A RETROSPECTIVE STUDY ON PRESCRIBING PATTERNS OF HEMATINICS AND BLOOD TRANSFUSION THERAPY IN A TERTIARY CARE HOSPITAL

A Dissertation submitted to
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CHENNAI – 600 032.

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

MASTER OF PHARMACY
IN
PHARMACY PRACTICE

Submitted By
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KOMARAPALAYAM – 638 183

OCTOBER– 2018
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<td>--------------</td>
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<td></td>
</tr>
<tr>
<td>WHO</td>
<td>world health organization</td>
<td></td>
</tr>
<tr>
<td>Hgb /Hb</td>
<td>Hemoglobin</td>
<td></td>
</tr>
<tr>
<td>RBCs</td>
<td>Red Blood Cells</td>
<td></td>
</tr>
<tr>
<td>Hct</td>
<td>Hematocrit</td>
<td></td>
</tr>
<tr>
<td>G6PD</td>
<td>Glucose -6- Phosphate Dehydrogenase</td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Gastro Intestinal</td>
<td></td>
</tr>
<tr>
<td>HR</td>
<td>Heart Rate</td>
<td></td>
</tr>
<tr>
<td>SV</td>
<td>Stroke Volume</td>
<td></td>
</tr>
<tr>
<td>HF</td>
<td>Heart Failure</td>
<td></td>
</tr>
<tr>
<td>MCV</td>
<td>Mean Cells Volume</td>
<td></td>
</tr>
<tr>
<td>MCH</td>
<td>Mean cells Hemoglobin</td>
<td></td>
</tr>
<tr>
<td>MCHC</td>
<td>Mean cells hemoglobin Concentration</td>
<td></td>
</tr>
<tr>
<td>FL</td>
<td>Femtolitre</td>
<td></td>
</tr>
<tr>
<td>Pg</td>
<td>Picogram</td>
<td></td>
</tr>
<tr>
<td>TIBC</td>
<td>Total Iron Binding Capacity</td>
<td></td>
</tr>
<tr>
<td>RDW</td>
<td>Red blood cells Distribution Width</td>
<td></td>
</tr>
<tr>
<td>ctHb</td>
<td>Concentration of Hemoglobin</td>
<td></td>
</tr>
<tr>
<td>ICSH</td>
<td>International Committee Standardization in Hematology</td>
<td></td>
</tr>
<tr>
<td>HICN</td>
<td>Hemoglobincyanide</td>
<td></td>
</tr>
<tr>
<td>Nm</td>
<td>Nanometer</td>
<td></td>
</tr>
<tr>
<td>Abbreviation</td>
<td>Definition</td>
<td></td>
</tr>
<tr>
<td>--------------</td>
<td>------------</td>
<td></td>
</tr>
<tr>
<td>Mg</td>
<td>milligram</td>
<td></td>
</tr>
<tr>
<td>Kg</td>
<td>kilogram</td>
<td></td>
</tr>
<tr>
<td>US</td>
<td>United States</td>
<td></td>
</tr>
<tr>
<td>IV</td>
<td>Intravenous</td>
<td></td>
</tr>
<tr>
<td>IDA</td>
<td>Iron Deficiency Anemia</td>
<td></td>
</tr>
<tr>
<td>ID</td>
<td>Iron Deficiency</td>
<td></td>
</tr>
<tr>
<td>CBC</td>
<td>Complete Blood Count</td>
<td></td>
</tr>
<tr>
<td>FCM</td>
<td>Ferric Carboxy Maltose</td>
<td></td>
</tr>
</tbody>
</table>
INTRODUCTION

I. ANAEMIA

Anaemia is not one disease but a condition that result from number of different pathogenesis. It can be defined as a reduction from normal haemoglobin quantity in the blood.

According to WHO Anaemia is a condition it which number of red blood cells or their oxygen – carrying is insufficient to meet physiologic needs, which vary by age, sex, attitude, smoking and pregnancy status.

Iron deficiency is thought to be the most common cause of anaemia globally, however other conditions, such as folate, vitamin B₁₂ and Vitamin A deficiencies, chronic inflammation parasitic infections and inherited disorders can all cause anaemia.

II. HAEMGLOBIN

Haemoglobin is an iron rich protein inside red blood cells that carries oxygen from lungs to tissues and organs in the body and carries carbon dioxide back going and gives blood litres red colour.

Therefore, total Haemoglobin concentration primary depends on the number of RBCs in the blood sample. Medical conditions that impact the number of RBCs will also affect Haemoglobin concentration.

Reference Range:

Males – 14-18 g/dL (or) 140-180 g/dL

Females – 12-16 g/dL (or) 120-160 g/L
Red Blood cell indices

RBC indices (also known as wintrobe indices) are useful in the classification of anaemia. These indices include,

(a) The MCV

(b) The mean cell haemoglobin (MCH)

(c) And the MCHC which calculated as following equations:

a) \[ \text{MCV} = \frac{Hct \times 1000}{RBC \text{ (in millions/µL)}} = 76-100 \text{ (in µm}^3) \]

b) \[ \text{MCH} = \frac{Hgb \text{ (in g/dL} \times 10)}{RBC \text{ (in millions/µL)}} = 27-33 \text{ (in pg)} \]

c) \[ \text{MCHC} = \frac{Hgb \text{ (in g/dL)}}{Hct} = 33-37 \text{ (in g/dL)} \]

A) Mean Cell Volume

The MCV detects changes in cell size. A decreased MCV indicates a microcytic cell, which can result from iron-deficiency anaemia.

A large MCV indicates macrocytic cell which can be caused by vitamin B12 or folic acid deficiency.

B) Mean cell haemoglobin

The MCHC is more reliable index of RBC Hgb than MCH. MCHC measures the concentration of Hbg where as MCH measures the weight of Hgb in the average RBC.

