKSHI**	**40**	**F**	**SURENDER**	**38**	**M**
94	**PHANINDRA**	**55**	**M**	**IRFAN PATHAN**	**33**	**M**
95	**BHUMIKA**	**38**	**F**	**SUBAYAMMA**	**56**	**F**
96	**ARJUN**	**49**	**M**	**ASTHA LAKSHMI**	**43**	**F**
97	**VASAVI**	**46**	**F**	**ANKITHA**	**54**	**F**
98	**SULEKHA**	**33**	**F**	**RAMESH**	**44**	**M**
99	**INDRANI**	**47**	**F**	**VISWAJITH**	**50**	**M**
100	**MUSTAFA**	**50**	**M**	**ANANDITHA**	**30**	**F**