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A PROSPECTIVE OBSERVATIONAL STUDY OF DIFFERENT TYPES OF ANEMIAS AND THEIR MANAGEMENT AT A TERTIARY CARE HOSPITAL

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Abstract:

Aim and Objectives: *To study and assess the pattern of anemia, utilization pattern of drugs, its management and compliance with the treatment.*

Materials and Methods: *In a prospective study spanning six months, the data of 100 patients was collected from In-Patient department of the General Medicine Department of Osmania General Hospital, Hyderabad.*

Results: *About 29.4% of the anemic patients were from the age group 21-30 years while 23.5% belonged to the age group 31-40 years. The cases of anemia in the females (36.2%) were found to be higher than in males (31.8%). The body mass index of each anemic patient showed that 25.4% of the anemic patients were under-weight according to their BMI. Among all the co morbid conditions, hepatic conditions were most prevalent (23.5%) among anemic patients. The female medical history showed 39.6% of females with a history of menorrhagia. 10.7% of the total cases of anemia were drug induced making flouroquinolones with the highest number of cases of drug induced anemia. More than half of the cases were of severe anemia (62.7%). About 52.7% of the cases included blood transfusion to treat the anemia.*

Conclusion: *Females were found to be the most prevalent group of patients to have developed anemia with some underlying gynecological and obstetric history. The in patients mostly had a severe form of anemia and treatment was done through blood transfusion and intravenous supplies. The clinical manifestations among all the subjects vary widely and an in depth analysis of each case of anemia will give more detailed information on the treatment pattern and other parameters associated.*

Keywords: *Anemia, Clinical Manifestations, Medical History, Compliance Management, Observation.*

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1. INTRODUCTION:

Approximately 610 million people across the world suffer with anemia every year. This accounts for 8.8 % of the total world population. Of these, the most common type of anemia is iron deficiency anemia which affects 310 million people. Anemias are a group of diseases characterized by a decrease in either the hemoglobin (Hb) or the volume of red blood cells (RBC), which results in decreased oxygen-carrying capacity of the blood.[1]

Anemias are generally a sign of underlying pathology, therefore, determining the cause of the anemia is important. Possible consequences of chronic anemia include reduced-quality-of-life, decreased survival and increased risk of cardiac complications, neurologic dysfunction and surgical complications. Awareness of anemia, its detection, investigation and management must be raised [2]

Anemias are a group of diseases characterized by a decrease in either Hb or red blood cells (RBCs), resulting in reduced oxygen carrying capacity of the blood. Anemias can result from inadequate RBC production, increased RBC destruction or blood loss. They can be manifestation of a host of systematic disorders, such as infections. Because anemias are often a sign of underlying pathology, rapid diagnosis of the cause is essential. [1]

1.1 DEFINITION

According to the World Health Organization (WHO), anemia is defined as a hemoglobin level of less than 13 g/dL in men and less than 12 g/dL in women.
However, normal Hb distribution varies not only with sex but also with ethnicity and physiological status. New lower limits of normal Hb values have been proposed, according to ethnicity, gender, and age. Anemia is often multifactorial and is not an independent phenomenon. For the classification and diagnosis the hematologic parameters, the underlying pathological mechanism and patient history should be taken into account. [3]

1.2 TYPES OF ANEMIAS

Anemia is classified by two methods:
1. Morphological classification
2. Etiological classification [4]

1. MORPHOLOGICAL CLASSIFICATION

Morphological classification depends upon the size and color of RBC. Size of RBC is determined by mean corpuscular volume (MCV). Color is determined by mean corpuscular hemoglobin concentration (MCHC). By this method, the anemia is classified into four types;
a) Normocytic Normochromic Anemia
Size (MCV) and color (MCHC) of RBCs are normal. But the number of RBC is less.
b) Macrocytic Normochromic Anemia
RBCs are larger in size with normal color. RBC count is less.
c) Macrocytic Hypochromic Anemia
RBCs are larger in size. MCHC is less, so the cells are pale (less colored).
d) Microcytic Hypochromic Anemia
RBCs are smaller in size with less color. [4]

2. ETIOLOGICAL CLASSIFICATION

On the basis of etiology (study of cause or origin), anemia is divided into five types:
a) Hemorrhagic anemia
b) Hemolytic anemia
c) Nutrition deficiency anemia
d) Aplastic anemia
e) Anemia of chronic diseases.

a) HEMORRHAGIC ANEMIA
Hemorrhage refers to excessive loss of blood. Anemia due to hemorrhage is known as hemorrhagic anemia. It occurs both in acute and chronic hemorrhagic
conditions.
Acute hemorrhage
Acute hemorrhage refers to sudden loss of a large quantity of blood as in the case of accident. Within about 24 hours after the hemorrhage, the plasma portion of blood is replaced. However, the replacement of RBCs does not occur quickly and it takes at least 4 to 6 weeks. So with less number of RBCs, hemo-dilution occurs.
Chronic hemorrhage
It refers to loss of blood by internal or external bleeding, over a long period of time. It occurs in conditions like peptic ulcer, purpura, hemophilia and menorrhagia. Due to continuous loss of blood, lot of iron is lost from the body causing iron deficiency. This affects the synthesis of hemoglobin resulting in less hemoglobin content in the cells. The cells also become small. Hence the RBCs are microcytic and hypochromic[5]

b) HEMOLYTIC ANEMIA
Hemolysis means destruction of RBCs. Anemia due to excessive hemolysis which is not compensated by increased RBC production is called hemolytic anemia. It is classified into two types Extrinsic hemolytic anemia and Intrinsic hemolytic anemia.

c) NUTRITIONAL DEFICIENCY ANEMIA
Anemia that occurs due to deficiency of a nutritive substance necessary for erythropoiesis is called nutrition deficiency anemia. The substances which

are necessary for erythropoiesis are iron, proteins and vitamins like C, B12 and folic acid. The types of nutrition deficiency anemia are. [6]

d) APLASTIC ANEMIA

Aplastic anemia is due to the disorder of red bone marrow. Red bone marrow is reduced and replaced by fatty tissues. Bone marrow disorder occurs in the following conditions:

i. Repeated exposure to Xray or gamma ray radiation.
ii. Presence of bacterial toxins, quinine, gold salts, benzene, radium, etc.
iii. Tuberculosis.
iv. Viral infections like hepatitis and HIV infections.

In aplastic anemia, the RBCs are normocytic and normochromic [7]

e) ANEMIA OF CHRONIC DISEASES

Anemia of chronic diseases is the second common type of anemia (next to iron deficiency anemia). It is characterized by short lifespan of RBCs, caused by disturbance in iron metabolism or resistance to erythropoietin action. Anemia develops after few months of sustained disease. RBCs are normocytic and normochromic.

1.3 EPIDEMIOLOGY OF ANEMIA

The WHO Global Database on Anemia is the only source of anemia estimates at country, regional and global level. The indicator used is the blood concentration of haemoglobin and the thresholds to establish normal ranges of haemoglobin for the different physiological groups of populations (children, adolescents, adults and pregnant women) were defined at a WHO Expert Consultation held in Geneva in 1992.

The global prevalence of anemia for the general population is 24.8% and it is estimated that 1620 million people are affected by anemia.

For pregnant women the prevalence is slightly lower; however, its distribution by region follows the same trend as the one observed for preschool-age children. The highest prevalence is in Africa (57.1%) and in South-East Asia (48.2%), followed by the Eastern Mediterranean (44.2%), Western Pacific (30.7%), and the European Americas regions, 25% and 24.1% respectively. Overall, 56.4 million pregnant women are anaemic (41.8% prevalence globally).

In non-pregnant women, the prevalence of anemia is slightly lower than in pregnant women. Overall, 468.4 million non-pregnant women are anaemic (30.2% prevalence globally). The highest prevalence is found in Africa (47.5%) and in South-East Asia (35.7%). In the Eastern Mediterranean region, the prevalence is 32.4%, 20.5% in the Western Pacific region, 19% in the European region, and 17.8% in the Americas. [8]

1.4 PATHOPHYSIOLOGY OF ANEMIA

It should be noted that, although there are many adjustments that can be made, one that cannot is decrease in the tissue requirement for oxygen. Actually, overall body oxidative metabolism increases in anemia because of the energy requirement of the compensatory activities.

Decreased hemoglobin oxygen affinity

Increased oxygen extraction of anemic blood by the tissues produces increased concentration of deoxyhemoglobin in the RBC, which stimulates the production of 2, 3-diphosphoglycerate (2,3-DPG). 2, 3-DPG shifts the hemoglobin-oxygen dissociation curve to the right, thus allowing the tissues to more easily strip the hemoglobin of its precious electron-accepting cargo.