In normochromic anemias changes in size of RBCs (MCV) are associated with corresponding changes in weight of Hgb (MCH), but the concentration of haemoglobin (MCHC) remains normal.
### TABLE – 1

#### CLASSIFICATIONS OF ANEMIA

<table>
<thead>
<tr>
<th>I. PATHOPHYSIOLOGIC (Classifies anemias based on pathophysiologic presentation)</th>
</tr>
</thead>
</table>
| 1. Blood Loss:  
   Acute: Trauma, ulcer, haemorrhoids  
   Chronic: Ulcer, vaginal bleeding, aspirin indigestion. |
| 2. Inadequate RBCs production:  
   Nutritional deficiency: Vitamin B12, folic acid, iron  
   Erythroblast deficiency: Bone marrow failure, Caplastic anemia, irradiation, chemotherapy, folic acid antagonists or bone marrow infiltration, leukemia, lymphoma, myeloma, metastatic solid tumors, mylofibrosis |
| 3. Endocrine deficiency:  
   Pituitary, adrenal, thyroid, testicular  
   Chronic disease: Renal, liver, infection, granulomatous, collagen vascular. |
| 4. Excessive RBC destruction:  
   Intrinsic factors: Heredity (G6PD), abnormal haemoglobin synthesis.  
   Extrinsic factors: auto immune reactions, drug reactions, infection (endotoxins) |

<table>
<thead>
<tr>
<th>II. MORPHOLOGIC (classifies anemia by Pad blood cell size) and haemoglobin content</th>
</tr>
</thead>
</table>
| 1. Macrocytic: Detective maturation with decreased production.  
  Megaloblastic: Pernicious (vitamin B12 deficiency), folic acid deficiency. |
| 2. Normochromic, normocytic  
  Recent blood loss  
  Hemolysis  
  Chronic disease  
  Renal failure  
  Autoimmune  
  Endocrine |
| 3. Microcytic, hyperchromic  
  Iron deficiency  
  Genetic abnormalities: Sickell cell, thalassemia |
Chapter 1 Introduction

IV. EPIDEMIOLOGY

Anaemia is one of the most common condition in the world and results in significant morbidity and mortality in the developing world.\textsuperscript{[1]}

In 2011, 29% (496 million) of non-pregnant women and 38% (32 million) of pregnant women aged 15-49 years where anaemic.\textsuperscript{[5]}

Anaemia is major health problem for adult by affecting 55% of women and 24% of men. The prevalence of Anaemia for ever married women in age group of 15-49 has increased from 52% in NFHS-2 to 56% in NFHS -3 and this anaemia confines to be a serious problem in India\textsuperscript{[6]}.

V. AETIOLOGY

The low haemoglobin level that defines anaemia results from two different mechanisms.

- **Increased haemoglobin loss do to either**
  
  Haemorrhage (Red cell loss) or

  Haemolysis (Red cell destruction)

- **Reduced haemoglobin synthesis due to**

  Lack of nutrient (or)

  Bone marrow failure

  Each of these mechanisms includes a number of disorder that required specific and appropriate therapy
A. GENTIC ETIOLOGY INCLUDE THE FOLLOWING

- Haemoglobin pathies
- Thalassemia
- Enzyme abnormalities of the glycolytic pathways
- Detects of the RBC cytoskeleton
- Congenital dyserythropoietic anaemia
- Rh null disease
- Heredity xerocytosis
- Abetalipoproteinemia
- Fanconi anemia

B. NUTRITIONAL ETIOLOGIES INCLUDE THE FOLLOWING

- Iron deficiency
- Folate deficiency
- Vitamin B₁₂ deficiency
- Starvation and generalized malnutrition

C. PHYSICAL ETIOLOGIES INCLUDE THE FOLLOWING

- Trauma
- Burns
- Frostbite
- Prosthetic value and surfaces
D. CHRONIC DISEASE AND MALIGNANT ETIOLOGYIES INCLUDE THE FOLLOWING

- Renal Disease
- Hepatic Disease
- Chronic infections
- Neoplasia
- Collagen vascular diseases

E. INFECTIOUS ETIOLOGYIES INCLUDE THE FOLLOWING

- Viral- hepatitis, infectious mononucleosis, cytomegalovirus
- Bacterial- clostridia, gram negative sepsis
- Protozoal- malaria, leishmaniasis, toxoplasmosis.

Thrombotic thrombocytopenic purpura (TTP) and haemolytic –aremic syndrome may be cause anaemia. Heredity spherocytosis either may present as a server hemohytic anaemia (or) a symptomatic with hemolysis also deficiency in G6PD may manifest as chronic haemolytic anaemia (or) exist without anaemia until an oxidant medication given to the patient.

In emergency department, acute hemorrhage is by far the most common etiology for anaemia. Drugs (or) chemical commonly cause the aplastic and hypoplastic group of disorders which certain are dose related while others are idiosyncratic.
Such as: Being exposed to a sufficient dose of

- Inorganic arsenic
- Bezene
- Racliation
- Or chemotherapeutic agents for treatment of neoplastic diseases which develops bone marrow depression.

VI. SIGNS AND SYMPTOMS

Signs and symptoms of anemia vary with the degree of RBC reduction and with the time interval during which it develops. In tissue hypoxia caused by the decreased oxygen carrying capacity of reduced RBC mass. Which result in perfusion of tissues. Such as

- Skin
- Mucous membrane
- Extremities

Slowly developing anaemia can be a symptomatic initially or include symptoms

Such as:

- Slight exertional dyspnea
- Increased anagina
- Fatigue
- Or malaise
Uncorrected Hypoxia can lead to a number of complications in

Cognition, quality of life, and respiratory and GI systems. In severe anaemia (hgb<8g/dL) stroke volume and heart rate increased in order to attempt oxygen delivery improvement to tissue. These changes in HR and SV can result in systole murmurs, argina pectorios, high output, HF, pulmonary congestion, ascites and edema.

