PREVALANCE AND AETIOLOGY OF ANEMIA IN TERITARY CARE HOSPITAL IN KANYAKUMARI DISTRICT - OBSERVATIONAL STUDY

Dissertation

Submitted to

THE TAMILNADU Dr. M.G.R MEDICAL UNIVERSITY

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

M.D GENERAL MEDICINE

May2018

CERTIFICATE

This is to certify that this dissertation entitled "**PREVALANCE AND AETIOLOGY OF ANEMIA IN TERITARY CARE HOSPITAL IN KANYAKUMARI DISTRICT"** is a bonafide record of the work done by **Dr.JINEESH RAJ** during the period 2015-2018. This has been submitted in the partial fulfilment of the award of M.D. Degree in GENERAL MEDICINE by the Tamilnadu Dr. MGR Medical University Chennai.

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DECLARATION

I Dr. JINEESH RAJ, hereby submit the dissertation titled "**PREVALANCE AND AETIOLOGY OF ANEMIA IN TERITARY CARE HOSPITAL IN KANYAKUMARI DISTRICT**" done in partial fulfilment for the award of the degree **M.D General medicine** in Sree Mookambika Institute of Medical Sciences, Kulasekharam. This is an original work done by me under the guidance and supervision of Dr. Krishnan kutty and Dr. R.V.Mookambika.

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ACKNOWLEDGEMENT

My foremost thanks goes to the God Almighty who has blessed me abundantly with his grace, for giving me the strength that I needed to complete this thesis.

I extend my sincere heartfelt thanks to **Dr. Velayuthan Nair**, Chairman and **Dr. Rema. V. Nair**, Director, for providing facilities to accomplish my dissertation work. I also thank the Principal of the Institution **Dr. Padmakumar** for his valuable support extended to me.

I express my utmost gratitude and heartfelt thanks to **Dr.Kaniraj Peter** HOD Professor Department of medicine for all your help and guidance throughout.

I express my sincere thanks and gratitude to my professor, mentor and guide **Dr.Krishnan kutty**, Associate Professor, for his valuable and constant guidance, supervision and support throughout the study. His constant motivation has helped me overcome all the challenges and difficulties that I came across this research work.

I am very much grateful to my co-guide **Dr. R.V.Mookambika,** Assistant Professor, for her tremendous help, valuable support and guidance in carrying out the study. Her constant encouragement helped me to overcome obstacles during the study.

I wish to express my sincere thanks to **Dr.Rajendran,**Assistant Professor, for his help and valuble suggestions throughout the study period.

I thank **Dr.Thilagar,** Assistant Professor, for the help and support throughout the study period.

I thank **Dr.Mohandas,** Assistant Professor, for the help and support throughout the study period.

I wish to express my sincere thanks to **Dr.Jayaram J.K,**Senior resident, for his help and valuble suggestions.

I also thank my **Co-Post graduates,Junior Post Graduates** for their help and support.

I am very grateful for the support, encouragement and care given by **my parents** and **friends** whenever I needed the most.

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ABBREVATIONS

WHO	**WORLD HEALTH ORGANISATION**
IDA	**IRON DEFICIENCY ANEMIA**
UNICEF	**UNITED NATIONS CHILDREN'S FUND**
UNU	**UNITED NATIONS UNIVERSITY**
HIV	**HUMAN IMMUNO DEFICIENCY VIRUS**
AIDS	**AUTO-IMMUNE DEFICIENCY SYNDROME**
MMR	**MATERNAL MORTALITY RATIO**
G6PD	**GLUCOSE 6 PHOSPHATE DEHYDROGENASE**
CFU-E	**COLONY FORMING UNIT-ERYTHROCYTES**
GIT	**GASTROINTESTINAL TRACT**
CBC	**COMPLETE BLOOD COUNT**
Hb	**HEMOGLOBIN**
LFT	**LIVER FUNCTION TEST**
RFT	**RENAL FUNCTION TEST**
ESR	**ERYTHROCYTE SEDIMENTATION RATE**
CRP	**C-REACTIVE PROTEIN**

BMI	**BODY MASS INDEX**
Hb1AC	**GLYCOSYLATED HEMOGLOBIN**
T2DM	**TYPE 2 DIABETES MELLITUS**
EDTA	**ETHYLENE DIAMINE TETRA-ACETIC ACID**
TC	**TOTAL COUNT**
RBC	**RED BLOOD CELLS**
MCV	**MEAN CORPUSCULAR VOLUME**
MCH	**MEAN CELL HEMOGLOBIN**
MCHC	**MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION**
RDW	**RED CELL DISTRIBUTION WIDTH**
SD	**STANDARD DEVIATION**
CKD	**CHRONIC KIDNEY DISEASE**

ABSTRACT

BACKGROUND: Anaemia is a major public health problem in India. According to WHO, there are two billion people with anaemia in the world and half of the anaemia is due to iron deficiency. The prevalence of anaemia is disproportionately high in developing countries like India which attributes to poverty, inadequate diet, certain diseases, pregnancy and lactation, and poor access to health services. Nutritional anaemia constitutes the most important cause of anaemia. The purpose of this study was to study the laboratory profile of patients with anemia and to study the hematological types of anemia in such patients as well as the closest possible etiological profile.

Objectives:

To study the prevalence of anaemia and its aetiology in patients attending Sree Mookambika Institute Of Medical Sciences and to study the proportion of patients with nutritional anaemia.

Materials and methods:

A cross sectional study was done in a tertiary health care hospital in kanyakumari district ,SREE MOOKAMBIKA INSTITUTE OF MEDICAL SCIENCES.A total of 160 patients who came to medicine department of the same who fulfilled our inclusion criteria were included in the study.The study took place for a period of 18 months.Statasticial analysis was done using percentage,standard error of proportion and chi-square test.

Results:

The overall prevalence of anemia was found to be 58.1% of which 28.1% has moderate,18.1% has mild and 11.9% has severe anemia. A significant association of anemia was found with age, socio economic status, chronic illness and history of blood transfusion.

Conclusion:

The high prevalence of anemia(58.1%) indicates that anemia continues to be a major public health problem in India. Age, socio economic status, literacy are the major determinants that contribute to the problem of anemia. High prevalence despite the easy availability and access to medical care, indicates the level of ignorance and indifferences to health needs.

INTRODUCTION

Anemia is a serious global public health problem affecting both developing and developed countries having an impact on human health as well as social and economic development. It occurs at all stages of the life, but is more common in pregnant women and young children. Globally, over 3.5 billion people, more than half the world's population, are affected by iron deficiency in the year 2014 as compared to 1.62 billion people in the year 2008. Prevalence of iron deficiency in developing countries are staggering (44% of women, 42% of preschoolers, 53% of school-aged children and 33% of men), with Asia and Africa having the highest prevalence [1]

In 2014, prevalence of nutritional anemia among adolescent girls was found to be high (47.6% to 90%) in different parts of in North India and the prevalence found to be (78.7%) in the state of TamilNadu[2].

In 1992, World Health Organization global estimates of anemia prevalence averaged 56%, with a range of 35–75% depending on geographic location World Health Organisation 1992[3].

The third National Family Health Survey (NFHS) 2005–2006 revealed that at least 80% of Indian children aged 12 to 23 months were anemic[4]. Anemia was especially prevalent among rural children, and the majority of India's population (72.2%) is rural. However, despite recent economic development and the existence of a national anemia-control program, the prevalence of anemia in India between 2000 and 2005 increased from 75.3% to 80.9% in children aged 6 to 36 months[5,6,7,8].

Infants and young children having high risk to develop iron deficiency due to higher requirements of iron for their rapid growth[9]. Iron deficiency is the primary cause for anemia in children.

ETIOLOGY

❖ NUTRITIONAL ANEMIA

Nutritional Anemia was defined (WHO)as "a condition in which the hemoglobin content of the blood is lower than normal as a result of a deficiency of one or more essential nutrients, regardless of the cause of such deficiency."[10]

- IRON DEFICIENCY

Iron deficiency is the most common preventable nutritional problem despite continued global goals for its control. Globally, the most significant contributor to the onset of anaemia is iron deficiency so that iron deficiency anemia and anaemia are often used synonymously, and the prevalence of anaemia has often been used as a proxy for iron deficiency anemia. It is generally assumed that 50% of the cases of anaemia are due to iron deficiency(IDA).[11]

The singular importance of iron deficiency was restated with more confidence by Baker and DeMaeyer: "The major factor responsible [for nutritional anemia] is a deficiency of iron, with folate deficiency also playing a role in some population groups, especially in pregnant women."

Anemia was considered an indicator of iron deficiency rather than iron deficiency being considered a contributing cause of anemia (WHO/UNICEF/ UNU, unpublished). However, iron deficiency is not the only cause of anemia.

Hook worm infestations, HIV / AIDS, malaria, chronic infections also results in Iron deficiency anemia[12].

Worm infestation is a major public health problem in children of developing countries because of poor socioeconomic conditions and lack of good hygienic living. It causes nutritional deficiencies and anaemia[13].

- OTHER CAUSES OF NUTRITIONAL ANEMIAS

➢ deficiency of folic acid, vitamin B12 (megaloblastic anemias)[14].

❖ INHERITED DISORDERS OF HAEMOGLOBIN

Of the large group of anemias due to inherited disorders, Thalassemias, particularly major disease of beta thalassemia are a considerable health problem in india and contribute significantly to mortality and morbidity with its increasing prevalence.

❖ ANEMIAS OF CHRONIC DISEASE

The most common chronic diseases associated with development of anemia are chronic kidney disease and chronic liver failure.

AGE GROUPS AFFECTED

Anemia is a common condition. It occurs in all age, racial, and ethnic groups.

Among children, female children were more susceptible to anemia and undernutrition was attributed as the major reason for nutritional anemia.

Among adolescents, girls constitute a vulnerable group, particularly in developing countries where they are traditionally married at an early age and exposed to a greater risk of reproductive morbidity and mortality. The nutritional anemia in this group attributes to high MMR, high incidence of low-birth weight babies, high perinatal mortality and fetal wastage and consequent high fertility rates. Also can be attributed to lack of education, and sanitation.[15]

Among adults, both men and women can have anemia. From teenage to socially active and productive age groups of population, anemia is almost exclusively prevalent in females. Especially, women of childbearing age are at higher risk for the condition.[16]

Estimates of anemia prevalence among the elderly range from 2.9% to 61% in elderly men and from 3.3% to 41% in elderly women[17]. Most anemia in older individuals results from iron deficiency, chronic inflammation, or chronic kidney disease, or it may be unexplained[18].

EFFECTS ON HEALTH

- Effects on pregnancy

Severe anemia during pregnancy is associated with a woman's increased risk of death and moderate to severe anemia is associated with an increased risk of low birth weight and preterm delivery

- Effects on growth and development

Anemia may compromise pubertal growth spurt. It may also reduce physical work capacity and cognitive function.

In infants with IDA, Language and mental developmental are affected.

- Effects on elderly age group

Anemia in older individuals is associated with a very wide range of complications such as,

1. increased risk for mortality,
2. cardiovascular disease,
3. cognitive dysfunction,
4. longer hospitalization for elective procedures and comorbid conditions,
5. reduced bone density, and falls and fractures.
6. Not surprisingly, anemia also has a significant effect on quality of life (QOL) in the elderly.

SOCIOECONOMIC FACTORS

Status of literacy and wealth of parents have strong negative association with the status of anemia of the children. The most vulnerable groups are the children of illiterate parents, maternal anemia, and those belonging to the poor families in the rural areas.

Economically deprived women having multiple, closely spaced pregnancies are at exceptionally high risk of developing anemia.

SCIENTIFIC JUSTIFICATION OF THE STUDY

Anemia is a common concern in older people ,females and children with associated risk factors and can have significant morbidity and mortality. Because anemia is a sign, not a diagnosis, an evaluation is almost always needed to identify the underlying cause. The purpose of this study was to study the laboratory profile of

patients with anemia and to study characteristics of hematological types of anemia in such patients as well as the closest possible etiological profile.

Early detection of the same has a better prognosis and brings out a better outcome.

In India, the prevalence of anaemia is high because of

- Low dietary intake, poor iron (less than 20 mg /day) and folic acid intake (less than 70 micrograms/day)
- Poor bio-availability of iron (3-4 percent only) in phytate fibre-rich Indian diet
- Chronic blood loss due to infection such as malaria and hookworm infestations.

This research investigates the prevalence of anemia among people in attending the OPD of tertiary centre in the district of kanyakumari. The main objective for assessing anaemia in this part of the country is that the knowledge about anemia and its prevalence in this area is low and there are not enough data available to inform decision-makers on the type of measures to be taken to prevent and control anaemia. This research will help provide the necessities to build up a base on which work can be done to overcome the problem of anemia.

AIMS & OBJECTIVES

1. Study the prevalence of anaemia and its aetiology in patients attending Sree Mookambika Institute Of Medical Science.

2. To find out the proportion of patients with nutritional anaemia.

REVIEW OF LITERATURE

HEMATOPOIESIS

ROLE OF ERYTHROPOIETIN:

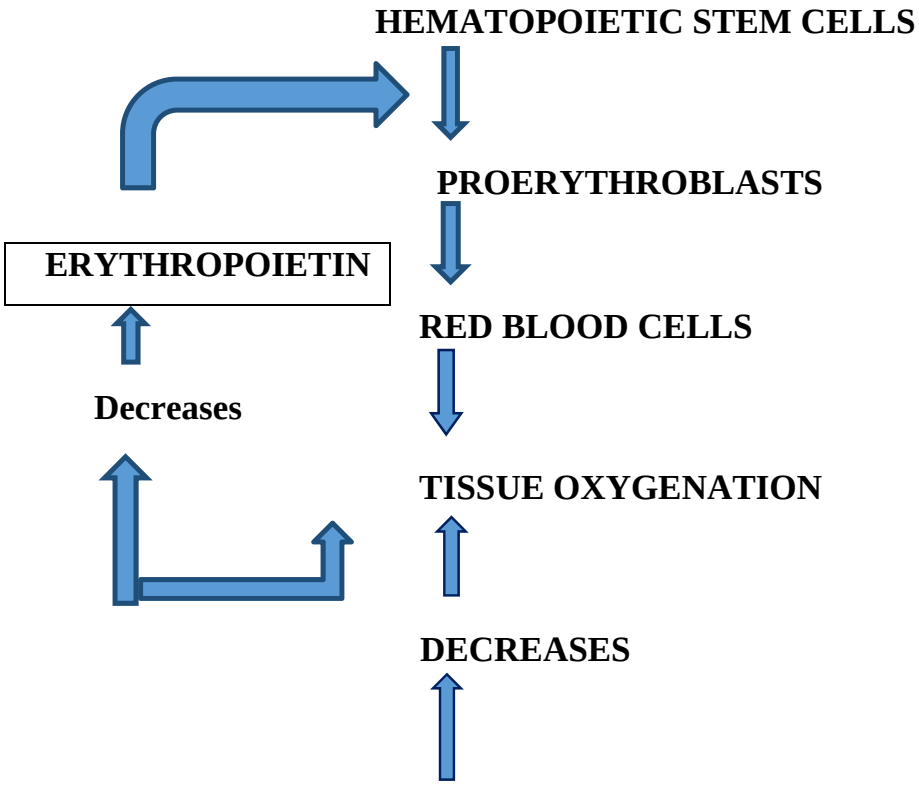
Hematopoiesis is the process by which blood elements are produced. The process is regulated through a series of steps beginning from pluripotent hematopoietic stem cell. Hematopoietic Stem cells are capable of forming red cells, granulocytes, monocytes, platelets, and the lymphocytes which are the cells of the immune system. Following lineage commitment, hematopoietic progenitor and precursor cells come under the regulatory influence of growth factors and hormones.

For red blood cell production, erythropoietin is the primary regulatory hormone. Erythropoietin is a glycoprotein molecule[19]. Erythropoietin is required for the maintenance of the committed erythroid progenitor cells, which undergo programmed cell death (apoptosis) in the absence of the hormone.

Erythropoietin is the principal hormone that regulates erythrocyte differentiation in mammals. This physiological regulator of red blood cell production is produced and released by the peritubular capillary cells which are highly specialized epithelial-like cells, lining the kidney. A small amount of Erythropoietin is also produced by the hepatocytes. The regulation of the production of Erythropoietin is linked to tissue oxygenation. The fundamental stimulus for Erythropoietin production is the availability of O_2 to meet the tissue metabolic needs.