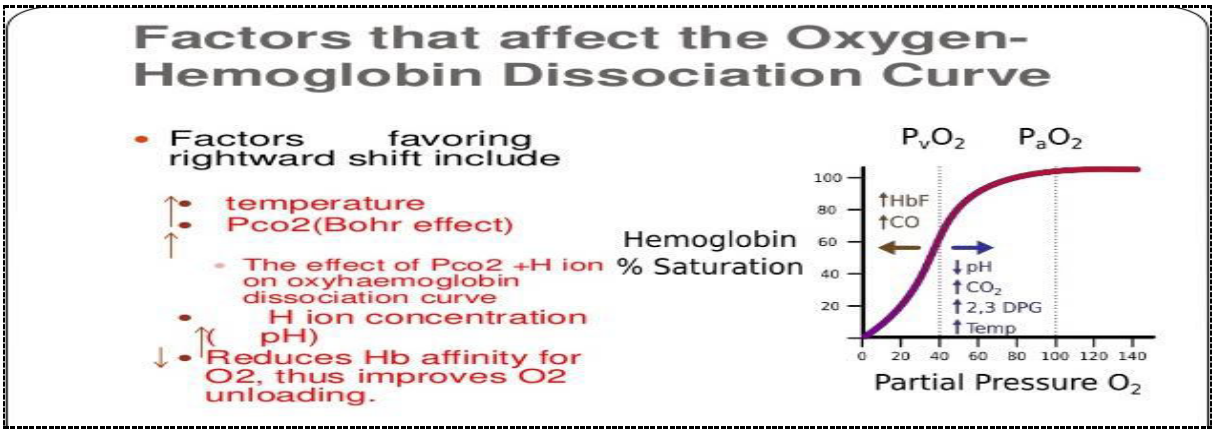


Figure no. 1 FACTORS AFFECTING OXYGEN-HEMOGLOBIN DISSOCIATION CURVE

Redistribution of blood flow

In anemia selective vasoconstriction of blood vessels subserving certain nonvital areas allows more blood to flow into critical areas. The main donor sites who sacrifice their aerobic lifestyle are the skin and kidneys. Shunting of blood away from cutaneous sites is the mechanism behind the clinical finding of pallor, a cardinal sign of anemia. Although the kidney can hardly be thought of as a nonvital area, it receives (in the normal state) much more blood flow than is needed to meet its metabolic requirements. Although (by definition) total body red cell mass is decreased in anemia, in the chronically anemic patient the total blood volume paradoxically is increased, due to increased plasma volume. It is as if the body were trying to make up in blood quantity what it lacks in quality.

Increased cardiac output

The heart can respond to tissue hypoxia by increased cardiac output. The increased output is matched by decreased peripheral vascular resistance and decreased blood viscosity (thinner blood flows more freely than thick blood), so that cardiac output can rise without an increase in blood pressure. Generally, anemia must be fairly severe (hemoglobin < 7 g/dL) before cardiac output rises. [9]

1.5 CLASSIFICATION OF ANEMIA

Anemia can be classified based on three systems of classifications- [10]

1. Morphology
2. Etiology
3. Pathophysiology

1 MORPHOLOGICAL CLASSIFICATION

This type of classification is based on the size of the red blood cells (RBC's). it includes the following types-

a) Microcytic –

Microcytic anemia is defined as the presence of small, often hypochromic, red blood cells in a peripheral blood smear and is usually characterized by a low MCV (less than 83 micron 3). Iron deficiency is the most common cause of **microcytic anemia**.

Sickle cell anemia, thalassemia and other hemoglobin abnormalities also come under this category. [11]

b) Macrocytic-

Macrocytosis is a term used to describe erythrocytes that are larger than normal, typically reported as mean cell volume (MCV) greater than 100 fL. The amount of hemoglobin increases proportionately with the increase in cell size. Therefore, if the increase in MCV is not related to macrocytic anemia, the mean cell hemoglobin concentration (MCHC) also increases in proportion.

This category includes Vitamin B12 deficiency anemia and Folic Acid deficiency anemia. [12]

c) Normocytic-

Normocytic anemias may be thought of as representing any of the following: a decreased production of normal-sized red blood cells (e.g., anemia of chronic disease, aplastic anemia); an increased destruction or loss of red blood cells (e.g., hemolysis, post hemorrhagic anemia); an uncompensated increase in plasma volume (e.g., pregnancy, fluid overload); or a mixture of conditions producing microcytic and macrocytic anemias. [13]

2. ETIOLOGICAL CLASSIFICATION

Based on the cause, anemias can be divided into:

a) Deficiency- This could include a deficiency of Iron, Vitamin B12, Folic acid or Pyridoxine.

b) Central- This type is caused due to impaired bone marrow function.

E.g. anemia of chronic disease, anemia of the elderly and malignant bone marrow disorders.

c) Peripheral- These types of anemias are caused either due to bleeding (hemorrhage) or hemolysis (hemolytic anemias).

3. PATHOPHYSIOLOGICAL CLASSIFICATION

Based on the pathophysiological mechanism involved, anemia can be classified into the following:

a) Excessive blood loss –This may be due to recent hemorrhage, trauma, peptic ulcer, gastritis, hemorrhoids, etc.

b) Chronic hemorrhage –It may be caused due to vaginal bleeding, peptic ulcer, intestinal parasites, Aspirin and other non- steroidal anti-inflammatory agents (NSAID's).

c) Excessive RBC destruction –This may be due to certain drugs, RBC antibodies, physical trauma to RBC's or excessive sequestration in the spleen.

d) Inadequate production of mature RBC's

1.6 CLINICAL MANIFESTATIONS

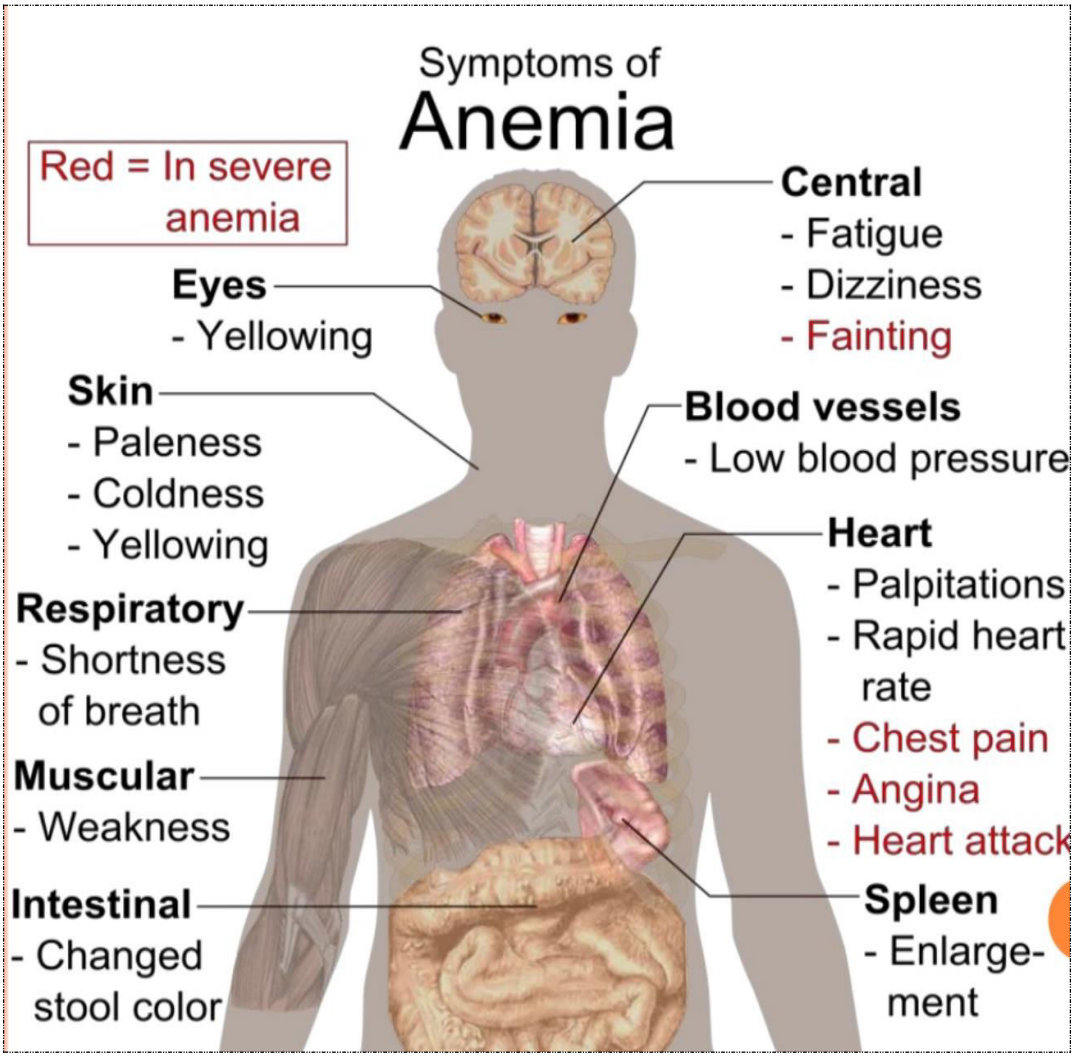


Figure no. 2 CLINICAL MANIFESATIONS OF ANEMIA

1.7 DIAGNOSIS

1. GENERAL PRESENTATION

History, physical examination and laboratory testing are used in the evaluation of anemia. The workup determines if the patient is bleeding and investigates potential causes of the anemia, such as increased RBC destruction, bone marrow suppression, or iron deficiency. Occupation, social habits, travel history, and diet all can be important in identifying causes of anemia. Additionally, information about concurrent non-hematologic disease states and a drug ingestion history are essential when evaluating the cause of the anemia. [14]

2. LABORATORY EVALUATION

Based on laboratory test results, anemia can be categorized into three functional defects:

a) RBC production failure (hypoproliferative)

b) Cell maturation ineffectiveness

c) Increase in RBC destruction Table 104–3 lists normal hematologic values, although these values may differ in certain populations, such as individuals living at high altitudes and endurance athletes. [15]

Table no. 1 NORMAL HEMATOLOGIC VALUES

	Reference Range			
Test	**2–6**	**6–12**	**12–18**	**18–49**
Hemoglobin (g/dL)	11.5–15.5	11.5–15.5	M 13.0–16.0 F 12.0–16.0	M 13.5–17.5 F 12.0–16.0
Hematocrit (%)	34–40	35–45	M 37–49 F 36–46	M 41–53 F 36–46
MCV (fL)	75–87	77–95	M 78–98 F 78–102	80–100
MCHC (%)	–	31–37	31–37	31–37
MCH (pg)	24–30	25–33	25–35	26–34
RBC (million/mm^3)	3.9–5.3	4.0–5.2	M 4.5–5.3	M 4.5–5.9
Reticulocyte count, absolute (%)				0.5–1.5
Serum iron (mcg/dL)		50–120	50–120	M 50–160 F 40–150
TIBC (mcg/dL)	250–400	250–400	250–400	250–400
RDW (%)				11–16
Ferritin (ng/mL)	7–140	7–140	7–140	M 15–200 F 12–150
Folate (ng/mL)				1.8–16.0[a]
Vitamin B_{12} (pg/mL)				100–900[a]
Erythropoietin (mU/mL)				0–19

F, female; M, male; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; RBC, red blood cell; RDW, red blood cell distribution; TIBC, total iron-binding capacity.
[a]Varies by assay method.