However, anaemia is generally not tolerated in patients with

- Cariac disease
- Skin and mucous membrane pallor
- Jaundice
- Smooth (or) beety tongue
- Cheilosis
- And spoon shaped nail (koilonychias)

Also may be associated with severe anaemia of different etiologies thus, acute onset anemia is characterized by cardiorespiratory symptoms such as

- Tachy cardia
- Lightheadedness
- And breathlessness

While chronic anaemia is characterized by

- Weakness
- Fatigue
• Headache
• Vertigo
• Faintness
• Cold sensitivity
• Pallor
• Loss of skin tone due to hypoxia

In case of iron deficiency anemia is characterized by

• Glossal pain
• Smooth tongue
• Reduced salivary flow
• Pica (compulsive eating of non-food items)
• And pagophagia (compulsive eating of ice)

These symptoms are not usually seen until Hgb concentration is less than 9g/dL. While vitamin B₁₂ and folate deficiency anaemia are characterized by

• Pallor
• Incerus
• And gastric mucosal atrophy

Vitamin B₁₂ anemia is distinguished by neuropsychiatric abnormalities (e.g., numbness, paresthesias, irritability) which are absent in folate deficiency anemia patients.
RISK FACTORS

There are a number of conditions that can increase your risk of anaemia. Some examples are:

- Low diet in iron, vitamins, and minerals.
- Chronic diseases such as kidney disease, gastritis, colitis, rheumatoid arthritis, and related diseases, thyroid disease, and heart failure.
- Blood loss during surgery or injury, excessive blood draws, heavy menstrual cycle.
- Family history of inherited anaemia (e.g., sickle cell or thalassemia).
- Inability to absorb iron caused by intestinal disorders such as Crohn's disease, celiac disease, or gastric bypass surgery.
- Adult 65 years of age and older.
- Women of childbearing age.
- Infant under 2 years old.

VII. DIAGNOSIS

Initial evaluation of anaemia involves a complete blood cell count, reticulocyte index, and examination of stool for occult blood.
TABLE-2 NORMAL HEMATOLOGIC VALUES

<table>
<thead>
<tr>
<th>TEST</th>
<th>REFERENCE RANGE (YEARS)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hgb (g/dL)</td>
<td>18 – 48</td>
</tr>
<tr>
<td></td>
<td>M:13.5 -17.5</td>
</tr>
<tr>
<td></td>
<td>F:120 – 16.0</td>
</tr>
<tr>
<td>HCT (%)</td>
<td>M: 41-53</td>
</tr>
<tr>
<td></td>
<td>F:36-46</td>
</tr>
<tr>
<td>MCV (fl)</td>
<td>80-100</td>
</tr>
<tr>
<td>MCH(%)</td>
<td>31-37</td>
</tr>
<tr>
<td>MCH(pg)</td>
<td>26-34</td>
</tr>
<tr>
<td>RBC (Million/mm³)</td>
<td>4.5-5.9</td>
</tr>
<tr>
<td>Reticulocyte count, absolute (%)</td>
<td>0.5-1.5</td>
</tr>
<tr>
<td>Serum iron (mcg/dL)</td>
<td>M:56-160</td>
</tr>
<tr>
<td></td>
<td>F:40-150</td>
</tr>
<tr>
<td>TIBC (mcg/dL)</td>
<td>250-400</td>
</tr>
<tr>
<td>RDW(%)</td>
<td>11-16</td>
</tr>
<tr>
<td>Ferritin (ng/mL)</td>
<td>M:15-200</td>
</tr>
<tr>
<td></td>
<td>F:12-150</td>
</tr>
<tr>
<td>Folate (ng/mL)</td>
<td>1.8-16.0</td>
</tr>
<tr>
<td>Vitamin B12 (pg/mL)</td>
<td>100-900</td>
</tr>
<tr>
<td>Erythropoietin (milliunits/mL)</td>
<td>0-19</td>
</tr>
</tbody>
</table>

VARIES BY ASSAY METHOD

- Macro anemia are characterized by increased in MCV (110 – 140 fl )

- Vitamin B₁₂ and folate concentrations can be measured to differentiate between the two deficiencies anemias.
➤ A vitamin B12 value of less than 150pg/ml together with appropriate peripheral smear and clinical symptoms, is a diagnostic vitamin B12 deficiency anemia

➤ A decreased RBC folate concentration (less than 150ng/ml) appears to be a better indicator of folate deficiency anemia than a decreased serum folate concentration (less than 3ng/ml)[8]

➤ Iron deficiency is the most common cause of microcytic anemia, up to 40 percent of patients with iron deficiency anemia will have normocytic erythrocytes (10). As such, iron deficiency should still be considered in all cases of anemia unless the MCV is greater than 95fl. Because this cut off has a sensitivity of 97 percent [11]

➤ The most sensitive laboratory for iron deficiency anemia is decreased serum ferritin (storage iron), which should be interpreted in conjunction with decreased transferring saturation and increased TIBC. White Hgb, HCT and RBC indices remain normal until later stages of iron-deficiency anemia.

➤ Hemolytic anemias tend to be normocytic and normochromic and to have increased levels of reticulocytes, lactic dehydrogenase and indirect bilirubin.

➤ Elderly patients with symptoms of anemia should undergo a complete blood cell count will peripheral smear and reticulocyte count, and other laboratory studies as needed to determine the etiology of anemia.[8].
Table 3: Haemoglobin levels to diagnose anemia (g/L)

<table>
<thead>
<tr>
<th>Population</th>
<th>Non anemia</th>
<th>Anemia</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Mild</td>
</tr>
<tr>
<td>Non-pregnant women (15 years</td>
<td>120 (or)</td>
<td>110-119</td>
</tr>
<tr>
<td>of age and above)</td>
<td>higher</td>
<td></td>
</tr>
<tr>
<td>Pregnant women</td>
<td>110 or</td>
<td>100-109</td>
</tr>
<tr>
<td></td>
<td>higher</td>
<td></td>
</tr>
<tr>
<td>Men (15 years of age and</td>
<td>130 or</td>
<td>110-129</td>
</tr>
<tr>
<td>above)</td>
<td>higher</td>
<td></td>
</tr>
</tbody>
</table>

MEASUREMENTS OF ctHb

1. Historical Perspective

The first test of Hb measurement devised more than a century ago\(^{[13]}\) involved adding drops of distilled water to a measured volume of blood units until it colour matched that of an artificial colour standard.

