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AN OBSERVATIONAL STUDY OF ETIOLOGY AND CLINICAL MANIFESTATIONS OF ANAEMIA IN RURAL HOSPITAL

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ABSTRACT

Background: Anemia is very commonly encountered in general clinical practice among all age groups. Although the etiology for such is truly myriad, with a comprehensive history, physical examination, and directed laboratory evaluation, a specific diagnosis can often be arrived at. A comprehensive history, physical examination, and directed laboratory evaluation will help to identify a specific cause for anemia. So, the present study was conducted for clinical and laboratory evaluation of anemia in local population and to find out its etiology for appropriate treatment and preventive strategies.
Methods: The present study was done over one year in a rural hospital. All patients who were anaemic during the study period were enrolled. Patients below 18 years, who did not give consent for the study were excluded. Complete detailed history was taken and general physical examination is done for all patients.
Results: out of 230 patients there was female preponderance with 163 females and 37 males. There was more number of patients in age group 21-30 yrs about 59.among the symptoms fatigue was followed by shortness of breath and weight loss.
Conclusion: The mean age of the study patients was 44.86 ± 15.7 with age range of 21 – 74 years and the gender ratio female: male of the study was 2.43:1. Fatigue or generalized weakness was the most common symptoms

Keywords: anaemia, fatigue, sob, weakness, microcytic

INTRODUCTION

Anemia is a condition in which the number of red blood cells or their oxygen- carrying capacity is insufficient to meet physiologic needs, which vary by age, sex, altitude, smoking, and pregnancy status. Anemia was a public health problem that affects low, middle and high-income countries and has significant adverse health consequences, as well as adverse impacts on social and economic development.[1-3] Although the most reliable indicator of anemia at the population level is blood hemoglobin concentration, measurements of this concentration alone do not determine the cause of anemia. Anemia may result from a number of causes, with the most significant contributor being iron deficiency.[1] Approximately 50% of cases of anemia are considered to be due to iron deficiency, but the proportion probably varies among population groups and in different areas, according to the local conditions.[4,5] Other causes of anemia include other micronutrient deficiencies (e.g. folate, riboflavin, vitamins A and B12), acute and chronic infections (e.g. malaria, cancer, tuber Tulosis and HIV), and inherited or acquired disorders that affect hemoglobin synthesis, red blood cell production or red blood cell survival (e.g. haemoglobinopathies).[6,7] Anemia resulting from iron deficiency adversely affects cognitive and motor development, causes fatigue and low productivity[8] and, when it occurs in pregnancy, may be associated with low birth weight and increased risk of maternal and perinatalmortality.[9,10] Anemia in its severe form, is associated with fatigue, weakness, dizziness and drowsiness. Pregnant women and children are particularly vulnerable. In developing regions,

maternal and neonatal mortality were responsible for 3.0 million deaths in 2013 and are important contributors to overall global mortality. [11,12] It hasbeen further estimated that 90,000 deaths in both sexes and all age groups are due to iron deficiency anemia alone. [13] Any strategy implemented to prevent or treat anemia should be tailored to local conditions, taking into account the specific etiology and prevalence of anemia in a given setting and population group.

AIM AND OBJECTIVES

To study the clinical and laboratory profile of anemia in local population and to find out its etiology. To study the demographic profile and clinical presentation of anemia To study the clinical and laboratory evaluation of anemia. To find out the etiology of anemia. To study the relationship of socio-demographic determinants with various grades of anemia.

Table 1: Classification of Anemia according to the Mean Corpuscular Volume (MCV)

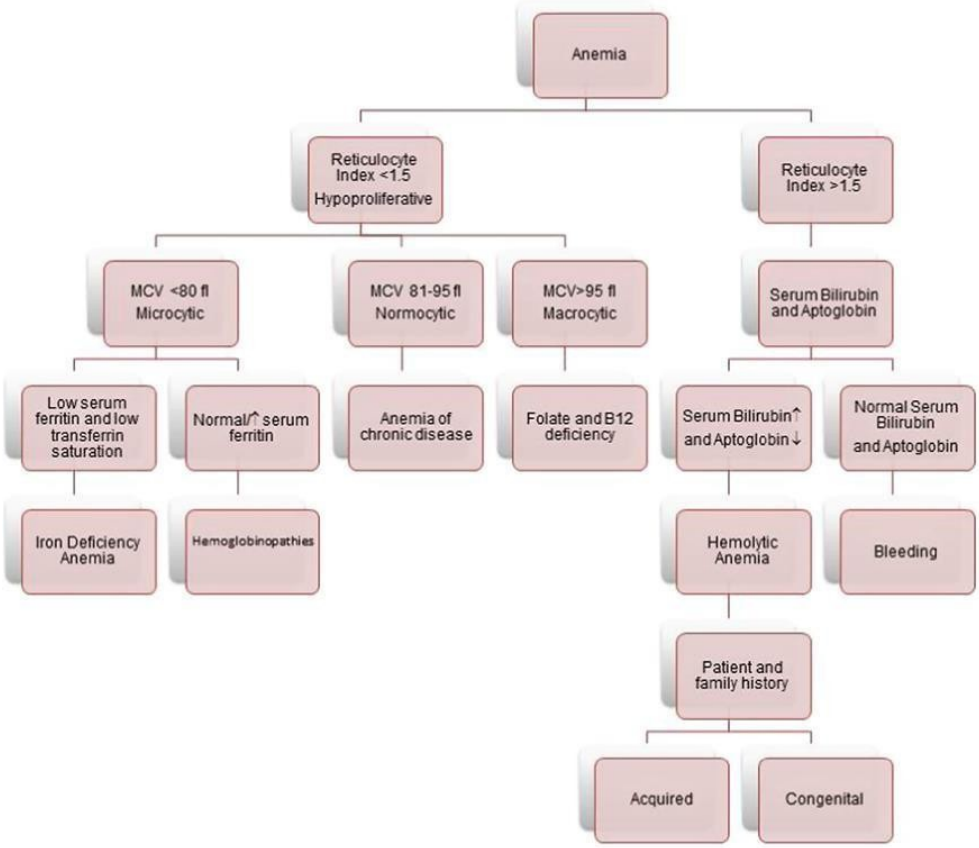
Low MCV (o80 fL)	Normal MCV (80-99 fL)	High MCV (4100 fL)
Thalassemic syndromes*	ACD	Folate or vitamin B_{12} deficiency
IDA	Anemia of CKD	Alcohol
IRIDA	Sickle cell disease	Chronic liver disease
Sideroblastic anemia	Myelodysplasia	MDS
	Combined deficiency (for example iron þ folate)	Reticulocytosis