1.8 TREATMENT

1. IRON DEFICIENCY ANEMIA

DIETARY SUPPLEMENTATION AND THERAPEUTIC IRON PREPARATION:

Treatment of IDA usually consists of dietary supplementation and administration of therapeutic iron preparations. Iron is poorly absorbed from vegetables, grain products, dairy products, and eggs; it is best absorbed from meat, fish, and poultry. Beverages have been shown to affect iron absorption. Meat, orange juice, and other ascorbic acid–rich foods should be included with meals, whereas milk and tea should be consumed in moderation between meals. In most cases of IDA, oral administration of iron therapy with soluble Fe2+ iron salts is appropriate.

Table no. 2 ORAL IRON PRODUCTS

Salt	Elemental Iron Percentage	Elemental Iron Provided
Ferrous sulfate	20%	60–65 mg/324–325 mg table 18 mg iron/5 mL syrup 44 mg iron/5 mL elixir 15 mg iron/0.6 mL drop
Ferrous sulfate (exsiccated)	30%	65 mg/200 mg tablet 60 mg/187 mg tablet 50 mg/160 mg tablet
Ferrous gluconate	12%	36 mg/325 mg tablet 27 mg/240 mg tablet
Ferrous fumarate	33%	33 mg/100 mg tablet 63–66 mg/200 mg tablet 106 mg/324–325 mg tablet 15 mg/0.6 mL drop 33 mg/5 mL suspension
Polysaccharide iron complex	100%	150 mg capsule 50 mg tablet 100 mg/5 mL elixir
Carbonyl iron	100%	50 mg caplet

Table no. 3 IRON-SALT DRUG INTERACTIONS

Drugs That Decrease Iron Absorption	Object Drugs Affected by Iron
Al-, Mg-, and Ca^{+2}-containing antacids	Levodopa ↓ (chelates with iron)
Tetracycline and doxycycline	Methyldopa ↓ (decreases efficacy of methyldopa)
$Histamine_2$ antagonists	Levothyroxine ↓ (decreased efficacy of levothyroxine)
Proton pump inhibitors	Penicillamine ↓ (chelates with iron)
Cholestyramine	Fluoroquinolones ↓ (forms ferric ion–quinolone complex)
	Tetracycline and doxycycline ↓ (when administered within 2 hours of iron salt)
	Mycophenolate ↓ (decreases absorption)

PARENTERAL IRON THERAPY

When evidence of iron malabsorption or intolerance to orally administered iron is seen or long-term nonadherence is suspected, parenteral iron therapy may be warranted. Patients with significant blood loss who refuse transfusions and cannot take oral iron therapy also may require parenteral iron therapy.

Table no. 4 COMPARISON OF PARENTERAL IRON PREPARATIONS

	Sodium Ferric Gluconate	Iron Dextran	Iron Sucrose
Amount of elemental iron	62.5 mg iron/5 mL	50 mg iron/mL	20 mg iron/mL
Molecular weight	Ferrlecit: 289,000–444,000 daltons	InFeD: 165,000 daltons DexFerrum: 267,000 daltons	Venofer: 34,000–60,000 daltons
Composition	Ferric oxide hydrate bonded to sucrose chelates with gluconate in a molar rate of 2 iron molecules to 1 gluconate molecule	Complex of ferric hydroxide and dextran	Complex of polynuclear iron hydroxide in sucrose
Preservative	Benzyl alcohol 9 mg/5 mL 20% (975 mg in 62.5 mg iron)	None	None
Indication	Treatment of iron-deficiency anemia in patients undergoing chronic hemodialysis who are receiving supplemental erythropoietin therapy	Treatment of patients with documented iron deficiency in whom oral therapy is unsatisfactory or impossible	Treatment of iron-deficiency anemia in patients undergoing chronic hemodialysis who are receiving supplemental epoetin alfa therapy
Warning	No black box warning; hypersensitivity reactions	Black box warning: anaphylactic-type reactions	Black box warning: anaphylactic-type reactions
IM injection	No	Yes	No
Usual dose	125 mg (10 mL) diluted in 100 mL normal saline, infused over 60 minutes; also can be administered as a slow IV injection (rate of 12.5 mg/min).	100 mg undiluted at a rate not to exceed 50 mg (1 mL) per min	100 mg into the dialysis line at a rate of 1 mL (20 mg of iron) undiluted solution per minute
Treatment	8 doses × 125 mg = 1,000 mg	10 doses × 100 mg = 1,000 mg	Up to 10 doses × 100 mg = 1,000 mg
Common adverse effects	Cramps, nausea and vomiting, flushing, hypotension, rash, pruritus	Pain and brown staining at injection site, flushing, hypotension, fever, chills, myalgia, anaphylaxis	Leg cramps, hypotension

Table no. 5 EQUATIONS FOR CALCULATING DOSES OF PARENTERAL IRON

In patients with iron deficiency anemia:

Adults + children >15 kg

Dose (mL) = 0.0442 (Desired Hb − Observed Hb) × LBW + (0.26 × LBW)

LBW males = 50 kg + (2.3 × inches over 5 ft)

LBW females = 45.5 kg + (2.3 × inches over 5 ft)

Children 5–15 kg

Dose (mL) = 0.0442 (Desired Hb − Observed Hb) × W + (0.26 × W)

In patients with anemia secondary to blood loss (hemorrhagic diathesis or long-term dialysis):

mg of iron = blood loss × hematocrit,

where blood loss is in milliliters and hematocrit is expressed as a decimal fraction.

Hb, hemoglobin; LBW, lean body weight; mL, milliliter; W, weight.

Iron dextran must be processed by macrophages for the iron to be biologically available. The absorption and metabolism vary with the route and amount of drug given.

2. VITAMIN B12 DEFICIENCY ANEMIA

The goals of treatment for vitamin B12 deficiency include reversal of hematologic manifestations, replacement of body stores, and prevention or resolution of neurologic manifestations. Patients should be counseled on the types of foods high in vitamin B12 content.

Table no. 6 GOOD SOURCES OF VITAMIN B12

Food	Serving Size	Amount (mcg)
Beef liver, cooked	3 oz	60
Breakfast cereal, fortified (100%)	¾ cup	6
Rainbow trout, cooked	3 oz	5.3
Sockeye salmon, cooked	3 oz	4.9
Beef, cooked	3 oz	2.1
Breakfast cereal, fortified (25%)	¾ cup	1.5
Haddock, cooked	3 oz	1.2
Clams, breaded and fried	¾ cup	1.1
Oysters, breaded and fried	6 pieces	1
Tuna, canned in water	3 oz	0.9
Milk	1 cup	0.9
Yogurt	8 oz	0.9

3. FOLIC ACID DEFICIENCY ANEMIA

Therapy for folic acid deficiency consists of administration of exogenous folic acid to induce hematologic remission, replace body stores, and resolve signs and symptoms.

Table no. 7 GOOD SOURCES OF FOLATE

Food	Serving	Amount (mcg)
Chicken liver	3.5 oz	770
Cereal	½ to 1½ cups	100–400
Lentils, cooked	½ cup	180
Chickpeas	½ cup	141
Asparagus	½ cup	132
Spinach, cooked	½ cup	131
Black beans	½ cup	128
Pasta	2 oz	100–120
Kidney beans	½ cup	115
Lima beans	½ cup	78
White rice, cooked	¾ cup	60
Tomato juice	1 cup	48
Brussels sprouts	½ cup	47
Orange	1 medium	47

2. AIMS AND OBJECTIVES

- To identify and observe the management of different types of anemias along with co-morbid conditions.
- To assess the incidence of adverse reactions occurring in patients undergoing blood transfusion for severe

anemia.

NEED OF THE STUDY

Approximately 610 million people across the world suffer with anemia every year. This accounts for 8.8 % of the total world population. Of these, the most common type of anemia is iron deficiency anemia which affects 310 million people.

In developing countries like India, where malnourishment is one of the biggest challenges, diagnosis and treatment of nutritional anemia is of utmost importance and hence, becomes the need of our study.

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

3. METHODOLOGY

An Observational and prospective study was performed in the Department of General Medicine, Osmania General Hospital for a period of 6 months with a sample size of 100 patients.

3.1 PROCEDURE FOR DATA COLLECTION AND ANALYSIS: In a prospective study spanning six months, we collected and analyzed the cases of 100 patients of anemia from the General Medicine Department of Osmania General Hospital, Hyderabad.