Alter modification\(^{[14]}\) involved first saturating blood with coal gas (carbon monoxide) to convert hemoglobin to the more stable carboxyhemoglobin. Modern hemoglobinometry dates from the 1950, following development of spectrophotometry and the hemiglobincyanide (cynamethemoglobin) method.

Adaptation of this method and others for use in automated hemotology analyzers followed. Over the past two decades advances have focused on development of methods which allow point-of-care testing (POCT) of haemoglobin.

This section deals with consideration of some of the methods currently used in the laboratory and then with those POCT methods done outside the laboratory.

Hemiglobincyanide – A spectrophotometric method
Nearly 40 years after it was first adopted as the reference method for measuring haemoglobin by ICSH \cite{15}, The HICN test remains the recommended method of the ICSH \cite{16} against which all new ctHb methods are judged and standardized.

i) Test Principle

Blood will be diluted with a solution containing potassium ferricyanide and potassium cyanide. In which potassium ferricyanide oxidizes iron in heme to the ferric state to form methemoglobin, which is converted to HICN by potassium cyanide.

HICN is a stable colored product, which in a solution has an absorbance maximum at 450 nm and strictly obeys bear-lambert’s law.

The diluted sample absorbance at 540 nm is compared with absorbance at the same wave length of a standard HICN solution whose equivalent haemoglobin concentration is known.

Most haemoglobin derivatives such as oxyhemoglobin, methemoglobin and carboxyhemoglobin but not sulfhemoglobin are converted to HICN and therefore measured by this method.

a) Reagent diluents (modified Drab kin Solution)\cite{17}

- Potassium ferricyanide (K3Fe(CN)6) 200mg
- Potassium cyanide (KCN) 50mg
- Dihydrogen potassium phosphate (KH2PO4) 140mg
- Non-ionic detergen (e.g. Tritonx-100) 1 ml
- Above diluted to 1000ml in distilled water.
b) Manual method

24 HL of blood is added to 5.0 ml reagent, mixed and left for 3 minutes. Absorbance is read at 540 nm against a reagent blank. The absorbance of HICN standard is measured in the same way.

(c) ICSH HICN standard

The standard HICN solution manufactured and assigned a concentration value according to very precise criteria laid down and reviewed periodically by ICSH. [16]

(d) Advantages of HICN

- International standard – accurate.
- Easily adapted by automated hematology analyzer.
- Well established and thoroughly investigated ICSH recommended.
- Inexpensive reagent

d) HICH Disadvantages

- Manual process requires accuracy in popetting and spectrophotometer.
- Reagent hazardous (Cyanide)
- Subject to interference from raised lipid, plasma proteins and leucocytes numbers.

PATHOPHYSIOLOGY

Anemias can be classified on the basis of RBC morphology, etiology or pathophysiology (Table 1)
Morphologic classifications are based on cell size. Macrocytic cells are larger than normal and associated with deficiencies of vitamin B₁₂ or folate. While microcytic cells are smaller whereas normocytic anaemia may be associated with recent blood loss (or) chronic disease.

Iron-deficiency anemia can be caused by inadequate dietary intake, inadequate GI absorption increased iron demand (e.g. pregnancy), blood loss and chronic disease.

Vitamin B₁₂ and folate – deficiency anemias can be caused by inadequate dietary intake, decreased absorption, and inadequate utilization, also intrinsic factor can cause decreased absorption of vitamin B₁₂ (i.e., pernicious anemia).

Folate deficiency anemia can be caused by hyper utilization due to pregnancy, haemolytic anemia, myelofibrosis, malignancy, chronic inflammatory disorders, long-term dialysis or growth spurt.

Drugs can cause anemia by reducing absorption of folate such as phenytoin or by interfering with corresponding metabolic pathways such as methotrexate.

Anemia of chronic disease is associated with chronic infectious or inflammatory processes, tissue injury or conditions that release pro inflammatory cytokines.

This pathogenesis is based on:

- Shortened RBC survival.
- Impaired marrow response.
- And disturbance of iron metabolism
Age related reduction in bone marrow reserve can render the elderly patient more susceptible to anemia that’s caused by multiple minor and unrecognized disease (e.g. nutritional deficiencies) which negatively effect erythropoiesis.

Hemolytic anemia results from decreased RBC survival time due to destruction in the spleen or circulation.

Most common etiologies are:

- RBC defects (e.g. heredity spherocytosis)
- Alter Hgb solubility or stability (sickle cell anemia)

Some drugs cause direct oxidative damage to RBCs such as phenytoin and Phenobarbital. [8]

**Erythropoiesis**

Erythropoietin is a hormone produced by the cells of the renal cortex. The kidney respond to hypoxia and anemia by increasing the production of erythropoietin. The red cells progenitors BFU-E and CFU-E have receptors on their faces.

When erythropoietin binds to these receptors, it promotes differentiation and division and consequently increased erythropoiesis. [1]
Figure 1: Simplified diagram of some of the stages within erythropoiesis.

TREATMENT:

A) IRON-DEFICIENCY ANEMIA:

Oral iron therapy with soluble ferrous iron salts which are not enteric coated and are not slow or sustained release, daily recommended at a daily dosage of 200 mg in two or three divided doses.

Diet plays significant role because iron is poorly absorbed from, the vegetables, grain products, dairy products and eggs; iron is best absorbed from: meat, fish and poultry. whereas the typical daily requirements of iron are:

<table>
<thead>
<tr>
<th>Table No-4 : Daily Iron requirements[^1]</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Category</strong></td>
</tr>
<tr>
<td>Infant (0-4 months)</td>
</tr>
<tr>
<td>Adolescent male</td>
</tr>
<tr>
<td>Adolescent female</td>
</tr>
<tr>
<td>Adult male</td>
</tr>
<tr>
<td>Menstruating female</td>
</tr>
<tr>
<td>In pregnancy</td>
</tr>
<tr>
<td>Postmenopausal female</td>
</tr>
</tbody>
</table>
Administration of iron with a meal decreases absorption by more than 50 percent but may be needed to improve tolerability.