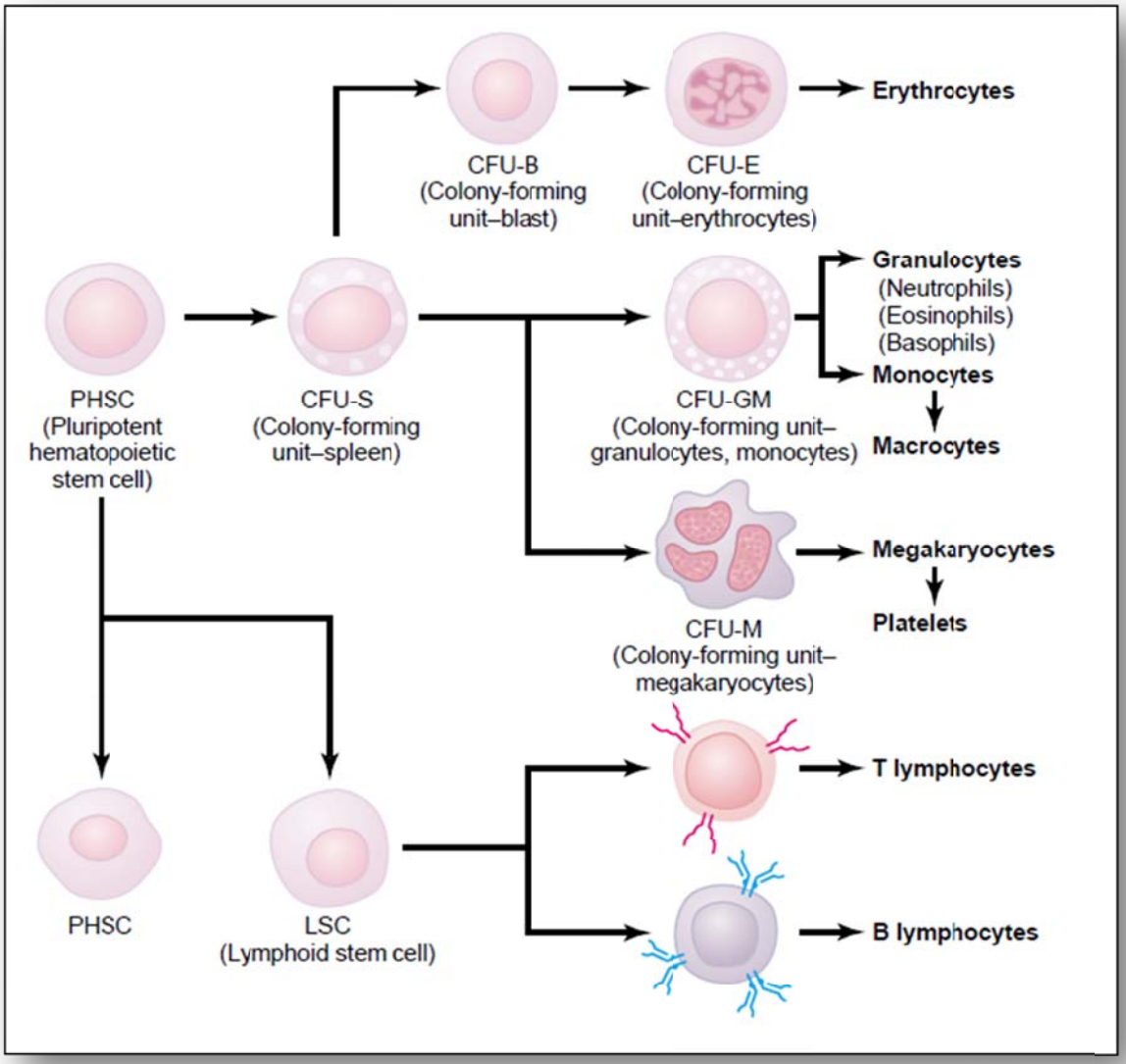
Impaired O_2 supply to the kidney can be due to anemia, hypoxemia, or rarely due to a entity called renal artery stenosis. The key to Erythropoietin gene regulation is HIF - 1 α, i.e. hypoxia-inducible factor. In the presence of Oxygen HIF- l

α is hydroxylated and degraded. If O_2 supply becomes impaired, the critical hydroxylation step does not occur, thus HIF- l α partner with other proteins and get translocated to the nucleus and upregulate the expression of the Erythropoietin gene and production of erythrocytes. Thus the process of hematopoiesis requires normal renal production of Erythropoietin, a functioning erythroid bone marrow for it to act, and adequate supply of substrates for the synthesis of hemoglobin. A defect in any one of the key components can lead to anemia.[20]



1) **LOW BLOOD VOLUME**
2) **ANEMIA**
3) **LOW HEMOGLOBIN**
4) **POOR BLOOD FLOW**
5) **PULMONARY DISESASE**

GENESIS OF BLOOD CELLS


Erythrocytes
CFU-B
(Colony-forming
unit–blast)
CFU-E
(Colony-forming
unit–erythrocytes)
Granulocytes
(Neutrophils)
(Eosinophils)
(Basophils)
Monocytes
Macrocytes
PHSC
(Pluripotent
hematopoietic
stem cell)
CFU-S
(Colony-forming
unit–spleen)
CFU-GM
(Colony-forming unit–
granulocytes, monocytes)
Megakaryocytes
Platelets
CFU-M
(Colony-forming unit–
megakaryocytes)
T lymphocytes
B lymphocytes
PHSC
LSC
(Lymphoid stem cell)

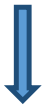
STAGES OF DIFFERENTIATION OF RED BLOOD CELLS

PROERYTHROBLAST

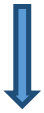
BASOPHIL ERYTHROBLAST

POLYCHROMATOPHIL ERYTHROBLAST

ORTHOCHROMATIC ERYTHROBLAST



RETICULOCYTE



ERYTHROCYTES

The production of different types of blood cells which includes formation, development, and differentiation of blood cells is called hematopoiesis and red cell production is called erythropoiesis.[21] The first cell that belongs to the erythroid series is the proerythroblast, Once the proerythroblast is formed, it divides multiple times, eventually forms many mature red blood cells. The proerythroblast is followed by cells called basophil erythroblasts, succeeding generations are polychromatophil erythroblast, orthochromatic erythroblast, reticulocytes and erythrocytes . During the reticulocyte stage, the cells pass from the bone marrow into the blood capillaries by a process called diapedesis (squeezing through pores of the capillary membranes). The basophilic material contained in the reticulocyte will normally disappear within 1 to 2

days, and the cell is then known as mature erythrocyte. The reticulocyte maturation time is a variable who's prolongation indicates the severity of anemia.[22]

FORMATION OF HEMOGLOBIN

Hemoglobin is made of four protein molecules known as globulin chains that are joined together. The normal adult hemoglobin abbreviated as HbA contains two alpha globulin chains and two beta globulin chains. Synthesis of hemoglobin begins in the proerythroblasts stage and ends in the reticulocyte stage. First, succinyl-CoA, from the Krebs cycle binds with the glycine molecule to form a pyrrole molecule. Four pyrroles molecules in turn combine to form protoporphyrin IX, which then joins with iron to form what is called the heme molecule. Finally, each heme molecule combines with a globin chain that are synthesized by ribosomes to form a hemoglobin chain. The four different types of hemoglobin chains are alpha chains, beta chains, gamma chains, and delta chains.[23]

ANEMIA

By definition, Anemia is defined as the reduction in the total circulating red cell mass below normal limits for the age and sex. Anemia decreases the oxygen-carrying capacity of the red blood cells, leading to tissue hypoxemia. Anemia is also defined as a reduction in the hemoglobin concentration of the blood, to the levels below the **normal limits for the age and sex**.[24]

The classification of anemia has been unsatisfactory till date. Attempts has been made to classify anemias according to the etiology involved, morphology, and severity. The different classifications of anemia are:[25]

❖ Morphologic characters include :

(a) red cell size (normocytic, microcytic, or macrocytic);

(b) degree of hemoglobinization, reflected in the color of red cells (normochromic or hypochromic).

These provide etiologic clues for the cause of anemia.

The above classification is based on the Red cell indices which are as follows:

- Mean cell volume: the average volume of a red cell (fL)
- Mean cell hemoglobin: the average amount of hemoglobin per red cell, in pictograms(pg).
- Mean cell hemoglobin concentration: the concentration of hemoglobin in a given volume of packed red cells, expressed as grams per deciliter(dL).
- Red cell distribution width: the coefficient of variation of red cell volume.

NORMAL RANGE:

Any value below the recommended WHO criteria is anemia. Adult reference ranges for red cell indices are as follows[26]

MEASUREMENT(units**)**	**MEN**	**WOMEN**
HEMOGLOBIN(gm/dL)	13.6-17.2	12.0-15.0
HEMATOCRIT(%)	39-49	33-43
RED CELL COUNT(x 10^6/μL)	4.3-5.9	3.5-5.0
RETICULOCYTE COUNT (%)	0.5-1.5	
MEAN CELL VOLUME(fL)	82-96	
MEAN CELL HEMOGLOBIN(pg)	27-33	
MEAN CELL HEMOGLOBIN CONCENTRATION(gm/dL)	33-37	
RED CELL DISTRIBUTION WIDTH	11.5-14.5	

- Anemias can also be classified based on the underlying mechanism / etiology by which it develops.
- These are divided into three main groups[27]:

- Anemia caused due to blood loss
- Anemia caused due to decreased or faulty red blood cell production
- Anemia caused due to destruction of red blood cells

CLASSIFICATION OF ANEMIAS:

MECHANISM	**EXAMPLES**
BLOOD LOSS	
1. ACUTE BLOOD LOSS **2.** CHRONIC BLOOD LOSS	Trauma Gastrointestinaltract lesions,gynecologicl disturbances
INCREASED RED CELL DESTRUCTION	
INHERITED GENETIC DEFECTS 1. RED CELL MEMBRANE DISORDERS 2. ENZYME DEFICIECY • HEXOSE MONOPHOSPHATE SHUNT ENZYME DEFICIENCIES • GLYCOLYTIC ENZYME DEFICIENCIES	Heriditary spherocytosis,hereditary elliptocytosis G6PD

	deficiency,glutathione synthetase deficiency
HEMOGLOBIN ABNORMALITIES 1. DEFICIENT GLOBIN SYNTHESIS 2. STRUCTURALLY ABNORMAL GLOBINS(HEMOGLOBINOPATHIES)	Pyruvate kinase deficiency,hemokinase deficiency
AQUIRED GENETIC DEFECTS 1. DEFICIENCY OF PHOSPHATIDYL-LINKED GLYCOPRPOTIENS	Thalassemia syndromes
ANTIBODY-MEDIATED DESTRUCTION	Sicle cell disease,unstable hemoglobins
MECHANICAL TRAUMA 1. MICROANGIOPATHIC HEMOLYTIC ANEMIA 2. CARDIAC TRAUMATIC HEMOLYSIS 3. REPETETIVE PHYSICAL TRAUMA	Paroxysmal nocturnal hemoglobinuria
INFECTIONS OF RED CELLS	Haemolytic diseases of the newborn,transfusion reactions,drug induced,auto-immune disorders
TOXIC OR CHEMICAL INJURY	
MEMBRANE LIPID ABNORMALITIES	

SEQUESTRATION	
DECREASED RED CELL PRODUCTION	
INHERITED GENETIC DEFECTS 1. DEFECTS LEADING TO STEM CELL DEPLETION 2. DEFECTS AFFECTING ERYTHROBLAST MATURATION	Fanconi anemia,telomerase defects
NUTRITIONAL DEFICIENCIES 1. DEFICIENCIESAFFECTINGDNA SYNTHESIS 2. DEFICIENCIES AFFECTING HEMOGLOBIN SYTHESIS	Thalassemia syndromes B_{12} and folate deficiencies
ERYTHROPOIETIN DEFICIECY	Iron deficiency anemia Renal failure,anemia of chronic diseases
IMMUNE MEDIATED INJURY OF PROGENITORS	
INFLAMATION MEDIATED IRON SEQUESTRATION	Aplastic anemia,pure red cell aplasia
PRIMARY HEMATOPOIETIC NEOPLASMS	
SPACE OCCUPYIING MARROW LESIONS	Anemia of chronic diseases
INFECTIONS OF RED CELL POGENITORS	Acute leukemiamyelodysplasia,

UNKNOWN MECHANISM	myeloproliferative disorders Metastatic neoplasms, granulomatous disease Parvovirus B19 infection Endocrine disorders, hepatocellular liver disease

CLINICAL FINDINGS:

Pallor, Weakness, malaise, and easy fatigability are the usual complaints. The low oxygen supply by the circulating blood leads to dyspnea on mild exertion. Fatty change in the liver, myocardium, and kidney is caused by hypoxia.

The most serious complications of severe anemia arise due to tissue hypoxia. Shock, hypotension, or coronary and pulmonary insufficiency can occur. This is more common in older individuals with underlying pulmonary insufficiency and cardiovascular disease. Sufficiently severe fatty changes in the myocardium are, leads to cardiac failure and compound the tissue hypoxia caused by the deficiency of O2 in the blood. The myocardial Hypoxia also can manifests as angina, particularly when complicated by pre-existing coronary artery disease[28]. With acute blood loss and shock, oliguria and anuria can develop as a result of renal hypoperfusion. Central nervous system hypoxia can cause headache, dimness of vision, and faintness.

ACUTE BLOOD LOSS

The effects of acute blood loss are mainly due to the loss of intravascular volume, which leads to reduction in oxygenation of tissues. This reduction in oxygenation leads to increase in secretion of erythropoietin from the kidney, stimulating the proliferation of erythrocytes (CFU-E) in the marrow and which appear as reticulocytes in the peripheral blood (known as reticulocytosis*)* but the change in hematocrit value after hemorrhage usually occurs slowly. And also during the bleeding episodes, there is a rapid increase in the numbers of circulating leukocytes and platelets.[29]

HEMOLYTIC ANEMIAS

Hemolytic anemias are defined as anemias resulting from an increase in red cell destruction. Normally red cell undergo destruction at the end of their life span of 120 days within the reticuloendothelial (RE) system cells, such as spleen and extravascular hemolysis, and hemoglobin is not liberated into the plasma in appreciable amounts. Hemolytic anemia presents as acute or chronic anemia, reticulocytosis, or jaundice.

Hemolytic anemias has the following features:

i. A shortened red cell life span below the normal 120 days.
ii. Elevated erythropoietin levels and a compensatory increase in erythropoiesis.
iii. Accumulation of hemoglobin degradation products that are produced during the process of red cell hemolysis.

There are two types of hemolytic anemia: Extravascular hemolysis and intravascular hemolysis.

The more common extravascular hemolysis is the destruction of red blood cells within the macrophages of the spleen and liver. The triad of symptoms of extravascular hemolysis: anemia, splenomegaly, and jaundice.

Intravascular hemolysis is the destruction of red blood cells in the circulatory system with the release of the contents of red cell into the plasma, caused by mechanical injury, complement fixation, intracellular parasites (e.g., falciparum malaria), or exogenous toxins. [30]

Intravascular hemolysis is manifested as anemia, hemoglobinemia, hemoglobinuria, hemosiderinuria,and jaundice. Unlike extravascular hemolysis, splenomegaly is not a feature.

TYPES OF EXTRAVASCULAR HEMOLYSIS

1) Hereditary Spherocytosis

In Hereditary spherocytosis the primary defect in hereditary spherocytosis is the loss of membrane surface area due to defects in the membrane proteins ankyrin, band 3, β spectrin, α spectrin, leading to reduced deformability and vulnerability to splenic sequestration and destruction. HS is caused by diverse mutations that leads to an insufficiency in the membrane skeletal components.[31]

2) Hemolytic Disease Due to Red Cell Enzyme Defects:

Abnormalities in the hexose monophosphate (HMP) shunt or glutathione metabolism resulting from an impaired enzyme function, reduce the ability of red

cells to protect themselves from oxidative injuries/ damage and lead to their destruction (hemolysis). The most important of all these enzyme derangements is the hereditary deficiency of glucose-6-phosphate dehydrogenase, i.e. G6PD deficiency. Oxidants can cause both intravascular and extravascular hemolysis in G6PD-deficient individuals. The level of severity of the enzyme deficiency often doesnot correlate with the severity of anemia clinically.[32]

3) Sickle Cell Disease

Sickle cell disease is the commonest hereditary hemoglobinopathy. Hemoglobin polymerisation is the central pathophysiology of this disease, leading to erythrocyte rigidity and vaso-occlusive symptoms.[33]

4) Thalassemia Syndromes

Thalassemia is a group of inherited hematologic disorders caused by defects in the synthesis of one or more hemoglobin chains leading to anemia, tissue hypoxia, and red cell hemolysis related to the imbalance in globin chain synthesis. Reduction or absence of synthesis of alpha globin chains is the cause of Alpha thalassemia and Reduction or absence of synthesis of beta globin chains is the cause of beta thalassemia. [34]

Clinical Syndromes	Genotype	Clinical Features	Molecular Genetics
β-Thalassemias			
β-Thalassemia major	Homozygous β-thalassemia (β^0/β^0, β^+/β^+, β^0/β^+)	Severe; requires blood transfusions	Mainly point mutations that lead to defects in the transcription, splicing, or translation of β-globin mRNA
β-Thalassemia intermedia	Variable (β^0/β^+, β^+/β^+, β^0/β, β^+/β)	Severe but does not require regular blood transfusions	
β-Thalassemia minor	Heterozygous β-thalassemia (β^0/β, β^+/β)	Asymptomatic with mild or absent anemia; red cell abnormalities seen	
α-Thalassemias			
Silent carrier	$-/\alpha\ \alpha/\alpha$	Asymptomatic; no red cell abnormality	Mainly gene deletions
α-Thalassemia trait	$--/\alpha\ \alpha/\alpha$ (Asian) $-/\alpha\ -/\alpha$ (black African, Asian)	Asymptomatic, like β-thalassemia minor	
HbH disease	$--/\alpha$	Severe; resembles β-thalassemia intermedia	
Hydrops fetalis	$--/--$	Lethal in utero without transfusions	

5) Paroxysmal Nocturnal Hemoglobinuria

Paroxysmal nocturnal hemoglobinuria (PNH) is a disorder of the hematopoietic stem cell resulting in the clonal production of blood cells which are defective in the surface proteins called glycan-phosphatidylinositol (GPI)-linked surface proteins and are abnormally susceptible to lysis or injury by complement[35].