Data collection for in-patients was done through demographics, admission notes, past medical history and patient's attender, keeping in mind the clinico-laboratory parameters and diagnosis of the patients which show a deficiency of hemoglobin as a hematological abnormality. Diagnosis and medications prescribed were recorded from daily reviews of clinicians' notes and treatment charts respectively. For all anemic patients, a documentation form was designed and interviews by prescribing clinician, patients and/or by their attendant where necessary. The demographic data, diagnosis (laboratory values), type of anemia and the drugs prescribed were recorded.

Figure no. 3 PLAN OF WORK

3.2 PLAN OF WORK

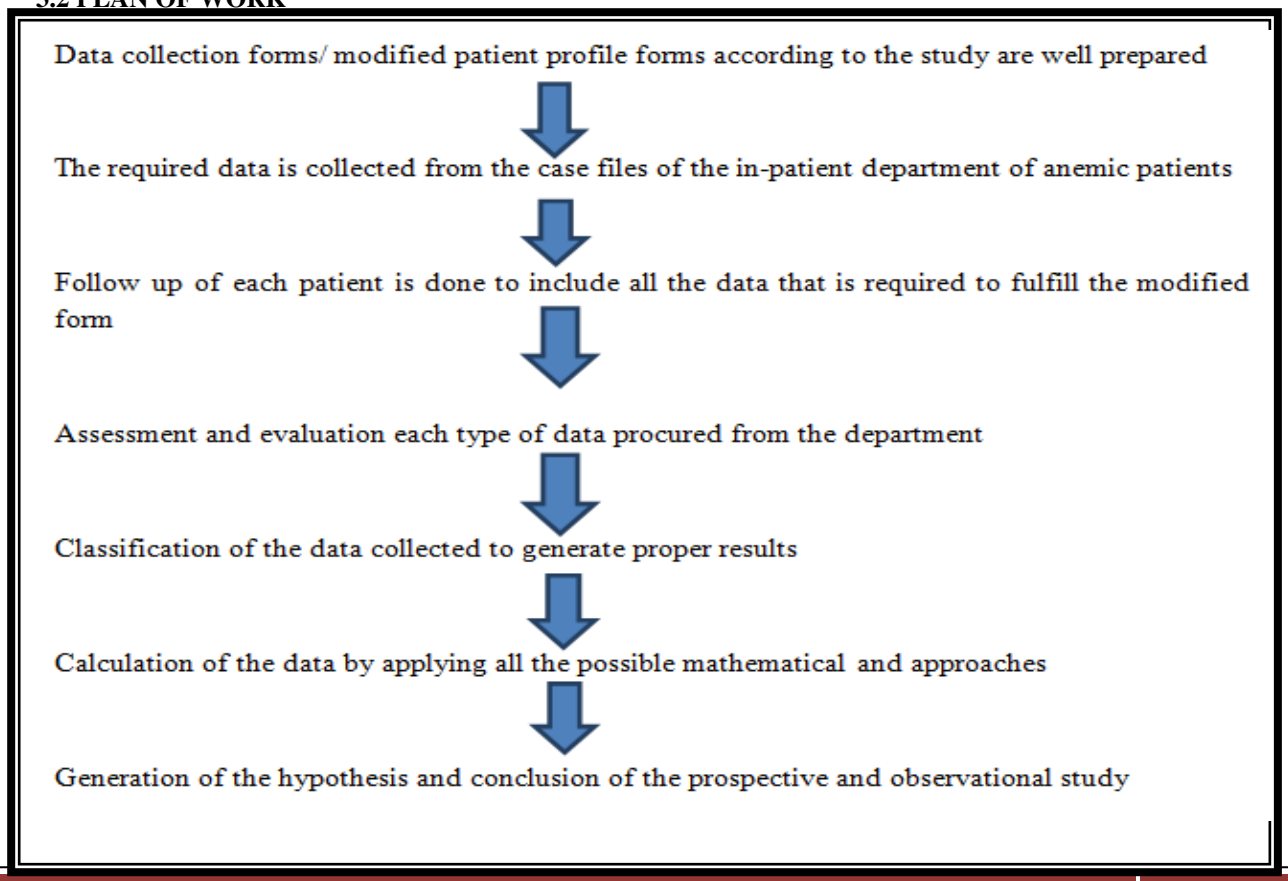
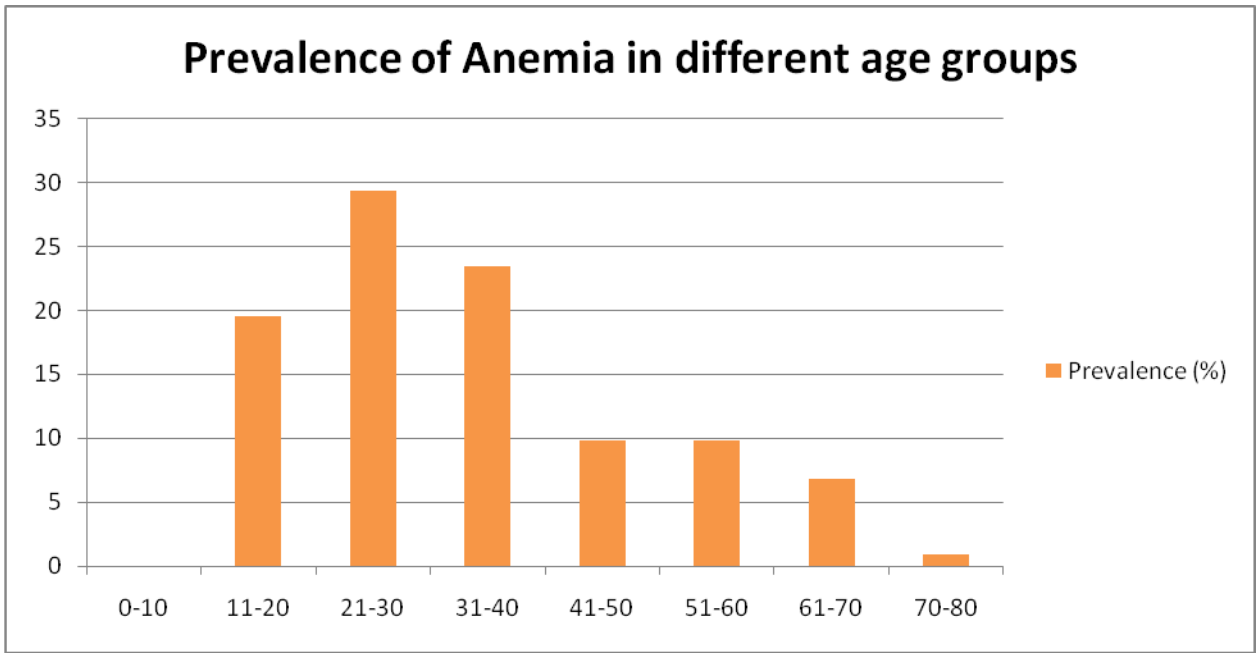


Table no. 8 PREVALENCE OF ANEMIA IN DIFFERENT AGE GROUPS

AGE GROUPS	NO. OF CASES (out of 100)	% PREVALENCE
0-10	0	0
11-20	20	19.6
21-30	30	29.4
31-40	24	23.5
41-50	10	9.8
51-60	10	9.8
61-70	7	6.8
71-80	1	0.9

Figure no. 4 PREVALENCE OF ANEMIA IN DIFFERENT AGE GROUPS

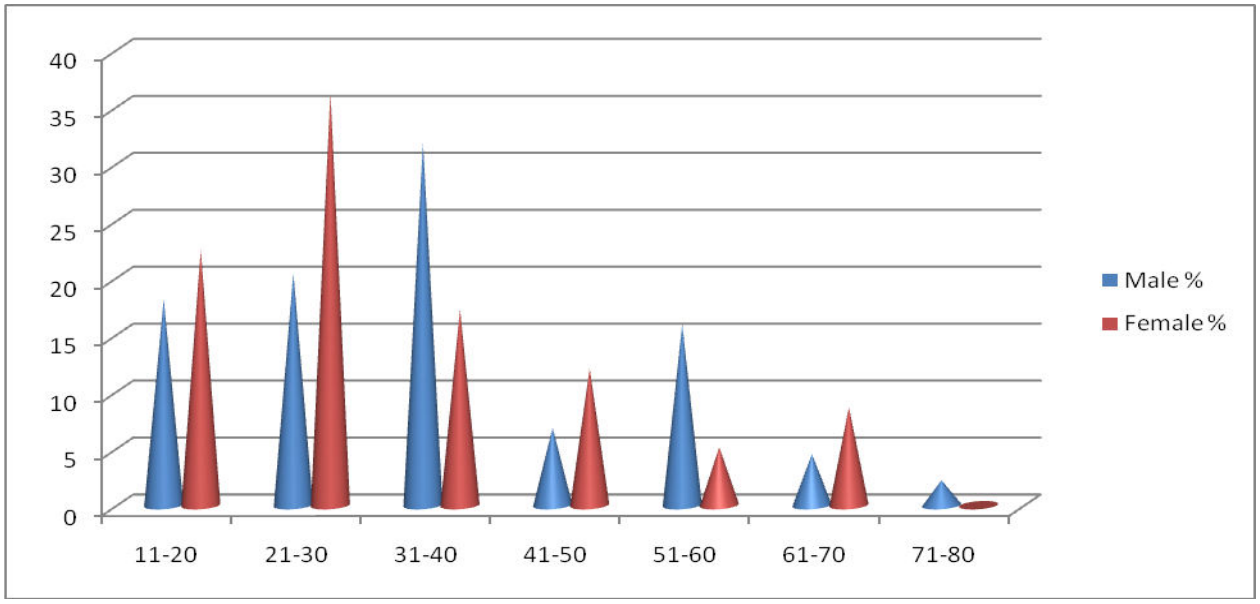


4.2 AGE AND SEX DISTRIBUTION OF ANEMIA

Table no. 9 AGE AND SEX DISTRIBUTION OF ANEMIA

AGE GROUP	MALE (%)	FEMALE (%)
11-20	8 (18.1%)	13 (22.4%)
21-30	9 (20.4%)	21 (36.2%)
31-40	14 (31.8%)	10 (17.2%)
41-50	3 (6.8%)	7 (12%)
51-60	7 (15.9%)	3 (5.1%)
61-70	2 (4.5%)	5 (8.6%)
71-80	1 (2.2%)	0 (0%)