**Table No. 4: Oral iron products:**

<table>
<thead>
<tr>
<th>Salt</th>
<th>Elemental iron (%)</th>
<th>Elemental iron provided</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ferrous sulfate</td>
<td>20</td>
<td>60-65 mg/324-325 mg tablet</td>
</tr>
<tr>
<td></td>
<td></td>
<td>18 mg iron/5 ml syrup</td>
</tr>
<tr>
<td></td>
<td></td>
<td>44 mg iron/5 ml syrup</td>
</tr>
<tr>
<td></td>
<td></td>
<td>15 mg iron/0.6 ml drop</td>
</tr>
<tr>
<td>Ferrous sulfate (exsiccated)</td>
<td>30</td>
<td>65 mg/200 mg tablet</td>
</tr>
<tr>
<td></td>
<td></td>
<td>60 mg/187 mg tablet</td>
</tr>
<tr>
<td></td>
<td></td>
<td>50 mg/160 mg tablet</td>
</tr>
<tr>
<td>Ferrous gluconate</td>
<td>12</td>
<td>36 mg/325 mg tablet</td>
</tr>
<tr>
<td></td>
<td></td>
<td>27 mg/240 mg tablet</td>
</tr>
<tr>
<td>Ferrous fumarate</td>
<td>33</td>
<td>33 mg/100 mg tablet</td>
</tr>
<tr>
<td></td>
<td></td>
<td>63-66 mg/200 mg tablet</td>
</tr>
<tr>
<td></td>
<td></td>
<td>106 mg/324-325 mg tablet</td>
</tr>
<tr>
<td></td>
<td></td>
<td>15 mg/0.6 ml drop</td>
</tr>
<tr>
<td></td>
<td></td>
<td>33 mg/5 ml suspension</td>
</tr>
<tr>
<td>Polysaccharide iron complex</td>
<td>100</td>
<td>150 mg capsule</td>
</tr>
<tr>
<td></td>
<td></td>
<td>50 mg tablet</td>
</tr>
<tr>
<td></td>
<td></td>
<td>100 mg/5 ml elixir</td>
</tr>
<tr>
<td>Carbonyl iron</td>
<td>100</td>
<td>50 mg caplet</td>
</tr>
</tbody>
</table>

Parentral iron maybe required for patients with: iron malabsorption, intolerance of iron therapy and incompialnce . furthermore the replacement dose depends on etiology of anemia and hemoglobin concentration.
Available Parenteral iron preparations have similar efficacy but different pharmacologic, pharmacokinetics and safety profiles. The new products are, sodium ferric gluconate and iron sucrose which appeared to be well tolerated when compared to iron dextran.[8]

**B) Folate- Deficiency Anemia :**

Folate-deficiency anemia usually managed by replacement therapy. The duration of treatment depends on the cause of the deficiency. Changes in dietary habits or removal of any precipitating factor can be considered.

The normal daily Requirement of folic acid is approximately 100µcg a day; although the usual doses are given are 5-15 mg a day, and because of these large doses sufficient folate is absorbed. Therefore Parenteral folic acid treatment is not normally required, Treatment for 4 months will normally be sufficient to ensure the folate deficient red cells are replaced.[1]

Oral folate 1 mg daily for 4 months should be sufficient to overcome folate-deficiency anemia, if malabsorption is present, the daily dose should be elevated and increased to 5mg.[8]

**C) Vitamin B₁₂-Deficiency Anemia :**

The majority of patients with vitamin B₁₂ deficiency require lifelong replacement therapy. since the anemia has developed slowly the cardiovascular system does not tolerate blood transfusion very well and is easily overloaded.

The standard treatment is **hydroxocobalamin** 1 mg intramuscularly three times a week for two weeks then 1 mg every 3 months. where there is neurological
involvement, slightly higher regimen is recommended, 1 mg every 2 months. Its retain in the body longer than *cyanocobalamin* and reaction to it is very rare. US texts recommend *cyanocobalamin* rather than hydroxocobalamin because the fear that some patients appear to develop antibodies to the vitamin B\textsubscript{12} transport protein complex in the serum.

A small amount of passive absorption of vitamin B\textsubscript{12} is occurred from the gastro-intestinal tract. High (1 mg) daily oral and sublingual doses of *cyanocobalamin* are absorbed in sufficient quantities to manage the pernicious anemia.

The Red cells return to normal and the platelets count rises to normal (or even higher) after 7-10 days. The hemoglobin takes much longer to returns to normal, it should rise approximately 2-3 g/dL each fortnight. Neurological damage maybe irreversible. \[1\]

**C) Anemia Of Chronic Disease:**

Treatment of anemia of chronic disease is less specific than that of other anemias and should focus on correcting reversible causes. Iron therapy is not effective when inflammation is present. RBC TRANSFUSIONS are effective but should be limited to episodes of inadequate oxygen transport and Hgb of 8 to 10 g/dL.

Epoetin alfa can be considered, especially if cardiovascular status is compromised, but the response can be impaired in patients with anemia of chronic disease (off-label use). The initial dosage is 50-100 units/kg three times weekly.
Hgb does not increase after 6-8 weeks, the dosage can be increased to 150 units/kg three times weekly. Epoetin alfa is usually well tolerated.

D) Other Types Of Anemias:

Patients with other types of anemias require appropriate supplementation depending on the etiology of anemia. In patients with anemia of critical illness, parenteral iron is often utilized but is associated with a theoretical risk of infection. Routine use of epotein alfa or RBC transfusions is not supported by clinical studies.

Anemia of prematurity is usually treated with RBC transfusions, the use of epotein alfa is controversial. In the pediatric population, the daily dose of elemental iron administered as iron sulfate is 3 mg/kg for infants and 6 mg/kg for older children for 4 weeks if response is seen, iron should be continued for 2-3 months to replace storage iron pool.