*β- and α-thalassemia.
ACD: anemia of chronic disease; CKD: chronic kidney disease; IDA: iron deficiency anemia; IRIDA: iron refractory iron deficiency anemia; MDS: myelodysplastic syndromes; HbE, Hb Lepore.

Table 2: Classification of Anemia according to the Reticulocyte Index (RI)

RI o 1% Hypoproliferative Anemias	RI o 1% Maturation Abnormalities	RI 4 1%*
ACD	Vitamin B_{12} deficiency	Immune hemolytic anemias
Anemia CKD	Folate deficiency	Infectious causes of hemolysis
IDA	MDS	Membrane abnormalities
Congenital dyserythropoietic Anemias	Sideroblastic anemia	Mechanical hemolysis
Drugs or toxins		Hemoglobinopathies
Endocrine anemias		Red blood cell enzyme abnormalities
Marrow replacement		

*Appropriate response to increased red blood cell destruction, blood loss, nutritional supplementation.
ACD: anemia of chronic disease; CKD: chronic kidney disease; IDA: iron deficiency anemia; MDS: myelodysplastic syndromes.



A simple work-up to classify and diagnose different forms of anemia based on RIis represented

Fig 1: Diagnostic work-up for anemia

PATHOPHYSIOLOGY

Nutritional anemias

Nutritional anemias result from insufficient bioavailability of haemopoietic nutrients needed to meet the demands of hemoglobin and erythrocyte synthesis.[14,15] As human diets have shifted over time from hunter-gatherer to more cultivated cereal-based diets with more heat exposure during food preparation, there has been a large drop inbioavailable haemopoietic nutrients (iron, vitamin B12, and folic acid) and absorption enhancers such as vitamin C. This situation is compounded by increased intake of otherdietary factors that reduce the bioavailability of non-haem iron, such as polyphenols (eg, tea, coffee, and spices such as cinnamon), phytates (whole grains, legumes), and calcium (dairy products).[16,17] Moreover, absorption of nutrients that promote haemopoiesis can be affected by physiological and pathophysiological factors—for example, Helicobacterpylori is associated with reduced iron stores through several different mechanisms.[18,19]

Iron deficiency

Iron deficiency occurs when the intake of total or bioavailable iron is inadequate tomeet iron demands, or to compensate for increased losses. Iron has an important role in the function of several biological processes, and is an integral part of the hemoglobin molecule wherein Fe^{2+} is bound to the protein protoporphyrin IX complex to form haem. Lack of available iron thereby results in low haem concentrations and hypochromic microcytic anemia. Periods of rapid growth, especially during infancy and pregnancy, result in substantial demands for iron, which accounts for the physiological vulnerability of children and women. Recurrent menstrual loss accounts for roughly 0·48 mg per day,with wide variation depending on menstrual flow.[20] During pregnancy, expansion of the red-cell mass and development and maintenance of the maternal-placental-fetal unit results in a substantial increase in iron requirements that range from 0·8 mg per day in the first trimester to 7·5 mg per day in the third trimester.[21,22] Even in developed countries,anemia during pregnancy is common;[23] however, in developing countries this occurrence is compounded by early onset of childbearing, high number of births, short intervals between births, and poor access to antenatal care and supplementation.[24] The effects of pregnancy and parity on iron status are long-lasting, with differences in iron stores as measured by serum ferritin between nulliparous, uniparous, and multiparous women detectable after menopause.

Folic acid deficiency

Folic acid is required for the synthesis and maturation of erythrocytes, and low serum and erythrocyte concentrations of folate can lead to changes in cell morphology and intramedullary death of erythrocytes and reduced erythrocyte lifespan. Folic acid deficiency contributes to megaloblastic anemia, a condition characterized by cells with large and malformed nuclei resulting from impaired DNA synthesis. During pregnancy, folate demands increase, and women entering pregnancy with poor folate status often develop megaloblastic anemia; furthermore, lactation places additional demands with preferential uptake of folate by mammary glands over maternal requirements.[25]

Vitamin B12 deficiency

Vitamin B12 is synthesized only by microorganisms, and its primary source is from ingestion of animal products. Absorption of vitamin B12 involves a complex process by which gastric enzymes and acid facilitate its release from food sources, before being bound by an intrinsic factor secreted by gastric parietal cells, followed by uptake in the distal ileum. Vitamin B12 deficiency can result in a megaloblastic macrocytic anemia, which is more common in severe vitamin B12 deficiency.[26]

Soil-transmitted helminths

Infectious diseases can contribute to anemia through impaired absorption and metabolism of iron and other micronutrients or increased nutrient losses. Of soil-transmitted helminth infections, hookworms (*Necator americanus and Ancylostoma duodenale*) are the major cause of anemia, and are commonly found in sub-Saharan Africa and southeast Asia, with an estimated 576–740 million infections.[27,28] In the tropics and subtropics, where ecological conditions allow larval development, hookworm infections are overdispersed or highly aggregated in areas of poverty, where poor water, sanitation, and infrastructure result in endemicity, often concentrated in small populations within these areas. Co-infection with several species is common.[29]