Figure no. 5 AGE AND SEX DISTRIBUTION OF ANEMIA

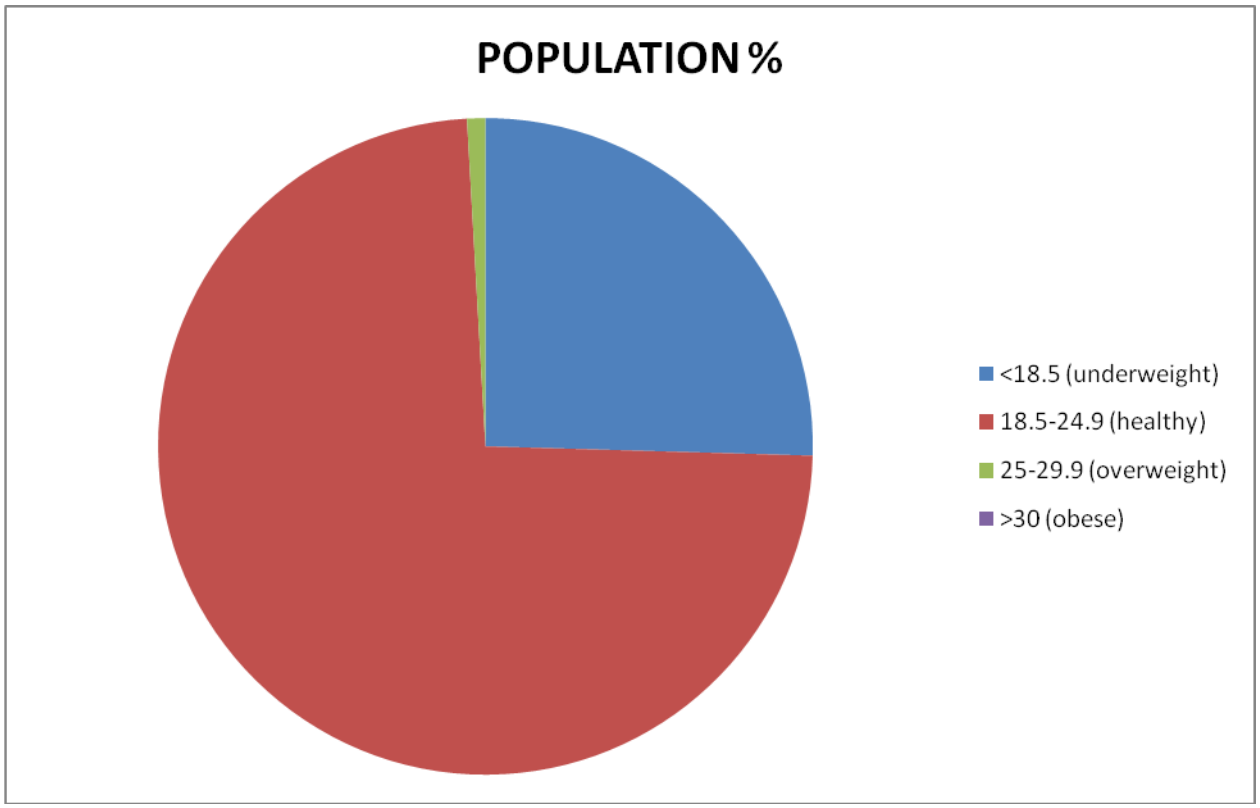


4.3 CORRELATION OF BODY MASS INDEX (BMI) WITH INCIDENCE OF ANEMIA

Table no. 10 CORRELATION OF BMI WITH INCIDENCE OF ANEMIA

BODY MASS INDEX	NO. OF CASES (out of 102)	% PREVALENCE
<18.5 (Underweight)	26	25.4%
18.5-24.9 (Healthy)	75	73.5 %
25-29.9 (Overweight)	1	0.1%
>30 (Obese)	0	0%

Figure no. 6 CORRELATION OF BMI WITH INCIDENCE OF ANEMIA

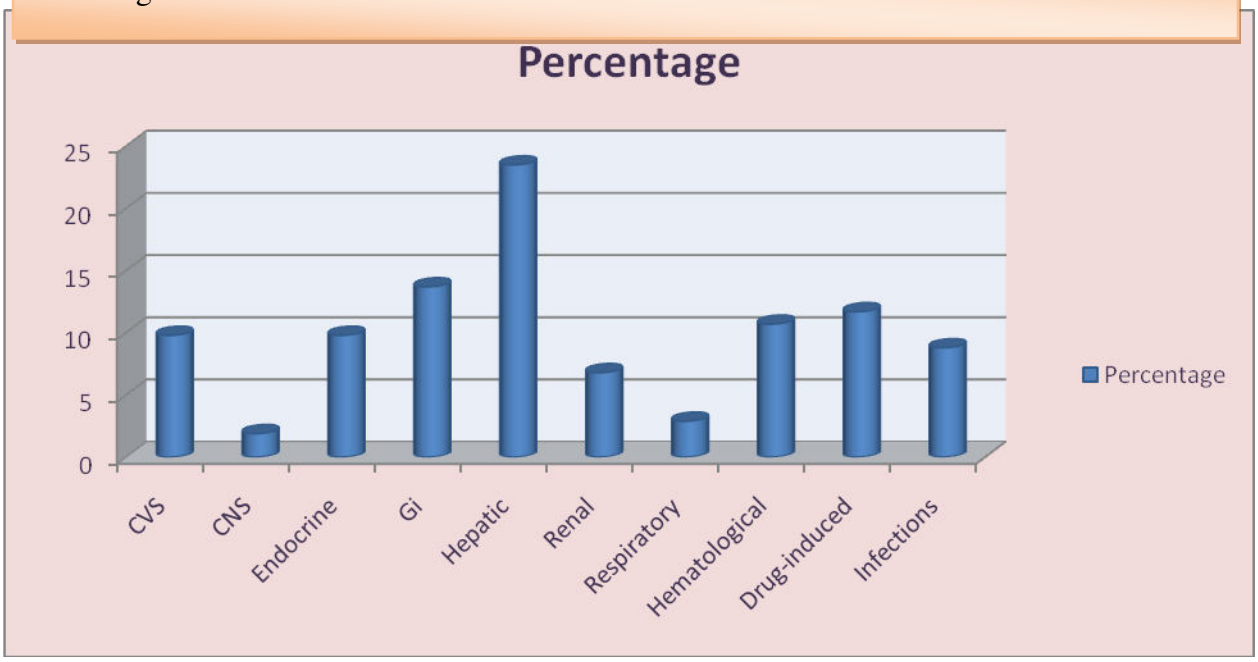


4.4 PREVALENCE OF ANEMIA WITH COMORBID CONDITIONS

Table no. 11 PREVALENCE OF ANEMIA WITH COMORBID CONDITIONS

COMORBIDITY	NO. OF CASES (out of 102)	% PREVALENCE
Cardiovascular	10	9.8
Central Nervous system	2	1.9
Endocrine	10	9.8
Gastro-intestinal	14	13.7
Hepatic	24	23.5
Renal	7	6.8
Respiratory	3	2.9
Hematological	11	10.7
Drug-induced	12	11.7
Infectious Diseases	9	8.8

Figure no. 7 PREVALENCE OF ANEMIA WITH COMORBID CONDITIONS

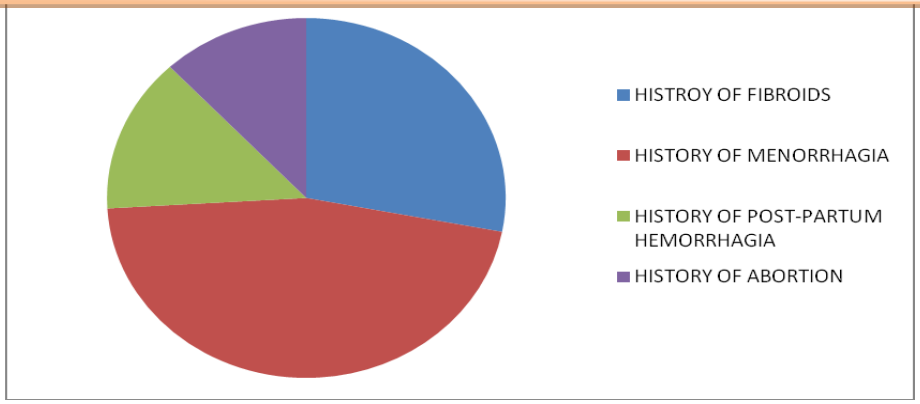


4.5 ANEMIA IN RELATION WITH GYNECOLOGICAL/ OBSTETRIC HISTORY

Table no. 12 ANEMIA IN RELATION WITH GYNECOLOGICAL/ OBSTETRIC HISTORY

PARAMETERS	NO.OF CASES (Out of 102)	% PREVALENCE
History of Fibroids	14	24.1
History of Menorrhagia	23	39.6
History of Post-partum Hemorrhage	7	12
History of Abortions	6	10.3

Figure no. 8 ANEMIA IN RELATION WITH GYNECOLOGICAL/ OBSTETRIC HISTORY

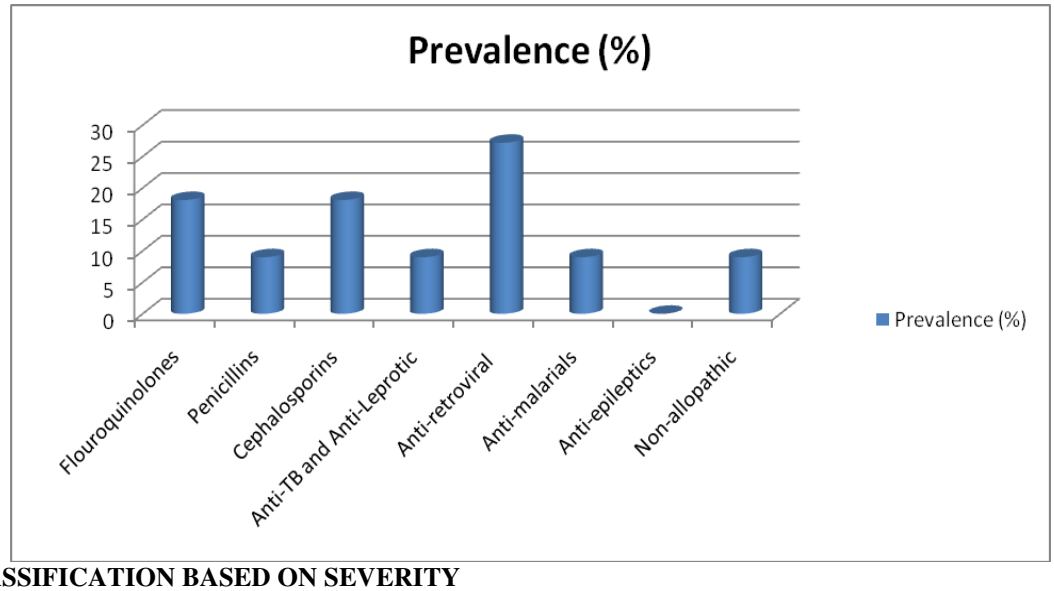


4.6 DRUG INDUCED ANEMIA

Table no. 13 DRUG INDUCED ANEMIA

CLASS OF DRUGS	NO. OF CASES (out of 102)	% PREVALENCE
Fluoroquinolones	2	18.1
Penicillins	1	9
Cephalosporins	2	18.1
Anti-TB and Anti-Leprotic	1	9
Anti-retroviral	3	27.2
Anti-malarials	1	9
Anti-epileptics	0	0
Non-allopathic	1	9

Figure no. 9 DRUG INDUCED ANEMIA

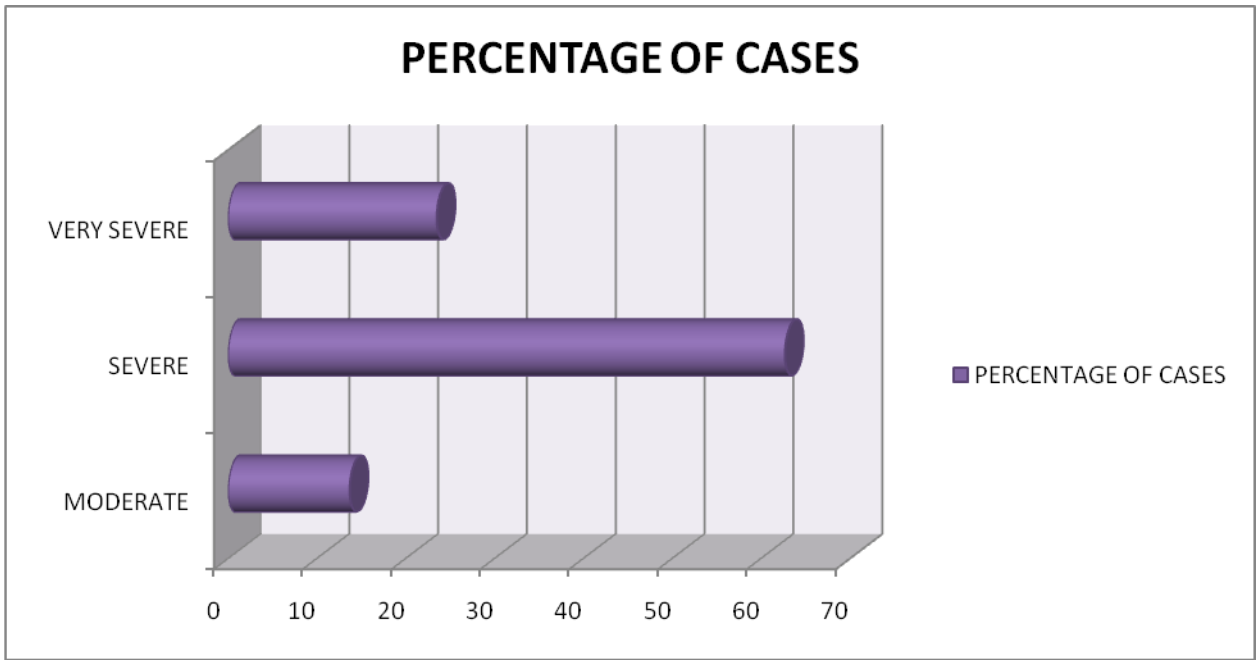


4.7 CLASSIFICATION BASED ON SEVERITY

Table no. 14 CLASSIFICATION OF ANEMIA BASED ON SEVERITY

HEMOGLOBIN RANGE	NO. OF CASES (out of 102)	% PREVALENCE
Moderate (7-10.9 gm %)	14	13.7
Severe (4-6.9 gm %)	64	62.7
Very severe (less than 4 gm %)	24	23.5

Figure no. 10 CLASSIFICATION OF ANEMIA BASED ON SEVERITY



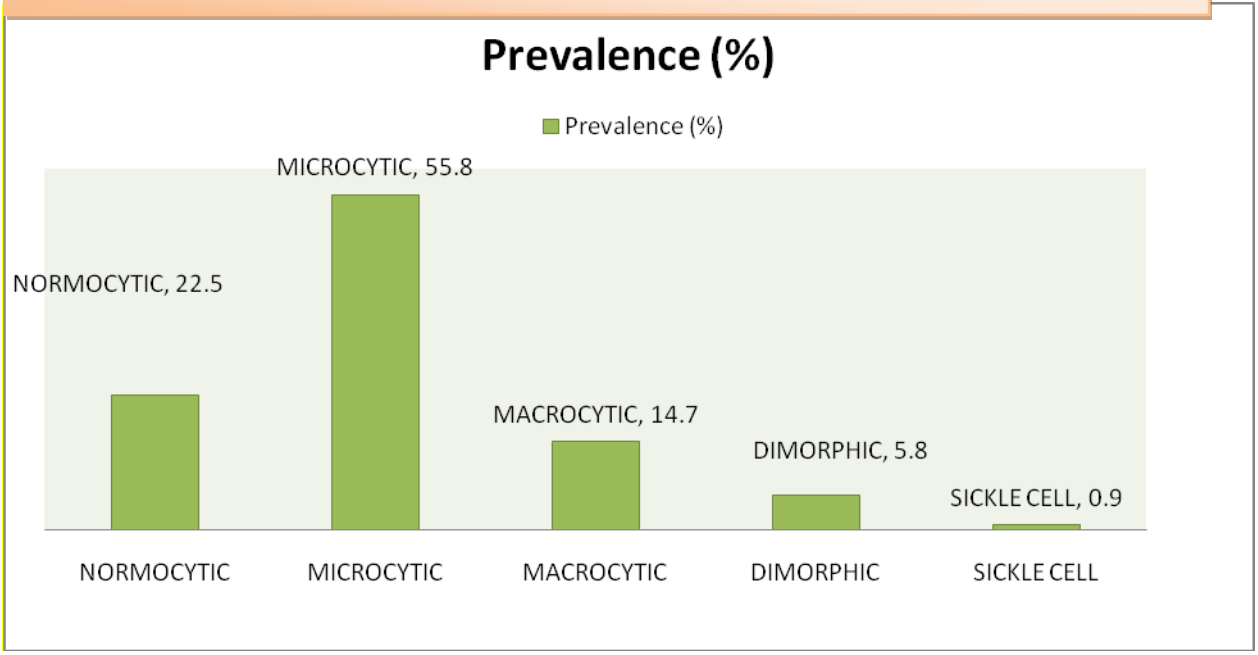
4.8 CLASSIFICATION BASED ON MORPHOLOGY (CELL TYPE)

Table no. 15 CLASSIFICATION BASED ON MORPHOLOGY (CELL TYPE)

CELL TYPE	NO. OF CASES (out of 102)	% PREVALENCE
Normocytic	23	22.5
Microcytic	57	55.8
Macrocytic	15	14.7
Dimorphic	6	5.8
Sickle cell	1	0.9

Based on morphology, it was found that 23 cases (22.5%) were normocytic, 57 cases (55.8%) were microcytic, 15 cases (14.7%) were macrocytic, 6 cases (5.8%) were dimorphic and 1 case (0.9%) was sickle shaped.

Figure no. 11 CLASSIFICATION BASED ON MORPHOLOGY (CELL TYPE)

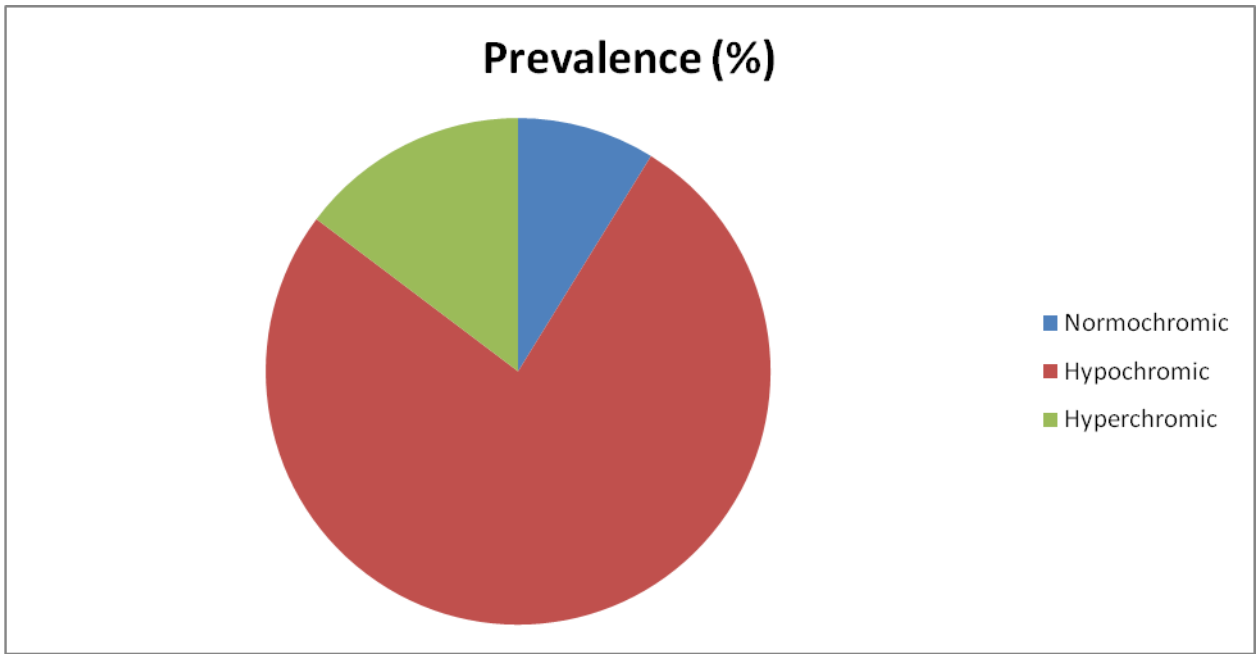


4.9 CLASSIFICATION BASED ON MORPHOLOGY (PIGMENTATION)

Table no. 16 CLASSIFICATION BASED ON MORPHOLOGY (PIGMENTATION)

PIGMENTATION	NO. OF CASES (out of 102)	% PREVALENCE
Normochromic	9	8.8
Hypochromic	78	76.4
Hyperchromic	15	14.7

Figure no. 12 CLASSIFICATION BASED ON MORPHOLOGY (PIGMENTATION)

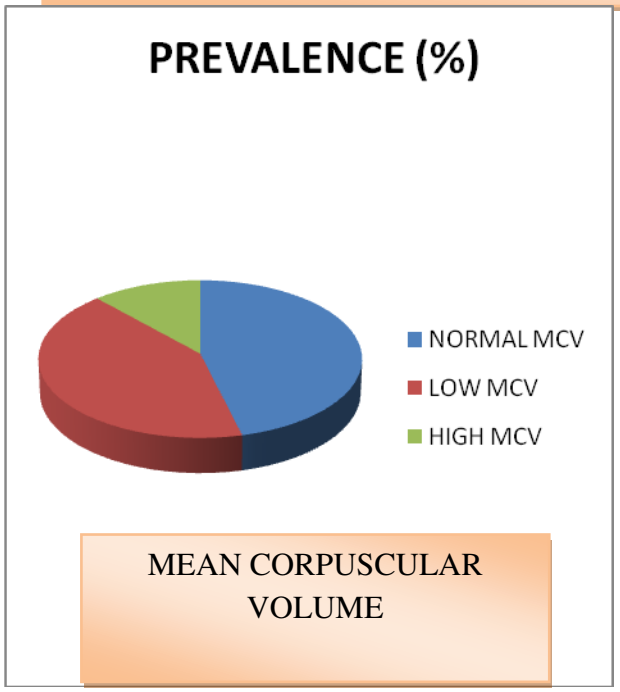


4.10 CLASSIFICATION BASED ON MEAN CORPUSCULAR VOLUME (MCV) AND MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION (MCHC)

Table no. 17 CLASSIFICATION BASED ON MCV AND MCHC

	NO. OF CASES (out of 102)	% PREVALENCE
NORMAL MCV	47	46
LOW MCV	43	42.1
HIGH MCV	12	11.7
NORMAL MCHC	71	69.6
LOW MCHC	21	20.5
HIGH MCHC	9	8.8

Figure no. 13 COMPARISON OF PREVALENCE OF MEAN CORPUSCULAR VOLUME (MCV) AND MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION (MCHC)

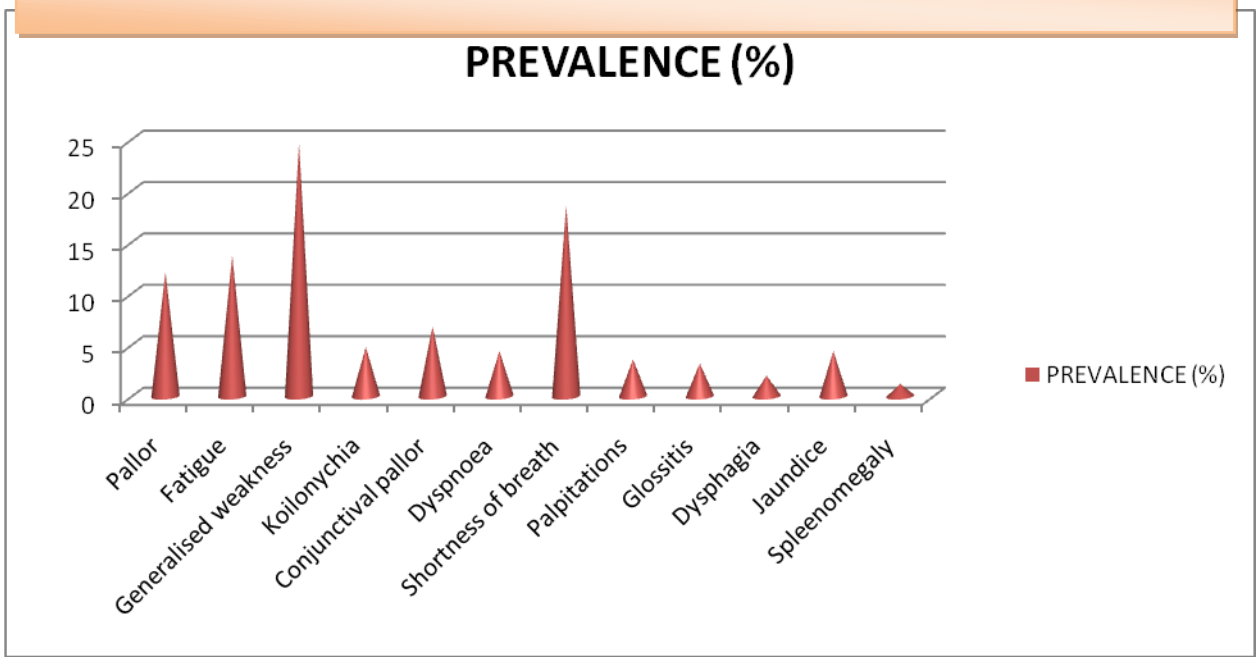


4.11 PREVALENCE OF CLINICAL MANIFESTATIONS OF ANEMIA

Table no. 18 PREVALENCE OF CLINICAL MANIFESTATIONS OF ANEMIA

CLINICAL MANIFESTATIONS	NO. OF CASES (out of 102)	% PREVALENCE
Pallor	30	12.1
Fatigue	34	13.7
Generalised weakness	61	24.6
Koilonychia	12	4.8
Conjunctival pallor	17	6.8
Dyspnoea	11	4.4
Shortness of breath	46	18.6
Palpitations	9	3.6
Glossitis	8	3.2
Dysphagia	5	2
Jaundice	11	4.4
Splenomegally	3	1.2