While treatment of hemolytic anemia should be focusing on correcting the underlying cause. There is no specific therapy for glucose-6-phosphate dehydrogenase deficiency, so treatment consists of avoiding oxidants medications and chemicals. steroids, other immunosuppressants, and even splenectomy can be indicated to reduce RBC destruction.[8]

During acute episodes of haemolysis, the patient should be kept well dehydrated to ensure good urine output to prevent hemoglobin damaging the kidney, blood transfusions maybe necessary. Vitamin E (an antioxidant) appears to have little clinical benefits in preventing haemolysis.

The common drugs implicated in causing haemolysis in G6PD deficiency:
1] Drugs to be avoided in all variants:

- Ciprofloxacin
- Dapsone
- Methylene blue
- Primaquine (reduced dose may be used in milder variants)
- Nalidixic acid
- Sulphonamides (including co-trimoxazole)

2] Drugs to be avoided in more severe variants:

- Aspirin (low dose under supervision)
- Chloramphenicol
- Chloroquine (may be acceptable in acute malaria)
- Menadione
- Probenecid
- Quindine
- Quinine (acceptable in acute malaria)
LITERATURE REVIEW

Quin Y et al., (2013) conducted a study aimed to investigate the relationship of anemia and body mass index (BMI) among adult women in Jiangsu province, China. Data was collected in sub-national cross-sectional survey and 1,537 women aged more than 20 years were included in the analysis. 31.1% of Chinese women were anemic. The prevalence of overweight, obesity and central obesity was 34.2%, 5.8% and 36.2% respectively. The study concluded that women with overweight/obesity or central obesity were less likely to be anemic as compared to normal weight women.

Rakesh PS et al., (2017) conducted a study aimed to identify the prevalence of anemia in Kerala and to comment on its trend across previous 25 years using google scholar searches and PubMed and scanning of reference lists are used to identify studies. All studies based on population on anemia from kerala, irrespective of it design since first of January 1990 to 31st December 2015. The prevalence of anemia was found to be around 30% among adolescent girls from recent study report and prevalence of sever anemia was less than 1% in all studies, anemia among tribal women and children were in range of 78.3% to 96.5%. The study concluded that current prevalence of anemia in Kerala is unclear and these results cannot be combined due to non uniform hemoglobin estimation method.

Kumari R et al., (2017) conducted a study to find out the prevalence of IDA in adolescent girls in a tertiary care hospital. Using cross-sectional study was done in biochemistry laboratory of Indira Gandhi Institute of Medical Sciences, Patna, Bihar, India for a period of six months duration, study result was out of 200 girls were found to be anemic. 50% adolescent girls were found to be anemic, of the
total 43.3% were mildly, 3.3% were moderately and 3.3% were severely affected by anemia. the study concluded that as the prevalence of anemia is 50%, it needs intervention for its prevention and control.

Sherwani RK\(^ {21}\) et al.,(2008) has conducted a study for the prevalence of iron deficiency anemia in chronic diarrhea and celiac disease. it includes 45 children between 1-12 years with chronic diarrhea who underwent investigation hemoglobin with GBP, RBC indices, serum iron and TIBC. 30 cases (66.7%) were male and 15 cases (33.3%) females. If no noted improvement after 2 weeks of treatment they will be subjected to duodenal biopsy, if biopsy showed features of celiac disease then antiendmysial antibody test was performed to substantiate the diagnosis 8 (17.8%) cases had watery diarrhea and 20 (44.4%) semi formed and 17 (37.8%) had watery and semi formed diarrhea. bloody diarrhea was present in 5 (11.1%) and abdominal distension in 21 cases (46.7%). pallor is seen in all the cases. The study conclusion was a better awareness of the profile and early detection of celiac disease would help detection of management of iron deficiency associated with celiac disease.

Mehta BC\(^ {22}\) et al.,(2007) has conducted a study about nutritional anemia (NA) which is common in India while iron deficiency (ID) is a well recognized cause of NA, so the students were given a questionair to elicit anemia related symptoms. blood was collected for CBC test, serum ferritin, folic acid and vitamin B\(_{12}\). Students of polytechnic received hematinics at bed time during their menstrual cycle whereas inmate of students whome received hematinics at bed 3 days a week.

After 6 months blood test were repeated once again in those who completed treatment students were divided into three groups. 1. Control group with Hb 12.0 g/dl or more and ferritin15 ng/ml or more, 2. ID group with Hb 12.0 g/dl or more
Chapter 2 Literature Review

and ferritin less than 15.0 ng/ml and Iron Deficiency Anemia (IDA) group with Hb
less than 12.0 g/dl and ferritin less than 15.0 ng/ml. Result was found that Median
age-16 years (range 10–25). Anemia (Hb < 12.0 g/dl)-94 (34.6%); MCV < 80 fl-153
(56.3%); MCH < 27 pg-167 (61.4%); Ferritin < 15.0 ng/ml-161 (59.2%); Folic acid
< 3.5 ng/ml-34 (12.5%); Vitamin B12 < 258 pg/ml-133 (48.9%) Pre-therapy: (1) Hb, MCV, MCH and ferritin significantly lower in ID and IDA Groups compared to
control group. (2) Hb, MCV, MCH and Ferritin significantly lower in IDA Group as
compared to ID Group.

Nutritional anemia is common amongst asymptomatic young female
students, Deficiencies of iron, folic acid and vitamin B12 are common and coexist,
105 mg elemental iron for 3 days in a week for 6 months is not adequate to correct
IDA. 105 mg iron for 3 days in a week is enough to correct ID, Non-anemic
individuals with ID have iron deficient erythropoiesis, Non-anemic individuals
without ID, in this cohort, also had iron deficient erythropoiesis.

Jayabose S23 et al.,(1993) conducted a study to evaluated the safety and
efficacy of a new transfusion regimen for children with severe anemia, 22
consecutive patients with severe anemia (hemoglobin < 5 g/dl) of gradual onset
requiring transfusion of packed red blood cells (PRBC) were studied. The
transfusion regimen consisted of continuous infusion of PRBC at the rate of 2
cc/kg/h until the desired volume was given. Throughout the transfusion, the patients
were closely monitored for any clinical signs of heart failure.