6) Immunohemolytic Anemias

Hemolytic anemia is caused by antibodies that identify and bind to red cells antigen, leading to their premature destruction. These disorders are commonly referred to as autoimmune hemolytic anemias.

WARM ANTIBODY TYPE(IgG ANTIBODIES ACTIVE AT 37^0C) • PRIMARY(IDIOPATHIC) • SECONDARY **i.** Auto immune disorders(particularly systemic lupus erythematosus) **ii.** Drugs **iii.** Lymphoid neoplasms
COLD AGGLUTININ TYPE(IgM ANTIBODIES ACTIVE BELOW 37^0C) • ACUTE • CHRONIC i. Idiopathic ii. Lymphoid neoplasms
COLD HEMOLYSIN TYPE(IgG ANTIBODIES ACTIVE BELOW 37^0C) Rare;occurs mainly in childer following viral infections

Warm agglutinin type autoantibodies of autoimmune hemolytic anemia are usually IgG, but can also be IgM or IgA that are active at 37 ^{0} C. They are usually Rh specific. Cold-type antibodies are both IgM or IgG known as Donath-Landsteiner [DL] antibodies. [36]

TYPES OF INTRAVASCULAR HEMOLYSIS

1) Hemolytic Anemia Resulting from Trauma to Red Cells*:*

The shear forces produced by the turbulence of blood flow and the pressure gradients across damaged valves or lumen narrowing causes hemolysis. Causes of mechanical injury such as trauma caused by blood flow across the cardiac valves, thrombotic narrowing of the microcirculation, or repeated physical trauma (e.g., marathon running).

2) Complement fixation

Complement fixation occurs in many of the situations in which antibodies recognize and bind to the red cell antigens.

3) Exogenous toxic factors

Toxic injury due to Clostridial sepsis, causes massive hemolysis and leads to anemia.[37]

ANEMIAS OF DIMINISHED ERYTHROPOIESIS

The most commonest and important group of anemias associated with red cell underproduction are those due to nutritional deficiencies, followed by those that arise secondary to renal failure and chronic inflammation. The less common disorders such

as generalized bone marrow failure (aplastic anemia, primary hematopoietic neoplasms and infiltrative disorders that lead to marrow replacement- metastatic cancer and disseminated granulomatous disease) also lead to anemia.

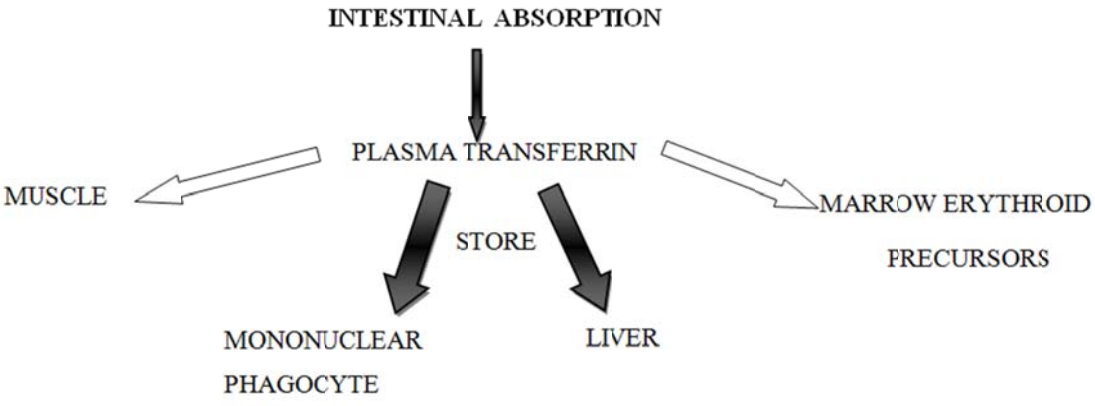
NUTRITIONAL ANEMIAS

A review of the literatures suggests that iron deficiency anemia is widespread throughout the world, especially in developing countries. The immediate cause of iron deficiency anemia is an inadequate diet.[38]

Iron Deficiency Anemia

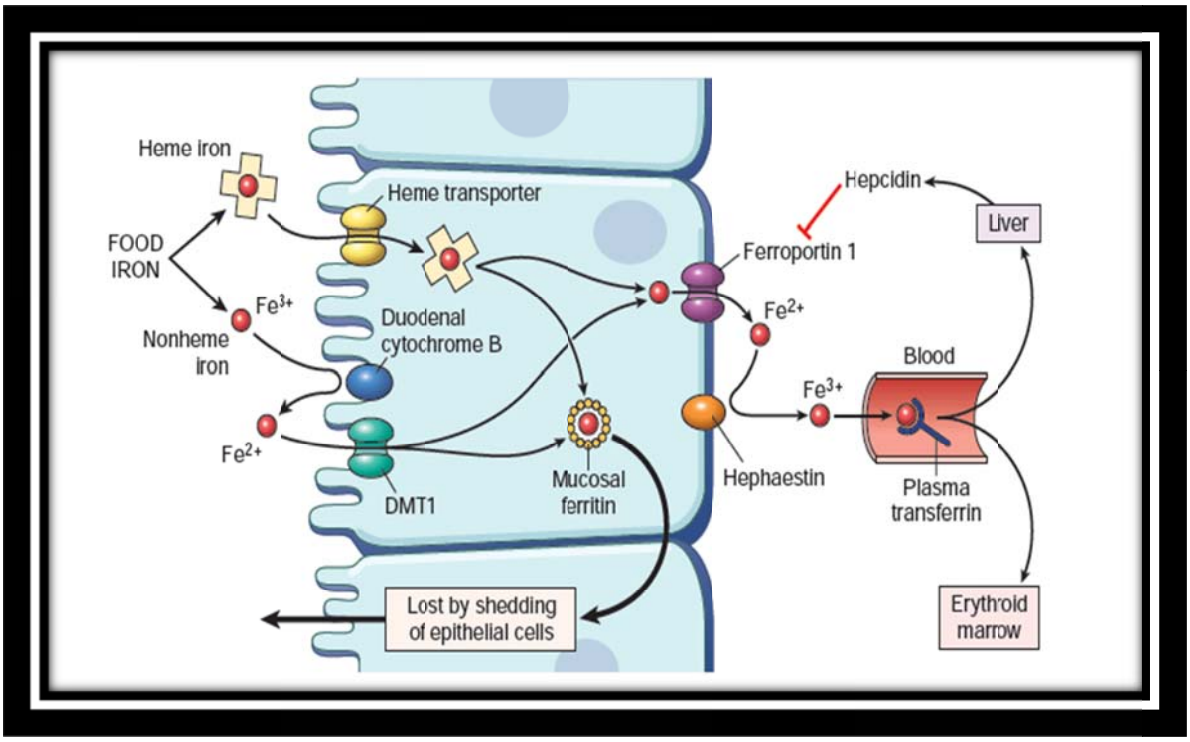
Deficiency of iron is the most common nutritional disorder in the world, especially in developing countries and results in clinical signs and symptoms that are related mostly to inadequate hemoglobin synthesis.

IRON METABOLISM



Free iron is highly toxic and it is therefore important that storage iron be sequestered. This is achieved by binding of iron in the storage pool (Liver) to either ferritin or hemosiderin.

Intestinal absorption of iron is regulated by *hepcidin*, a small circulating peptide that is synthesized and released from the liver in response to increases in intrahepatic iron levels. Hepcidin inhibits iron transfer from the enterocyte to plasma by binding to ferroportin and causing it to be endocytosed and degraded. Thus, when the body is replete with iron, high hepcidin levels inhibit its absorption into the blood. Conversely, with low body stores of iron, hepcidin synthesis falls and this in turn facilitates iron absorption.[39]



Etiology : Iron deficiency can result from:

(1) inadequate dietary intake,

(2) impaired absorption from GIT,

(3) increased requirement by the body,

(4) chronic blood loss such as gastric blled in APD.

Nutritional iron deficiency arises when the physiological requirements cannot be met by the iron absorption from diet.

Impaired absorption is found in sprue, other causes are gastrectomy, fat malabsorption (steatorrhea), and chronic diarrhea.

Increased requirement is an important cause of iron deficiency in growing infants, children, and adolescents, as well as premenopausal women, particularly during pregnancy.

Chronic blood loss such as External hemorrhage or chronic bleeding into the gastrointestinal tract, genitourinary tracts, depletes the iron stores. Iron deficiency anemia produces a microcytic hypochromic type of blood picture.[40]

CLINICAL FEATURES SPECIFIC TO IRON DEFICIENCY

In severe and long-standing iron deficiency, depletion of iron-containing enzymes in cells throughout the body also causes other changes, including koilonychia, alopecia, atrophic changes in the tongue and gastric mucosa, and intestinal malabsorption. In the brain, iron is bound to ferritin, the levels of which are decreased by iron deficiency and increased by iron supplementation. A Low ferritin levels in childhood have been reported to affect the development of the nervous system, leading to mental retardation and behavioral disorders like pica.[41]

MEGALOBLASTIC ANEMIAS

The megaloblastic anemia causes impairment of DNA synthesis that leads to ineffective hematopoiesis and distinctive morphologic changes, including abnormally large erythroid precursors and red cells. The spectrum of etiologies associated with macrocytic anemia includes nutritional deficiencies (e.g: vitamin B12 and folate), drugs (eg: methotrexate), primary bone marrow disorders (e.g: myelodysplasia and leukemia) and other chronic illnesses. Macrocytic morphology due to vitamin B12 deficiency and folate deficiency is a direct result of ineffective or dysplastic erythropoiesis.[42]

ANEMIA OF CHRONIC DISEASE

Anemia of chronic disease may be secondary to infections, autoimmune disorders, chronic renal failure, or malignancies. It is characterized by an immune activation with an increase in inflammatory cytokines and resultant increase in hepcidin levels. Inappropriate erythropoietin levels or hyporesponsiveness to erythropoietin hormone and reduced red blood cell survival contribute to the anemia.[43]

APLASTIC ANEMIA

Aplastic anemia is a group of disorders characterized by pancytopenia, variable bone-marrow hypocellularity, and the absence of underlying malignant or myeloproliferative disease. Due to diverse causes, such as exposures to toxins and

radiation, reactions to drugs and viruses, and inherited defects in telomerase and DNA repair.[44]

Causes of Pure red cell aplasia*:*

- Acute: due to Parvovirus B19 infection (may persist in immunosuppressedpatients)
- Chronic: Associated with thymoma, large granular lymphocytic leukemia, neutralizing antibodies against erythropoietin, and other autoimmune phenomenon.

INVESTIGATING ANEMIA

Initially a complete blood count (CBC) is performed and the values evaluated. Further evaluation will depend on the altered parameters in CBC. Hb value is low then will be evaluated with peripheral smear and reticulocyte count.

Further proceedings is based on bone marrow examination which may reveal some conditions such as, marrow infilteration, myeloma, aplastic anemia, myelodysplasia, etc.

MANAGEMENT OF IRON DEFICIENCY ANEMIA

The most common anemia is nutritional anemia, of which iron deficiency is the front runner. Management of iron deficiency is mainly by supplementation of iron. The treatment modalities for managing Iron deficiency will depend on the underlying

cause. Once the cause has been ascertained, oral iron therapy is commonly prescribed to the patient to correct the iron deficiency. The treatment of iron deficiency should always start with oral iron, both to correct anemia as well as to replenish the body stores. When this fails because of conditions such as large blood losses, iron malabsorption, or intolerance to oral iron, parenteral iron can be prescribed using iron dextran, iron gluconate or iron sucrose. Parenteral iron therapy is necessary for patients with intolerance or unresponsiveness to oral iron therapy. Blood transfusions is reserved for patients with or who are at risk of cardiovascular instability due to their degree of anemia. The ultimate goal in the management of anemia is to correct anemia, maintain the iron status parameters within the recommended levels and to improve the patient survival rate.[45]

Latest studies have revealed that, Erythropoietin combined with parenteral iron is an effective treatment for moderate and severe iron deficiency anemia, with minimal adverse or side effects. It is an effective alternative to blood transfusion, or may be used in cases of resistant anemia that are not been effectively treated by iron supplementation alone.[46]

STUDIES CONDUCTED ON ANEMIA

Study done by VaniSrinivas et al Department of Community Medicine, Grant Medical College, JJ Hospital Campus, Byculla, Mumbai, Maharashtra, India in 2015 showed that the overall prevalence of anaemia was 78.3%. Prevalence of mild, moderate and severe anemia was 64.2%, 36.2% and 0.6% respectively. [47]

Debjit Chattopadhyay et al in 2013 in eastern India, showed that of the total, 15.08 % anemic cases were less than 13 years of age i.e. paediatric age group, 7.46%

cases belonged to 13-18 years age group, 24.60% cases belonged to the age group of 18 -30 years (young adults), 17.50% cases belongs to the age group of 30 – 40 years, 15.29% cases belonged to 40-50 years age group, 10.8% cases belonged to 50-60 years age group, 6.51% cases fall in the 60-70 years age group, and 2.61% cases belongs to old age (greater than 70 years). The study also revealed that 1.78% cases had severe anaemia (Hb less than 7g%) and 31.75% cases had mild to moderate anaemia(Hb 7-10g%). And out of total anemic patients more than 87% were females.[48]

Bharati et al in 2015 in India observed that, the highest and the lowest prevalence of anemia have been found to be in the central and the northeast zones, respectively. The vulnerable groups were the children belonging to illiterate families and to the low socioeconomic strata in the rural areas.[49]

Prevalence of nutritional anemia in pregnant woman in India is a major problem. 33-89% among pregnant women are anemic. Among adolecent girl 60%s are anemic as observed by Toteja G.S et al in a study conducted in 16 districts of India in 2006.[50]

The development of anemia was largely influenced by literacy, occupation, consumption of iron, vit B12, folate, parity, and fertility, and the Weekly administration of iron-folate turned out to be a practical and effective strategy for preventing anemia in adolescent girls. This was also seconded by K.N. Agarwal et al [51].

In a study by Verma M. et al in 1998 in Punjab, prevalence of anemia was 51.5%. Girls had a significantly higher prevalence of anemia, exception- at 5 years and 10-12 years age group. More menarcheal girls were anemic as compared to non-menarcheal ones. Compared to non-vegetarians (38%), more vegetarians (65.9%) were anemic[52].

In a Community Based Study study conducted by Dr. Mallikarjuna M et al in 2015 in India, the prevalence of anemia among adolescent girls was 56.3%. About 48.3% of the normal and 45.8% of the anemic girls were aged between 10 – 13 years. The age of 29.1% of the adolescent anemic girls were aged between 14 – 16 years and 24.5% were aged between 17 – 19 years. More than half of adolescent girls in this study belonged to Class IV socio – economic state and nuclear families[53].

The prevalence of anemia in children according to Zuffo CR et al in 2016 in Rio, was 34.7%. the major factors associated with anemia were: maternal age less than 28 years, male child, children less than 24 months, and children who did not consume iron food sources such as meat, beans, and green leafy vegetables. There was no significant association between anemia and iron rich food intake. [54]

According to Mohammad Intekhab Alam Chand et al, the prevalence of anaemia among adolescent girls in the year 2016 in bihar was 75.74%. The percent of severely, moderately and mildly anemic girls were 5.44%, 32.18%, and 38.12% respectively. Percentage of anemia was high in adolescent girls belonging to joint family as compared to nuclear family. Mean BMI of anemic girls was 17.74. The prevalence was maximum among adolescent girls with illiterate mothers (85.71%).[55]

The prevalence of anemia according to a study on Anemia and nutritional status of pre-school children in Kerala by George in 2000 was 11.4%. The percentage of anemic children among male and female children were 10.25 and 12.55 respectively and statistical analysis showed that female children were more susceptible to anemia than male children. Normal nutritional status was seen among 46.7% of the children. There was significant association between moderate under nutrition and anemia and undernutrition was attributed as the major reason for nutritional anemia.[56]

Among the 1000 adolescent girls included in the study conducted by Deshpande NS et al in 2013 in India, 60% were anemic, 18.4% had mild anemia, 41.3% had moderate anemia, and 0.4% were having severe anaemia. The highest prevalence of anemia was noted among adolescent girls belonging to class III, IV, V of modified kuppuswamy classification and severely thin adolescent girls were found to be at a higher risk for developing anemia.[57]

A study conducted in the three districts of orissa by bulliya G et al in 2007, reaveled that prevalence of anemia was high in non school going girls. Of the total adolescence girls, 96.5% were anemic with 45.2% having mild, 46.9% having moderate and 4.4% having severe anemia[58].

According to a study on anemia and diabetes, conducted by Ranil et al, in Chennai in the year 2010, the prevalence of anemia in T2DM patients were 12.3%. Between the age 40- 49, the prevalence was higher in women than in men (26.4% v 10.3%). Men with anemia had double the risk of developing diabetic retinopathy than women. Anemia was associated with the development and progression of both microvascular and macrovascular complications of diabetes. Anemia also leads to a

falsely low HbA1c levels, which result in the under treatment of hyperglycemia and the progression of both microvascular and macrovascular diabetic complications[59].

Roosy Aulakh et al in the year 2009 studied the utility of red cell distribution width (RDW) in the diagnosis of iron deficiency among children with microcytic hypochromic anemia and concluded that RDW had limited specificity for diagnosis of Iron deficiency anemia among children with microcytic hypochromic anemia[60].

A study carried out by P.R. Deshmukh et al, in Maharashtra, India to study the 'effectiveness' of a weekly iron-supplementation regimen among urban-slum, rural, and tribal girls of the district in the year 2008 showed that the overall prevalence of anaemia came down significantly from 65.3% to 54.3%. The decline was significant in tribal girls (48.6% from 68.9%) and among rural girls (51.6% from 62.8%).[61]

Sabita Basu et al in 2005 showed that the overall prevalence of anemia (as per WHO Guidelines) was significantly higher among adolencent girls (23.9%) as compared to the opposite sex. Anemia was observed more in the rural part of India (25.4%) as compared to urban India (14.2%). Iron stores estimated by serum ferritin in the selected subjects were deficient in 81.7% of adolescent girls and 41.6% of the adolescent boys.[62]

Kumar et al showed that the prevalence of anemia (considering anemia as Hb concentration <11 g/dL) was 69.6%, whereas the prevalence of iron deficiency anemia was 31%. The main predictors for Hb concentration were the biomarkers such as plasma concentrations of serum transferrin, folate, vitamin B12 among. Length-for-age *Z* score and family income also predicted Hb concentration.[63]

A study on the prevalence of anaemia, conducted among the Scheduled Caste preschool children in the state of Punjab in 2002 by Sidhu S et al, revealed that the overall prevalence of anemia was 81.66%. The prevalence of anemia was maximum in age group >2 and minimum in age group >5.[64]

According to a community trial carried out by Jolly R et al in Vellore District, Tamil Nadu in 2000, the prevalence of anaemia was 44.8% with severe anaemia being 2.1%, moderate being 6.3% and mild anaemia being 35.5%. The prevalence of anaemia was 40.7% in prepubertal girls compared to 45.2% in postpubertal girls. A significant association was found between the haemoglobin concentration and the girl's and maternal literacy status.[65]

A study conducted by Sampathkumar et al in 1997, in one of the many rural blocks of tamil nadu in 1997 investigated the prevalence of both anemia and hookworm infestation in female adolescents in rural part of Tamil nadu. The prevalence of anemia was found to be 76.6%. Of the total, 63% had hookworm infestation and 48.5% of the girls did not wear slippers when they went outside.[66]

A study by Bharathi et all in 2009, that investigated the severity and distribution of anemia among Indian adolescent girls of age group 10 to 19 years and the association of anemia with socioeconomic and demographic factors, showed that the highest prevalence of anemia (99.9%) was observed in Jharkhand in the eastern part of India. The prevalence of anemia in the northeastern states were relatively low. The highest prevalence rates were observed among older girls (15 to 19 years), illiterate girls living in rural areas, girls from illiterate households and households with low standard of living, girls from Scheduled Tribes and those living in west India, and married girls.[67]

MATERIALS AND METHODS

STUDY DESIGN

Cross-sectional study

STUDY SUBJECT

Patients attending medicine department in Sree Mookambika Institute of Medical Sciences,Kulasekharam.

INCLUSION AND EXCLUSION CRITERIA

INCLUSION CRITERIA

- All the new patients attending in Medicine Department(outpatient and inpatient) in Sree Mookambika Intitute of medical Sciences with haematological problems

- age group 15-85 years.

EXCLUSION CRITERIA

- elderly age(>85years)

- any contraindication for invasive evaluation

STUDY AREA

Sree Mookambika Institute Of Medical Sciences

STUDY PERIOD

18months(January 2016-june 2017)

SAMPLE SIZE CALCULATION

Sample size is calculated by using the formula

$$N = \frac{4pq}{(d^2)}$$

p- Prevalance of anemia=55%(Gerardo Alvarez-Uria et al study in South India)

q-100-p=45

d-15% of p=15% of 55=8

N-SAMPLE SIZE

MINIMUM SAMPLE SIZE-154

SAMPLE SIZE - 160

SAMPLING TECHNIQUE

By convenient sampling,the patients who came to medicine department who fulfilled our inclusion criteria.

PROCEDURE IN DETAIL

ETHICAL CLEARANCE

Clearance from the instutional ethical committee was obtained. Permission to conduct the study was obtained from the Head of the institute of Sree Mookambika

Institute of medical sciences. Informed consent was obtained from the patients who were included in the study.

A brief history was obtained. The history included socio-demographic factors,dietary habits,history of previous diagnosis of anemia and utilisation of health services for the same(blood transfusion), menstrual history,history of any bleeding manifestations,history suggestive of any chronic illness,history of alcohol intake and any drug history.

Data collected by laboratory investigations was to be entered on the same data collection tool.

LABORATARY INVESTIGATIONS:

All the laboratory investigations were done in CENTRAL LAB,SREE MOOKAMBIKA INSTITUTE OF MEDICAL SCIENCES. Blood samples were collected for laboratory evaluation of CBC by a trained laboratory technician. The left dorsum of hand was cleaned with cotton soaked in spirit and allowed to dry.With the help of a 3mL syringe blood is withdrawn and collected in 2 containers one for hematology the other for serology.

COMPLETE BLOOD COUNT :

(1) Hb,PCV,TC,PLATELET,RBC,MCV,MCH,MCHC,RDW

The blood sample collected in the red container(hematology) is allowed to mix properly by placing it on the mixer attached to the automated CBC machine. This EDTA sample is placed in an automated machine “MINDRAY BC-500” through which we get the complete CBC reprt in the computer connected to the machine.

(2)ESR

The EDTA sample collected is kept in an automated machine "VES MATIC" and ESR value is obtained.

RETICULOCYTE COUNT:

The EDTA sample obtained is mixed with equal amounts of reticulocyte dilution fluid and is incubated for half an hour at 37◦C. Then a smear is made by taking a drop of incubated sample on a slide and holding another slide as spreader at an angle of 30-45◦ and sliding it forward rapidly over the slide. This smear is looked at with a microscope after staining it with leishman's stain,followed by adding distilled water(double the amount of stain) and number of reticulocyte is counted by a pathologist under oil immersion lens of microscope.

CRP:

CRP is estimated by turbidometry method by the machine "TURBODYNE SC"(TULIP GROUP) .It is done by first inserting the card,and following the steps being shown on the machine screen,ie, first 20µL of reagent 1 is taken followed by 20µL of blood sample and kept in incubator at 37^{o}C for 5 minutes following which the incubated sample is placed in the machine and 20 µL of Reagent 2 is added and the value is obtained.

PERIPHERAL SMEAR:

Peripheral semar is done by slide method. A drop of blood is placed in the centre 1-2cm from one end. Another slide is used as a spreader,holding the same in 30-45◦ near the drop of blood.The spreader is moved backwards so that it makes contact with the drop of blood.The spreader is then moved forward rapidly over the slide.A thin peripheral blood film is prepared.it is dried and then stained using leishman's stain. Then distilled water is pored over the stained film doule the amount

of stain used.The slide is washed after 1-2 minutes,dried and examined under oil immersion lens of the microscope.

SERUM IRON:

Clotted sample is used to obtain Serum iron values by using the automated machine “BECKMAN COULTER”.

SERUM FERRITIN:

Serum Ferritin values are obtained by doing hormonal assay (CLIA method) by an automated machine “BECKMAN COULTER”

SERUM TIBC:

Clotted sample is used to obtain TIBC values by using the automated machine “BECKMAN COULTER”.

STOOL:

Stool sample is collected in a container by the patient. By card test stool is tested for blood,ova by using a buffering solution.

VITAMIN B12:

Vitamin B12 values are obtained by hormonal assay done in an automated machine “BECKMAN COULTER”.

RFT:

Clotted sample collected is taken and serum is separated and the seperataed serum is passed through the automated machine “BECKMAN COULTER”.

LFT:

Clotted sample collected is taken and serum is separated and the seperataed serum is passed through the automated machine "BECKMAN COULTER".

DEFINITIONS

1.) ANEMIA

WHO criteria was used for the diagnosis of anemia

POPULATION	**NON ANEMIA(Hb in gms/dl)**	**ANEMIA(Hb in gms/dl)**		
		MILD	MODERATE	SEVERE
Children 6-59 months of age	11 or higher	10-10.9	7-9.9	<7
Children 5-11 years of age	11.5 or higher	11-11.4	8-10.9	<8
Children 12-14 years of age	12 or higher	11-11.9	8-10.9	<8
Non-pregnant women(15 years and above)	12 or higher	11-11.9	8-10.9	<8
Pregnant women	11 or higher	10-10.9	7-9.9	<7
Men(15 years and above)	13 or higher	11-12.9	8-10.9	<8

2.) SOCIO ECONOMIC CLASSIFICATION

The socio economic statuses of subjects were estimated by using modified B.G Prasad's scale. The classification is based on the consumer price index 274 during May 2017

INCOME	SOCIO ECONOMIC CLASS
>6254	Upper class
3127-6253	Upper middle class
1876-3126	Middle class
938-1875	Lower middle class
<938	Lower class

RESULT

SOCIODEMOGRAPHIC CHARACTERISTICS

AGE DISTRIBUTION

The distribution of age in the study participants ranges from 18 to 71 years. The mean age of study participants were 37.88 years (95% CI is 34.998, 40.242) with a SD of 16.63 years.

Distribution according to age of participants

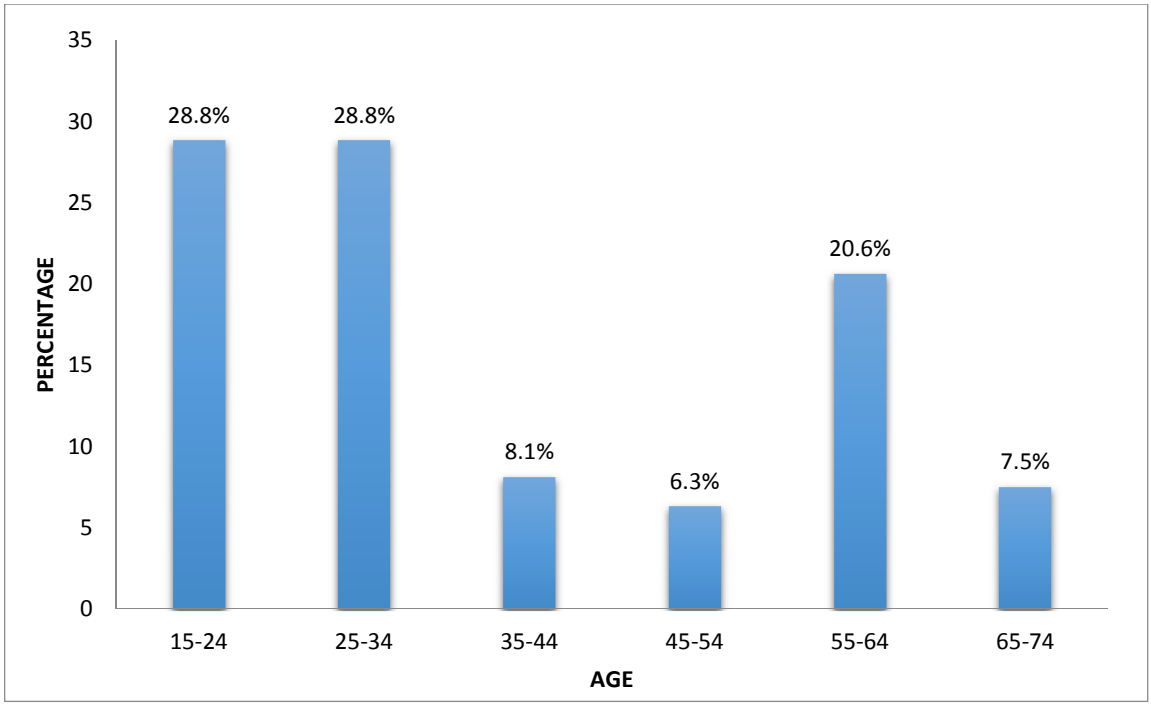
Age characteristics	Value
Minimum	18
Maximum	71
Mean	37.88
Standard deviation	16.63

Majority of the study participants were 15-24 and 25-34 years of age group (28.8%), followed by 55-64 years of age group (20.6%).

Age distribution in the study population

Age	Frequency	Percent
15-24	46	28.8
25-34	46	28.8
35-44	13	8.1
45-54	10	6.3
55-64	33	20.6
65-74	12	7.5
Total	160	100

Age of the study participants



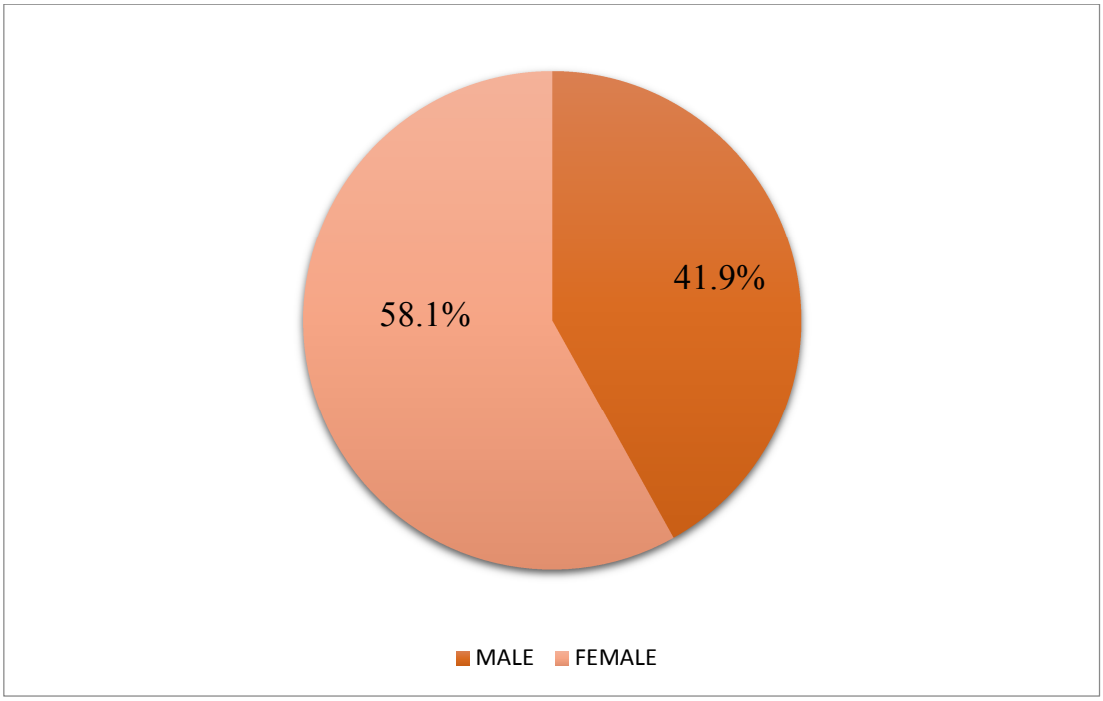
GENDER

Majority of the study participants were females (58.1%).

Distribution of gender

Gender	Frequency	Percent
Male	67	41.9
Female	93	58.1
Total	160	100

Gender of the study participants



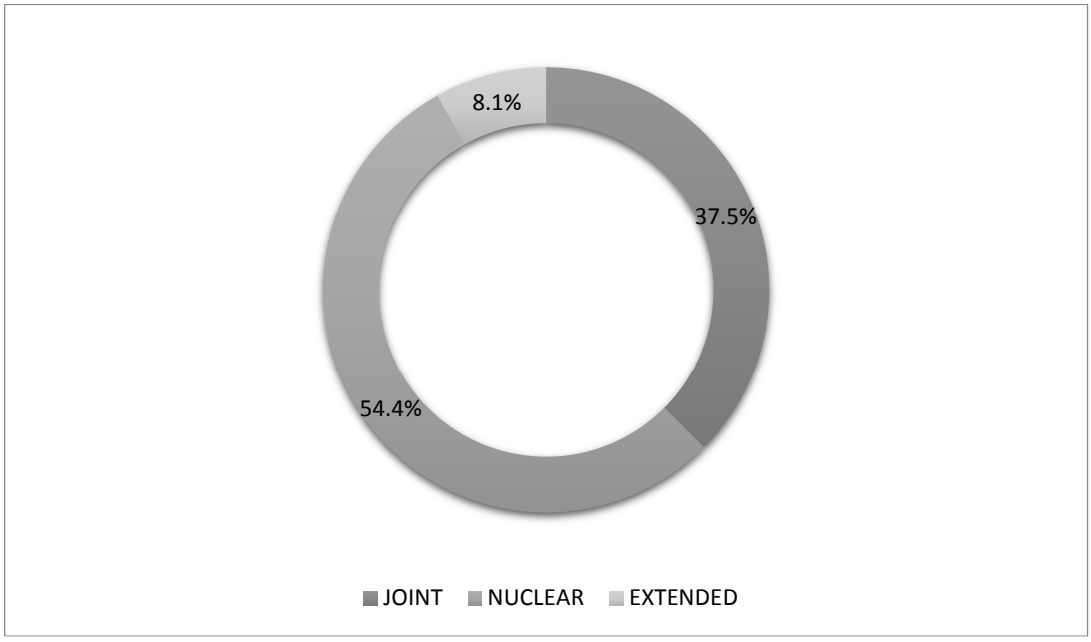
TYPE OF FAMILY

Most of the study participants were from nuclear family (54.4%)

Distribution of type of family

Type of family	Frequency	Percent
Joint	60	37.5
Nuclear	87	54.4
Extended	13	8.1
Total	160	100

Type of family



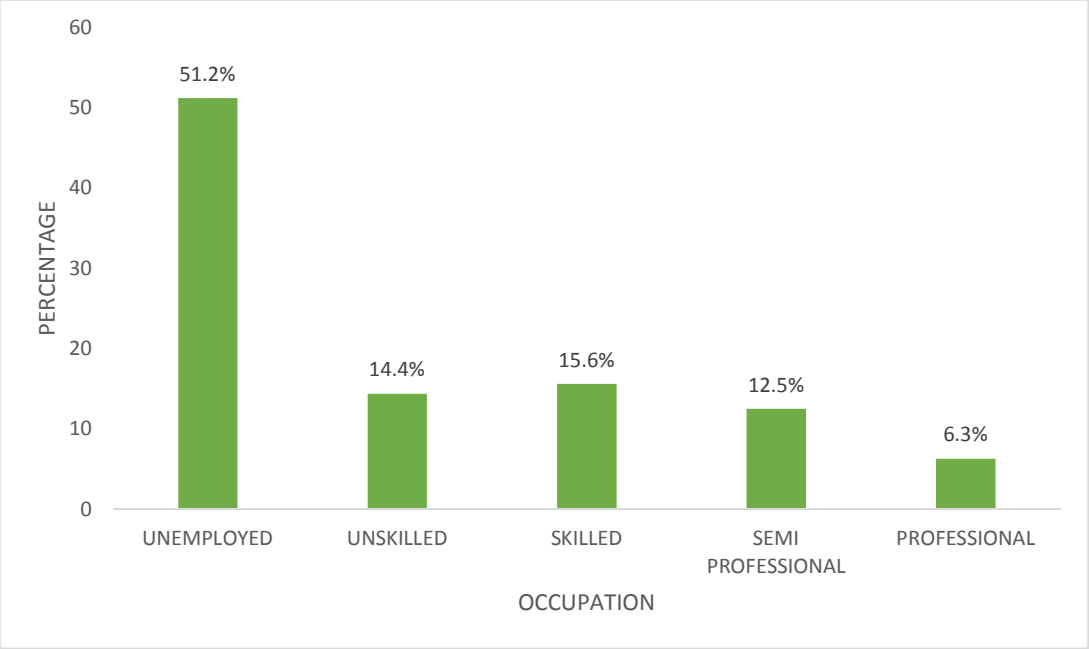
OCCUPATION

51.2% (82) of the study participants were unemployed followed by 15.6% (25) were skilled.

Occupation of the study participants

Occupation	Frequency	Percent
Unemployed	82	51.2
Unskilled	23	14.4
Skilled	25	15.6
Semi professional	20	12.5
Professional	10	6.3
Total	160	100

Occupational status of the study participants



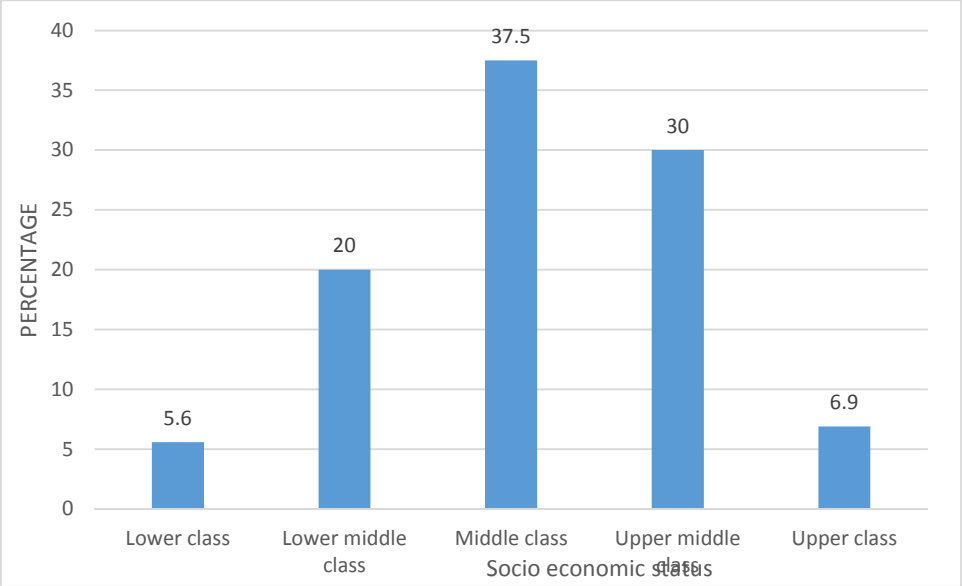
SOCIO ECONOMIC STATUS

37.5% (60) of the study participants belongs to middle class family followed by 30% (48) belongs to upper middle class family

Distribution of socio economic status

Socio Economic Status	Frequency	Percent
Lower class	9	5.6
Lower middle class	32	20.0
Middle class	60	37.5
Upper middle class	48	30.0
Upper class	11	6.9
Total	160	100.0

Socio economic status of the study population



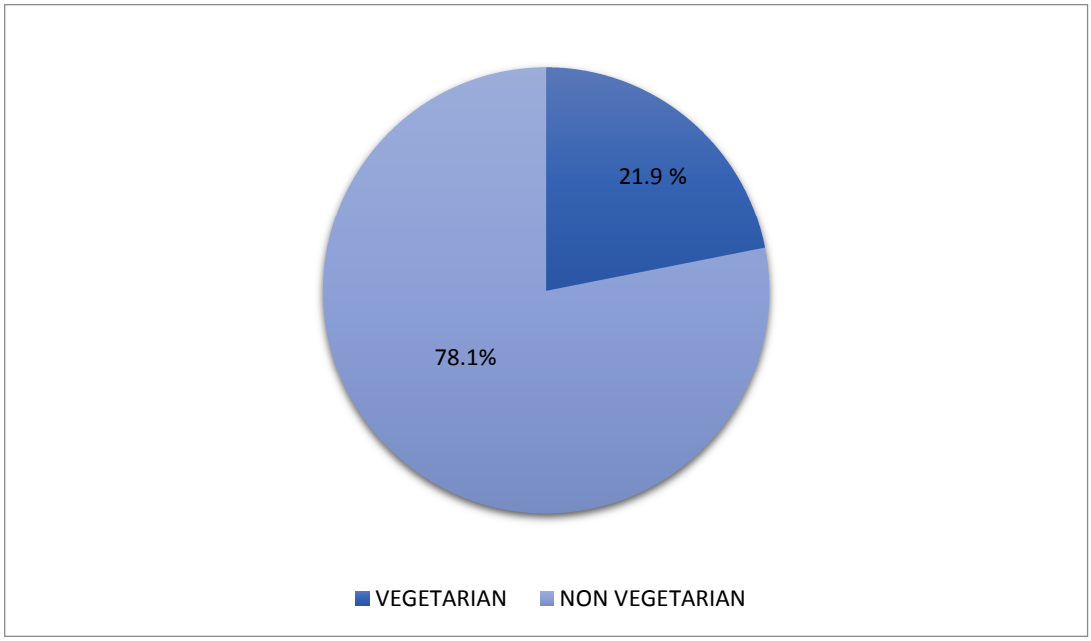
DIET

Most of the study participants were non vegetarians (78.1%)

Distribution of food habits

Food Habits	Frequency	Percent
Vegetarian	35	21.9
Non vegetarian	125	78.1
Total	160	100.0

Food habits of the study population



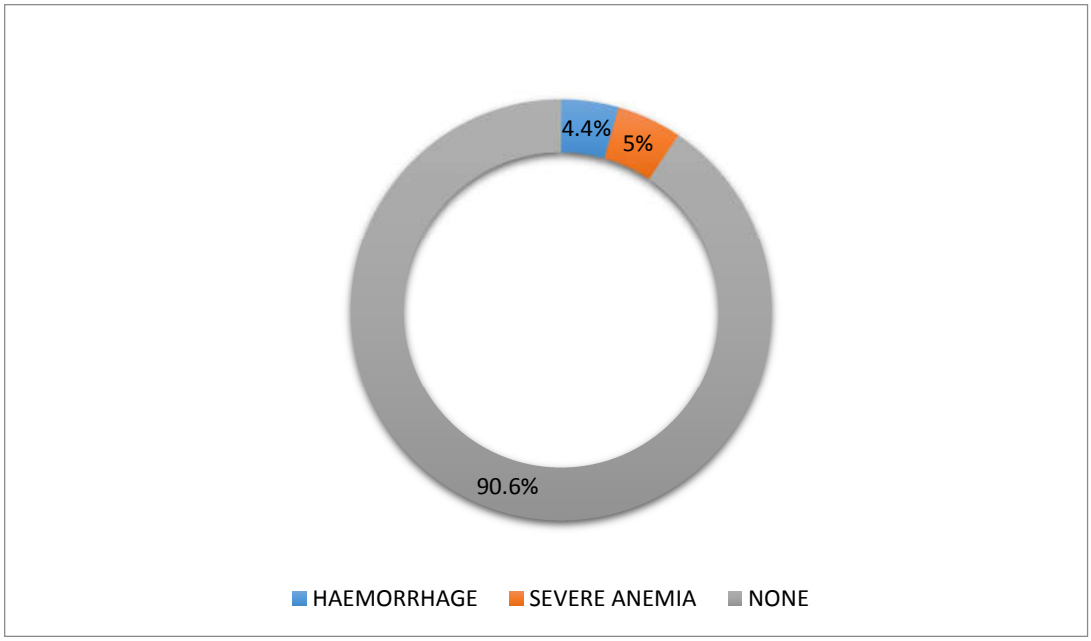
REASON FOR BLOOD TRANSFUSION

4.4% has taken blood transfusion due to haemorrhage and 5% has taken blood transfusion due to severe anaemia.

Distribution of reason for blood transfusion

Reason for Blood Transfusion	Frequency	Percent
Haemorrhage	7	4.4
Severe anaemia	8	5.0
None	145	90.6
Total	160	100

Reason for blood transfusion in the study population



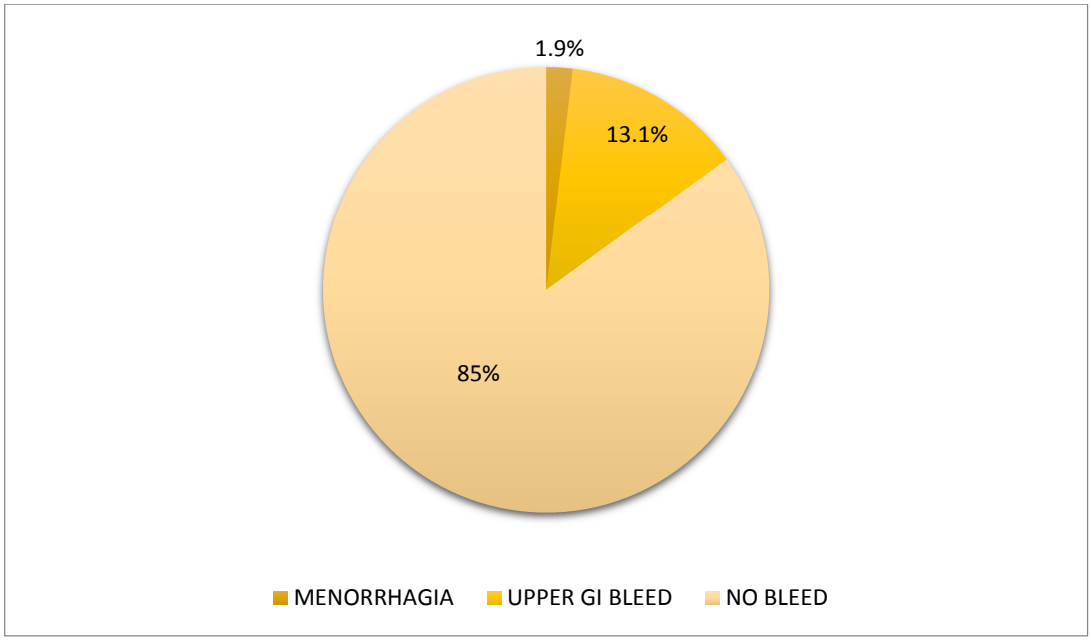
EXCESSIVE BLEEDING

13.1% (21) suffered from excessive bleeding due to upper GI bleed and only 1.9% (3) suffered from excessive bleeding due to menorrhagia.

Distribution of excessive bleeding

Excessive Bleeding	Frequency	Percent
Menorrhagia	3	1.9
Upper GI bleed	21	13.1
No bleed	136	85.0
Total	160	100

Excessive bleeding



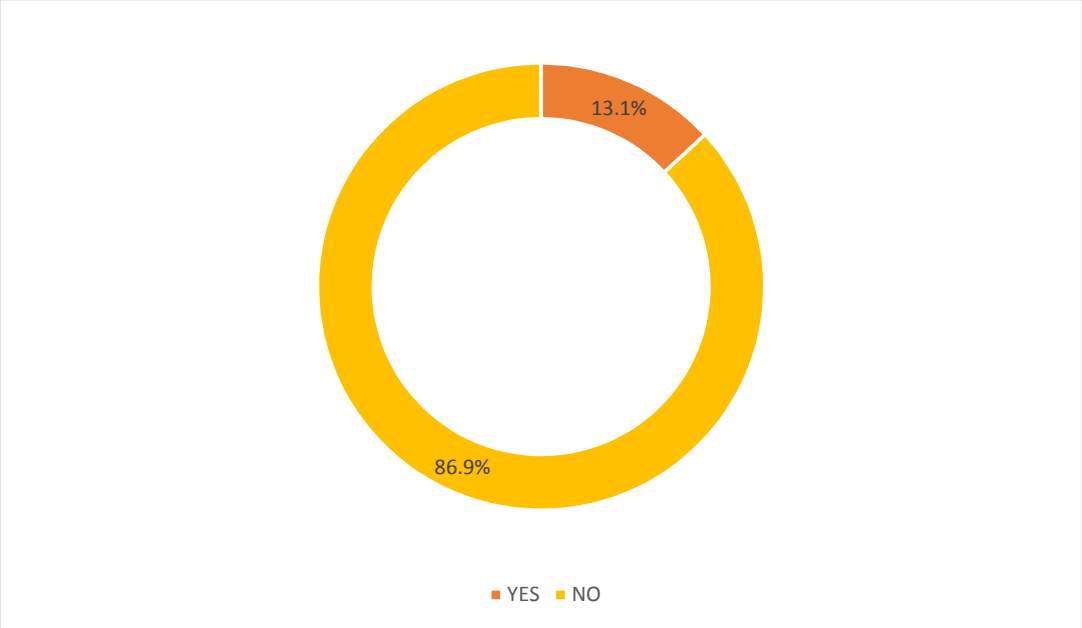
ALCOHOL

Only 13.1% (21) of the study participants were alcoholic.

Distribution of alcohol consumption among study population

Alcohol Consumption	Frequency	Percent
Yes	21	13.1
No	139	86.9
Total	160	100

Alcohol consumption in the study population



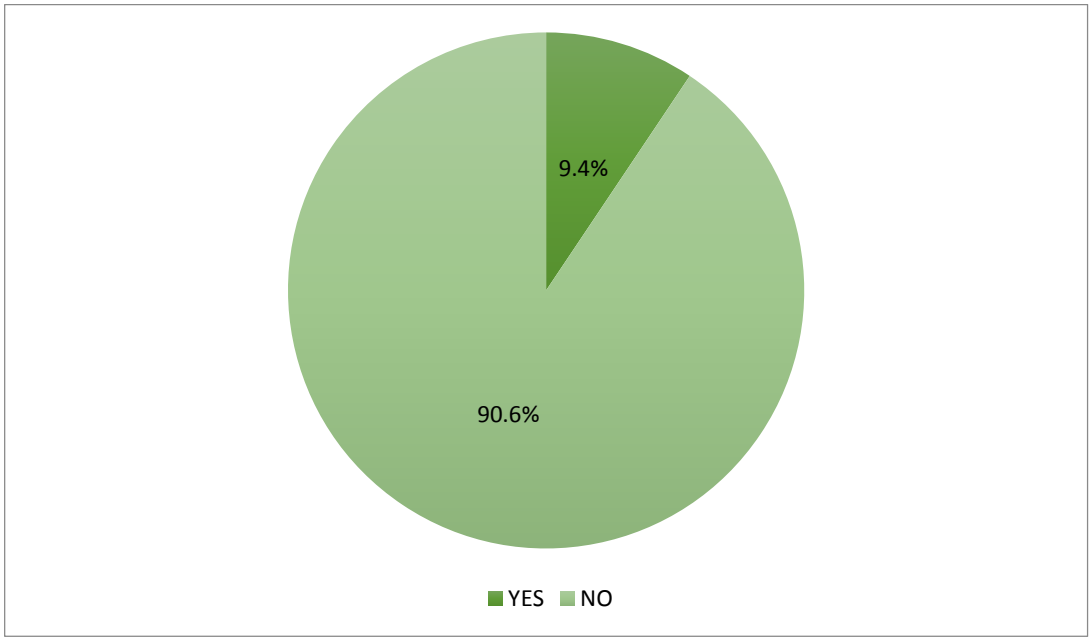
CHRONIC ILLNESS

Only 9.4% (15) of the study participants suffered from chronic illness.

Distribution of chronic illness

Chronic Illness	Frequency	Percent
Yes	15	9.4
No	145	90.6
Total	160	100.0

Chronic illness in the study population



DRUG HISTORY

There is no history of drug intake among the study participants.

HAEMOGLOBIN

The distribution of haemoglobin in the study participants ranges from 2.5 to 17. The mean age of study participants were 9.8 years (95% CI is 9.242, 10.358) with a SD of 3.6.

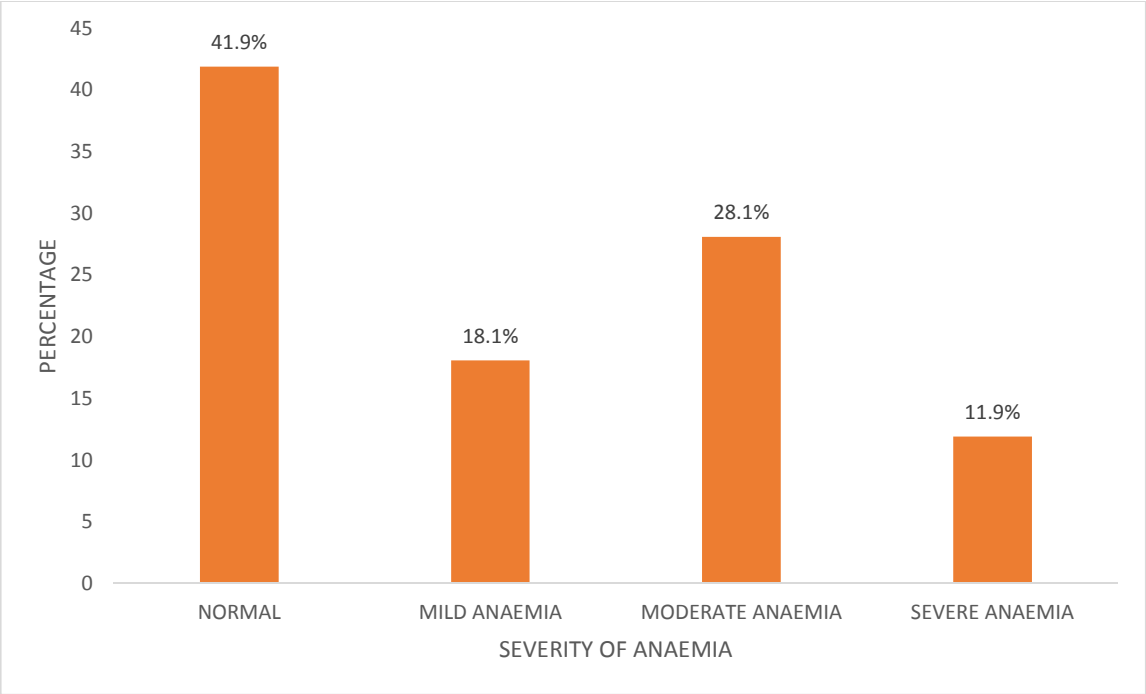
PREVALENCE OF ANAEMIA

58.1% (93) of the study participants were anaemic. Among them 28.1% (45) had moderate, 18.1% (29) had mild and 11.9% (19) severe anaemia.

Prevalence of anaemia in the study population

	Frequency	Percent
Normal	67	41.9
Mild	29	18.1
Moderate	45	28.1
Severe	19	11.9
Total	160	100

Prevalence of anaemia in the study population



CRP

Only 60 patients was tested for CRP. Out of this 14 participants were positive for the test. Among them 57.1% (8) had mild anaemia and the remaining had moderate anaemia (42.9%)

Prevalence of anaemia among CRP positive patients

	Frequency	Percent
Mild	8	57.1
Moderate	6	42.9
Total	14	100

FACTORS RELATED TO ANAEMIA

In our study there was a statistically significant association between prevalence of anaemia and age

Distribution of anaemia by age

Age	Anaemia		Total
	Anaemic N (%)	Non anaemic N (%)	
15-24	15 (32.6)	31 (67.4)	46
25-34	24 (52.2)	22 (47.8)	46
35-44	4 (30.8)	9 (69.2)	13
45-54	6 (60)	4 (40)	10
55-64	32 (97)	1 (3)	33
65-74	12 (100)	0	12
TOTAL	93 (58.1)	67 (41.9)	160
Chi-square = 52.358		p-value = 0.001*	

*p <0.05 is statistically significant

The study revealed there was no statistically significant association between prevalence of anaemia and gender.

Distribution of anaemia by gender

Sex	Anaemia		Total
	Anaemic N (%)	Non anaemic N (%)	
Male	41 (61.2)	26 (38.8)	67
Female	52 (55.9)	41 (41)	93
Total	93 (51.8)	67 (41.9)	160
Chi-square = 0.446		p-value = 0.504	

*p <0.05 is statistically significant

There was no statistically significant association between prevalence of anaemia and type of family

Distribution of anaemia by type of family

Type of family	Anaemia		Total
	Anaemic N (%)	Non anaemic N (%)	
Joint	38 (63.3)	22 (36.7)	60
Nuclear	51 (58.6)	36 (41.4)	87
Extended	4 (30.8)	9 (69.2)	13
Total	93 (58.1)	67 (41.9)	160
Chi-square = 4.674		p-value = 0.097	

*p <0.05 is statistically significant

There was a statistically significant association between prevalence of anaemia and occupation

Distribution of anaemia by occupation

Occupation			Total
	Anaemic N (%)	Non anaemic N (%)	
Unemployed	45 (54.9)	37 (45.1)	82
Unskilled	15 (65.2)	8 (34.8)	23
Skilled	20 (80)	5 (20)	25
Semi professional	11 (55)	9 (45)	20
Professional	2 (20)	8 (80)	10
Total	93 (51.8)	67 (41.9)	160
Chi-square = 11.694		p-value = 0.018*	

*p <0.05 is statistically significant

There was a statistically significant association between prevalence of anaemia and socio economic status.

Socio economic status			Total
	Anaemic N (%)	Non anaemic N (%)	
Lower class	9 (100)	0	9
Lower middle class	23 (71.9)	9 (28.1)	32
Middle class	37 (61.7)	23 (38.3)	60
Upper middle class	22 (45.8)	26 (54.2)	48
Upper class	2 (18.2)	9 (81.8)	11
Total	93 (51.8)	67 (41.9)	160
Chi-square = 19.832		p-value = 0.000*	

*p <0.05 is statistically significant

There was no statistically significant association between prevalence of anaemia and food habits.

Distribution of anaemia by food habits

Diet	Anaemia		Total
	Anaemic N (%)	Non anaemic N (%)	
Vegetarian	22(62.9)	13(37.1)	35
Non vegetarian	71(56.8)	54(43.2)	125
Total	93(58.1)	67(41.9)	160
Chi-square = 0.412		p-value = 0.521	

*p <0.05 is statistically significant

There was no statistically significant association between prevalence of anaemia and alcohol consumption

Distribution of anaemia by alcohol consumption

Alcohol	Anaemia		Total
	Anaemic N (%)	Non anaemic N (%)	
Yes	9 (42.9)	12 (57.1)	21
No	84 (60.4)	55 (39.6)	139
Total	93 (58.1)	67 (41.9)	160
Chi-square = 2.315		p-value = 0.128	

*p <0.05 is statistically significant

There was a statistically significant association between prevalence of anaemia and chronic illness.

Distribution of anaemia by chronic illness

Chronic illness	Anaemia		Total
	Anaemic N (%)	Non anaemic N (%)	
Yes	15 (100)	0	15
No	78 (53.8)	67 (46.2)	145
Total	93 (58.1)	67 (41.9)	160
Chi-square = 11.924		p-value = 0.001*	

*p <0.05 is statistically significant

There was a statistically significant association between prevalence of anaemia and reason for blood transfusion.

Distribution of anaemia by reason for blood transfusion

Reason for blood transfusion	Anaemia		Total
	Anaemic N (%)	Non anaemic N (%)	
Haemorrhage	7 (100)	0	7
Severe anaemia	8 (100)	0	8
None	78 (53.8)	67 (46.2)	145
Total	93 (58.1)	67 (41.9)	160
Chi-square = 12.532 p-value = 0.001*			

*p <0.05 is statistically significant

There was a statistically significant association between prevalence of anaemia and reason for blood transfusion.

Distribution of anaemia by excessive bleeding

Excessive bleeding	Anaemia		Total
	Anaemic N (%)	Non anaemic N (%)	
Menorrhagia	3 (100)	0	3
Upper GI bleed	21 (100)	0	21
No bleed	69 (50.7)	67 (49.3)	136
Total	93 (58.1)	67 (41.9)	160
Chi-square = 24.091		p-value = 0.000*	

*p <0.05 is statistically significant

DISCUSSION

The overall prevelance of anemia in our present study was found to be 58.1% of which 28.1% has moderate,18.1% has mild and 11.9% has severe anemia.A lower prevelance of anemia(35.1%) was noted by Sanjeev M Chaudhary et al[68]. The study done by the same, had none who was suffering from severe anemia.A higher prevelance of anemia was noted by J.Rajaratnam et al in Tamil Nadu[69]. Toteja.G.S et al found 90% prevelance of anemia among adolescent girls in 16 distriucts of India,with 7.1% having severe anemia.[50]

The highest prevelance of anemia was seen in the age group of 55-64 years in our study ie,34.4% of the total anemic patients,while a study done by Gerardo Alvarez-Uria Et al found that the most commonly affected age group with anemia is in those less than 10 years, especially in those less than 5 years followed by women and older adults[70].Another study done by Sanjay Gupta et al found a high number 42% of anemia cases in the age group of 11-25 years.[71]

In our study there was no statistical significant association of anemia with gender. In our study it was found that out of the total patients with anemia majority were females,55.9%.Likewise in a study done by Sanjay Kumar Gupta et al, among the cases of anemia 82% was females and 18% affected was males[71].In another study done by Nasrin A Qureshi et al, it was noted that mild anemia was more common in

males-52.21% and moderate and severe anemia was more common among females-50.98% and 51.67% respectively.[72]

The majority of subjects with anaemia in the present study has microcytic hypochromic picture in peripheral smear; suggestive of iron deficiency anemia while 20.4% of the anaemic people have dimorphic picture. Sanjeev M Chaudhary et al also found majority of subjects with anaemia (25.4%) had microcytic hypochromic picture while 4.7% had dimorphic picture in peripheral smear[68]. Whereas Khandhuri et all found peak incidence of megaloblastic anemia in the age group of 10-30 years old(48%)[74].

The high prevalence of anemia in child bearing age group is of important public health indication. It is found that anemia accounts for 12.8% of Maternal mortality in Asia[75].Iron requirements are more in pregnancy and iron deficiency is associated with low birth weight,pre-term delivery and maternal death.[76]

There was significant association between anemia and chronic illnesses. In our study all the 15 patients who were suffering from a chronic illness was found to have anemia,ie a 16% of the total anemic patients. The basic pathology behind it being decreased availability of iron,decrease in life span of RBC from 120 days to 70-80 days,and erythropotein deficiency.[77] Out of the 15 patients,12 were CKD patients. The study done by Obrador et al showed that 88% of those with advanced stages of CKD who required renal replacement therapy had a PCV less than 30mg/dl.[78] Anemia is not common in earlier stages of CKD,the prevalence of anemia was 5.2% in

patients with stage III,increasing to 44.1% in stage IV disease.[79] In our present study too,the majority of CKD patients who had anemia were in the advanced stage(IV-V).

SUMMARY

- The present study was an observational study done for the duration of 18months carried out in 160 patients who came to the medicine department of SREE MOOKAMBIKA INSTITUTE OF MEDICAL SCIENCES.

- The overall prevelance of anemia was found to be 58.1%, more of females were found to be anemic .

- The average age group affected was 55-64 years.

- Significant association was found between anemia and age, socio economic status,chronic illnesses and history of blood transfusion.

- Majority of the people were affected by iron deficiency anemia.

CONCLUSION AND RECOMMENDATIONS

The high prevalence of anemia(58.1%) indicates that anemia continues to be a major public health problem in India. Age, socio economic status, literacy are the major determinants that contribute to the problem of anemia. High prevalence despite the easy availability and access to medical care, indicates the level of ignorance and indifferences to health needs.

A major shift in the programme to mandatory regular supply of iron and folic acid and better coverage of the programmes in the rural setup covering people of all age and socio economic groups can bring down the current prevalence of anemia. We have to rectify the nutritional deficiencies with food fortification. Timely interventions for reducing the burden of worm infestations and other chronic illnesses can bring down the number of people affected. Above all, awareness creation and nutrition education on the importance of taking iron supplementation and nutritional counselling on consumption of iron rich foods can prevent anemia especially in the young females.

LIMITATIONS

- The cross sectional study covered only the patients attending to medicine department in a tertiary health hospital in kanyakumari district,which donot exactly give an idea about the overall prevalence of the anemia in the entire patients attending to other departments of the same hospital.

- The study was done only on patients above 15 years,which didn't cover a major proportion of paediatric age group especially the neonates who significantly contribute to the high prevelance of anemia in a national level

- Dietary history couldnot be elicited in detail due to time constraints

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INSTITUTIONAL HUMAN ETHICS COMMITTEE

SREE MOOKAMBIKA INSTITUTE OF MEDICAL SCIENCES,
KULASEKHARAM, TAMILNADU

Communication of Decision of the Institutional Human Ethics Committee(IHEC)

SMIMS/IHEC No:1 /Protocol no: 46/ 2016

Protocol title: PREVALENCE AND ETIOLOGY OF ANAEMIA IN TERTIARY CARE HOSPITAL IN KANAYAKUMARI DISTRICT
Principal Investigator: Dr.Jineesh Raj
Name& Address of Institution: Department of General Medicine Sree Mookambika Institute of Medical Sciences, Kulasekharam ☑ New review ☐ Revised review ☐ Expedited review
Date of review (D/M/Y): 15.12.2016 Date of previous review , if revised application: 10/12/2016
Decision of the IHEC: ☑ Recommended ☐ Recommended with suggestions ☐ Revision ☐ Rejected
Suggestions/ Reasons/ Remarks:
Recommended for a period of :six months

Please note*

- Inform IHEC immediately in case of any Adverse events and Serious adverse events.
- Inform IHEC in case of any change of study procedure, site and investigator
- This permission is only for period mentioned above. Annual report to be submitted to IHEC.
- Members of IHEC have right to monitor the trial with prior intimation.

Reneegalyangadhar

Signature of Member Secretary IHEC

SREE MOOKAMBIKA INSTITUTE OF MEDICAL SCIENCES ★ KULASEKHARAM ★
IHEC
Date 21/12/16

CONSENT FORM

PART 1 OF 2

INFORMATION FOR PARTICIPANTS OF THE STUDY

Dear Volunteers,

We welcome you and thank you for your keen interest in participating in this research project. Before you participate in this study, it is important for you to understand why this research is being carried out. This form will provide you all the relevant details of this research. It will explain the nature, the purpose, the benefits, the risks, the discomfort, the precautions and the information about how this project will be carried out. It is important that you can read and understand the contents of the form carefully. This form may contain certain scientific terms and hence, if you have any doubts or if you want more information, you are to ask the study personnel or the contact person mentioned below before you give your consent and also at any time during the entire course of the project.

1. Name of the Principal Investigator **:** Dr.Jineesh Raj
Postgraduate-M.D General Medicine
SMIMS, Kulasekharam
Mob:9585953436
Email-drjineeshraj@gmail.com

2. Name of the Guide **:** Dr.K Krishnan kutty.
Associate Professor
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SMIMS, Kulasekharam
Mob No: 9443404424
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3. a. Name of the co-guide **:** Dr.R V Mookambika
Asst Professor
Department of Medicine
SMIMS, Kulasekharam
Mob No: 9894278335

Email: drmookambika86@gmail.com

4. Institute: details with Address : Sree Mookambika Institute Of
Medical Science
Kulasekharam,
Kanyakumari District-629161
Tamil Nadu

5. Title of the study:

"Prevalence and etiology of anaemia in tertiary care hospital in kanyakumari district".

6. Background Information:

Hematological disorder include non malignant like various anaemia, bleeding disorder, plateletdisorder,hemoglobinopathies malignant include leukemia, lymphoma, plasma cell dyscrasia,myeloproliferativeneoplasm.The prevalence of anaemia is disproportionately high in developing countries like India. It has mainly been ascribed to poverty, inadequate diet, certain diseases, pregnancy and lactation, and poor access to health services in developing countries. Nutritional anaemia constitutes the most important cause of anaemia. It is mainly due to deficiency of Iron, Vitamin B12 and Folate. Young people are particularly susceptible to develop anaemia because of their rapid growth and associated high iron requirements. This study aims at finding the prevelance of anaemia and the possible causes which helps in the management of anaemia.

7. Aims and Objectives:

1.To study the prevalence of anaemia and its aetiology in patients attending Sree Mookambika Institute Of Medical Science.

2.To find out the proportion of patients with nutritional anaemia.

9. Scientific justification of the study:

Anemia is a common concern in older people ,females and children with associated and can have significant morbidity and mortality. Because anemia is a sign, not a diagnosis, an evaluation is almost always needed to identify the underlying cause. The purpose of this study was to study the laboratory profile of patients with anemia and to study characteristics of hematological types of anemia in such patients as well as the closest possible etiological profile.

Early detection of the same has a better prognosis and brings out a better outcome.

In India, the prevalence of anaemia is high because of

- Low dietary intake, poor iron (less than 20 mg /day) and folic acid intake (less than 70 micrograms/day)
- Poor bio-availability of iron (3-4 percent only) in phytate fibre-rich Indian diet
- Chronic blood loss due to infection such as malaria and hookworm infestations

10. Procedure of the study:

After acceptance of thesis by the ethics committee an informed consent will be obtained from those patients who have abnormal bloodcount parameters,visiting to SMIMS.

History taking performa is used for documenting age/sex/address/clinical information/symtoms/predisposing factors,drug history,socioeconomic status and previous history of treatment.

Investigation-Complete Blood Count(CBC),peripheral smear,reticulocyte count.Results will be analysed and further relevant investigations will be done as per the proforma.

All data collected will be charted and analysed to find the aetiology and prevalence of anaemia,also the proportion of nutritional anaemia wil be obtained.

11.Expected risk of the participants: There are no expected adverse events or risks.

12.Expected Benefits of the Research for the participants: direct benefit available to participants is early detection of the disease.once the patients are detected of various disorders treatment plan can be started at the earliest.

13.Maintenance of confidentiality: All data collected for the study will be kept confidentially. No personal details will be revealed.

14.Why have I been chosen to be in this study: you have abnormal values of blood counts which needs evaluation.So you fulfil the criterion for selection.

15.How many people will be in the study **:** 154

16.Agreement of compensation to the participants **:** No

17.Anticipated prorated payment, if any, to the participants of the study **:** Nil

18.Can I withdraw from study at any time during the study period **:** Yes

19.If there is any new finding/information, would I be informed **:** Yes

20.Expectedduration of the participantsparticipation in the study **:** 12 months

21.Whom do I contact for further information :

For any study related queries,you are free to contact

Dr Jineesh Raj
Post Graduate – M.D General Medicine
Department of General Medicine
SreeMookambika Institute of Medical Sciences,
Kulasekharam, Tamil Nadu -629161
Mobile Number: 9585953436
e-mail I.D:drjineeshraj@gmail.com

-

Place:

Date:

Signature of Participant **Signature**

CONSENT FORM

PART 2 OF 2

PARTICIPANTS CONSENT FORM

The details of the study have been explained to me in writing and details have been fully explained to me. I am aware that the results of the study may not be directly beneficial to me but will help in the advancement of medical sciences. I confirm that I have understood the study and had the opportunity to ask questions. I understand that my participation in the study is voluntary and that I am free to withdraw at any time, without giving any reasons, without the medical care that normally is provided by the hospital being affected. I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s). I have given details of the study. I fully consent to participate in the study titled **"Prevalence and Etiology of anaemia in tertiary care hospital in kanyakumari district."**

Serial no/Reference no:

Name of the participant:

Address of the Participant:

Contact number of the Participant:

Signature/Thumb impression of the participant/Legal guardian

Witness

1.

2.

Date:

Place:

സമ്മത പത്രം – ഭാഗം – 1

പഠനവുമായി സഹകരിക്കുന്ന വ്യക്തികളുടെ അറിവിലേയ്ക്ക്

പ്രിയപ്പെട്ട സന്നദ്ധ സേവകരേ,

ഞങ്ങൾ നിങ്ങളെ സ്വാഗതം ചെയ്യുന്നു. അതോടൊപ്പം ഈ പഠനവുമായി സഹകരിക്കാനുള്ള സന്നദ്ധതയോട് നന്ദി രേഖപ്പെടുത്തുന്നു. നിങ്ങൾ ഈ പഠനത്തിൽ പങ്കെടുക്കുന്നതിനു മുൻപ് ഈ പഠനം എന്തിനാണ് നടത്തപ്പെടുന്നത് എന്ന് അിറയേണ്ടതുണ്ട്. അതിനാൽ ഈ ഫോറത്തിൽ ഗവേഷണ പഠനത്തിന്റെ വിവരങ്ങളും മറ്റും വിശദമായി രേഖപ്പെടുത്തിയിരിക്കുന്നു. ഈ പഠനത്തിന്റെ രീതി, ഉദ്ദേശം, പ്രയോജനം, അപകടസാദ്ധ്യത, ക്ലേശം, മുൻകരുതൽ, എങ്ങനെ ഈ പഠനം മുൻപോട്ടു കൊണ്ടുപോകുന്നു എന്നിങ്ങനെ എല്ലാ വിവരങ്ങളും ഫോറത്തിൽ രേഖപ്പെടുത്തിയി രിക്കുന്നു. സദയം ഈ വിവരങ്ങൾ വായിച്ചു മനസ്സിലാക്കുവാൻ അഭ്യർത്ഥിക്കുന്നു. ഈ വിവരങ്ങളിൽ ശാസ്ത്രപരമായ പദങ്ങൾ ഉള്ളതിനാൽ സംശയനിവാരണത്തിനു പ്രധാന പഠനകർത്താവിനോടോ താഴെ രേഖപ്പെടുത്തി യിരിക്കുന്ന വ്യക്തികളോടോ ഫോറം ഒപ്പിടുന്നതിനു മുൻപോ അല്ലെങ്കിൽ ഈ പഠനത്തിന്റെ കാലാവധി തീരുന്നതുവരേയോ സമീപിക്കാവുന്നതാണ്.

1. സൂത്രധാരൻ : **ഡോ. ജിനേഷ് രാജ്**
ബിരുതാനന്തര ബിരുത വിദ്യാർത്ഥി,
ജനറൽ മെഡിസിൻ വിഭാഗം
ശ്രീ മൂകാംബിക ഇൻസ്റ്റിറ്റ്യൂട്ട് ഓഫ് മെഡിക്കൽ സയൻസ്,
കുലശേഖരം - 629 161.

2. പ്രധാന മാർഗ്ഗദർശി : **ഡോ. കൃഷ്ണൻ കുട്ടി**
പ്രൊഫസർ
ജനറൽ മെഡിസിൻ വിഭാഗം,
ശ്രീ മൂകാംബിക ഇൻസ്റ്റിറ്റ്യൂട്ട് ഓഫ് മെഡിക്കൽ സയൻസസ്,
കുലശേഖരം.

3. സഹ മാർഗ്ഗ ദർശി : **ഡോ. മൂകാംബിക**
വ്യക്ക വിഭാഗം,
ശ്രീ മൂകാംബിക ഇൻസ്റ്റിറ്റ്യൂട്ട് ഓഫ് മെഡിക്കൽ സയൻസസ്,
കുലശേഖരം.

4. ഇൻസ്റ്റിറ്റ്യൂട്ട് : **ശ്രീ. മൂകാംബിക ഇൻസ്റ്റിറ്റ്യൂട്ട് ഓഫ് മെഡിക്കൽ സയൻസസ്**
പടനിലം, കുലശേഖരം, കന്യാകുമാരി - 629 161.
തമിഴ്നാട്.

5. പഠന വിഷയം:

കന്യാകുമാരി ജില്ലയിലെ ത്രിതീയ ആശുപത്രികളിൽ വിളർച്ച സാധ്യതയുടെ ആധിക്യവും, അവയുടെ കാരണങ്ങളും.

6. പഠന പാശ്ചാത്തലം?

രക്തരോഗങ്ങളെ രണ്ടായി തരം തിരിക്കാം. ക്യാൻസർ, ക്യാൻസർ അല്ലാത്തവ, ക്യാൻസർ അല്ലാത്ത രക്തരോഗങ്ങൾ പലതരമുണ്ട്. പലയിന വിളർച്ചകൾ ബ്ലീഡിംങ് അസുഖങ്ങൾ , ഹീമോഗ്ലോബിനോ പതിയസ്, ക്യാൻസർ തന്നെ പലതരമുണ്ട്. രക്താർബുദം, കഴലകൾ, പ്ലാസമാസെൽ ഡിസ്ക്രോസിയ മയിലോപ്രോലിഫിറേറ്റിവ് അസുഖങ്ങൾ .

വളർച്ച എന്നത് വികസ്വര രാജ്യങ്ങളിൽ കൂടുതലായി കാണുന്നു എന്നാണ്. അതിന് കാരണമായത് ദാരിദ്ര്യവും, രോഗാവസ്ഥകളും, ഭക്ഷണകുറവും, ഗർഭം, മുലയൂട്ടൽ രോഗാവസ്ഥയെ തിരിച്ചറിഞ്ഞ് ചികിത്സിക്കുന്നു. സമയകൂടുതലു എന്നിവയെല്ലാമാണ്. പോഷക ആഹാരങ്ങളുടെ കുറവാണ് വിളർച്ചയുടെ ഒരു പ്രധാന കാരണം. ഇതിൽ ഇരുമ്പു സത്ത് വിറ്റാമിൻ ബി12, ഫോളേറ്റ് എന്നി പദാർത്ഥങ്ങൾ ആഹാരത്തിൽ കുറയുന്നതോടെ

വിളർച്ചയുണ്ടാകും. യുവജനങ്ങളിൽ വിളർച്ച അധികമായി കണ്ടുവരുന്നതുകാരണം അവരുടെ വളർച്ചയ്ക്ക് അത്യാവശ്യമായ ഇരുമ്പു സത്ത് കിട്ടുന്നില്ല.

രക്താർബുദത്തിൽ രണ്ടു തരമായ AML, ALL എന്നിവയിൽ AML അധികമായ പത്ത് വയസ്സിനുമേൽ പ്രായമുള്ളവരിൽ കണ്ടെത്തുകയും അഘ എന്ന അവസ്ഥ 65 വയസ്സിന് മേലുള്ളവരിൽ അധികമായി കാണപ്പെടുന്നു. പരമ്പരാഗതമായ അസുഖങ്ങൾ , റേഡിയേഷൻ, ചുറ്റുമുള്ള അന്തരീക്ഷം , മരുന്നുകളോടുള്ള സാമീപ്യം വയറസ്സുകൾ എന്നിവയാണ് രക്താർബുദത്തിന് പ്രധാന കാരണങ്ങൾ.

7. ലക്ഷ്യങ്ങൾ

1. ശ്രീ മൂകാംബിക ഇൻസ്റ്റിറ്റ്യൂട്ട് ഓഫ് മെഡിക്കൽ സയൻസിലെ വിളർച്ച സാധ്യതയുള്ള രോഗികളിൽ അതിന്റെ
ആധിക്യവും, കാരണങ്ങളും തിരിച്ചറിയാനുള്ള പഠനം.
2. പോഷക ആഹാര കുറവുകൊണ്ടുണ്ടാകുന്ന വിളർച്ചയുടെ അനുപാതത്തിന്റെ പഠനം.

8. പഠനത്തെക്കുറിച്ചുള്ള ശാസ്ത്രീയ ന്യായീകരണം

- ഇന്ത്യയിൽ, വിളർച്ചയുടെ വ്യാപ്തി കൂടുതലാണ് അവയുടെ കാരണങ്ങൾ
- ഭക്ഷണക്കുറവ്, ഇരുമ്പുസത്ത് കുറവ് (< 20 എം.ജി/ ദിവസവും) കൂടാതെ ഫോളിക്ക് ആസിഡ് കുറവ് (< 70എം.ജി/ ദിവസവും)
- ഇന്ത്യൻ ഭക്ഷണ രീതിയിൽ ഇരുമ്പു സത്ത് 3-4 ശതമാനം മാത്രമേ ഉള്ളു.
- മലമ്പനി കൊക്കോപ്പുഴു എന്നീ രോഗങ്ങൾ കാരണമുള്ള രക്തക്കുറവ്.

ഈ പഠനത്തിലൂടെ വിളർച്ചയുടെ വ്യാപ്തി ശ്രീ മൂകാംബികാ മെഡിക്കൽകോളേജിലെ രോഗികളിൽ എത്രമാത്രം എന്ന് കണ്ടെത്തുക എന്നതാണ് ഉദ്ദേശം

9. പഠനരീതി

- ഈ പഠനം ശ്രീ മൂകാംബിക ഇൻസ്റ്റിറ്റ്യൂട്ടിൽ വച്ചാണ് നടത്തുന്നത്. ആവശ്യമുള്ള വിവരങ്ങൾ രോഗികളിൽ നിന്നും ശേഖരിക്കുന്നതാണ്.

- രോഗിയുടെ വയസ്സും, മേൽവിലാസവും, രോഗവിവരങ്ങളും അപകട ഘടകങ്ങളും , ചികിത്സാ വിവരങ്ങളും കുറിച്ചു വയ്ക്കുന്നു.

- പരിശോധനകൾ രക്തകോശ അളവുകൾ മജ്ജയിൽ കുത്തിയെടുത്ത് പരിശോധിക്കുക, ഉദരത്തിന്റെ സ്കാൻ, ഇരുമ്പിന്റെ ദാതുകളുടെ പഠനം.

10. പഠനം മൂലം പങ്കെടുക്കുന്ന ആൾക്ക് ഉണ്ടാകാൻ ഇടയുള്ള അപകട സാധ്യത ? ഒന്നുമില്ല

11. പഠനം മൂലം പങ്കെടുക്കുന്ന ആൾക്ക് ഉണ്ടാകുന്ന നേട്ടം ?

വളരെ നേരത്തേ രോഗിയുടെ രോഗാവസ്ഥ കണ്ടെത്തുകയും അവയെ തിരിച്ചറിഞ്ഞ് നേരത്തേ തന്നെ ചികിത്സാരീതി തുടങ്ങാനും സാധിക്കും.

12. വിവരങ്ങൾ രഹസ്യമായി സൂക്ഷിക്കുന്നുണ്ടോ ?

നിങ്ങളിൽ നിന്നും ശേഖരിക്കുന്ന എല്ലാ വിവരങ്ങളും രഹസ്യമായി സൂക്ഷിക്കുന്നതാണ്.

13. ഈ പഠനത്തിൽ നിങ്ങളെ തിരഞ്ഞെടുക്കുന്നതെന്തിന് ?

രക്തപരിശോധനയിൽ അളവുകൾക്ക് വ്യത്യാസമുള്ളതിനാൽ അതിനെ വിലയിരുത്തേണ്ടതാണ്.

14. എത്ര ആളുകൾ ഈ പഠനത്തിൽ ഉൾപ്പെടുന്നു. 154

15. ഈ പഠനത്തിൽ പങ്കെടുക്കുന്നതിന് പ്രതിഫലം ലഭിക്കുമോ? ഇല്ല

16. **പങ്കെടുക്കുന്നവർ പ്രതീക്ഷിക്കുന്ന തുക** – ഇല്ല

17. **എപ്പോൾ വേണമെങ്കിലും എനിക്ക് ഈ പഠനത്തിൽ നിന്ന് പിന്മാറാമോ?** - അതേ

18. **ഈ ഗവേഷണത്തിന്റെ ഫലമായി പുതിയ എന്തെങ്കിലും കണ്ടെത്തലുകളുണ്ടെങ്കിൽ അത് എന്നെ അറിയിക്കുമോ ?**

തീർച്ചയായും, ഈ പഠനത്തിന്റെ കണ്ടെത്തലുകൾ ഈ പഠനാവസാനം നിങ്ങളെ അറിയിക്കുന്നതായിരിക്കും.

19. **ഈ പഠനത്തിന്റെ സമയ ദൈർഘ്യം എത്രയാണ് ?** 12-18 മാസം വരെ

20. **വേറെ ഏതെങ്കിലും വിവരങ്ങൾ ?** ഇല്ല

21. **കൂടുതൽ വിവരങ്ങൾക്കായി താഴെ പറയുന്നവരെ നിങ്ങൾക്ക് ബന്ധപ്പെടാവുന്നതാണ്.**

ഡോ. ജിനേഷ് രാജ്
ബിരുതാനന്തര ബിരുത വിദ്യാർത്ഥി,
ജനറൽ മെഡിസിൻ വിഭാഗം
ശ്രീ മൂകാംബിക ഇൻസ്റ്റിറ്റ്യൂട്ട് ഓഫ് മെഡിക്കൽ സയൻസ്,
കുലശേഖരം - 629 161.
മൊബൈൽ നമ്പർ : 9585953436
ഇ-മെയിൽ ഐഡി: drjineeshraj@gmail.com

സ്ഥലം: കുലശേഖരം മുഖ്യ ഗവേഷകന്റെ ഒപ്പ്

തീയതി : പങ്കെടുക്കുന്ന ആളുടെ ഒപ്പ്

സമ്മതപത്രം

ഭാഗം – 2

ഈ പഠനത്തെക്കുറിച്ചുള്ള വിശദാംശങ്ങൾ എഴുത്തുമൂലമായി എന്നെ അറിയിച്ചുട്ടുണ്ട്. എനിക്ക് ഈ പഠനം കൊണ്ട് നേരിട്ടു ഗുണം ഇല്ലെങ്കിലും ഇത് മെഡിക്കൽ സയൻസിന് വളരെ ഗുണകരമായ ഒന്നാണ്. എനിക്ക് പഠനം പൂർണ്ണമായി മനസ്സിലായതിനാൽ ഞാൻ സ്വയം ഈ പഠനത്തിൽ പങ്കുചേരുകയാണ്. ഏതു സമയത്തും എനിക്ക് ഇതിൽ നിന്നും പിൻമാറാനുള്ള അവകാശം ഉള്ളതായും എന്ന് എനിക്ക് ബോധ്യപ്പെട്ടിരിക്കുന്നു. അതെന്റെ ചികിത്സയിൽ ഏതുവകയിലും തടസ്സമാകാൻ കാരണമാവുകയുമില്ല. ഈ പഠനത്തിൽ നിന്നുമുള്ള ഫലങ്ങൾ വിജ്ഞാനപരമായി മാത്രമായി ഉപയോഗിക്കു എന്നുള്ളതും എനിക്കു ബോധ്യമായി. ഈ പഠനത്തിനെക്കുറിച്ചുള്ള മുഴുവൻ രേഖകളും അടങ്ങിയ പത്രം എനിക്കു ലഭിച്ചു.

ഞാൻ **"കന്യാകുമാരി ജില്ലയിലെ ത്രിതീയ ആശുപത്രികളിൽ വിളർച്ച സാധ്യതയുടെ ആധിക്യവും, അവയുടെ കാരണങ്ങളും."** അന്വേഷണത്തിൽ ഞാൻ പൂർണ്ണമായും സമ്മതിച്ചുകൊണ്ട് പങ്കെടുക്കുന്നു.

സീരിയൽ നമ്പർ / റഫറൻസ് നമ്പർ :
പങ്കെടുക്കുന്ന ആളിന്റെ പേര് :

മേൽവിലാസം :

☐ഒപ്പ് / വിരലടയാളം

സാക്ഷി :

സ്ഥലം :

തീയതി

ஒப்புதல் படிவம்

பகுதி – 1

ஆய்வில் பங்குபெறுவோருக்கான தகவல்கள்

அன்பார்ந்த தன்னார்வலர்களே!

எங்கள் ஆய்வில் தாங்கள் ஆர்வத்துடன் பங்கேற்றமைக்கு நன்றி கூறி வரசேற்க்கிறோம். இதில் கலந்து கொள்வதற்கு முன் இந்த ஆய்வு எதற்காக நடத்தப்படுகிறது என அறிந்திருக்க வேண்டியது முக்கியமாகும். இந்த படிவத்தில் தங்களுக்கு தேவையான அனைத்து தகவல்களும் இருக்கிறது. இந்த ஆய்வின் தன்மை, தேவை, நன்மைகள், ஆபத்துகள் ஆகியவை இந்த படிவத்தில் அடங்கும். இந்த படிவத்தில் அறிவியல் சார்ந்த வார்த்தைகள் இருப்பதால், தங்கள் சந்தேகங்களை ஆய்வின் எந்த நேரத்திலும் கீழே குறிப்பிட்டுள்ள நபரிடம் கேட்டு தெரிந்துக்கொள்ளலாம்.

1. முதன்மை ஆய்வாளரின் பெயர் : டாக்டர். ஜினீஷ் ராஜ்
முதுகலை பொது மருத்துவம்
ஸ்ரீ முகாம்பிகா மருத்துவ கல்லூரி
மருத்துவமனை
குலசேகரம்
அலைபேசி எண்: 9585953436
மின்னஞ்சல் – drjineeshraj@gmail.com

2. வழிகாட்டியின் பெயர் : டாக்டர் கிருஷ்ணன் குட்டி
பேராசிரியர்
மருத்துவத்துறை
ஸ்ரீ முகாம்பிகா மருத்துவ கல்லூரி
மருத்துவமனை
குலசேகரம்
அலைபேசி எண்: 9447043863
மின்னஞ்சல் – vkumardr@yahoo.co.in

3. துணை வழிகாட்டியின் பொயர் : டாக்டர். மூகாம்பிகா
இணை பேராசிரியர்
மருத்துவத்துறை
ஸ்ரீ முகாம்பிகா மருத்துவ கல்லூரி
மருத்துவமனை
குலசேகரம்
அலைபேசி எண்: 9443380740
மின்னஞ்சல் –

4. கல்லூரியின் பெயர் : ஸ்ரீ முகாம்பிகா மருத்துவ கல்லூரி
மருத்துவமனை குலசேகரம்
கன்னியாகுமரி மாவட்டம் – 629161
தமிழ்நாடு.

5. ஆய்வின் தலைப்பு :

6. பின்னணி தகவல்கள்:

இந்தியா போன்ற வளரும் நாடுகளில் இறத்த சோகையின் அளவு அதிகமாகி வருகிறது. இறந்த வறுமை, சரியான உணவின்மை, சில நோய்கள், கர்பம், தாய்ப்பால் கொடுத்தல் மற்றும் சரியான மருத்துவ வசதிகள் இல்லாமல் இருத்தல், உணவு குறைப்பாட்டால் வரும் இரத்தசோகையே மிக முக்கியமான காரணமாக அமைகிறது. இரும்பு சத்து, B_{12} உயிர்சத்து, ∴போலேட் முதலிய குறைபாடுகளே முக்கியமானவை ஆகும். இற்த ஆய்வானது இரத்தசோகை ஏற்படும் விகிதத்தையும் அதன் காரணத்தையும் அறியவே பயன்படுகிறது.

7. ஆய்வின் குறிக்கோள்:

1.

2.

8. ஆய்வின் அறிவியல் விளக்கம்:

இந்தியாவில் இரத்த சோகையின் விகிதம் மிக அதிகம் ஏனெனில்,

- உணவு குறைவாக எடுத்துக்கொள்ளுதல், இரும்புசத்து மற்றும் ∴போலிக் அமிலச்சத்து குறைபாடு (70 mrg – க்கும் குறைவாக)
- குறைந்த பையோ அவைலபலிட்டி – இரும்பு நார்சத்து
- மலேரியா மற்றுத் கொக்கிபுழு தொற்று காரணமாக ஏற்ப்படும் இரத்த இழப்பு

9. ஆய்வின் செய்முறை

10. பங்கு பெறுவோர்களின் எதிர்பார்க்கர்டும் ஆபத்து : ஏந்த ஆபத்தும் இல்லை

11. பங்கு பெறுவோர்களின் நன்மைகள் : நோயை சீக்கிரம் கண்டறியலாம். நோயை சீக்கிரம்

குணப்படுத்தும் வழிமுறைகளை
கண்டறியலாம்.

12. ஆய்வின் இரகசியத்தன்மை : ஆய்வில் சேகரிக்கப்படும் தகவல்கள் அனைத்தும்

இரகசியமாக பாதுகாக்கப்படும்.

13. நான் ஏன் இந்த ஆய்விற்கு தேர்தெடுக்கப்பட்டேன் :

தேர்ந்தெடுப்பதற்க்கான தகுதிகளை நீங்கள் பூர்த்தி செய்கிறார்கள்.

14. ஆய்வில் பெறுவோர்களின் எண் : 154

15. பெறுவோர்களுக்கு வழங்கப்படும் ஒப்பந்தம் : இல்லை

16. பெறுவோர்களுக்கு ஏதேனும் தொகை வழங்கப்படுமா : இல்லை

17. ஆய்வின் போது நான எந்தநேரமும் விலகிக்கொள்ளலாமா : ஆம்

18. ஏதேனும் புது தகவல்கள் கிடைத்தால் தெரிவிக்கப்படுமா? : ஆம்

19. ஆய்வு நடத்தப்படும் நேரம் : 12–18 மாதங்கள்

20. ஏதேனும் தகவல்கள் : இல்லை

21. மேலும் தகவல்களுக்கு யாரை அணுக வேண்டும் : டாக்டர். ஜினீஷ் ராஜ்

முதுகலை பொது மருத்துவம்

ஸ்ரீ முகாம்பிகா மருத்துவ கல்லூரி

மருத்துவமணை. குலசேகரம்

அலைபேசி எண்: 9585953436

மின்னஞ்சல்–

drjineeshraj@gmail.com

இடம்:

நாள்:

பங்கேற்பாளரின் கையொப்பம் ஆய்வாளரின் கையொப்பம்

ஒப்புதல் படிவம்

பகுதி – 2

பங்கேற்பாளர் ஒப்புதல் படிவம்

ஆய்வு குறித்த தகவல்கள் எழுத்தின் மூலம் எனக்கு தெரிவிக்கப்பட்டது. ஆய்வின் முடிவுகள் நேரடியாக எனக்கு எந்த நன்மையும் தரப்போவதில்லை என்பதை அறிவேன். ஆய்வு குறித்து முழுவதும் அறிந்துகொண்டேன். ஆய்வின் எந்நேரத்திலும் நான் விலகிக் கொள்ளலாம் என்பதை அறிவேன். ஆறிவியல் காரணங்களுக்காகவே இந்த ஆய்வு நடத்தப்படுகிறது என்பதையும் அறிவேன். "ஸ்ரீ மூகாம்பிகா மருத்துவ கல்லூரி மருத்துவமனை நோயாளிகளில் இரத்த சம்பந்தமான நோய்களின் விகிதம்.

வரிசை எண் :

பங்கேற்பாளரின் பெயர் :

பங்கேற்பாளரின் முகவரி :

சாட்சி 1 :

பங்கேற்பாளரின் கையொப்பம்

நாள் :

இடம் :

PROFORMA

AGE : **SEX:**

OCCUPATION: **PLACE:**

DIET HABBITS:

HISTORY OF BLOOD TRANSFUSIONS:

REASON FOR TRANSFUSION:

HISTORY OF EXCESSIVE BLEEDING:

H/O ALCHOHOL INTAKE OR LIVER DISEASE:

DRUG HISTORY:

HB:

PCV:

TC:

PLATELET COUNT:

MCV:

MCH:

MCHC:

RDW:

PERIPHERAL SMEAR:

RETICULOCYTE COUNT:

IF MCV LOW (a) S IRON

(b) FERRITIN

(c) TIBC

(d) HB ELECTROPHORESIS(if needed)

IF MCV HIGH (a) VIT B12

(b)FOLIC ACID

(c)LFT(if needed)

IF RETICULOCYTE COUNT HIGH (a) LDH

(b)COOMBS TEST

(c)G6PD(if needed)

IF RETICULOCYTE COUNT LOW (a)S CREATININE

(b)TSH

(c)ESR,CRP

BONE MARROW ASPIRATION &BIOPSY(if needed)

KEY TO MASTER CHART

1) Age

2) Sex: Male (1)
Female(2)

3) Occupation: Unskilled(1)
Skilled(2)
Semi-professional(3)
Professional(4)
Unemployed(5)

4) Income/monthly

5) Dietary Habit: VEGETARIAN (1)
NON VEGETARIAN(2)

6) History of blood transfusion: HAEMORRHAGE(1)
THALASSEMIA(2)
SEVERE ANEMIA(3)
NONE(4)

7) History of excessive bleeding: MENORRHAGIA(1)
UPPER GI BLEED(2)
LOWER GI BLEED(3)
OTHER BLEEDING MANIFESTATION(4)
NO BLEED(5)

8) History of alcohol intake: YES(1)
NO(2)

9) Chronic illnesses: YES(1)
NO(2)

10) Drug history: YES(1)
NO(2)

11) Hb: HAEMOGLOBIN

12) TC: TOTAL COUNT

13) PLATELET COUNT

14) MCV: MEAN CORPUSCULAR VOLUME

15) MCH: MEAN CELL HEMOGLOBIN

16) MCHC: MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION

17) RDW: RED CELL DISTRIBUTION WIDTH

18) PERIPHERAL SMEAR:

NOT DONE(0)

MICROCYTIC HYPOCHROMIC(1)
MACROCYTIC HYPOCHROMIC(2)
NORMOCYTIC NORMOCHROMIC(3)
DIMORPHIC (4)
LEUKEMIC AND OTHERS(5)

19) RETICULOCYTE COUNT

20) RED BLOOD CELL COUNT

21) ESR:ERYTHROCYTE SEDIMENTATION RATE

22) CRP: C-REACTIVE PROTEIN

23) PERIPHERAL SMEAR

24) SERUM IRON

25) SERUM FERRITIN

26) TIBC: TOTAL IRON BINDING CAPACITY

27) STOOL: NOT DONE(0)
NORMAL(1)
OCCULT BLOOD(2)
CYST,OVA(3)

28) VIT B12: VITAMIN B12

29) FOLIC ACID

30) RFT: RENAL FUNCTION TEST: NOT DONE(0)
NORMAL(1)
IMPAIRED(2)

31) LFT: LIVER FUNCTION TEST: NOT DONE(0)
NORMAL(1)
IMPAIRED(2)

32) TSH: THYROID STIMULATING HORMONE

33) LDH:LACTATE DEHYDROGENASE

34) COOMB'S TEST

35) G6PD: GLUCOSE-6-PHOSPHATE DEHYDROGENASE

NOTDONE(0)