Figure no. 14 PREVALENCE OF CLINICAL MANIFESTATIONS OF ANEMIA

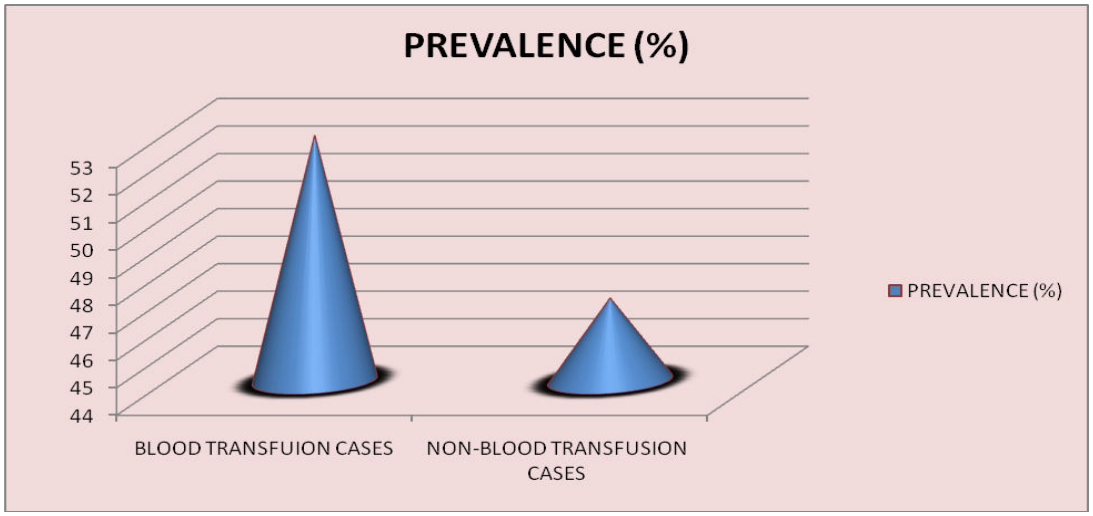


4.12 FREQUENCY OF BLOOD TRANSFUSION IN ANEMIA PATIENTS

Table no. 19 FREQUENCY OF BLOOD TRANSFUSION IN ANEMIA PATIENTS

	NO. OF CASES (out of 102)	% PREVALENCE
Blood Transfusion Cases	54	52.9
Non-blood Transfusion Cases	48	47.1

Figure no. 15 FEQUENCY OF BLOOD TRANSFUSION IN ANEMIA PATIENTS

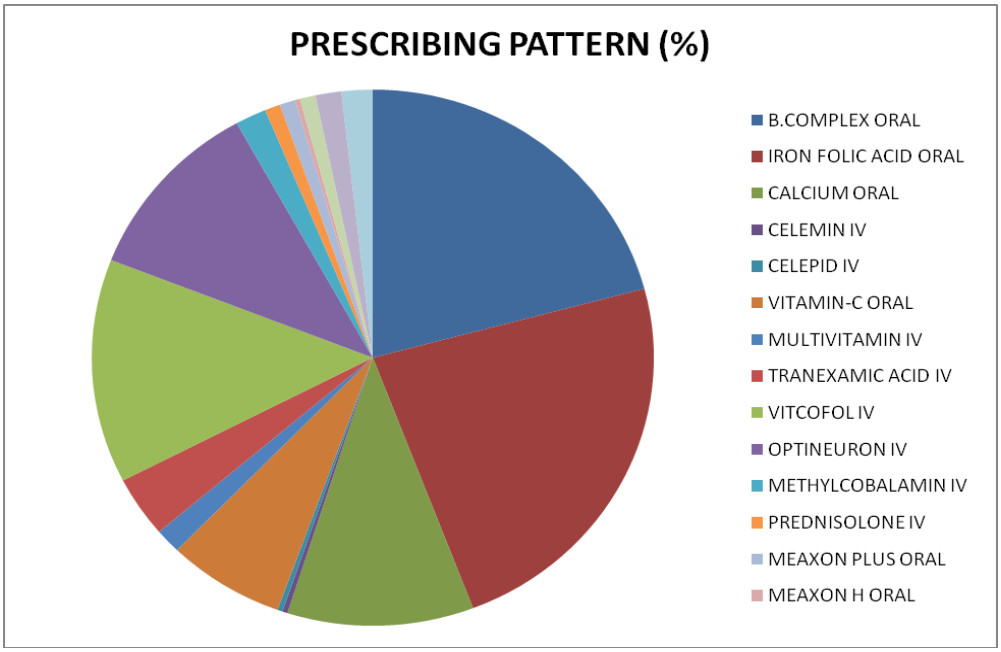


4.13 PRESCRIBING PATTERN IN ANEMIA AT A TERTIARY CARE HOSPITAL

Table no. 20 PRESCRIBING PATTERN IN ANEMIA AT A TERTIARY CARE HOSPITAL

MEDICATIONS	NO. OF CASES (out of 102)	PRESCRIBING PATTERN
B.complex	68	66.6
Iron folic acid	76	74.5
Calcium	35	34.3
Celemin	1	0.9
Celepid	1	0.9
Vitamin-c	22	21.5
Multivitamin	5	4.9
Tranexamic acid	12	11.7
Vitcofol	44	43.1
Optineuron	36	35.2
Methylcobalamin	6	5.8
Methyl Prednisolone	3	2.9
Meaxon plus	3	2.9
Meaxon h	1	0.9
Hepamers	3	2.9
Neurobion plus	5	4.9
Vitamin k	6	5.8
Prednisolone	2	1.9

Figure no. 16 PRESCRIBING PATTERN IN ANEMIA AT A TERTIARY CARE HOSPITAL



5. DISCUSSION:

The following criteria were asserted and evaluated keeping in context prior prospective and observational studies done on anemia –

It was observed that the prevalence of anemia was highest in the age group 21-30 years (29.4%) followed by the age group 31-40 years (23.5%).The prevalence of anemia was found highest in the females of age group 21-30 years 36.2% which was followed by males of age group 31-40 years (31.8%). This study also aimed to investigate the relationship of anemia and body mass index among the subjects in the health institution. Subjects were classified by body mass index (BMI) categories as underweight, normal weight, overweight and obese according to the WHO standard. Anemia was found to prevalent in 73.80% patients with a BMI of 18.5-24.9 and 25.40% of patients were underweight and anemic.

Cappellini et al. (2015), in their study, estabilished that "Anemia is often multifactorial and is not an independent phenomenon. For the classification and diagnosis the hematologic parameters, the underlying pathological mechanism and patient history should be taken into account."

Also, Joosten et al (2015) carried out a study, "Iron deficiency anemia and anemia of chronic disease in geriatric hospitalized patients: How frequent are comorbidities as an additional explanation for the anemia?" To investigate whether comorbidities as an additional explanation for the severity of the anemia are frequent, and might help to explain the anemia severity in older patients with iron deficiency anemia (IDA) and the anemia of chronic disease (ACD)."

And in contrast to these previously carried out studies we developed a relationship between anemia and other presenting clinical manifestations and other pathological conditions (co morbidity). The presence of anemia was highest in hepatic conditions (23.5%) like – Cirrhosis of liver, alcoholism, viral hepatitis etc. The second most common conditions that prevailed along with anemia were gastrointestinal conditions (13.7%) like – Peptic ulcer disease, gastrointestinal bleeding due to ulcers, Crohn's disease and gastritis etc.

As it was already established that anemia was most prevalent in women compared to men, a perspective was formed pertaining to the gynecological and obstetric history in women and the result was that the women with history of menorrhagia were found to be most anemic (39.6%) and women with history of fibroids and PCOS were found to be second most anemic (24.1%).

Many drugs have a tendency of causing blood disorders hence this criteria was also undertaken and- Of all the cases of anemia that were observed, 10.7% cases were found to be drug induced. Of these, 18.1% were induced by Fluoroquinolones, 18.1% by Cephalosporins and 27.2% by Anti-retrovirals. 9% of the drug induced cases were contributed by penicillin, 9% by Anti-TB drugs, 9% by anti-malarial drugs and 9% by non-allopathic drugs.

According to the severity of anemia another classification was obtained and the results were - Of all the anemic patients, 13.7% were moderately anemic, 62.7% were severely anemic and 23.5% were very severely anemic.

The data obtained from the hemogram report of each patient and the results were – Based on morphology, it was found that 22.5% cases were normocytic, 55.8% cases were microcytic, 15% cases were macrocytic, 5.8% were dimorphic and 0.9% was sickle shaped. Of all the cases of anemia, 8.8% were normochromic, 76.4% were hypochromic and 14.7% were hyperchromic.

Anemia as a condition or comorbidity or a symptom itself, has many other clinical manifestations. All the clinical manifestations in the subjects of our study were observed, noted and a standard set of symptoms were found to be present in almost all cases. The results were -generalized weakness (24.6%), shortness of breath (18.6%), fatigue (13.7%), pallor (12.1%), followed by conjunctival pallor (6.8%).

The treatment of anemia is done either by supplementation with drugs or by transfusion of blood in severe cases or cases of trauma and emergency. So, in accordance to this the results concluded that, the treatment of 52.9% cases included blood transfusion whereas the remaining 47% cases did not receive blood transfusion.

Iron folic acid was prescribed in 23.5% of the cases, followed by Vitamin B complex in 21% of the cases. Vitcofol (combination of Nicotinamide, Folic acid and Cyanocobalamine) was prescribed in 13.6% of the cases. Prednisolone was prescribed in all drug-induced anemia cases. Tranexamic acid was prescribed in all anemia cases caused due to bleeding (3.7%).

6. CONCLUSION:

Anemia being neglected in most of the cases until presented with serious symptoms or signs needs to be addressed with full coherence in the population

where anemia might be the leading cause of any disease or disorder. One of the most important aspects when we talk about hemoglobin deficiency is its treatment. Within our study we have crucially observed the different scenarios where treatment requires pharmacotherapy (oral or parenteral) and blood transfusion. Anemia is not only nutritional but also has a relation with the ethnicity of the population groups. Hence, consideration of anemia in a particular group of people of a certain class or ethnicity or gender or economic background is crucial. So, demographic and epidemiological exposure and gain of knowledge pertaining to social factors apart from medical factors serves as an important outcome.

The study has helped us develop a certain level of responsibility towards usage, prescription, dispensing of different dosage forms and doses in the treatment of anemia. Basically, GOOD CLINICAL PRACTICE and rational use of drugs is also one of the outcomes of our study. To be able to interpret the hematological data in itself is a great virtue. Hence, a proper understanding of several types of biochemical and hematological reports too is an outcome of our study. This will enhance our credibility and our wisdom as a CLINICAL PHARMACIST.

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