The result was found to be No patient developed any signs of cardiac failure
or increase in the heart rate during or after the completion of transfusion. All
patients had a decrease in the heart rate by the completion of transfusion. The mean
Chapter 2 Literature Review

The decrease in the heart rate was 28% of the pretransfusion heart rate (range 12-44%), they concluded that children with severe anemia of gradual onset requiring transfusion therapy, continuous transfusion of PRBC at the rate of 2 cc/kg/h is safe and effective regimen resulting in the hematocrit level.

Kaur et al., (2018) has conducted a study which was aimed to evaluate dietary intake, prevalence, and the effect of anemia on various morphophysiological variables among postmenopausal women, its a community-based sample survey. A total of 250 postmenopausal women aged 45-80 years from various parts of North India participated in the study. Anthropometric measurements, hemoglobin concentration, and bone mineral density (BMD) were assessed. The prevalence of anemia was reported to be 85.2% among postmenopausal women. The intake of nutrients such as calcium, iron, and protein and energy was lower among anemic women than nonanemic women. Anemia was not only the result of aging but also unbalance and inadequate dietary intakes.

Mishra et al., (2018) conducted a study to evaluate the safety and efficacy of FCM in treating anemia in patients of menorrhagia. Thus avoiding blood transfusion, It was an open, single arm observational study including 90 women of age more than 30 years with definitive diagnosis of menorrhagia with IDA and hemoglobin (Hb) levels between 4 gm% and 11 gm%. Intravenous ferric carboxymaltose (FCM) (500-1500 mg) was administered, and the improvement in blood indices was assessed. Blood indices measured pre-FCM and 3 weeks post-FCM showed a mean increase in Hb from 8.33±1.10 to 10.89±1.02 with a statistically significant \( P < 0.01 \). There was a statistically significant rise of packed cell volume, serum ferritin, and serum iron in
the post-FCM blood levels after three weeks. It is concluded that the Intravenous FCM is an effective and safe treatment option for IDA with a single administration of high dose without serious adverse effects obviating the need for blood transfusion before surgery.
AIM AND OBJECTIVES

The study is conducted and concerned with monitoring of haematinics and blood transfusion therapy prescribing patterns in order to:

To analyze the prescribing patterns of drugs used for anemia treatment.

To assess blood transfusion therapy for anemic patients.
PLAN OF STUDY

PHASE I:

1] PRELIMINARY LITERATURE SEARCH

2] DESIGNING STUDY PROTOCOL

3] DATA COLLECTION FORM

PHASE II:

1] LITERATURE SURVEY

2] DATA COLLECTION

3] DATA ANALYSIS

PHASE III:

RESULT AND DISCUSSION
METHODOLOGY

STUDY DESIGN:

Retrospective observational study.

STUDY LOCATION:

The study was carried out in a tertiary hospital, on inpatients medical records from medical ward.

SAMPLE SIZE:

Sample size in this study is 63 patients who diagnosed with anemia and got treatment at the hospital.

STUDY DURATION:

Six months.

INCLUSION CRITERIA:

Male and female patients.

Patients received oral / IV haematins.

Patients received blood transfusion therapy.

Patients over 18 years of age.

EXCLUSION CRITERIA:

Patients under 18 years of age.

Pregnant patients were excluded from the study.

SOURCE OF DATA:

The data was collected from patients case reports at medical report department (MRD).
RESULT AND DISCUSSION

AGE:

The study included 63 patients, out of these patients, 31 patients was found under age category between 18 – 40 years of age (49.2%), while 18 patients were above 60 years of age (28.6%) and last 14 patients under age category between 41-60 years of age (22.2%).

**TABLE-1 : AGE WISE DISTRIBUTION**

<table>
<thead>
<tr>
<th>Age</th>
<th>Frequency</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>18-40</td>
<td>31</td>
<td>49.2</td>
</tr>
<tr>
<td>41-60</td>
<td>14</td>
<td>22.2</td>
</tr>
<tr>
<td>60+</td>
<td>18</td>
<td>28.6</td>
</tr>
</tbody>
</table>

**Figure -1 : Age vs Frequency**

![Age vs Frequency Chart]
GENDER:

Out of 63 patients, 43 patients were females (68.26%) while remaining 20 patients were male (31.74%). The majority of patients in this study were females.

**TABLE-2: GENDER WISE DISTRIBUTION**

<table>
<thead>
<tr>
<th>Gender</th>
<th>Frequency</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female</td>
<td>43</td>
<td>68.26</td>
</tr>
<tr>
<td>Male</td>
<td>20</td>
<td>31.74</td>
</tr>
</tbody>
</table>

**Figure – 2: Gender vs Frequency**

![Gender vs Frequency Chart](image-url)
BLOOD TRANSFUSION THERAPY:

Anemic Patients who treated with blood transfusion therapy are 50 patients, whome are the majority of the study (79.36%), while remaining 13 patients were treated with hematins oral or IV supplements (20.64%).

**TABLE-3: BLOOD TRANSFUSION THERAPY**

<table>
<thead>
<tr>
<th>Category</th>
<th>Frequency</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood transfusion therapy</td>
<td>50</td>
<td>79.36%</td>
</tr>
<tr>
<td>Hematinics supplements</td>
<td>13</td>
<td>20.64%</td>
</tr>
</tbody>
</table>

**Figure - 3: Category vs Frequency**
BLOOD GROUPING:

Blood groups were found in this study, 18 patients were identified as O group (36%), where as 13 patients were identified as A group (26%), while 14 patients were identified as B group (28%), and 5 patients were identified as AB group (10%).