Hookworms can cause chronic blood loss, with the severity dependent on the intensity of infection, the species of hookworm (*A.duodenale* is more invasive than *N. americanus*), host iron reserves, and other factors such as age and comorbidity. Systematic review of 12 studies of deworming during pregnancy showed that women withlight hookworm infection had a standardised mean difference of hemoglobin that was 0·24lower (95% CI –0·36 to –0·13) than in those with no hookworm. Adult parasites invade and attach to the mucosa and submucosa of the small intestine, causing mechanical andchemical damage to capillaries and arterioles. By secreting anticlotting agents, the parasite ingests the flow of extravasated blood, with some recycling of lysed erythrocytes and blood. Hookworm disease results when chronic blood loss exceeds iron reserves,inducing iron-deficiency anemia.[30,31]

HIV/AIDS

Anemia is the most common haematological complication associated with HIV infection, and is a marker of disease progression and survival. The burden of HIV/AIDS-related anemia falls predominantly on sub-Saharan Africa, where women and children are most at risk. The mechanism of HIV/AIDS-related anemia is multifactorial, resulting from HIV infection and the induced anemia of chronic disease, AIDS-related illnesses, and antiretroviral treatment. Further research is needed to assess the burden and effect of strategies for HIV/AIDS prevention and treatment on anemia.[32,33]

Leukemias

Anemia occurs in leukemia because of marrow infiltration by leukemic cells resulting in bleeding due to thrombocytopenia or erythroid failure or in part due to decreased survival of erythrocytes. Red cells in these anemias are usually normocytic. Autoimmune phenomenon is a well-known complication of lymphoproliferative diseases and in particular of CLL. Three autoimmune hematological conditions frequently associated with CLL are, Auto immune hemolytic anemia, ITP and pure red cell aplasia. Of these, Auto immune hemolytic anemia is the most common. Elderly males with active CLL are more prone to these.[34]

Anemias of Renal Disease

In renal insufficiency, normochromic anemia develops which is very similar to anemias of chronic disease (ACD). In this condition, there is a deficiency of erythropoietin production. Metabolic injury to erythrocytes is the postulated mechanism, and there is evidence that the administration of erythropoietin, when the anemia is severe, can partially improve the condition. Improvement is usually not complete, as non-specific substances accumulate in renal insufficiency that suppress hematopoiesis. In addition, specific inhibitors may accumulate in uremia. However, in patients on hemodialysis, erythropoietin is very effective in correcting the anemia, as these inhibitors are removed.It is important to correct any concomitant nutritional deficiencies such as those involving iron or folic acid, which are common in renal diseases. In rare cases, blood transfusions may be necessary to protect the patient's cardiovascular status. The smear is normocytic normochromic and burr cells are sometimes prominent.[35,36]

Anemias of Liver Disease

Hepatocellular disease is responsible for a host of anemias, especially when accompanied by alcoholism and poor diet. It accounts for many of the macrocytic anemias. Folate deficiency, marrow suppression, hypersplenism, bleeding, and alteration of the red cell membrane by the bile salts contribute to anemia. The smear shows considerable poikilocytosis with spiculated red cells and some macrocytes. If folate deficiency occurs, a megaloblastoid picture is superimposed.[37] In liver disease, however, the serum iron is increased and there is an increase in transferrin saturation. Serum ferritin is also increased and often reflects the total body ironoverload.

Anemias of Endocrine Disorders

Endocrine disorders that commonly produce anemia are hypothyroidism, hypopituitarism, and adrenal insufficiency. In all these cases, the anemia is normocytic and normochromic but, it may sometimes be macrocytic.

Anemias of Collagen Vascular Diseases

Anemias of chronic diseases appear in collagen vascular diseases such as Rheumatoid arthritis, Polyarteritis, Dermatomyositis, Systemic lupus erythematosus, Polymyalgia rheumatica and Temporal arteritis. The latter is essentially a disease of geriatric patients.

"Unexplained anemia" (UA) of elderly people

A mild, normochromic normocytic anemia with a hemoglobin concentration usuallybetween 11 and 12 g/dL has been reported in people over the age of 70 years. This anemia cannot be accounted for by any underlying disease or deficiency, and the bone marrow does not contain ringed sideroblasts. This unexplained anemia is said to accountfor over 30 percent of the anemias in this age group. It is associated with low neutrophil, lymphocyte, and platelet

counts, and there is an increased red blood cell 2, 3 DPG level implying that, this condition is not merely a normal age-related variant. The significance of this type of anemia is presently unknown, but it could probably be myelodysplastic syndrome.[38]

SYSTEMIC MANIFESTATIONS OF ANEMIA

Skin

Pallor can be the most evident sign of anemia, but many factors other than hemoglobin concentration affect the color of the skin. These factors include the degree ofdilatation of the peripheral blood vessels, the degree and nature of pigmentation, and the nature and fluid content of the subcutaneous tissues. Other changes sometimes observed in the anemic patients include loss of normal skin elasticity along with brittle or broken nails.[39]

Cardiovascular System

In many patients, respiratory and circulatory symptoms are noticeable only after exertion or excitement. However, when anemia is sufficiently severe, dyspnea and awareness of vigorous or rapid heart action may be noted even at rest. When anemia develops rapidly, shortness of breath, tachycardia, dizziness or faintness (particularly upon rising from a sitting or recumbent posture) and extreme fatigue are prominent. In chronic anemias, only moderate dyspnea or palpitation may occur, but in some patients,congestive heart failure, angina pectoris, or intermittent claudication can be the presenting manifestations. In severe and prolonged anemia, hyperdynamic cardiac failure may supervene, along with salt and water retention, edema and even ascites. Eventually, left ventricular hypertrophy may occur.

Gastro-intestinal system

Disturbances of the gastro-intestinal tract (GIT) are rather common in anemia. Some of them, such as duodenal ulcer, carcinoma of the gastro-intestinal tract, glossitis and atrophy of the tongue papillae as seen in pernicious anemia may be the manifestations of the disorder underlying the anemia. Reduced perfusion of the intestinalmucous membranes may in turn cause nausea, anorexia and malabsorption.

Central Nervous System

Headache, vertigo, tinnitus, faintness, scotoma, lack of mental concentration, drowsiness, restlessness and weakness are common symptoms of severe anemia. Paraesthesia is common in pernicious anemia and may be associated with other symptoms and signs of peripheral neuropathy.

Renal System

In mild to moderate anemia, the renal function is not affected. However, in severe chronic anemia, renal blood flow may decrease and result in impaired renal function. The resulting fluid retention causes renal edema, which may in turn aggravate cardiacinsufficiency. Severely impaired renal function resulting in deficient erythropoietin production itself may cause anemia.[40]

Respiratory System

One of the compensatory mechanisms in anemia is, a rise of the respiratory rate in an attempt to increase blood oxygenation. The patient experiences shortness of breath, a symptom which most often develops in a state of exertion, when reduced blood oxygen meets increased oxygen requirements of the exercising muscles.

Immune System

In patients with iron deficiency anemia, impairment of the immune system has been observed based on a decrease in B and T cell function. Additionally, the decreasedperfusion of skin and mucous membranes in anemia may contribute to a higher rate of infections, as it has been shown that anatomic compartments with poor perfusion are prone to infections.[41]

Ocular Manifestations

Certain ophthalmic findings have been observed in anemic patients. About 20% of such patients have flame-shaped hemorrhages, hard exudates, cotton wool spots and venous tortuousness affecting the retina. The Retinal hemorrhages occur even in the absence of thrombocytopenia.[42]

Genito-urinary System

Slight proteinuria is not uncommon in patients with significant anemia. Microscopichematuria is also seen. These genitourinary symptoms arise frequently in patients with anemia and may partly result from impaired secretion of sexual hormones. Menorrhagia, irregular menstrual cycles and amenorrhea are among the most common symptoms reported by women. Males may suffer from impotence.[43]

MATERIAL AND METHODS

STUDY DESIGN: Hospital based Cross-Sectional study
STUDY PERIOD: The study was carried out over a period of one year
STUDY PLACE: The present study will be conducted at Department of General Medicine, in rural hospital
STUDY POPULATION: Patients with anemia who came to OPD in General Medicine. In rural Hospital.
SAMPLE SIZE: Based on the previous study on anemia among the general population, theprevalence of anemia was 44.98%.
So, for the present study the estimated prevalence of anemia was taken at 45%Estimated prevalence (p) = 45%
Confidence interval (CI) = 95%
Relative precision (d) = 15% of p = 15 × 45 /100 = 6.75
Using the standard formula for proportion = $[(Z\alpha/2)^2 \times p \times q] / d^2$ Sample size (n)= $(Z\alpha/2)^2 \times p \times (1\text{-}p) / d^2$ = (1.96×1.96)×45×55 / 6.75 x 6.75 = 9504 / 45.5 = 208.8
Taking additional 10% cases, the final sample size for the present study wasrounded to 230.

INCLUSION CRITERIA

1. Age more than 18 years.
2. Patients who were diagnosed anemic.
3. Patients who gave consent for the study.

EXCLUSION CRITERIA

1. Patients with acute blood loss due to trauma, surgery.
2. Pregnant women.
3. Patients who didn't give consent for the study.

ETHICAL CLEARENCE AND CONSENT

Institutional ethical committee clearance was obtained from ethical committee prior to the start of study. A written and

informed consent was taken from patients who were participated inthe present study.

METHOD OF COLLECTION OF DATA

Complete detailed history was taken and general physical examination is done for all patients. Socioeconomic status classification was done according to modified BG Prasad classification 2018 (annexure). Hemoglobin level, Serum iron, Serum ferritin, Transferrin ratio and TIBC was measured. Biochemical investigations – Vitamin B12 levels, Thyroid profile, Liver Function Test, Renal Function Tests was also carried out. Complete stool examination is done for all anemia diagnosed patients.

Hemoglobin was estimated by Sahli's acid hematin method with Sahli's Hellige hemoglobinometer. Total red cell count was done using Hayem's diluents and cells werecounted on the improved Neubauer's chamber. Packed cell volume was done by centrifuging exalate blood drawn from a vein in a Wintrobe haematocrit tube at 3000RPMfor 30minutes.

Reticulocyte count was done by 0.3% alcoholic solution of Brilliant cresyl blue by the technique described by Wintrobe. Reticulocyte index was calculated by multiplying reticulocyte count with ratio of patient PCV with normal PCV and that was multiplied by the ratio of one by maturation time.

Reticulocyte index greater than two reflect adequate marrow function while less than two reflect hypo plastic marrow. Mean corpuscular volume, mean corpuscular hemoglobin, and mean corpuscular hemoglobin concentration was also calculated. Total leucocyte count was done by improved Neubauer counting chamber. Normal value is 5000-10000 cells per cumm of blood. Differential leucocyte count was done by preparing a thin and even film and staining with Leishman's stain. Special notes were made regarding the percentage of five or more lobed neutrophils and malaria parasites.

Platelet count was done by standard method described. ESR was done by using Westergren's method. Red cell osmotic fragility test was based on creeds technique. Peripheral blood film was prepared by prickling the left middle or ring finger tip.

It was stained using Leishman's stain. The air dried film was covered withLeishman's stain (0.15gm% in 100ml of methyl alcohol) for a minute. An equal volume ofbuttered water at pH 6.8 was added. This was allowed to remain for 7-10 minutes and washed with buffered water thoroughly. This was dried and examined under light microscopy using both low power and oil immersion lens. Bone marrow aspiration cytology was done using standard methods. Stool was examined microscopically for 4-5 consecutive days. Benzedrine test was used for detecting occult blood in stool. Complete urine examination was also done. Serum proteinand A/G ratio was done. SGOT/SGPT was done to rule out liver disease. Blood urea andserum creatinine was done to rule out renal disease. Chest X ray PA view was done routinely in all cases.

DATA ANALYSIS: Data collected was entered into MS-Excel 2013 spreadsheet. The collected data was analyzed using IBM statistical package for social sciences (IBM SPSS) version 23 software (trail version).

Statistical Tests

1. Continuous variables was reported as mean ± standard deviation (SD) whilecategorical variables was expressed as absolute values and percentages.
2. Microsoft Excel 2013 was used for generating charts and diagrams.
3. Bivariate analysis was performed using chi-square test and p-value less than 0.05 was considered statistically significant.

RESULTS

Table 3: Distribution of Patients According to Age

AGE GROUP	NUMBER	PERCENTAGE (%)
21 – 30 Yrs	59	25.7
31 – 40 Yrs	35	15.2
41 – 50 Yrs	53	23.0
51 – 60 Yrs	38	16.5
> 60 Yrs	45	19.6
Total	230	100.0

AGE	Years
Mean	44.86
SD	15.7
Range	21 – 74

The mean age of the study patients was 44.86 ± 15.7 with age range of 21 – 74 years. 25.7% of the study population belong to 21 – 30 Yrs followed by 23% in 41 – 50 Yrs. 19.6% of the study population were above 60 years.

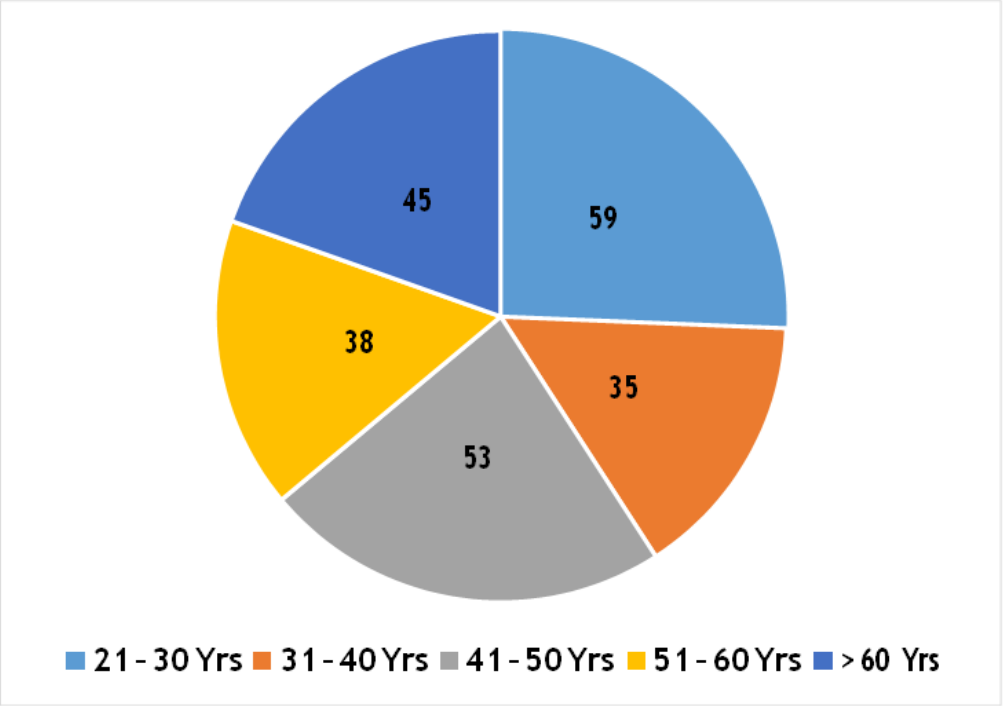
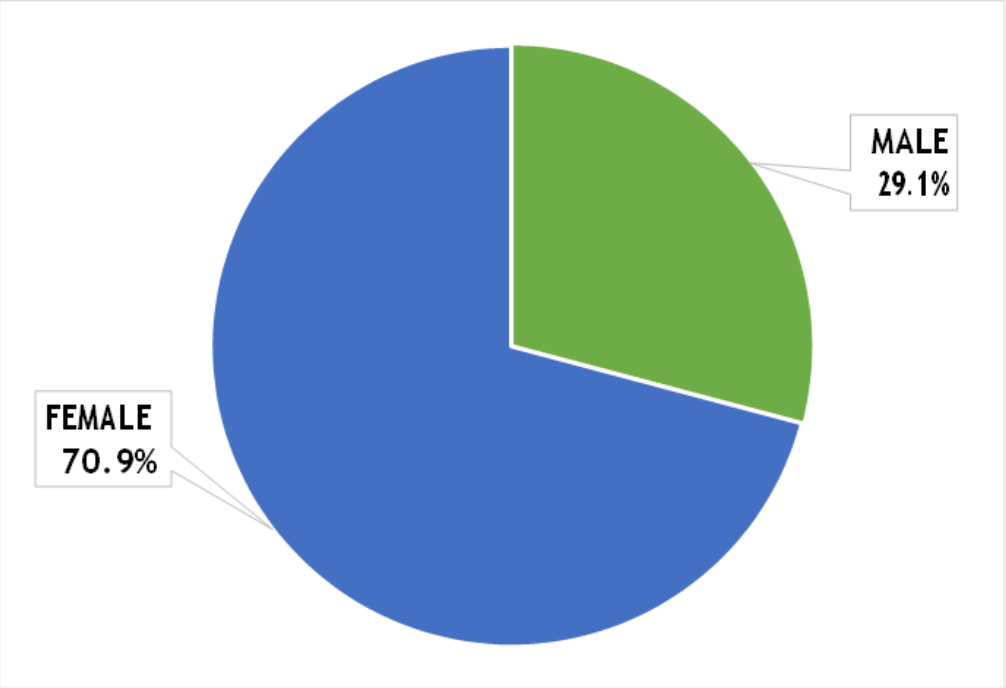
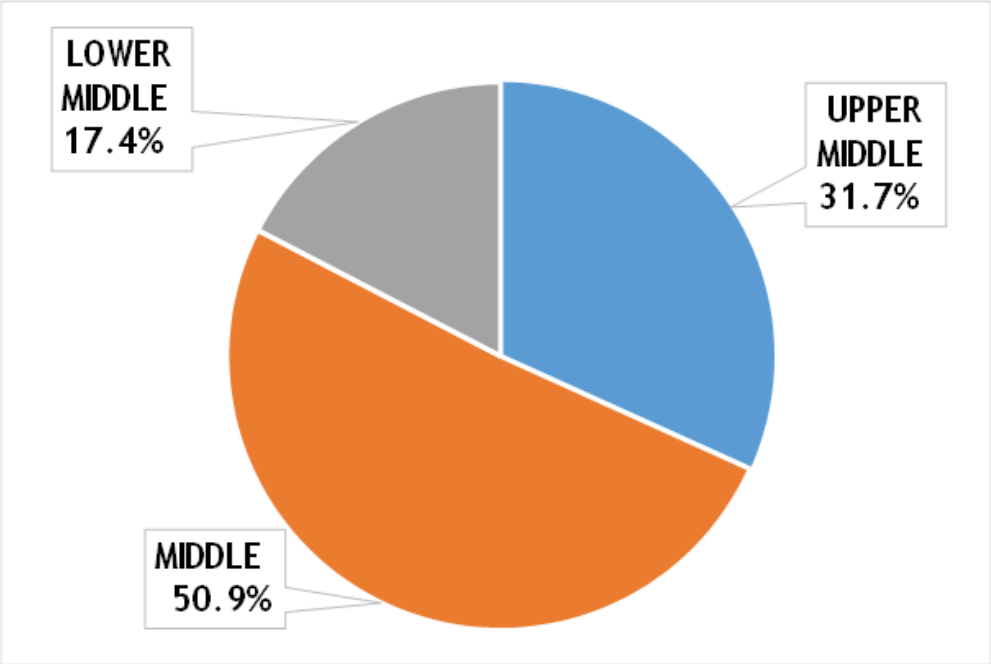


Fig 2: Age Distribution of Patients



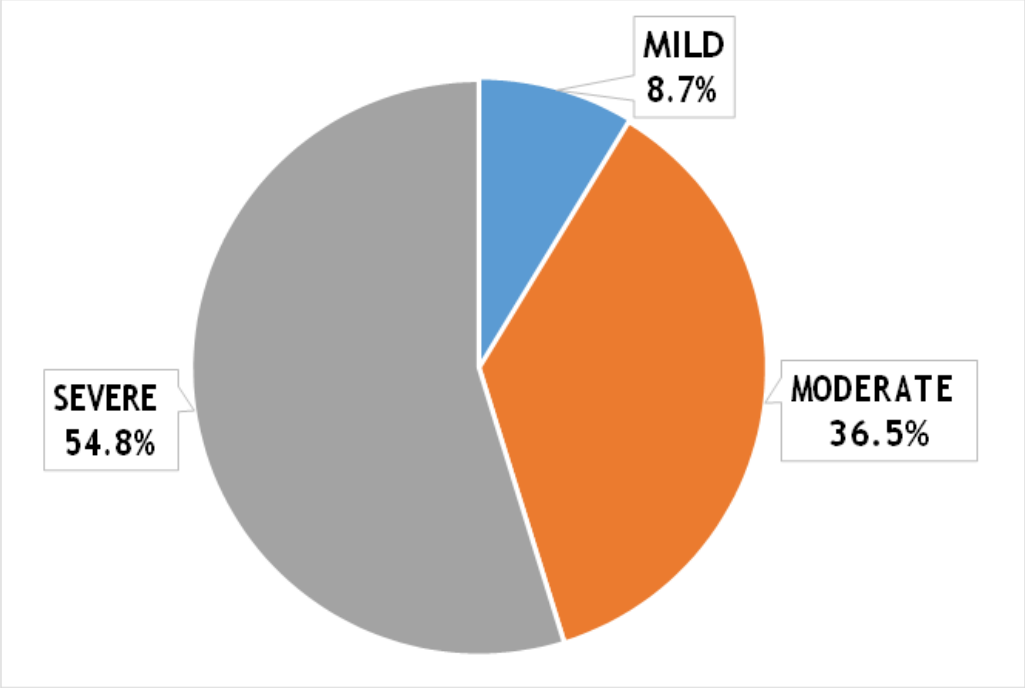
Among the 230 study subjects majority were female 70.9% and 29.1% were male. The gender ratio female: male of the study was 2.43:1.

Fig 3: Gender Distribution of Patients



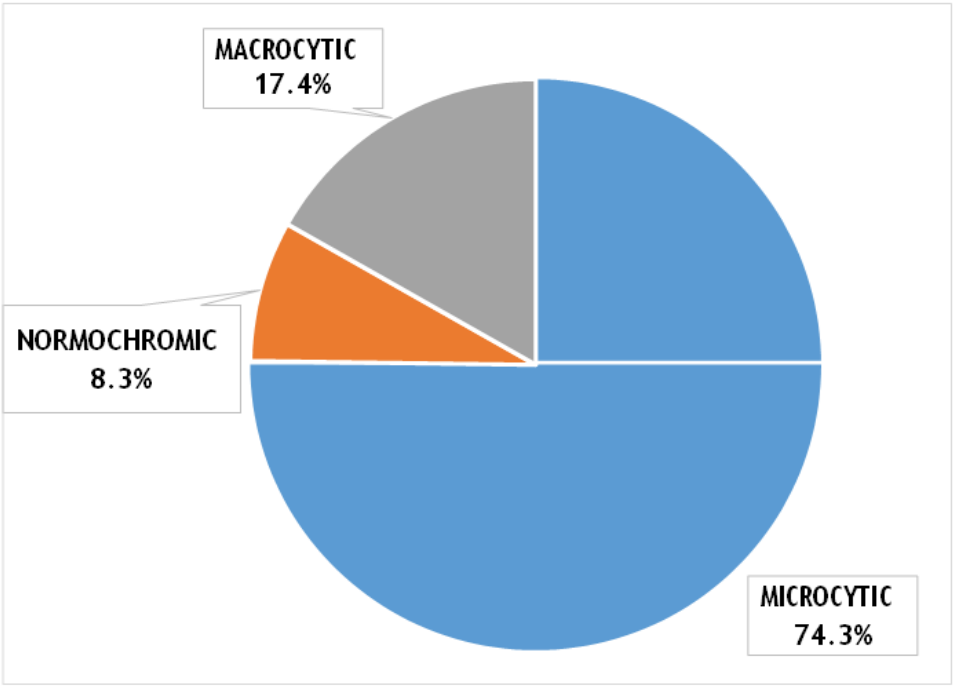
According to modified BG Prasad socioeconomic classification 2018, majority 50.9% of the study population belong to middle class. In the rest 31.7% belong to upper middle and 17.4% belong to lower middle class.

Fig 4: SES Classification Distribution of Patients



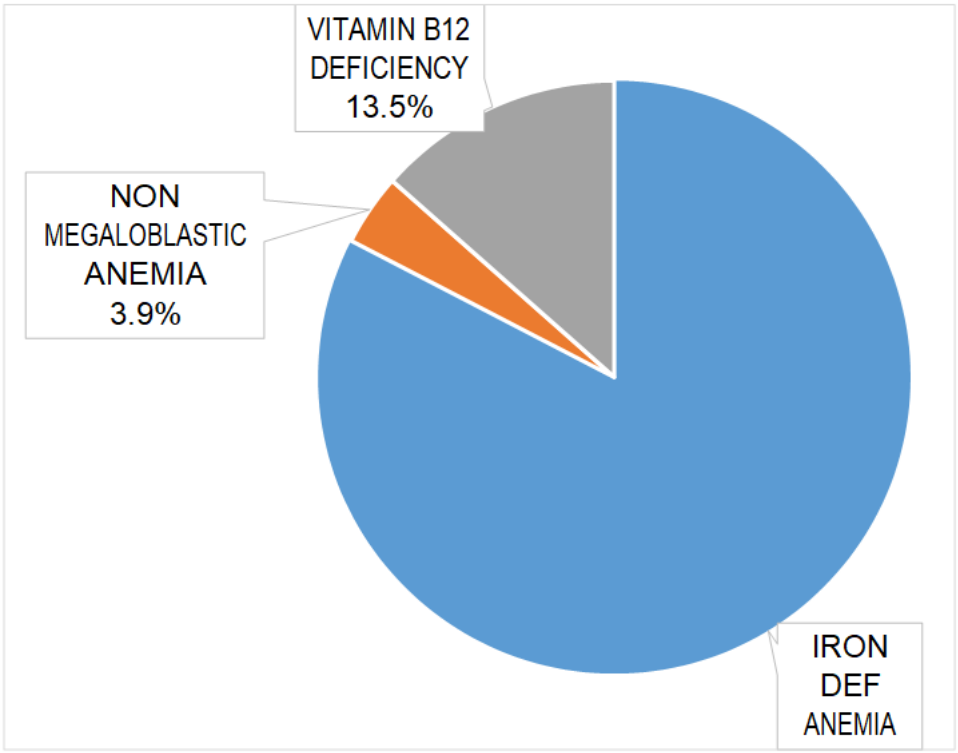
Among the 230 study population 54.8% had severe, 36.5% had moderate and8.7% had mild degree of anemia.

Fig 5: Distribution of Patients According to Hemoglobin Level



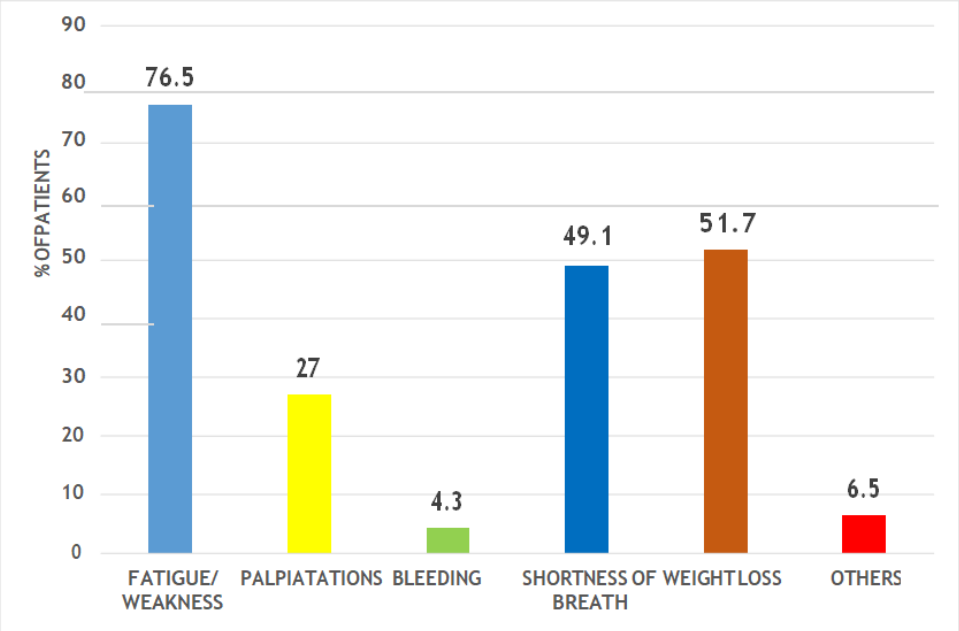
Out of 230 patients, 74.3% had microcytic anemia. 17.4% had macrocytic and8.3% had normocytic anemia.

Fig 6: Distribution of Patients According to Morphological Classification



Among the 230 study population 82.6% had iron deficiency anemia, 13.5% hadvitamin B12 deficiency anemia and 3.9% had non-megaloblastic anemia.

Fig 7: Distribution of Patients According to Diagnosis



Fatigue or generalized weakness was the most common symptom 76.5% in the patients. Other common symptoms at presentation was 51.7% weight loss, 49.1% had shortness of breath.

Fig 8: Distribution of Patients According to Presenting Symptoms

DISCUSSIONS

The present study was a hospital based cross-sectional study which was done among patients with anemia who came to OPD in General Medicine department in rural hospital.

The study was conducted among 230 patients for a period of one year with an aim to study the clinical and laboratory profile of anemia in local population and to find out its etiology.

In the present study among 230 anemia cases 54.8% had severe, 36.5% had moderate and 8.7% had mild degree of anemia. As the present study was a hospital based study and conducted among anemia patients only and people having mild anemiawithout other comorbidities rarely seek medical suggestions and high percentage of severe anemia cases may be due to the fact that most of the patients present to the hospital with symptoms which present in moderate to severe anemia cases. The mean Hb of the study population in the present study was 7.5 ± 1.79 g/dL.

Anil Raina et al.,[39] had reported that among the study population 21.4% had severe anemia, 72.6% had moderate anemia and 6% had mild anemia in which there was low severity when compared to the present study and this discordance maybe due to different study settings. Nidhi Gupta et al.,[55] had reported that 22.8% had severe, 50%had moderate and 27.1% had mild degree of anemia.

In the present study the mean age of the study patients was 44.86 ± 15.7 with age range of 21 – 74 years. Majority of the study population 25.7% belong to 21 – 30 Yrs followed by 23% in 41 – 50 Yrs. 19.6% of the study population were elderly. There wasno statistical significant association found between age and degree of anemia (p > 0.05).

The mean age of patients in the Joshi R et al.,[40] was 52.61 ± 15.44 years which was slightly high when compared to the present study. They also reported that anemia was more common in the age group 20-39 years which was similar to the present study. Lamsal K et al.,[41] reported that the commonest age group was 40-49 years, followed by 50-59 years and this may be due to the cases which are commonly referred to their center had alcoholic liver disease. Milind Chandurkar et al.,[42] had reported that 64% of cases were >40 yrs.

The mean age of the Syed Zulfiquar Ali Shah et al.,[43] was 48.87±7.85 yrs which was similar to the present study and majority belong to 20 – 29 years. Maximum numberof cases were seen in age group of 21-30 years (20.6%) followed by age group 51-60 years (20.3%) as reported by Pandey A et al.,[44] with mean age of 43.65±17.25 years which was similar to the present study.

Table 4: Comparing mean age with previous studies

STUDY	MEAN AGE (Years)
Present study	44.86 ± 15.7
Joshi R et al.,[45]	52.61 ± 15.44
Syed Zulfiquar Ali Shah et al.,[46]	48.87 ± 7.85
Pandey A et al.,[47]	43.65 ± 17.25
Nidhi Gupta et al.,[48]	45 ± 10.7

Among the 230 study subjects majority were female 70.9% and 29.1% were male.The gender ratio female: male of the study was 2.43:1. Among severe anemia cases, 76.2% were female and 23.8% were male and no statistical significant association found between gender and degree of anemia. (p > 0.05).

In the study conducted by Joshi P et al.,[49] also reported that anemia was more common in females and in all age groups, outnumbered males which was similar to the present study. In the study conducted by Ather Akhtar Pasha et al.,[50] 38% of the study population were male and 62% were female and the gender ratio was similar to the present study.

Milind Chandurkar et al.,[51] had reported that 46% patients of anemia were male and 54% were female in which the gender ratio was low when compared to the present study. However Syed Zulfiquar Ali Shah et al.,[52] and Chauhan R et al.,[53] reported male predominance over female which was in contrast to the present study. Gupta et al.,[55] had reported that 23.3% had koilonychia which was similar to the present study

SUMMARY

In the present study among 230 anemia cases 54.8% had severe, 36.5% hadmoderate and 8.7% had mild degree of anemia. The mean Hb of the study population in the present study was 7.5 ± 1.79 g/dL. The mean age of the study patients was 44.86 ± 15.7 with age range of 21 – 74years and the gender ratio female: male of the study was 2.43:1. Out of 230 patients, 74.3% had microcytic anemia. 17.4% had macrocytic and8.3% had normocytic anemia. In 52.6% patient's nutritional factors was the cause for anemia. In the rest of the study population 26.1% anemia of chronic disease, 8.3% hook worm infestation were the other common causes. Fatigue or generalized weakness was the most common symptoms. In the present study, the mean MCV was 80.14 ± 17.65 fl, the mean MCH was 28.51 ± 6.75 pg and the mean MCHC was 31.47 ± 5.13 g/dL. There was no statistical significant association found between age, gender, hook worm infection and degree of anemia. There was statistical significant association found between socioeconomic statusand degree of anemia ($p < 0.05$) with majority of the patients belong to middle class. There was also a statistical significant association found between nutritionalfactors and degree of anemia ($p < 0.05$) with high prevalence of severe anemia innutritional deficiency. There was a statistical significant association found between hemoglobin indices and degree of anemia. ($p < 0.05$)

CONCLUSION

In majority of patients cause of anemia can be diagnosed by thorough investigations. In India, nutritional deficiency especially iron deficiency and less commonly, vitaminB12 and folic acid deficiency are still the leading causes of anemia. Most of these patients are amenable to nutritional therapy, infrequently require transfusion and have low mortality.

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