Table - 4: Blood Groups Wise Distribution

<table>
<thead>
<tr>
<th>Blood Group</th>
<th>Frequency</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>O</td>
<td>18</td>
<td>36</td>
</tr>
<tr>
<td>A</td>
<td>13</td>
<td>26</td>
</tr>
<tr>
<td>B</td>
<td>14</td>
<td>28</td>
</tr>
<tr>
<td>AB</td>
<td>5</td>
<td>10</td>
</tr>
</tbody>
</table>

Figure - 4: Blood groups vs frequency
COMPONENTS AND VOLUME OF BLOOD TRANSFUSION THERAPY:

Mainly two components where used for the treatment of anemic patients using either:

A] Whole human blood IP (350ml)

B] or concentrated RBCs IP (250 – 280 ml) or both are used for treatment.

Out of 50 patients who were treated by blood transfusion therapy, 29 of anemic patients received a Whole human blood IP (58%) at volume of 350 ml while 15 anemic patients received concentrated RBCs IP at volume of 250 – 280 ml (30%) , and 6 patients only received both components (12%) as blood transfusion therapy.

**TABLE -5: Components of Blood Transfusion Therapy**

<table>
<thead>
<tr>
<th>Components</th>
<th>Frequency</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Whole human blood IP [350 ml]</td>
<td>29</td>
<td>58</td>
</tr>
<tr>
<td>Conc. RBCs IP [250-280ml]</td>
<td>15</td>
<td>30</td>
</tr>
<tr>
<td>Both</td>
<td>6</td>
<td>12</td>
</tr>
</tbody>
</table>

**Figure 5 : Components of Blood Transfusion vs Frequency**

![Bar chart showing components of blood transfusion therapy](chart.png)
SEVERITY OF ANEMIA BASED ON HB LEVEL:

Out of the 63 patients, 36 anemic patients were identified as sever anemia (57.15%), while 19 anemic patients were observed as moderate anemia (30.15%) and 8 patients were identified as mild anemia (12.70%). Majority of patients were severly anemic patients.

Table -6: Severity level

<table>
<thead>
<tr>
<th>Severity level</th>
<th>Frequency</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>8</td>
<td>12.7</td>
</tr>
<tr>
<td>Moderate</td>
<td>19</td>
<td>30.15</td>
</tr>
<tr>
<td>Sever</td>
<td>36</td>
<td>57.15</td>
</tr>
</tbody>
</table>

Figure - 6 : Severity level vs frequency
BODY MASS INDEX (BMI):

Out of 63 anemic Patients, 45 of patients body mass index were found to be Normal weight (71.4%), whereas 9 patients were identified to be over weighted patients (14.2%), then 6 patients were found to be under the normal weight (9.5%) and 3 patients were found to be obese patients (4.9%).

Table-7 : BMI Wise Distribution

<table>
<thead>
<tr>
<th>BMI</th>
<th>Frequency</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Under normal weight</td>
<td>6</td>
<td>9.5</td>
</tr>
<tr>
<td>Normal weight</td>
<td>45</td>
<td>71.4</td>
</tr>
<tr>
<td>Over weight</td>
<td>9</td>
<td>14.2</td>
</tr>
<tr>
<td>Obese</td>
<td>3</td>
<td>4.9</td>
</tr>
</tbody>
</table>

Figure - 6 : BMI VS Frequency
**DURATION:**

Among 63 anemic patients 26 patients received the treatment within 1 day only (41.27%), followed by the duration of 2 days treatment with 11 patients (17.46%), duration of 3 days 9 patients (14.30%), duration of 4 days 5 patients were treated (7.93%), duration of 5 days were treated 4 patients (6.34%), duration of 7 days [a week] 4 patients (6.34%) were treated and finally in 10 days duration of time also 4 patients (6.35%) were treated at the hospital.

**TABLE-8 : DURATION**

<table>
<thead>
<tr>
<th>Duration (in days)</th>
<th>Frequency</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>26</td>
<td>41.27</td>
</tr>
<tr>
<td>2</td>
<td>11</td>
<td>17.46</td>
</tr>
<tr>
<td>3</td>
<td>9</td>
<td>14.30</td>
</tr>
<tr>
<td>4</td>
<td>5</td>
<td>7.93</td>
</tr>
<tr>
<td>5</td>
<td>4</td>
<td>6.34</td>
</tr>
<tr>
<td>7</td>
<td>4</td>
<td>6.34</td>
</tr>
<tr>
<td>10</td>
<td>4</td>
<td>6.35</td>
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</tbody>
</table>

**Figure - 7 : Duration VS Frequency**
CONCLUSION

The study is used to assess age group and the gender of the respondent involved in the study. Majority of anemic patients were females and in the age between 18 to 40 years.

The prescribed hematinics to anemic patients were fully insignificance, because most of these patients were readmitted for blood transfusion therapy which was the majority of the cases were treated with.

The anemic patients should be given a chart of nutrition and food that containing iron, Folate and Vitamin B₁₂ to increase the hemoglobin level along side with the prescribed hematinics.
BIBLIOGRAPHY


2] WHO topics anemia. available http://www.who.int/topics/anemia/en/


20] Prevalence of iron Deficiency and Iron Deficiency Anaemia in Adolescent Girls

In A Teritary Care Hospital, Rekha Kumari, Raushan Kumar Bharti, Kalpana


# DATA COLLECTION FORM

<table>
<thead>
<tr>
<th>Case no:</th>
<th>IP/OP NO:</th>
<th>Height:</th>
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<tbody>
<tr>
<td>Age</td>
<td>sex</td>
<td>Department:</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Admitted on:</th>
<th>Discharged on:</th>
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</table>

**Complaint at admission:**

**Past history:**

**Family history:**

**Drug allergies:**

**Diagnosis:**

<table>
<thead>
<tr>
<th>Brand name</th>
<th>Generic name</th>
<th>Dose &amp; Frequency</th>
<th>Date</th>
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</tbody>
</table>
Hematology:

RBcs:

Haemoglobin level (gm/dl):

Mcv: MCH: MCHC:

Hct:

Severity of Anemia:

□ sever
□ moderate
□ mild

blood transfusion therapy:

blood component:

blood group:

volume required: