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THE STUDY OF THE INCIDENCE OF IRON DEFICIENCY ANEMIA IN ELDERLY SUBJECTS OVER 65 YEARS IN RELATION TO THEIR DIETARY HABITS AMONG OUTPATIENT POPULATION IN A TERTIARY CARE HOSPITAL IN KOLKATA INDIA.

Preventive & Social Medicine

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ABSTRACT

Introduction: Anemia is one of the leading public health problems of the developing countries like India. Though it is the commonest haematological abnormality in old group of population, should never be thought as normal aging process. Inspite of the existence of various medical causes that lead to iron deficiency anemia, it has been observed that the most common dietary deficiency worldwide is iron, affecting half a billion of persons, the women and children being affected most and not the least the geriatric group of population. **Aims:** The main aim of this study is to find out the anemic individuals above 65yrs old irrespective of sex and health problems coming to the tertiary care outpatients department for check-up. The definite cause of the anemia is detected and diagnosed with particular relation with their dietary habits vegetarian and non-vegetarian. **Materials and Method:** This Study was conducted from August 2006 and July 2008 at rheumatology clinic of a tertiary care hospital.Total 100 patients were included in this study. **Result:** We observed that, the mean TLC 4000-10000/ Cumm was lower in Hemorrhagic [9150.0000± 3730.9910] compared to Acute on chronic disease [11798.1818± 5934.7218] but this was statistically significant (p=0.0012). **Conclusion:** Our study showed that, RBC Total (3, 8-5, 5) 10^6/ ml was more in Hypothyroid compared to Acute on chronic disease but this was not statistically significant.

KEYWORDS

Anemia, absorption, transferrin, ferritin, haemoglobin.

INTRODUCTION

Anemia is one of the leading public health problems of the developing countries like India. Though it is the commonest haematological abnormality in old group of population, should never be thought as normal aging process. Inspite of the existence of various medical causes that lead to iron deficiency anemia, it has been observed that the most common dietary deficiency worldwide is iron, affecting half a billion of persons, the women and children being affected most and not the least the geriatric group of population. In a country like India where different caste and creed, religion and culture co-exist with varied socio-economic status, wide dietary variation is expected. India, like many other Nation, is aging very fast. The aged population is particularly vulnerable to nutritional deficiencies due to various medical and social factors. The present study looks into the various causes of iron deficiency anemia with particular reference to nutritional factors and measures its incidence among all causes of anemia.

Anemia is defined as a state of decreased oxygen carrying capacity of the blood wherein the hemoglobin is less than 13 g/ml in men and less than12 g/ml in non pregnant women by WHO reference standards [1].The demographic transition with ageing of population is a global phenomenon and in recent years there has been an increasing international awareness of health issues relating to aging population[2,3]. According to a 2004 report of the United States National Health and Nutritional Examination Survey (NHANES) III, 10 % of Americans 65 years and older are anemic, rising to a 25 % of men and 20 % of women 85 years and older [4].The prevalence of anemia among elderly Indians , as reported in Indian cross sectional studies is between 6 and 30 % among men and between 10 and 20 % among women [5].Thus anemia represents an emerging global health problem producing a negative impact in the quality of life among the elderly and requiring greater allocation of health resources[6].

The Tertiary care Hospital wherefrom the study is done is situated in the southern part of Kolkata , West Bengal, India; which is inhabited by a wide range of people with upper, middle, and lower classes of socio-economic variety. As the study involves iron deficiency anemia; importance of iron, its sources, absorption, transport, storage, excretion, functions and the deficiency states are narrated below.

Iron is of great importance in human nutrition. It is an essential element of human beings as it is responsible to various biochemical reactions. The adult human body contains between (3-4) grams of Iron, of which about 60-70 percent is present in the blood as hemoglobin as circulating Iron needed to carry O2 through the blood and rest (1-1.5) gm as storage Iron; out of which some part is devoted to cellular processes like storing O2 (myoglobin), or performing energy-producing Redox reactions (cytochromes) and some circulates through the plasma, bound to transironrrin. Each gram of hemoglobin contains about 3.34 mg of Iron.

Those who are detected to be anemic, their particular dietary habits that is a normal daily dietary intake of foods to be noted and the total dietary iron intake is to be calculated.

As several other factors are also responsible for iron absorption and improvement of haemoglobin status daily intake of protein, folic acid, vit B12, vit-C also calculated. The factors which inhibit iron absorption like fibres, phytates, are also calculated.

Then the amount of iron absorbed is to be calculated as per protocol no.[7]The various types of food and medicines that causes decrease and increase iron absorption has to be kept in mind and calculated. The amount of iron that is absorbed varies from 1% to 5% from the regular diet in a normal individual, as studied using a radio-active isotope. Because of the multiplicity of factors affecting iron absorption, it is not possible to make a valid estimate of iron absorption from a meal.

The absorption varies with the food, being higher with a rice based diet and lower with millets. Germination and baking increase iron absorption, while it is reduced with tannins, phytates, caffines, oxalates and fibres.

Iron in meat and liver is better absorbed than that in eggs and leafy vegetables; with animal foods the mean iron absorption ranges from7% to 22%, and from fish about11% and 13% from liver. Animal protein in beef, pork, chicken or fish increases absorption of non haem iron from vegetable sources.

On rice based diet iron absorption increases with the addition of 4 gms of fish. Tea and coffee tannate has iron hence it interferes by decreasing iron absorption. Gastric acidity maintains the solubility of inorganic iron, which aids the formation of small molecules with ascorbic acid, citrate, fructose and amino acids. Citrate and ascorbate, being soluble, are more easily absorbed, while tannate, phytate and phosphate are not readily absorbed. Vitamin C reduces ferric iron to ferrous iron which remains soluble even at neutral ph and is better absorbed.

Even when the diet is poor in iron, vitamin C supplement with each meal enhances iron absorption Calcium inhibits iron absorption and antacids and PPI-s also inhibits iron absorption. Taking all the criteria of different mode of factors of iron absorption in concerned with various types of foods the criteria for absorbed amount of iron is formulated in many ways.

AIMS AND OBJECTIVES

The main aim of this study is to find out the anemic individuals above 65yrs old irrespective of sex and health problems coming to the hospital Outpatients Department for check-up. The definite cause of

the anemia is detected and diagnosed with particular relation with their dietary habits vegetarian and non-vegetarian.

Review Of Literature

Nutrient Deficient Anemias:

Nutrient deficient anemias are significant cause of anemia in the elderly. Iron, folate or vitamin B-12 deficiencies maybe seen, but mostly a combination of the three occurs [8].

Anemia Due To Iron Deficiency

Iron Metabolism

Iron is derived from diet either as heme iron contained in animal products or as non-heme iron in plant products like cereals, pulses and vegetables. Heme iron is absorbed better when compared to non heme iron. Iron in the body can be divided into storage and functional compartments. About 80% of the iron is in functional form as in hemoglobin, myoglobin and certain enzymes such as catalase and cytochromes, the remaining iron is stored in the body in the form of ferritin and hemosiderin, which is a permanent non reusable form.

Iron Balance

Dietary iron is absorbed in proximal duodenum. Luminal non heme iron is mostly in ferric state and must first be reduced to ferrous form by ferrireductases like b cytochromes[9]. Ferrous iron is then transferred across apical membrane by divalent metal transporter 1 (DMT 1). Heme iron is directly transported into enterocyte by a heme transporter. Iron can now be either circulated or stored. Iron that is destined for circulation is transported across the basolateral membrane by ferriportin, a process that is coupled to oxidation of ferrous iron to ferric form by hephastin and ceruloplasmin. Newly absorbed iron binds to transferrin and is rapidly delivered to erythroid progenitors.

Role Of Transferrin

Transferrin is a glycoprotein with a molecular weight of approximately 80 kD which has iron binding capacity, is synthesized by the liver and enables delivery of iron to cells including erythroid precursors. The eythroid precursors have high affinity receptors for transferrin which mediate iron absorption through endocytosis[9].

Role of Ferritin

Ferritin is a ubiquitous protein that is found in highest levels in the liver, bone marrow and spleen and serves to store iron as reusable, non toxic molecule while free iron is ionized and hence highly toxic. Apoferritin, is an apoprotein present in the intestinal mucosa, that binds to free ferrous iron forms ferritin and stores it in ferric state. Intracellular ferritin is located in cytosol and in lysosomes in which partially degraded protein shells of ferritin accumulate into hemosiderin granules. Under steady state conditions serum ferritin levels correlate with body iron stores so is the most effective way to diagnose anemia and when levels are below 16 ng/ml iron deficiency is highly likely [10].

Role of Hepcidin

It is a small circulating peptide that is synthesized and released from the liver in response to increases in intrahepatic iron levels and inhibits iron transfer from enterocyte and macrophages to plasma by binding to ferriportin causing it to be endocytosed and degraded. As hepcidin levels increase, iron becomes trapped within duodenal cells in the form of mucosal ferritin and is lost as these cells get sloughed.

Iron Deficiency

An estimated 16 % of anemic elderly have iron deficiency which occurs when the rate of iron utilization exceeds the rate of intestinal absorption and may be caused by

1. Inadequate intake
2. Impaired absorption
3. Increased requirement
4. Chronic blood loss

Anemia Due To Vitamin B-12 Deficiency

Vitamin B -12 is a complex organo-metallic compound called cobalamine essential for DNA synthesis, maturation and normal functioning of the nervous system. Micro-organisms are the ultimate source of this vitamin; vegetarian diets are deficient in this vitamin. Vitamin B 12 acts as cofactor in conversion of homocysteine to methionine, a reaction which yields tetrahydrofolate through several steps. Tetrahydrofolate is essential since it is required for conversion of deoxyuridine phosphate to deoxythymidine monophosphate, an immediate precursor of DNA. Deficiency of vitamin B 12 causes reduced availability of tetrahydrofolate leading to impaired DNA synthesis. Anemia due to vitamin B 12 and folate is termed megaloblastic anemia and is found to have an incidence of 14% according to NHANES III study [11].

Pathogenesis:

Absorption Of Vitamin B-12

This is done by the presence of a factor called the intrinsic factor which is secreted by the parietal cells of gastric fundic mucosa.B-12 from the diet gets bound to R- Binders, also called transcobalamin I or cobalophilin, in saliva. When it reaches the duodenum it gets dissociated from the R-binder and gets bound to intrinsic factor and remains bound till it reaches the ileum. In the ileum, it is endocytosed by ileal enterocytes and gets associated with a carrier protein transcobalamin II and is secreted into the plasma which delivers B – 12 to the required sites rapidly.

Pernicious Anemia

It is a disease of older adults uncommon in ages below 30 years with a suspected genetic predisposition and a prevalence of 0.1% in the general population and 1.9% in subjects over the age of 60 years. It is due to autoimmune attack on the gastric mucosa and there are three types of autoantibodies are present:

Type I which blocks the binding of B-12 to intrinsic factor and is the cause in 75% of the patients, type II prevents binding of IF-B12 complex to ileal receptor and type III which acts against α and β subunits of gastric proton pump and are found in 90 % of persons not specific for pernicious anemia. The autoantibodies are of diagnostic utility, they are not the primary cause of gastric pathology. The autoreactive T-cell response initiates gastric mucosal injury and triggers formation of autoantibodies which further exacerbate epithelial injury and cause the mass of intrinsic factor secreting cells to fall below a threshold, when anemia develops. Pernicious anemia is found to be associated with other autoimmune diseases such as Hashimotos thyroiditis, type 1 diabetes mellitus.

Other Causes Of B-12 Deficiency

1. Achlorhydria and loss of pepsin secretion.
2. Following gastrectomy IF is unavailable
3. Loss of exocrine pancreatic function, B-12 cannot be released from R- binder complex.
4. Ileal resection can remove or damage the site of IF-B12 absorption
5. Tapeworm infestation when present compete with the host for B 12 and induce deficiency
6. In conditions like hyperthyroidism, disseminated cancer, chronic infections an increased demand for B12 can produce a relative deficiency.

Anemia Due To Folate Deficiency

The causes for folate deficiency in elderly include .

1. Decreased intake or malabsorption
2. Hyperutilisation of folic acid by metabolic processes caused by malignancy, Crohn's disease, rheumatoid arthritis
3. Certain drugs that act as folic acid antagonists like methotrexate,(used for a variety of conditions in the elderly like rheumatoid arthritis, lupus, psoriasis, asthma) phenytoin, trimethoprim, chemotherapeutic drugs damage DNA or inhibit DNA synthesis through several mechanisms and cause megaloblastic changes.
4. The patients on dialysis for renal failure tend to lose folic acid in the dialysis fluid.

Anemia Of Chronic Inflammation:

It was formerly called anemia of chronic disease and it is a hypoproliferative anemia defined as having low serum iron and RBC levels despite normal levels iron stores occuring in the setting of chronic inflammation and is associated with decreased serum iron, decreased total iron binding capacity, increased serum ferritin levels and abundant stored iron in macrophages. The illnesses associated with this type of anemia include chronic microbial infections, chronic immune disorders and neoplasms. It is the most common cause of anemia in the elderly constituting 32 % of all etiologies according to NHANES III study.

Older adults tend to have higher levels of pro-inflammatory cytokines secondary to multiple co-morbid illnesses like atherosclerosis, diabetes mellitus and malignancies. The cytokines implicated in anemia of chronic inflammation include TNF α, IL 1, IFN α, IL-6. The

pathophysiology implicated in anemia of chronic inflammation has a number of mechanisms, the most important of which is through hepcidin. Hepcidin as discussed earlier serves as a primary regulator of iron homeostasis. It serves to inhibit ferroportin which transports iron out of storage units. IL- 6 is found to increase hepcidin expression, trapping iron within the macrophages and starving the erythroid precursors of the iron. In addition these progenitors do not proliferate adequately because erythropoietin levels are inappropriately low for the degree of anemia due to the action of IL-1, TNF α and TGFβ. TNF also inhibits the erythroid colony forming units thereby reducing erythropoeisis. Also cytokine IL-6 causes increased plasma volume thereby leading to a dilutional anemia. The erythrocyte survival is shortened in the setting of chronic inflammation which has been attributed to an extra corpuscular hemolysis. The cause for iron sequestration in setting of chronic inflammation is considered to be the body's defense in fending off certain bacteriadependant on iron for survival. The anemia is usually mild with either normocytic normochromic or microcytic hypochromic RBCs.

Renal Insufficiency

Chronic kidney disease is an important cause of anemia in elderly due to the fact that renal function declines with age[12,13] which is further decreased by diseases like diabetes mellitus and hypertension. Anemia in chronic renal disease is attributed mainly to decreased erythropoietin and reduced RBC survival.

Pathogenesis

Role Of Erythropoietin (EPO)

It is a glycoprotein which is a hematopoietic growth factor which serves primarily to regulate RBC production. Secretion of EPO mainly occurs in the interstitial fibroblasts in the kidney in close association with peritubular capillary and proximal convoluted tubule with a lesser contribution from perisinusoidal cells in the liver. Reduced tissue oxygenation potently induces a logarithmic increase in the EPO synthesis. EPO enhances erythrocyte production mainly by inhibiting apoptosis of erythroid progenitor cells and to a lesser degree by enhancing erythroid progenitor proliferation and differentiation. Serum erythropoietin levels have been shown to be inappropriately low at a creatinine clearance of < 40 %. Mild decreases in hemoglobin may be detected at a creatinine clearance of 40- 60 ml/min. Anemia is considered to be an indication of end stage kidney disease but women and diabetics may develop this complication at an earlier stage of the disease [14].

Reduced Red Cell Survival In Kidney Disease

Some studies have shown that plasma from uremic patients can depress heme synthesis or inhibit growth of erythroid colonies. Inhibitors of multipotential stem cell have also been found. Secondary hyperparathyroidism in renal failure patients may also be a reason for marrow suppression. Survival of RBC is found to be decreased and it has been discovered that the survival is related to the level of azotemia [15].

Anemia Due To Endocrinopathies

1. Variations In Thyroxine Levels

The prevalence of anemia in hypothyroid patients is 20 – 60 %. Thyroid hormones are necessary for hemoglobin synthesis in adults and maturation of hemoglobin in the fetus hence hypothyroidism results in anemia. The anemia in hypothyroidism is commonly normocytic but occasionally macrocytic. Both hypothyroidism and hyperthyroidism have been known to cause pernicious anemia[16].

2. Androgen Deficiency

Testosterone decreases with aging in men and the greater rate of fall hemoglobin levels in men than in women suggests that falling testosterone maybe one of the causes of unexplained anemia. Testosterone accentuates proliferation of erythroid burst forming units and colony forming units by stimulating specific nuclear receptors and this effect is abolished by pretreatment of marrow cells with cyperoterone flutamide which block binding of androgens to their nuclear receptors. The erythropoietic effects of testosterone is best explained by thefact that following orchidectomy or androgen deprivation therapy for prostatic carcinoma hemoglobin levels fall by 1.2 g/dl to 1.5 g/dl on an average[17,18].

Hypopituitarism

Anemia due to hypopituitarism is usually normocytic normochromic and results chiefly from deficiency of hormones of the target organs controlled by the pituitary. Panhypopituitarism produces effects on RBC production by decreasing oxygen consumption which in turn decreases erythropoietin secretion and red cell mass until new equilibrium is established between oxygen supply and demand.

Anemia Due To Myelodysplastic Syndromes (MDS)

Myelodysplastic syndromes represent a heterogeneous group of disorders characterized by clonal hematopoiesis and peripheral blood cytopenias resulting from bone marrow dysfunction. Myelodysplasia is more common in elderly patients, more than 75 % of patients are over 60 years of age with recent estimates of approximately 45000 new cases per year .Anemia in conjunction with macrocytosis, thrombocytosis or neutropenia in the absence of other causes should raise the suspicion of myelodysplasia. The prevalence of anemia due to MDS has been estimated between 9 and 16 % based on three studies, NHANES III, Chicago and Stanford hospital and Clinics, VA Palo Alto Health Care system.

Pathogenesis

The basic pathophysiology in MDS involves increased apoptosis and proliferation. The proliferation is due to clonal expansion of a multipotenthematopoietic progenitor that is capable of granulocyte, monocyte, erythrocyte and megakaryocyte differentiation. Also increased levels of apoptotic mediators are present including TNF – α , FAS antigen (CD -95) and calcium dependant kinase. The proliferation of progenitor cells results in a hypercellular marrow but failure to accumulate adequate number of mature cells results in cytopenia.The peripheral smear picture is that of macrocytosis or dimorphic RBC picture[19].

Anemia Due To Primary Hematologic Disorders

A variety of other primary hematologic disorders have anemia as a manifestation. Anemia in the elderly can be due to primary hematological malignancies like AML, CLL, aplastic anemia or Multiple myeloma.

Acute Myelogeneous Leukemia

In normal hematopoiesis, the myeloblast is an immature precursor of myeloid white blood cells; a normal myeloblast will gradually mature into a mature white blood cell. In AML, though, a single myeloblast accumulates genetic changes which "freeze" the cell in its immature state and prevent differentiation. Such a mutation alone does not cause leukemia; however, when such a "differentiation arrest" is combined with other mutations which disrupt genes controlling proliferation, the result is the uncontrolled growth of an immature clone of cells, leading to the clinical entity of AML. Anemia due to AML result from the growth of leukemic clone cells, which tends to displace or interfere with the development of normal blood cells in the bone marrow.

Chronic Lymphoid Leukemia

Chronic lymphoid leukemia (CLL), is the most common type of leukemia in adults affecting 2 to 6 individuals per 100< 000 per year. It is a neoplastic disease characterized by the accumulation of small mature-appearing CD5+ B lymphocytes in the blood, marrow, and lymphoid tissues. The causes of this disease are unknown, although genetic factors likely contribute to its development. Anemia develops due to crowding of bone marrow with proliferating lymphocytes thus decreasing erythroid precursors.

Multiple Myeloma

Multiple myeloma (MM) is a malignant plasma cell disorder that accounts for approximately 10% of all hematologic cancers. The disease is characterized by monoclonal proliferation of plasma cells together with overproduction of a monoclonal antibody, often accompanied by anemia, hypercalcemia, renal insufficiency, or bone lesions. Approximately 97% of MM patients develop anemia during the course of their illness, and 70% are anemic at diagnosis. The anemia is usually normocytic normochromic, serum-iron levels are normal to low, serum ferritin is high, and hemosiderin is prominent in bone marrow macrophages. This suggests than iron release from reticulo endothelial macrophages is impaired, consistent with anemia of inflammation[20].

Aplastic Anemia

It is a hypoproliferative state which can occur due to several causes some of which are toxins like benzene, drugs like antimetabolites, anthracyclines, autoimmune diseases and inherited causes like Fanconi's anemia and DiamondSchwachman syndrome.According to the International Aplastic Anemia and Agranulocytosis Study the overall incidence of aplastic anemia is 2 cases/ I million with a higher incidence in southeast asia. It commonly presents in the 2nd decade

with a smaller peak in incidence after the age of 60 years. Immune injury to the marrow after drug-, viral-, or toxin-induced marrow aplasia could result from induction of neoantigens that provoke a secondary T cell– mediated attack on hematopoietic cells causing reduced hematopoietic precursors leading to the pancytopenia.

Anemia Secondary To Bone Marrow Infiltrates – Myelophthisic Anemia:

This is a type of anemia that results from marrow infiltration by tumour cells or non hematopoietic tissue. The marrow environment is susceptible to infiltration by almost all cancers but the most common are lung, breast and prostate cancers [21]. The anemia is usually mild to moderate and peripheral smear may show presence of tear drop RBCs and nucleated RBCs a feature suggestive of marrow infiltration. Bone marrow biopsy is the most reliable procedure to confirm the diagnosis and should performed in all patients suspected of having myelophthisic anemia.

Anemia Due To Hemolytic Disease:

Hemolytic anemias are the result of RBC deficiency due to premature destruction of RBC either within blood vessels (intravascular) or elsewhere (extravascular) resulting in shortened RBC lifespan and the marrow is unable tocompensate for the loss resulting in anemia .They can be classified as inherited and acquired. Inherited causes for hemolysis include,

Intrinsic Membrane Defect

1. Hereditary Spherocytosis, Hereditary Elliptocytosis,:

In hereditary spherocytosis there is defect in proteins of erythrocyte membrane including ankyrin, band 3, β spectrin, α- spectrin and protein 4.2. This leads to increased membrane fragility leading to loss of membrane surface area relative to intracellular volume, accounting for the spheroidal shape and decreased deformability of red cell. The size of fenestrations in the venous sinuses in the spleen is small relative to the RBC size, and to pass through requires significant deformability of the red cell and its membrane. This, however, is a major problem for spherocytes, which have lost surface area and are dehydrated and get lysed as they pass through. It presents with chronically compensated mild to moderate hemolytic anemia and its diagnosis is based on presence of spherocytes in peripheral smear, a positive family history and a negative direct antiglobin test. The mean corpuscular hemoglobin concentration is frequently elevated [22].

Hereditary Elliptocytosis (HE) and Variants

Similar to HS, these group of disorders occur due to defects in RBC membrane proteins of which spectrin defect is commonest. The mutation impairs ability of spectrin dimers to self associate into dimers and tetramers thereby disrupting the membrane cytoskeleton and reducing the deformability of RBC s as they pass through splenic sinusoids.

Hereditary pyropoikilocytosis is a variant of HE and a severe congenital hemolytic anemia that presents with RBC fragments, poikilocytes and microspherocytes on peripheral smear.

2. Hemolysis Due To Abnormal Hemoglobins

This can be due to either reduced synthesis of globin chains (quantitative) or defective synthesis of globin chains(qualitative).

Quantitative Defects

Thalassemia

This is a group of congenital anemia in which there is deficient synthesis of one or more globin subunits of normal hemoglobin. There is either production of either α or β (called α thalassemia or β thalassemia respectively) chains as a result of which there is accumulation of free globin chains that cause lysis of RBC precursors. It can occur in three forms thalassemia major in which there is severe anemia requiring frequent transfusions, thalassemia minor or thalassemia intermedia in both cases there is mild to moderate anemia. The prevalence of anemia due to thalassemia in elderly ranges from 12 – 20 %.

Qualitative Defects

Sickle Cell Anemia

Sickle hemoglobin is a mutant hemoglobin in which valine has been substituted for the glutamic acid normally at the sixth amino acid of the β- globin chain. This hemoglobin becomes polymerized and becomes poorly soluble when the oxygen tension is lowered and red cells that contain this hemoglobin become distorted and rigid. Hemolysis in sickle cell anemia results from the lysis ofcomplement-sensitive red cells and Hb lost during sickling or shear-induced membrane fragmentation. Extravascular hemolysis may occur by two mechanisms: monocyte and macrophage recognition and phagocytosis of red cells that have undergone sickling- or oxidation-induced membraneand physical entrapment of rheologically compromised red cellsSickling and oxidation- induced membrane changes promote cell dehydration and clustering of membrane protein band 3 which leads to accumulation of IgG and complement on the sickle cell surface [23].

3. Erythrocyte Enzymopathies

Glucose-6-phosphate dehydrogenase (G6PD) is a highly conserved housekeeping enzyme and rate-limiting enzyme of the pentose phosphate pathway in all cells.The pentose phosphate pathway (PPP) converts glucose to ribose-5- phosphate, a precursor to RNA, DNA, ATP, CoA, NAD, and FAD and also provides reductive potential in the form of NADPH. G6PD is a ubiquitous enzyme that must be quite ancient in evolution because it has been found in all organisms, from prokaryotes to yeasts, to protozoa, to plants, and animals. G6PD deficiency results from mutations in the G6PD gene and is well-known common cause of hemolytic anemia in human. RBCs lack other pathways to generate NADPH so G6PD deficiency becomes especially lethal in red blood cells, where any oxidative stress will result in hemolytic anemia.

Acquired Causes Of Hemolytic Anemia

Acquired causes of hemolysis include

1. Hemolysis due to mechanical injury to RBCs
2. Immune mediated injury to RBCs
3. Drug induced injury to RBCs
4. Hemolysis due to infections

Mechanical Injury to RBCs Can Occur Due to Several Causes

(i) Cardiac hemolysis
(ii) Microangiopathic haemolytic anemia

Cardiac Hemolysis

This is due to injury to the red blood cells owing to turbulence in the heart chamber because of the pressure gradient created most commonly by prosthetic valves. It can also be caused due to aortic stenosis, mitral valve disease, coarctation of aorta.

Microangiopathic Hemolytic Anemia (MAHA)

This encompasses several etiologies, the common mechanism in these disorders is a microvascular lesion that results in luminal narrowing often due to deposition of fibrin and platelets. The causes of MAHA are

a. Thrombotic thrombocytopenic purpura (TTP)
b. Disseminated malignancies

TTP:

In this disease there is deficiency of the enzyme ADAMTS13 also designated as Von Willebrand Factor metalloprotease whose function of ADAMTS13 is to degrade the high molecular weight multimers of Von Willebrand Factor (vWF). In its absence the vWf multimers accumulate in plasma and promote platelet activation and aggregation. The deficiency can be eitheracquired or inherited where in the acquired form there is antibody against the ADAMTS13 and in the inherited form there is deficiency of ADAMTS1z.

Disseminated Malignancies

Hemolysis due to disseminated malignancies is common in pancreatic, lung and prostatic carcinomas. Hemolysis can occur either due to DIC or intravascular tumour emboli. In case of DIC, a protease found in mucin secreted by adenocarcinoma directly activates factor X. Subsequently the coagulation cascade gets activated resulting in deposition of fibrin, formation of intravascular hyaline thrombi which cause shearing of red cells. Intravascular tumor emboli disrupt the endothelium, promoting deposition of fibrin, adherence of platelets, intimal hyperplasia, and vascular hypertrophy.Peripheral smear shows presence of moderate to severe anemia, schistocytes, helmet cells, microspherocytes, polychromasia and nRBCs, normal to high WBC count with a left shifted differential count. Marrow aspiration and biopsy reveal erythroidhyperplasia, normal to high numbers of megakaryocytes and tumour invasion in 55 % of patients[24].

Immune Mediated Hemolysis

It is characterized by shortened RBC lifespan and presence of

antibodies directed against the red cells. The antibodies can either be warm antibody type or cold antibody type.

Warm Antibody Type
This type is predominantly due to IgG type immunoglobulin which acts best at a temperature of 370 C with or without complement proteins. Theautoantibody coated RBCs are trapped by macrophages in Billroth cords of spleen to a lesser extent by Kupffer cells in liver.

Cold Antibody Type
It is caused by IgM antibodies that bind red cells at low temperatures (00C– 40C). The IgM antibodies bind to red cells causing agglutination and rapid complement fixation. This deposits sublytic quantities of C3b which leads to removal of affected red cells.

Drug Mediated Hemolysis:
Injury to RBCs due to drugs can occur due to several mechanisms:
(i) Drug Adsorption Mechanism:
Drugs like penicillin binds firmly to proteins on RBC membrane which is recognized by IgG antibodies and these RBCs are sequestered in the spleen.
(ii) Ternary Complex Mechanism
In drugs like quinidine the antibodies recognize a complex of the drug and a membrane protein, fix complement and cause intravascular hemolysis. They can also act as opsonins and cause extravascular hemolysis.
(iii) Autoantibody Mechanism
Drugs like α-methyl dopa induce autoantibody in an unknown manner, particularly Rh blood group antigens.

Hemolytic Anemia Due To Infections
Several organisms can cause hemolysis by different mechanisms. Some like Plasmodium and Babesia colonize RBCs and cause hemolysis and yet others like Bartonella, Clostridium adhere to cell surface and such cells are removed bythe spleen. Other mechanisms involved maybe the action of antibodies against antigen coated cells leading to agglutination or to complement mediated lysis[25].

Unexplained Anemia
The traditional notion that anemia in geriatric patients signifies some serious underlying disorder is not always true. A proportion of elderly have anemia that does not meet any criteria for any etiology. Even with the advent of better tests, such as serum ferritin , methylmalonic acid, and soluble transferrin receptor, a significant portion of elderly persons with anemia will be diagnosed as having unexplained anemia.The pathophysiology of unexplained anemia is poorly understood and is primarily a diagnosis of exclusion. Whether unexplained anemia represents a spectrum of undiagnosed diseases is unclear. Several theories have been postulated to explain this phenomenon, including decreased production of hematologic factors, inflammatory cytokine presence, marrow abnormalities and androgen deficiencies[26].

Consequences Of Anemia In Geriatric Patients:
Morbidity and mortality related to anemia in the geriatric age group can occur from underlying disease or due to effects of the anemia itself and has the following consequences:

Muscle and Bone
Anemia and osteoporosis are both prevalent in the geriatric population with a negative relationship between low hemoglobin levels and bone mass. Also lowerhemoglobin levels are associated with lower ankle extension strength, lower muscle density and less muscle mass.

MOBILITY
Anemia has been associated with greater average decline in physical performance[108,109,110].The risk of mobility problems in persons with hemoglobin< 12 mg/dl is more than twice than that occurring in persons with hemoglobin > 13 mg/dl.

FALLS
Thirty percent of people over the age of 65 years fall at least once a year. Falls and fractures are significant causes of disability, admission to institutional care and mortality. Hemoglobin level less than 10g/dl is associated with a higher incidence of fall and injury to hip and head as compared to those who were not anemic.

Cognition
Anemia due to vitamin B 12 deficiency and anemia alone has been demonstrated to be a risk factor for functional and cognitive decline.

Cardiac Disease
Acute and chronic anemia can cause congestive cardiac failure in patients without heart disease and may precipitate congestive heart failure and angina in patients with underlying heart disease.

Hospital Admissions
Anemia in elderly has been associated with frequent hospital admission for various causes.

History And Physical Examination:
Medical History:
Recent hospitalization can cause anemia in the elderly as a result of multiple phlebotomies as well as the acute illness itself. Any history of recent surgery leads one to think of acute blood loss, also when the person needs transfusion during surgery it implicates the person was anemic before the surgery and the presence of another condition preventing an appropriate response to blood loss should be thought of. Diseases that cause anemia including malignancies, myelodysplastic syndrome, chronic kidney disease and rheumatologic diseases should be taken into account. Dietary history is important since strict vegan diets can raise the risk of B-12 deficiency and history of alcoholism should not be overlooked as it is a cause of B 12 and folate deficiencies.

History of previous transfusions is an indication of the presence of a chronic illness like thalassemia or other hemolytic diseases.

History of drug intake should be reviewed to rule out the drugs as a cause for anemia.

Symptoms:
Most symptoms of anemia are nonspecific; however a temporal relation between falling hemoglobin and exacerbation of symptoms is very useful.

General symptoms include the following:
- Fatigue
- Weakness
- Exertional dyspnoea
- Tinnitus
- Presyncope
- Palpitations
- Poor concentration Symptoms of iron deficiency:
- Blood loss- tarry stools, hematochezia, hematuria
- Pica
- Dysphagia
- Mouth and tongue soreness Symptoms of vitamin B 12 deficiency
- Neuropathy
- Ataxia
- Dementia

Symptoms Of Hemolysis
- Jaundice
- Dark coloured of urine

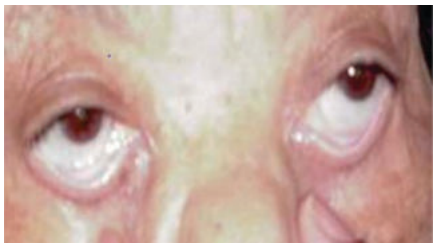
Physical Examination
Careful physical examination helps in revealing cause of the anemia hence justifying comprehensive physical examination. One should look for the following:
- Pallor (present in anemia due to any cause)(image 1)
- Icterus (seen in haemolytic anemia) (image 2)
- Koilonychia (in iron deficiency) (image 3)
- Lymphadenopathy (in metastasis due to malignancies) (image 4)
- Glossitis (image 5)
- Aphthous ulcers (image 6)
- Angular stomatis (image 7)
- Pedal edema (image 8)
- Tachycardia (in severe anaemia due to any cause)
- Cardiac murmurs (in severe anemia)
- Hepatomegaly (in haemolytic anemias)
- Splenomegaly (in haemolytic anemia)

Laboratory Assessment Of Anemia:
After obtaining history and initial physical examination laboratory evaluation should be done which includes:
- Complete blood count
- Stool for occult blood
- Iron profile

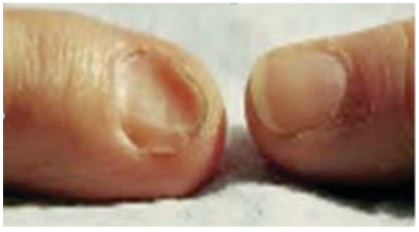
- Vitamin profile
- Erythropoietin level
- Peripheral smear examination

Complete Blood Count

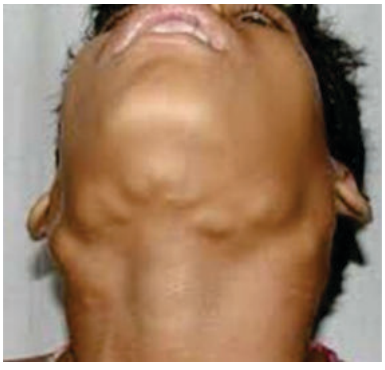
The CBC is important for the diagnosis of anemia and for monitoring disease progression and treatment efficacy. When assessing the elderly anemia patient, the most important components of the CBC are:



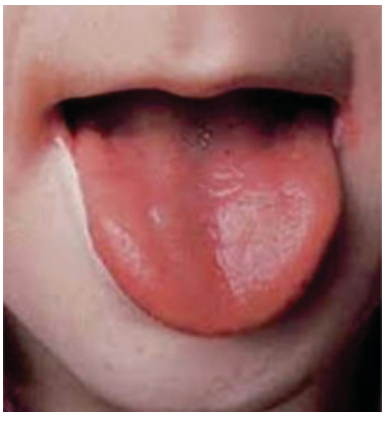
Bulbar Conjunctival Pallor



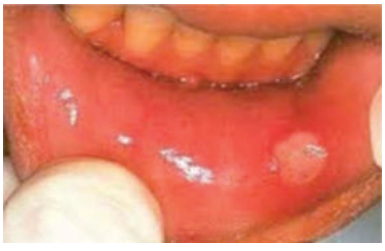
Koilonychia



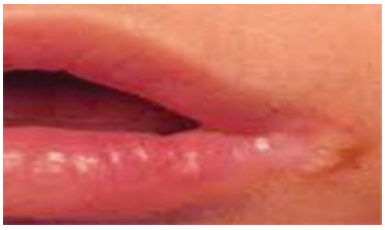
Lymphadenopathy



Glossitis



Apthous Ulcers



Angular Stomatitis

Erythrocyte (RBC) Count:

Reports the total number of RBCs per litre of whole blood.

- Normal range for men: 4.7–6.1 million cells/mcL
- Normal range for women: 4.2–5.4 million cells/mcL

Hemoglobin (HgB):

Measures the amount of hemoglobin present in the blood

- Normal range for men: 13–17 g/dL
- Normal range for women: 12–16 g/dL

Hematocrit (HCT):

Packed cell volume in proportion to blood volume.

- Normal range for men: 40% to 52%
- Normal range for women: 36% to 48%

Mean cell (Corpuscular) Volume (MCV):

It measures the average size of RBCs, a diagnostic parameter for evaluating anemia, and differentiates microcytic and normocytic anemia in the elderly.

- Normal range: 81–100 fL
- Macrocytosis: Greater than 100 fL with large RBCs
- Microcytosis: Less than 81 fL with small RBCs

Mean Cell Hemoglobin (MCH):

It is the average amount of Hb in a RBC.

- Normal range: 27–34 Hb/cell

Mean Cell Hemoglobin Concentration (MCHC):

It is the average concentration of Hgb in an RBC.

- Normal range: 30% to 36%

RBC Distribution Width (RDW-CV):

It measures variations in the size of RBCs.

- Normal range: 12% to 14%

Leukocyte (White Blood Cell) Count:

Reports the number of leukocytes in the blood; the differential includes different types of leukocytes (i.e., neutrophil, eosinophil, basophil, lymphocyte, monocyte).

- Normal range: 4,500–10,000 cells/ mcL

Thrombocytes /Platelet Count:

It indicates the number of platelets present.

- Normal range: 150,000–450,000 cells/ mcL

Stool Examination

Stool specimen for occult blood is another essential test in evaluating anemia in elderly as GI bleed is often a major etiologic factor. The bleed may be intermittent so three stool samples testing is recommended. Before the test, the patient is advised to stop iron supplements if any, to avoid red meat and food with red dye for 3 days before the test.

Endoscopic Evaluation

If the patient tests positive for occult blood in the stool sample a gastroenterology consult is ideal. The patient will need endoscopy or colonoscopy to identify the bleed. It might indicate haemorrhoids or less other serious problems, it might also indicate malignancy.

Iron Profile

Serum iron is not an adequate test to exclude iron deficiency. Numerous tests are available to diagnose iron deficiency including RBC, MCV, serum iron, serum transferrin, iron saturation, serum ferritin, soluble transferrin receptor (sTFR).

Serum ferritin is the most useful test for diagnosing iron deficiency. Low serum ferritin is highly specific for iron deficiency.As an acute

phase reactant, ferritin can be elevated in inflammation, complicating the diagnosis of iron deficiency anemia in the presence of inflammation. Values between 18 and 44 ng/ml are highly suggestive of iron deficiency in the elderly.

Alternative strategies to diagnose iron deficiency anemia have been studied, most commonly employing the sTFR. The sTFR is a truncated fragment of the membrane receptor that is increased in iron deficiency, when iron availability for erythropoiesis is low. STFR enhances diagnostic sensitivity in elderly persons compared with using a very low serum ferritin for iron deficiency anemia. Another method standardizes sTFR based on the serum ferritin. By using the log of the sTFR/ferritin, a ratio greater than 2.5 may indicate iron deficiency. However, raising the threshold of ferritin to less than 30-50 ng/mLaffords a similar or better diagnostic performance. Further, sTFR is less sensitive in detecting early iron deficiency compared with ferritin.[27]

The Iron Profile Will Measure:
Total Serum Iron
- Normal range for men: 60–176 mcg/dL
- Normal range for women: 45–170 mcg/dL

Total Iron Binding Capacity
- Normal range: 250–450 mcg/dL

Unsaturated Iron Binding Capacity
- Normal range: 100–400 mcg/dL

Transferrin Saturation
- Normal range: 20% to 50%

Serum Ferritin
- Normal range for men: 12–350 ng/mL
- Normal range for women: 12–200 ng/mL

Vitamin Profile:
The vitamin profile may include measurements of:

Folate (Vitamin B9)
- Normal range: 4–20 mcM

Vitamin B12 (cobalamin)
- Normal range: 200–900 pg/mL

Methylmalonic Acid
- Normal range: 73–271 nM

Homocysteine
- Normal range: 5.1–13.9 mcM

Vitamin B 12 and folate deficiencies though rare are essential to rule out as a cause of anemia as these are reversible causes. Low vitamin B-12 levels are frequent in older adults. If a level less than 200 pg/mL is detected, then cause of vitamin B-12 deficiency (eg, pernicious anemia, malabsorption) should be investigated and vitamin B-12 replenished. For equivocal levels of vitamin B-12, such as a level between 200 and 350 pg/mL, evaluate methylmalonic acid. An elevated methylmalonic acid level is evidence of a vitamin B-12 tissue deficiency. One must recognize that methylmalonic acid is also elevated in renal dysfunction. Homocysteine is elevated in vitamin B-12 deficiency and folate deficiency. If the methylmalonic acid is elevated in the setting of anemia and a low vitamin B-12, evaluate for vitamin B-12 deficiency and empirically treat the patient.

Erythropoietin Levels
Erythropoietin level is not ordered frequently, but it is used in patients with unexplained polycythemia which is a condition with abnormally elevated concentration of RBCs. The erythropoietin level is also reduced in chronic kidney disease and myelodysplasia. The normal range is 4.5–21.3 mU/mL. Measuring serum erythropoietin levels is of little diagnostic utility in patients with chronic kidney disease.

Peripheral Smear Examination
Peripheral blood smear is essential to determine the caue of anemia. The peripheral enables one to study the morphology of the three cell components and is highly subjective but highly rewarding.

Morphology Of Red Blood Cells
In a well spread dried and stained blood film the RBC have round smooth contours and diameters between 6.0 – 8.5 µm with a central pallor approximately one third of the area of the red blood cell.

Anisocytosis and Poikilocytosis
These terms mean more variation in size (anisocytosis) or shape (poikilocytosis) (image 9) than that usually expected in a smear. It is seen in iron deficiency anemia, megaloblastic anemia, thalassemias and myelodysplastic syndromes.

Macrocytes
Macrocytes are present classically in megaloblastic anemias and also in aplastic anemia, myelodysplastic syndromes and dyserythropoietic conditions.

Microcytes
Microcytes in a peripheral smear (image 10) occur in defects of hemoglobin synthesis and are seen in iron deficiency, thalassemia, severe cases of anemia of chronic disease.

Basophilic Stippling
The presence of basophilic granules that are distributed throughout the cell is termed basophilic stippling and is seen in conditions like thalassemia, liver disease, lead poisoning and megaloblastic anemias.

Hypochromia
Occurrence of the central pallor more than one third of the area of the cell helps one to identify hypochromia which occurs in conditions of reduced hemoglobin synthesis or unusually red cells. Iron deficiency anemia and sideroblasticanemias are some of causes of hypochromia.

Hyperchromasia
Unusually deep staining of cells with loss of central pallor is seen when theres presence of macrocytes and when are rounded. It is seen in megaloblastic anemias and spherocytosis.

Schistocytosis
Schistocytes are red cell fragments that are smaller than normal red cells with irregular or round contour and with hypochromasia or hyperchromasia. It can occur in conditions like thalassemias, congenital dyserythropoietic anemia, microangiopathic hemolytic anemia, cardiac hemolytic anemia and in thermal injury like burns.

Acanthocytosis
This describes red cells with irregular spicules of varying length, thickness and irregularly distributed over the cell surface and seen in abnormal phospholipid metabolism and in liver diseases[130]

Target Cells
This term refers to cells with centrally stained area and a peripheral rim of hemoglobinized cytoplasm separated by non stained or lightly staining cytoplasm and is present in thalassemias, hereditary hypolipoproteinemia and some cases of iron deficiency anemia.

Sickle cells
Sickle cells are present in freshly drawn samples of blood from individuals homozygous for hemoglobin S and can be observed when subjected to hypoxia.

Howell-jolly Bodies
Howell-jolly bodies are round cytoplasmic inclusions that stain purple with Romanowsky stains and are regularly present following splenectomy[28]

Morphology Of Leucocytes
Neutrophils
Normal neutrophils are 13µm in size with segmented nucleus having 2- 5 lobes connected by tapering chromatin strands and cytoplasm shows fine azurophilic granules when stained with Romanowsky stains. The nuclear chromatin is peripherally condensed with clumping.

Lymphocytes
The circulating lymphocytes are uniform in size about 9 µm in diameter with a thin rim of cytoplasm occasionally showing scant azurophilic granules.

Eosinophils
They are of size 12- 17 µm with a bilobed nucleus usually and have

bright orange granules in the cytoplasm.

Monocytes
Monocytes are the largest of circulating leucocytes of size 15- 18 µm with blue- grey cytoplasm having variable number of reddish granules and large curved nucleus that maybe folded or curved.

Basophils
They are the least number of leucocytes with variably sized dark blue granules in the cytoplasm that tend to obscure the segmented nucleus.

Bone Marrow Study
The absolute indications for bone marrow examination include evaluation of leucopenia, anemia, pancytopenia, leucoerythroblastosis and diagnosis of leukemia as well as monitoring the effects of therapy. Relative indications include evaluation of iron metabolism unexplained fever (PUO) or splenomegaly, and sampling for chromosomal analysis, immunophenotyping, or microbiological cultures[29,30].

Iron Deficiency Anemia
The bone marrow in iron deficiency is characterized by mild to moderate erythroid hyperplasia with striking nuclear distortions, resembling those found in dyserythropoietic anemias. Karyorrhexis and nuclear budding are particularly common, but multinuclearity, nuclear fragmentation, and even intranuclear bridging may be observed. The individual normoblasts appear small and may have scanty cytoplasm, often with irregular, ragged borders. Macrophage iron is absent or severely reduced in the marrow, spleen, and liver of iron-deficient subjects.

Megaloblastic Anemia
Aspirated marrow is cellular with striking megaloblastic changes, especially in the erythroid series. The high apoptosis rate of erythroid precursor cells in the marrow creates more globin lysis and, thus, jaundice. Sideroblasts are increased in number and contain increased numbers of iron granules. The ratio of myeloid to erythroid precursors falls to 1:1 or lower, and granulocyte reserves may be decreased.

Hemolytic Anemia
The marrow shows hyperplasia of the erythroid series with normoblasts constituting 25 -60 % of all nucleated cells.

Anemia Of Chronic Inflammation
The marrow in cases of anemia with chronic inflammation is moderately cellular with erythroid hyperplasia and normal erythroid maturation.

Myelodysplastic Syndrome
Marrow in myelodysplastic syndromes may be either normocellular or hypercellular. The erythroid series may show large or small erythroblasts, nuclear fragmentation and stippled erythroblasts. Granulocytic precursors show hypogranulation, monocytoid appearing granulocytes and Pelger-Huet anomaly of the neutrophils. Megakaryocytes appear micro megakaryocytes with unilobed or bilobed nuclei

Anemia In The Elderly
Typically, in persons 65 years of age or older there is an underlying etiology for the anemia such as chronic disease, iron deficiency, or myelodysplastic syndromes that can be identified by further investigation. In a study of 232 patients aged 65 to 98 (median 81) years of age, 24% were found to anemic of these, after a comprehensive workup 17% did not have an identifiable underlying cause. The major causes of anemia and their prevalence in the elderly are illustrated prevalence ranges from three studiesare for UAE (34–44%); iron deficiency (12–25%); CKD (4–8%); MDS (9–16%); malignant hematologic (e.g., chronic lymphocytic leukemia) disorder (2%); or inflammation (6–20%). Interestingly, folic acid deficiency has disappeared in the U.S. population, probably as a result of fortification of flour. While 10–20% of elderly patients have been described as Vitamin B12 deficient (defined by reduced serum levels of Vitamin B12), clinically significant Vitamin B12 deficiency is uncommon, diagnosed in only 1/19026 and 1/174 subjects in two studies, respectively. For emphasis that means that as a cause of macrocytosis, Vitamin B12 deficiency is much less common than MDS or ethanol abuse.

Studies at two academic institutions using comprehensive clinical and hematologic analyses have refined the prevalence and causes of anemia in the elderly. Iron deficiency anemia represented the most common cause of anemia in the elderly, at 25.3%. Iron deficiency may be due to nutritional deficiency or blood loss; in the relatively affluent Western world, blood loss is the major issue. The cause of blood loss must be identified since in this patient group, since iron deficiency could be a sign of a serious disorder such as colon cancer. Furthermore, in this patient group one cannot use simple laboratory tests based on transferrin saturation and ferritin levels to readily differentiate between iron deficiency and inflammation as a cause of anemia.

A number of studies have demonstrated that IL-6 levels increase with aging, and are correlated with the development of anemia in the elderly, including healthy women, the Framingham Heart Study, and the EPESE (Established Populations for Epidemiologic Studies of the Elderly. More recently, studies of IL-6 and hepcidin levels have not been found these to be elevated in elderly patients who do not have comorbid diseases. There may be several reasons for these discrepant results. First, if aging is associated with only small increases in IL-6, one would a need large sample size for detection. Additionally, currently these assays measure the monomeric form of IL-6, which also exists as a multimer complicating the analysis.

Evaluation Of Anemia
Commonly identified when the elderly are scheduled for elective surgical procedures. Anemia is a common condition in surgical patients and is independently associated with increased perioperative mortality.[31] When pre-admission testing prior to elective surgery reveals anemia, it should therefore be viewed as a significant and treatable medical condition, rather than as simply an abnormal laboratory value.[32] The diagnosis of an unexpected anemia in patients scheduled for elective surgery in which significant blood loss is anticipated, should be considered an indication for rescheduling elective surgery until evaluation and management of anemia is accomplished.

In traditionally taught approaches to evaluating a patient with anemia, the mean corpuscular volume (MCV) typically has been used as a starting indice, followed by biochemical analysis. The MCV has been shown to add value to the RDW (red cell distribution width) for evaluation of macrocytosis. But for microcytic anemias, MCV is of less value, particularly for patients with iron deficiency who have comorbidities. Twenty-two percent of elderly patients can be identified as having iron deficiency anemia by their response to a course of ORAL iron therapy, despite not having the typical laboratory findings of transferrin saturation less than 16% and ferritin less than 30 ng/mL. When transferrin saturation is low (<16%) and the ferritin level is high (>200ng/ml), the diagnosis of anemia of inflammation is generally considered. However, the MCV is normal in 70% of patients with anemia of inflammation, despite limited iron delivery to red cell precursors as indicated by the low transferrin saturation. This overlap of these two common causalities of anemia (iron deficiency and inflammation) has made the use of the traditional markers: MCV, transferrin saturation, and ferritin, difficult to interpret in routine practice.[33]

Iron-restricted erythropoiesis can cause anemia due to an absolute iron deficiency; iron sequestration which is mediated by hepcidin; and/or a functional iron deficiency due to erythropoietin-stimulated erythropoiesis. The evaluation of anemia must also consider unexpected diagnoses including chronic kidney disease (CKD) or occult malignancy. If absolute iron deficiency is diagnosed, in the elderly post-menopausal population it is mandatory to rule out gastrointestinal pathology, including malignancy as a source of chronic blood loss. Referral to a gastroenterologist may be the most effective way to proceed. However, in one-third to two-thirds of such patients, work-up of the gastrointestinal tract is negative. Serum creatinine and GFR must be determined in order to evaluate for CKD, in which case referral to a nephrologist may be appropriate. The suggested cut-off of glomerular filtration rate (GFR) < 60 mL/min for consideration that the anemia is secondary to end stage renal disease (ESRD), follows CMS guidelines on reimbursement for erythropoietic stimulating agent (ESA) therapy in patients with ESRD; but between 30–60 mL/min GFR, other causes for anemia may be possible.

When ferritin and/or iron saturation values rule out absolute iron deficiency, serum creatinine and glomerular filtration rate (GFR) determination may indicate chronic kidney disease (CKD) and the need for referral to a nephrologist.

When ferritin and/or iron saturation values are in determinant, further evaluation to rule out absolute iron deficiency versus inflammation/ chronic disease is necessary. A therapeutic trial of iron would confirm absolute iron deficiency. No response to iron therapy would indicate the anemia of chronic disease, suggesting that ESA therapy be initiated.

When serum ferritin and transferrin saturation values are inconclusive, further evaluation is necessary to rule out absolute iron deficiency or inflammation/chronic disease. As noted above, a response to a therapeutic trial of oral iron confirms absolute iron deficiency. However, lack of response to oral iron therapy may require a trial of IV iron, since in the presence of hepcidin, absorption of iron from the intestinal tract is impaired. In a study of patients with a clinical diagnosis of iron deficiency anemia based on ferritin and transferrin saturation assays, only 21% responded to 4 weeks of oral iron therapy compared to 65% of patients given IV iron therapy.[34]

A lack of response to iron therapy suggests a diagnosis of the anemia of inflammation or UAE. At this point, clinical evaluation for inflammatory conditions as well as measurement of C-reactive protein, fibrinogen, erythrocyte sedimentation rate (ESR), IL6, and hepcidin levels (if clinically available) may be useful. If abnormal, management of the underlying condition supplemented with an erythropoiesis stimulating agent (ESA) may be appropriate for further management.49 A careful history for alcohol use/abuse particularly in patients with an MCV>100 may be in order contributing either to poor marrow reserve or folate deficiency in the elderly. An early study by Cash and Sears of 90 patients (mean age 50.9 ± 16.5, not confined to elderly individuals) with anemia of chronic disease observed that there was a broader spectrum of associated diseases with ACD than had previously been recognized. A more recent study by Waalen et al.[35] compared a large cohort of UAE cases in the elderly with a matched, non-anemic control group and found that IL-6 and hepcidin levels did not differ significantly; whereas testosterone levels were lower in men and erythropoietin levels were inappropriately low for the degree of anemia.

The diagnosis of UAE is usually considered when other causes of anemia in the elderly have been eliminated. The diagnosis of UAE is based on the findings of a hypoproliferative anemia: a low reticulocyte index and an inadequate erythropoietin level for the degree of anemia. The management of these patients is a serious and recurrent issue, since in the absence of an etiology there is no proven efficacious intervention. When such patients are symptomatic, when they find themselves in clinical situations involving blood loss, or when surgical intervention is required, consideration of transfusion therapy is necessary, as described below.

Management Of Anemia

As detailed above, management of anemia is determined by results of the evaluation. While folate deficiency is increasingly being made a non-entity with folate supplementation in flour, particular attention needs to be paid in certain settings: e.g. poor diet combined with alcoholism, or compliance failure with folate supplementation in a dialysis patient. Similarly, Vitamin B12 deficiency may be infrequently diagnosed, but a diagnostic trial of Vitamin B12 therapy may be necessary if the constellation of symptoms and signs are consistent with Vitamin B12 deficiency Further assays for, methylmalonic acid and homocysteine may be helpful but can also be inconclusive.[36]

As indicated above, a diagnostic or therapeutic trial including IV iron therapy may be necessary to rule out absolute iron deficiency. Even the "gold standard" diagnostic bone marrow aspirate indicating the presence of some stainable iron may obscure a deficiency of storage iron, since in some of these cases the patients' anemia has been shown to be responsive to iron therapy.[37,38]

Thirdly, therapy with an erythropoiesis stimulating agent (ESA) may be indicated for treatment of one of several causes of anemia in the elderly. ESA's can be useful in treating the anemia in patients with end stage kidney disease; anemia in patients with inflammation/chronic disease who are scheduled for elective surgery; and in patients with MDS[39] who have moderate to severe anemia, in which the only other alternative may be chronic blood transfusions.

ESAs were first demonstrated and approved for use to increase the hemoglobin levels in patients with end-stage chronic kidney disease (CKD) undergoing dialysis[40] and subsequently in such subjects who did not require dialysis.[41] Based on prospective randomized trials that demonstrated reduced allogeneic blood transfusion, ESAs have subsequently been approved in patients undergoing elective surgery and in oncology patients with chemotherapy-induced anemia.

Subsequent to FDA approval, clinical trials were undertaken in an attempt to demonstrate long term improved patient outcomes with ESA therapy. lists five such clinical trials in patients undergoing elective spine surgery; patients with chronic kidney disease (predialysis); and in patients with stage II-IV (New York Heart Association) congestive heart failure.[42] Each of these studies were designed to evaluate aggressive anemia management (to a target Hgb within normal range) vs. conservative Hgb correction. In patients scheduled for elective spine surgery, outcomes were compared for ESA and placebo-treated cohorts who did not receive anticoagulation prophylaxis. In this study a higher incidence of deep vein thrombosis was found in patients receiving epoetin alfa compared to placebo. According to the subsequently revised prescribing information for ESA therapy, antithrombotic prophylaxis should be considered when ESA's are used in elective surgical patients who do not receive perioperative anticoagulation. In anemic patients with congestive heart failure (CHF) a large, randomized trial of patients whose median age was 72.0 (range 63–78), long-term ESA therapy was successful in correcting anemia and reducing blood transfusions, but did not improve long-term clinical outcomes (composite outcome of death or any cause of hospitalization from worsening CHF). The American Society of Hematology/American Society of Clinical Oncology Clinical Practice guideline update has recommended use of ESAs in patients with low risk MDS, in order to avoid blood transfusions. In clinical practice, the potential for increased risks of death and thromboembolic events should be balanced against the benefits of treatment with ESAs, including avoidance of allogeneic blood transfusions.[43]

Blood Transfusion Therapy

Patients with moderate anemia have few associated symptoms, due to significant compensatory mechanisms that preserve oxygen transport in the setting of a reduced hemoglobin. These compensatory mechanisms include increased blood flow due to decreased blood viscosity, increased oxygen unloading to tissues due to increased red cell bisphosphoglycerate, (2,3 BPG) increased plasma volume, and redistribution of blood flow.[44] Generally, symptomatic manifestations occur only when the hemoglobin is below two-thirds of normal (i.e., less than 9 to 10 g/dL) as basal cardiac output increases in the anemic patient and is manifested clinically by symptoms of fatigue, dyspnea and tachycardia.[45] However, the normal compensatory mechanisms of tachycardia and increased stroke volume may be impaired in the elderly, particularly in those with cardiovascular disease.

The recognition that transmission of non A, non B hepatitis was one of the complications for patients undergoing cardiac bypass surgery, along with the advent of HIV transmission by blood transfusion, led to the realization that rather than reacting to the cardiovascular compensatory response to anemia with blood transfusion therapy,[46,47] a more appropriate response for stable patients would be to allow the normal compensatory cardiovascular response to anemia to preserve oxygen transport in patients who were otherwise stable and did not have risk factors. Based on Level 1 evidence it is now generally agreed that a more restrictive transfusion practice is safe, with equivalent patient outcomes compared to transfusion practice to maintain hemoglobin levels above 10g/dL.

Blood transfusion strategies to treat anemia in the elderly and patients with cardiovascular disease, are poorly understood. For patients known to have risk factors such as age or cardiovascular disease (CVD), the odds adjusted ratio of postoperative mortality has been observed to increase significantly when compared with patients not known to have CVD illustrating that the management of anemia, including blood transfusion therapy, should be different for patients known to have CVD. In this study of 1,958 Jehovah's Witness patients undergoing surgery without blood transfusions, overall mortality was 3.2%. Five hundred forty seven (28%) of patients had Hgb <12 g/dl preoperatively. Ten deaths (16.1%) occurred within one day of surgery and 25 (40.3%) occurred within one week. Patients with cardiovascular disease had a 10.0% death rate, which was 4.3 times (95% CI, 2.6–7.1) higher than in patients without cardiovascular

disease. This assessment echoes a clinical practice guideline that concluded that 'the presence of coronary artery disease likely constitutes an important factor in determining a patient's tolerance to low hemoglobin.[48]

Acute Coronary Syndromes (ACS)

Perioperative myocardial ischemic episodes are associated with hematocrit levels less than 28% in patients undergoing radical prostatectomy. A retrospective, analysis of CMS (Center for Medicare Services) data on 79,000 elderly patients (> 65 years of age) hospitalized with acute myocardial infarction in the United States., found that blood transfusion in patients whose admission hematocrit values were less than 33%, were associated with significantly lower mortality rates.[49] Anemia (defined as a hematocrit of less than 39 percent) was present on admission to the hospital in nearly half the patients (43.4 percent) and was clinically significant (i.e., 33 percent or lower) in 10.4 percent of the patients. It was concluded that a more proactive management of anemia in elderly patients including blood transfusion therapy, was warranted.[50]

There is substantial variation in the use of blood transfusions in patients with ACS. An analysis of 24,000 patients in 27 countries found an association between non-ST segment ACS and a lower likelihood of blood transfusion. Rates of 30 day and 60 day clinical outcomes did not correspond to rates of transfusion, suggesting that the geographic variability in transfusion practices represented either underuse or the overuse of transfusions. A study of 44,000 patients with ACS in 400 U.S. hospitals found that 22.2% of patients were anemic and 10.4% received transfusions; of those with a nadir hematocrit ≤ 24%, transfusions had a beneficial impact on mortality; and for patients with hematocrit levels between 24–27%, transfusions had a neutral impact on mortality. However, more recently a systemic review of 10 published studies of blood transfusion strategies in patients with anemia associated with myocardial infarction, found that patients receiving blood transfusions had a higher all-cause mortality rate associated with blood transfusion vs no blood transfusion (18.2% vs 10.2%, risk ratio 2.9, p< 0.001). The difficulty in interpreting these retrospective, observational studies is that any conclusion that blood transfusions are causative, is confounded by both the underlying anemia and the underlying cause of the anemia as driving causes of the poor outcomes.[51]

A small pilot trial randomized 45 patients with acute myocardial infarction (MI) and hematocrit level ≤ 30% to a liberal cohort receiving transfusions to maintain hematocrit ≥30% or a conservative cohort (transfusions to maintain 24% to 27%).[52] The primary clinical safety measurement of in-hospital death, recurrent MI or worsening congestive heart failure occurred in 8 (38%) of the liberal cohort vs 3 (13%) in the conservative cohort (p=0.046). These results suggested that more liberal transfusions practices in patients with AMI were associated with worse clinical outcomes. An addendum to the British Committee for Standards in Haematology (BCSH) guidelines[53] dealt with measures to avoid over-transfusion, particularly transfusion-associated circulatory overload (TACO) in vulnerable patients such as the elderly. It was noted that there were complex issues including lack of attention to fluid balance and prescription of diuretics.

Lopez-Contreras MJ et al [54](2010) found thatto investigate iron status in institutionalized elderly subjects and to determine its association with different factors including: age, gender, body mass index, dietary intake, consumption of iron supplements, functional status and disease. A cross-sectional study.Seven public nursing homes.252 subjects, aged 65-96 years. Food intake was assessed by a 4-day weighed-food record. Iron status indices were measured. Barthel' s Index was used to evaluate functional status. Illnesses were ascertained from medical records. Anemia was found in 25.4% of subjects studied. Average dietary intakes fulfilled the amounts of Recommended Dietary Intake for Spanish elderly population, except for folate. A substantial percentage of subjects exhibited folate dietary deficit (89.2%). Mean (SD) BMI was 27.8 (6.4) kg/m2, and functional status 78.1 (26.5). Taking into account hematocrit, red blood cell count and serum iron concentration values, poor iron status was significantly more common in men (59.4, 61.4 and 16.8%, respectively) than in women (36.4, 36.4 and 6.0%, respectively). Hemoglobin concentration was positively associated with the energy and nutrient dietary intake, and negatively with age, BMI and functional status. Based on World Health Organization criteria, anemia was found in 25.4% of elderly subjects studied. Iron deficiency seems to be the main cause of anemia, and chronic disease the second cause of anemia. Dietary intake is not one of the principal causes of anemia in the study population, except for folate intake.

Karoopongse E et al [55](2013) found thatanemia is one of the most common health problems in the elderly in low and middle income countries. Evidence from studies in high income countries suggests that the presence of anemia may predict mortality. They aimed to estimate the prevalence of anemia and the determine the relationship of hemoglobin, mean corpuscular volume (MCV) and mortality in community dwelling Thai elderly. Data from subjects aged ≥ 60 years from the Fourth Thai National Health Examination Survey were analyzed. Comorbidity and hematologic indexes including MCV were obtained. The Cox proportional hazard model was applied to explore associations with mortality. Data from 8,935 subjects were obtained. The mean age of participants was 69.2 years (SD 6.8). 3446 (38.2%) of subjects had anemia; 1931(56%) of these were classified as mild and normocytic. With a total 51,268 person-year of follow up, 753 participants with anemia died, and the cumulative all-cause mortality was 38.5 per 1,000 person-years. The presence of anemia was associated with an increased risk of mortality with HR of 1.66 (95% CI = 1.50–1.84 , p < 0.001). Among subjects with low MCV, hemoglobin level < 10 g/dl in men and < 9 g/dl in women significantly increased the risk of mortality (HR of 2.71, 95% CI = 1.88–3.91 and HR of 3.14, 95%CI = 2.11–4.67, respectively) Persons with anemia and normal MCV, the association with mortality was evident at hemoglobin levels below 11 g/dl for both males and females. (HR of 1.98, 95% CI = 1.67–2.35). Anemia is a moderate to severe public health significant in the population for community dwelling elderly in Thailand. At the same level of Hemoglobin, low MCV population seem to have lower mortality rate than normal MCV. Systematic screening for anemia should be implemented to identify patients at increased risk of mortality. The future research should be focus on causes of anemia and factors contributing to increased mortality in normal to high MCV would be of interest. If this could lead to identifying modifiable causes, it would be beneficial for improving mortality risk among older people.

Busti F et al [56](2014) found thatiron deficiency (ID) is relatively common among the elderly population, contributing substantially to the high prevalence of anemia observed in the last decades of life, which in turn has important implications both on quality of life and on survival. In elderly subjects, ID is often multifactorial, i.e., due to multiple concurring causes, including inadequate dietary intake or absorption, occult bleeding, medications. Moreover, because of the typical multimorbidity of aged people, other conditions leading to anemia frequently coexist and make diagnosis of ID particularly challenging. Treatment of ID is also problematic in elderly, since response to oral iron is often slow, with a substantial fraction of patients showing refractoriness and requiring cumbersome intravenous administration. In the last decade, the discovery of the iron regulatory hormone hepcidin has revolutionized their understanding of iron pathophysiology. In this review, they revisit ID among elderly people in the light of the impressive recent advances on knowledge of iron regulation, and discuss how hepcidin may help in diagnosis and treatment of this common clinical condition.

Leibowitz D et al [57](2014) found thatthe global population is aging. Cardiovascular disease is the leading cause of death in both men and women older than 65 years. In particular, elderly patients have an increased prevalence of left ventricular hypertrophy (LVH) and chronic kidney disease (CKD), both of which predict increased cardiovascular morbidity and mortality. LVH and CKD frequently coexist in the elderly, and LVH is a powerful predictor of mortality in patients with end-stage renal disease. Several hemodynamic factors contribute to LVH and CKD in the elderly. Increased arterial stiffness in the elderly is associated with LVH and CKD. Studies using noninvasive measures of arterial stiffening have shown a correlation between these measures and LVH in patients with CKD. Hypertensive patients with an altered circadian blood pressure pattern such as nondippers have an increased incidence of LVH and CKD. Anemia is a risk factor for LVH in patients in all stages of CKD, and studies have shown correlations between age, anemia and LV mass. Non-hemodynamic factors include chronic inflammation, increased oxidative stress, and reduced autophagy, all of which are present in the elderly. Disordered mineral metabolism in the elderly with reduced levels of vitamin D and elevated levels of parathyroid hormone and phosphorus is associated with LVH and CKD. Multiple patho-

physiologic mechanisms contribute to the development of LVH and CKD in the elderly. Future research should be directed at interfering with this development and reducing the burden of cardiovascular and renal diseases in this growing population.

Stauder R et al [58](2014) found thatanemia in the elderly (defined as people aged > 65 years) is common and increasing as the population ages. In older patients, anemia of any degree contributes significantly to morbidity and mortality and has a significant effect on the quality of life. Despite its clinical importance, anemia in the elderly is under-recognized and evidence-based guidelines on its management are lacking.

Goodnough LT et al [59](2014) found thatanemia is now recognized as a risk factor for a number of adverse outcomes in the elderly, including hospitalization, morbidity, and mortality. What constitutes appropriate evaluation and management for an elderly patient with anemia, and when to initiate a referral to a hematologist, are significant issues. Attempts to identify suggested hemoglobin levels for blood transfusion therapy have been confounded for elderly patients with their co-morbidities. Since no specific recommended hemoglobin threshold has stood the test of time, prudent transfusion practices to maintain hemoglobin thresholds of 9–10 g/dl in the elderly are indicated, unless or until evidence emerges to indicate otherwise.

Bach V et al [60](2014) found thatanemia in later life is associated with increased morbidity and mortality. The purpose of this study was to evaluate the prevalence and possible causes of anemia in the elderly in a well defined hospital cohort. Participants in this cross-sectional, retrospective analysis included all inpatients and outpatients aged ≥64 years with complete blood counts treated at Innsbruck Medical University Hospital between October 1, 2004 and September 29, 2005 (n=19,758, median age 73 years). According to World Health Organization criteria, 21.1% of these patients were anemic, ie, 30.7% and 37.0% at 80+ years and 90+ years, respectively. The prevalence of anemia was significantly correlated with advanced age (r=0.21; P<0.001) and male sex (P<0.001). In anemic patients, renal insufficiency with a glomerular filtration rate <30 mL/min/1.73 m2 (11.3% versus 2.1%), hyperinflammation (62.1% versus 31.4%), absolute (14.4% versus 6.9%) or functional (28.2% versus 11.8%) iron deficiency, and folate deficiency (6.7% versus 3.0%) were observed significantly more often than in nonanemic subjects (P<0.001). The pathogenesis of anemia was multifactorial, with decreased renal function (glomerular filtration rate <60 mL/min/1.73 m2), signs of inflammation, and functional iron deficiency detected in 11.4% of anemic patients. Hemoglobin was significantly correlated with elevated C-reactive protein (r=–0.296; P<0.001) and low transferrin saturation (r=0.313; P<0.001). Mean corpuscular volume correlated only weakly with the various anemia subtypes. Cytopenias and morphologic alterations suggestive of underlying myelodysplastic syndromes were found in a substantial proportion of anemic patients, including thrombocytopenia (5.4%), leukopenia (8.26%), and macrocytic alterations (18.4%). Anemia was frequently diagnosed in this series of elderly patients. Partly treatable nutritional deficiencies, such as iron or folate deficiency, were identified as possible causes. A complex and heterogeneous interplay of chronic inflammation, functional iron deficiency, and renal impairment was identified in a large proportion of patients. A hitherto undiagnosed myelodysplastic syndrome can be assumed in a relevant proportion of patients. Morphologic classification based on mean corpuscular volume is inadequate from the standpoint of pathogenesis. New parameters are needed to differentiate the multifactorial pathogenesis of anemia in the elderly.

Raza S et al [61](2014) found thatover 10% of adults older than 65 years have World Health Organization defined anemia (Hemoglobin lower than13 g/dl in men and 12 g/dl in women). It is more prevalent with increasing age, exceeding 20% in the very elderly (85 years and older). Typical symptoms of anemia are nonspecific and often attributed to aging or to an exacerbation of another illness in the elderly. They present a case series of patients between ages 65-99 years who were followed at the Senior Health clinic and presented with nonspecific symptoms. All these patients were found to have life-threatening anemia requiring blood transfusions. Case series. All their elderly patients experienced good outcomes in terms of resolution of their symptoms and improvement in functional status. There was a significant difference in the total number of symptoms pre-transfusion compared with symptoms post-transfusion (p < 0.01). There were no adverse outcomes. Their case series suggests that symptoms of anemia in the elderly are often attributed to aging or other disease co-morbidities. Nonspecific symptoms like dyspnea, fatigue and confusion should not be ignored. Management decisions regarding anemia should involve functional assessment of the elderly subject. Immediate arrangements for transfusion must be made if the elderly patient is symptomatic regardless of the hemoglobin level. If monitored appropriately, blood transfusions can prolong survival; improve quality of life and functional status of the older individual.

Contreras-Manzano A et al [62](2015) found thatto describe de prevalence of iron deficiency (ID) and anemia in a sample of Mexican elderly population from the National Health and Nutrition Survey (Ensanut) 2012. 1 920 subjects ≥60 years of age were included. Hemoglobin, serum concentrations of ferritin and CRP were measured. The risk for ID and anemia adjusted for potential confounders was assessed in logistic regression models. The overall prevalence of anemia was 13.9%, 15.2% in males and 12.8% females. For ID, overall it was 4.2%, males 4.0% and females 4.3%. The greatest prevalence of ID was found in males and females over 80 years old (6.9 and 7.0%, respectively). ID was present in 1.5 of 10 Mexican elders with anemia. The prevalence of anemia was high in the elderly, however the prevalence of ID was low; there is a need to further investigate the causes of anemia in this age group.

Prakash KG et al [63](2015) found thatit is easy to overlook anemia in the elderly, since such symptoms as fatigue, weakness, or shortness of breath may be attributed to the aging process itself. Although the prevalence of anemia does increase with age, healthy aging is not usually associated with anemia. So, anemia should not be accepted as an inevitable consequence of aging, as a cause (often multifactorial) is identified in about 80% of the elderly patients. The purpose of this cross sectional study was to assess the clinical profile of elderly patients with anemia and to study characteristic hematological types of anemia and also to arrive at an appropriate etiological diagnosis. 50 patients aged 60years and above with Hb% <13gms% in males, and< 12 gms% in females were enrolled into the study. 64% patients were male and the mean age of patients was 66.65 years. The most common presentation was easy fatigability in 44(88%) patients, followed by dyspnoea in 35(70%) and giddiness in 30 (60%) patients. Etiologically, the most common cause of anemia in their study was anemia of chronic inflammation (26%), followed by iron deficiency anemia (24%) and hematological malignancies (18%), while B12 and Folate deficiency were responsible for 10% of anemias in the elderly. Morphologically, normocytic anemia (contributed mainly by diseases of chronic inflammation and hematological malignancies), was the most common type present in 26 (52%) patients, while 16 (32%) had microcytic anemia and 8 (16%) had macrocytic anemia with a p-value of 0.001, which was statistically significant. Anemia in elderly is not due to inevitable consequence of aging. They should be thoroughly investigated to define etiology and then appropriately treated.

Joosten E et al [64](2015) found thatanemia is an important clinical problem in older patients. The aim of the present study was 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). In the present prospective study, 191 consecutive hospitalized older patients with IDA and the ACD were investigated. A peripheral blood count, C-reactive protein, standard iron parameters, serum vitamin B12 and folate, and renal and thyroidal function tests were analyzed. The attending geriatrician was responsible for the medical diagnosis and follow up. A total of 56 patients with IDA and 135 with the ACD were investigated. Just 24 patients with IDA had normal serum folate, vitamin B12 and thyroid-stimulating hormone levels without laboratory evidence of inflammation or chronic renal failure, but one of these patients was diagnosed with hemolytic anemia. Hence, 23 patients (41%) were diagnosed with “IDA only”. “ACD only” was diagnosed in 104 patients (77%), and 22 patients (16%) with ACD had chronic renal failure. A myelodysplastic syndrome was found in two patients. Additional etiologies are often diagnosed in anemic older patients, but it remains unknown to what extent these diseases might influence the pathogenesis of the anemia. Individual and clinical judgment remain crucial to evaluating and treating older anemic patients.

Babaei M et al [65](2017) found thatdiagnosis and treatment of iron deficiency anemia in older subjects improves their quality of life. Serum ferritin as a marker of iron stores is an acute phase protein. In

older subjects who usually have many concomitant chronic medical conditions, serum ferritin may increase in response to inflammatory processes irrespective of iron stores. This study was performed to determine the diagnostic properties of serum ferritin in the diagnosis of iron deficiency anemia in older subjects. This case-control study included all the inhabitants of Amirkola town who participated in the Amirkola Health and Aging Project. Diagnosis of anemia was confirmed based on a hemoglobin level <13 g/dL in men and <12 g/dL in women and iron deficiency anemia by percent transferrin saturation <15%. A receiver operating characteristic curve was constructed to determine an optimal serum ferritin cutoff value to differentiate patients with and without iron deficiency anemia at the highest sensitivity and specificity. Eighty patients with iron deficiency anemia and 160 cases of anemia without iron deficiency (mean age: 72.9 ± 8 and 71.6 ± 7.6 years, respectively; p-value = 0.37) were analyzed. In iron deficiency anemia, the mean serum ferritin was significantly lower (p-value = 0.036) compared to patients without iron deficiency anemia. Serum ferritin with a cutoff level of 100 ng/mL differentiated patients with and without iron deficiency anemia with a sensitivity of 60% and specificity of 59% and area under the receiver operating characteristic curve of 0.615 ± 0.040 (95% confidence interval: 0.536-0.694; p-value = 0.004). These findings indicate that in elderly subjects, iron deficiency anemia may develop with higher levels of serum ferritin. Hence, the conventional cutoff of serum ferritin for the diagnosis of iron deficiency anemia in young adults is not appropriate for the elderly population.

Joosten E et al [66](2018) found thatanemia in older adults is a risk factor for numerous negative outcomes. There is no standard definition, but in most studies, anemia is defined as a hemoglobin value <12 g/dL for women and <13 g/dL for men. Absolute iron deficiency anemia is defined as the combination of anemia and the absence of total body iron. Serum ferritin is the most frequently used diagnostic parameter, but its concentration increases with age and in the presence of inflammatory diseases. Other laboratory tests, such as transferrin saturation, soluble transferrin receptor and the soluble transferrin receptor/ferritin index might provide useful information, but there is a wide variety in the cut-off values and interpretation of the results. Recent research regarding hepcidin as a central regulator of iron homeostasis is promising, but it has not been used yet for the routine diagnosis of iron deficiency anemia. In older iron deficiency anemia patients, an esophagogastroduodenoscopy and colonoscopy should be initiated in order to identify the underlying bleeding cause. CT colonography can replace a colonoscopy, and in specific cases, a video capsule is recommended. It remains crucial to keep in mind which potential benefits might be expected from these investigations in this vulnerable population, taking into account the comorbidity and life expectancy, and one should discuss in advance the possible therapeutic options and complications with the patient, a family member or a proxy. Oral iron administration is the standard treatment, but parenteral iron is a convenient and safe way to provide the total iron dose in one or a few sessions.0

Chatterjee P et al [67](2018) found thatblood is a fluid inside living creatures that carries vital substances to the cells and transports metabolic waste products away from the same cells. In vertebrates blood is composed of blood cells suspended in blood plasma. Plasma, which constitutes 55% of blood fluid, is mainly water by volume (92%). The adult human body contains approximately 5 liters of blood. This makes up about 7% of a human's weight. By the time a human is 5 or 6 years old, they have about the same amount of blood as adults do. However, their blood makes up a larger percentage of their body weight than it does in adults. The human body produces 41.6 milliliters of blood in one day; therefore 15184 milliliters of blood are produced in one year. One of the main proteins in blood plasma is the albumin protein. Its function is to regulate the osmotic pressure of the blood. Today, the concept of blood circulation seems obvious and proven, but people in the 17th century believed that blood was a static liquid in the human body. Blood could only be studied after the invention of the compound microscope by the Dutchman, Zacharias Janssen. Jan Swammerdam, another Dutchman, studied blood under the microscope and discovered 'ruddy globules' which they commonly call as red blood cells today. Now they know that the blood cells are mainly red blood cells (RBC), white blood cells (WBC), and platelets. Amongst these cells, RBC's are the most abundant. The RBC's contain hemoglobin, a tetramer protein composed of heme and globin, which binds oxygen. The function of the RBC is to deliver oxygen from the lungs to the tissues and carbon dioxide from the tissues to the lungs. If the hemoglobin is too low, then the cells in the body will not get enough oxygen and the organs would not get sufficient minerals to function properly. This condition is commonly called as Anemia which varies by age, sex, altitude, smoking, and pregnancy status. It develops when your blood lacks enough healthy red blood cells or hemoglobin; hence anemia can also be defined as the lowered ability of the blood to carry oxygen. It impairs the body's ability for gas exchange.

Stauder R et al [68](2018) found thatanemia is quite frequently diagnosed in older individuals and is a key indicator of various reactive and clonal conditions. Many underlying diseases, like myelodysplastic syndrome (MDS), develop preferentially in elderly individuals. The prevalence of anemia at older age is increasing, and this is mainly attributable to more frequently applied diagnostics and demographic changes in their societies. The etiology of anemia at older age is complex and ranges from bone marrow failure syndromes to chronic kidney disease, and from nutritional deficiencies to inflammatory processes including inflammaging in immunosenescence. In a smaller number of cases, no clear-cut etiology is identified. These patients are referred to as unexplained anemia or idiopathic cytopenia of unknown significance. In others, somatic mutations in leukocytes are found, but diagnostic criteria for MDS or other hematologic diseases are not fulfilled, a condition termed clonal cytopenia of undetermined significance. Management of anemias at older age depends on (1) the severity of the anemia, (2) underlying condition(s), and (3) patient-related factors, including comorbidities. Even a mild anemia may substantially affect physical and cognitive capacities and quality of life. An underestimated aspect is that because of age-related changes, organ function such as erythropoietin production in the kidney may become suboptimal. Management and treatment of anemia in older patients often require a multidisciplinary approach and detailed investigations of organ function. In this article, they review current concepts around anemias at older age, with special emphasis on etiologies, clinical implications, and innovative concepts in the management of these patients.

Andrès E et al [69](2018) found thatthis chapter, they report and discuss the current literature of nutrient-deficiency anemia, also called nutritional anemia, in elderly patients in developed countries. For this purpose, a review of medical literatures was conducted searching PubMed, textbooks of hematology, internal medicine and geriatrics, and information collected from international meetings were also included. The term nutrient deficiency or nutritional anemia covers any anemia, defined by a hemoglobin level <13 g/dL (<130 g/L) in men and <12 g/dL (<120 g/L) in women, resulting from a deficiency of materials essential for erythropoiesis. Patients with nutritional anemia often have mild to moderate anemia, with hemoglobin levels between 8 and 10 g/dL (80 and 100 g/L). In practice, nutritional anemia represents one-third of all anemia in elderly patients. About two-third of nutritional anemia is associated with iron deficiency and most of those cases are the result of chronic blood loss from gastrointestinal lesions. The remaining cases of nutritional anemia are usually associated with vitamin B12 (cobalamin), most frequently related to food-cobalamin malabsorption (especially in case of atrophic gastritis), and Biermer's disease (pernicious anemia); and/or vitamin B9 (folate) deficiency, most frequently related to inadequate dietary intake or malnutrition, several drugs (as methotrexate, cotrimoxazole) and chronic alcohol intake. In clinical practice, recognition of these disorders and deficiencies is essential for optimal treatment (nutrient-deficiency replacement).

Sturtzel B et al [70](2018) found thatiron deficiency is one of the most common causes of anemia in geriatric patients. Although the oral iron intake is often inadequate, the potential of iron dense foods in the daily meals of geriatric institutions is rarely considered. To test during a 1-year span whether an improved frequency of iron dense foods in the daily meals has an impact on the oral iron intake, the hemoglobin concentration and anemia prevalence of institutionalized geriatric patients. A parallel, open, pre-and post-oral nutrition intervention study. Two geriatric hospitals participated as intervention centers and one as comparison center. In the two intervention centers, a menu plan adapted with iron dense foods was applied. In the comparison center the regular meals provisions was continued. At months 1, 6 and 12 of the intervention time the routine blood-parameter hemoglobin was taken from the geriatric hospital's medical report. Component analysis assessed the nutrient density of the offered meals. 2-day-weighing records realized at month 1 and 6 of intervention-time assessed the iron intake. Ninety-nine geriatric patients in the intervention centers and 37 in the comparison center. All of them had multiple chronic diseases and an average age of 84 years. With the non-parametric Friedmann-Test

for repeated measurements, they establish differences within the groups. With the Mann-Whitney-U-Test, they establish differences between the groups. For dichotomous variables, the chi-square-test was used. A p-value of< 0.05 was considered statistically significant for all analyses. In the intervention centers the iron intake ($p < 0.001$) and the hemoglobin concentration ($p = 0.002$) improved significantly ($p < 0.001$). As in the comparison center the frequency of meat and sausage offerings was twice as much as recommended also the hemoglobin concentration improved ($p = 0.001$). Geriatric patients with anemia or low hemoglobin level benefit optimally from a diet rich in iron dense foods. Enhanced access to such can indeed correct iron deficiency anemia.

Vadakattu SS et al [71](2019) found thatthe data on the prevalence of nutritional anemia among the urban elderly population in India was limited. Hence, the present study was carried out with an aim to assess the prevalence of nutritional anemia and its association with vitamin B12, folate, ferritin and homocystine among the urban elderly population. A community- based cross-sectional study was carried out among 282 urban elderly (≥ 60 years) subjects (186 males and 96 females) in Hyderabad. Fasting blood samples were collected and hemoglobin (Hb) was estimated by cyanmethemoglobin method. Plasma Folic acid and vitamin B12 levels were estimated by RIA and homocysteine and ferritin levels were estimated by ELISA methods. The overall prevalence of anemia (Hb < 12 g/dL for females and < 13 g/dL for males) among the urban elderly was 20.6% and the prevalence was found to be increasing with the age. The prevalence of vitamin B12 (< 203 pg/mL), folic acid (< 4 ng/mL), ferritin (< 15 ng/mL) and hyperhomocysteinemia (≥12 μmol/L) in these subjects was 36.0%, 8.2%, 1.1% and 24.3% respectively. The prevalence of anemia due to deficiencies of iron (ferritin < 15 ng/mL), folate and vitamin B12 was 5.45%, 9.1% and 42.3% respectively. A significant association was observed between the prevalence of anemia with ferritin and hyperhomocysteinemia. In conclusion, the prevalence of anemia and nutritional anemia among the urban-based elderly was 20.6% and 56.85% respectively. The association of anemia with hyper-homocysteinemia needs further studies.

Pathania A et al [72](2019) found thatanemia is a common morbidity in elderly persons (aged 60 years or above). In India, in recent years, the number of old age homes (OAHs) and the residents living in them has increased significantly. The aim of this study was to estimate the prevalence of anemia among elderly persons living in OAHs. This was a cross-sectional study among individuals living in OAH in Delhi, India. Using combination of location and type of OAH, 28 clusters of almost equal sizes were created, of which 13 clusters were randomly selected, and all elderly persons living therein were selected for the study. Sociodemographic profile was recorded using a self-designed, semistructured interview schedule. Hemoglobin (Hb) was estimated using HemoCue Hb 201+ system. Binary Logistic regression was used to assess the socioeconomic determinants of anemia. The study included 334 elderly persons, with a mean (standard deviation [SD]) age of 75.2 (8.6) years and mean (SD) Hb of 11.6 (1.7) g/dL. The mean (SD) Hb in men was 12.1 (1.7) g/dL compared to 10.9 (1.5) g/dL among women ($P < 0.0001$). The overall prevalence of anemia was 68.7% (95% confidence interval 63.9, 73.4); among those who were anemic, 47.4% had mild anemia, 47.0% had moderate anemia, and 5.6% had severe anemia. The prevalence of mild anemia was 45% in men compared to 24.8% in women. The odd of anemia among ≥80 years was 2 times that among 60–69 years ($P < 0.029$). The prevalence of anemia among elderly persons in OAHs is high in Delhi, India and increased with age.

Barbind SR et al [73](2020) found thatanemia is one of the most common health problems in India. Understanding the variations among the prevalence of anemia between population groups in this large and heterogeneous country is crucial to inform relevant health policy and health service interventions. The present study was conducted to assess prevalence of anemia in the population of Nanded district, Maharashtra. All subjects between 13-70 years of age were enrolled in the study for screening of prevalence of anemia. Under complete aseptic conditions, 10 ml of blood was withdrawn from antecubital vein and 2 ml was used for complete blood count estimation and for further blood investigations as necessary. Various hematological parameters were tested in correlation to history and clinical examination of the patients. All data thus obtained was arranged in tabulated form and analyzed using SPSS software. There were a total of 2190 subjects evaluated, out of these 560 were anemic and 1630 were non-anemic. Out of 560 anemic subjects, 420 were of female gender and remaining 140 were male. The mean iron levels were 64±34 μg/dl. The mean screen ferritin levels were 201±183 ng/dl. There were 26% of subjects in their study who were anemic, and majority of them were females Although, Iron deficiency anemia revealed as a most common type of anemia, vitamin B12 and folic acid deficiencies also contributed significantly to nutritional anemias. The disease widely occurs and affects mostly children of growing age, females in reproductive ages and elderly population.

Burton JK et al [74](2020) found thatiron deficiency anaemia (IDA) is common in older adults and associated with a range of adverse outcomes. Differentiating iron deficiency from other causes of anaemia is important to ensure appropriate investigations and treatment. It is possible to make the diagnosis reliably using simple blood tests. Clinical evaluation and assessment are required to help determine the underlying cause and to initiate appropriate investigations. IDA in men and post-menopausal females is most commonly due to occult gastrointestinal blood loss until proven otherwise, although there is a spectrum of underlying causative pathologies. Investigation decisions should take account of the wishes of the patient and their competing comorbidities, individualising the approach. Management involves supplementation using oral or intravenous (IV) iron then consideration of treatment of the underlying cause of deficiency. Future research areas are outlined including the role of Hepcidin and serum soluble transferrin receptor measurement, quantitative faecal immunochemical testing, alternative dosing regimens and the potential role of IV iron preparations.

Vetrano DL et al [75](2020) found thatiron deficiency is a major cause of anemia in older people. Increasing the knowledge on the predictors of iron-deficiency anemia (IDA) may facilitate its timely diagnosis. To investigate the predictors of IDA in older people in four European countries.Retrospective longitudinal study. Primary care patients aged 65 or older (N=24,051) in four European countries. IDA predictors were estimated using multivariate Cox regression based on information gathered from national primary care databases: Italy (years 2002–2013), Belgium, Germany and Spain (years 2007–2012). Adjusted hazard ratios (aHR) with 95% confidence intervals (CIs) were estimated. In Spain and Belgium, men were at greater risk of IDA than women, while they had a lower risk in Italy. Weakness, irritability, alopecia and xerostomia were signs and symptoms significantly associated with IDA. Concurrent diseases, potential causes of anemia, positively associated with IDA were small bowel polyposis, stomach cancer, obesity, gastritis and peptic ulcer, esophagitis, Crohn's disease, celiac disease, lymphangiectasis, gastrectomy or gastric atrophy, gut resection or bypass, and cardiac prosthetic valve. Aspirin users had a 12–35% higher hazard of IDA than non-users. Similarly, corticosteroids and anti-acids were positively associated with IDA. A higher level of comorbidity was associated with an increased hazard of IDA in all countries. Specific signs and symptoms, chronic conditions, a greater comorbidity burden, and specific pharmacological treatments registered in primary care databases represent relevant predictors and correlates of incident IDA in older people in Europe. General practitioners might employ this information to obtain early diagnosis of IDA in community-dwelling older adults.

Romano AD et al [76](2020) found thatiron deficiency (ID) is the most frequent nutritional deficiency in the whole population worldwide, and the second most common cause of anemia in the elderly. The prevalence of anemia is expecting to rise shortly, because of an ageing population. Even though WHO criteria define anemia as a hemoglobin serum concentration <12 g/dL in women and <13 g/dL in men, several authors propose different and specific cut-off values for the elderly. Anemia in aged subjects impacts health and quality of life, and it is associated with several negative outcomes, such as longer time of hospitalization and a higher risk of disability. Furthermore, it is an independent risk factor of increased morbidity and mortality. Even though iron deficiency anemia is a common disorder in older adults, it should be not considered as a normal ageing consequence, but a sign of underlying dysfunction. Relating to the molecular mechanism in Iron Deficiency Anemia (IDA), hepcidin has a key role in iron homeostasis. It downregulates the iron exporter ferroportin, inhibiting both iron absorption and release. IDA is frequently dependent on blood loss, especially caused by gastrointestinal lesions. Thus, a diagnostic algorithm for IDA should include invasive investigation such as endoscopic procedures. The treatment choice is influenced by the severity of anemia, underlying conditions, comorbidities, and the clinical state of the patient. Correction of anemia and iron supplementation should be associated with the treatment of the causal

disease.

Ngum R et al [77](2021) found thatiron-deficiency anemia (IDA) is common in the elderly population. It is usually the result of chronic gastrointestinal diseases which could lead to iron losses, malabsorption, or both. IDA is most often the result of chronic gastrointestinal blood loss caused by esophagitis, gastritis, ulcer, colon cancer, pre-malignant polyps, or angiodysplasia. They are presenting a unique case that describes the unusual finding of intestinal helminthiasis in an elderly patient during endoscopic evaluation for IDA. It also touches on the risk factors, clinical manifestations, diagnosis, and treatment of enterobiasis.

Pivetta G et al [78](2022) found thatiron-deficiency anemia in the elderly may be due to numerous gastrointestinal conditions. Anemia is frequent in celiac disease (CD); however, the use of routine duodenal biopsies, independently of age or serology, is debated. To determine the diagnostic yield of routine duodenal biopsies in adult and elderly patients with no bleeding anemia, a cross-sectional study analyzing 7968 gastroscopies (2017–2020) was performed; 744 were for anemia and 275 were excluded (GI bleeding or without duodenal biopsies). Of the 469 included patients, clinical, endoscopic, and histological features were analyzed in groups with or without histopathological changes in the duodenal mucosa (DM). Univariate/multivariate analyses were performed. Of the 469 patients, 41 (8.7%) had DM histopathological changes, 12 (2.6%) had CD, 26 (5.5%) had duodenal intraepithelial lymphocytosis (DIL), and 3 had (0.6%) other conditions. They were younger compared to patients with normal DM. DM histopathology was significantly inversely correlated with age group, with prevalences of 27%, 20%, 12.5%, 10%, and 2.5%, in the <40–50, 51–60, 61–70, 71–80, and >80-year age groups, respectively ($p = 0.0010$). Logistic-regression models showed that anemic patients aged >60, >70, or >80 years with endoscopically normal DM had a progressively three- to four-fold higher probability of having normal duodenal histology. In adults, anemic patients without bleeding, age and endoscopically normal DM are predictors of normal DM histology. In >70-year anemic patients, negligible DM pathology was found. The results suggest that routine duodenal biopsies are questionable in elderly anemic patients.

MATERIALS AND METHODS

Thirty two patients of both sexes with Systemic Lupus Erythematosus (SLE), who attended the medical outpatient department, rheumatology clinic of The Tertiary Care Hospital in Kolkata or were admitted in the medical ward of the same, were selected during the period between August 2006 and July 2008. Informed and a written consent was taken from all the patients.

The tertiary care hospital situated in the southern part of Kolkata city. This hospital is a referral center for many health centers scattered over the southern part of West Bengal. Apart from that a good number of patients come from the northern part of West Bengal as well as from the eastern states of India.

Each of these 32 patients has satisfied the revised ACR classification criteria for SLE (1997 update) (4 out of 11 criteria at any time in his or her medical history).

Statistical Analysis

For statistical analysis data were entered into a Microsoft excel spreadsheet and then analyzed by SPSS (version 27.0; SPSS Inc., Chicago, IL, USA) and Graph Pad Prism version 5. Data had been summarized as mean and standard deviation for numerical variables and count and percentages for categorical variables. Two-sample t-tests for a difference in mean involved independent samples or unpaired samples. Paired t-tests were a form of blocking and had greater power than unpaired tests. One-way analysis of variance (one-way ANOVA) was a technique used to compare means of three or more samples for numerical data (using the F distribution). A chi-squared test ($\chi 2$ test) was any statistical hypothesis test wherein the sampling distribution of the test statistic is a chi-squared distribution when the null hypothesis is true. Without other qualification, 'chi-squared test' often is used as short for Pearson's chi-squared test. Unpaired proportions were compared by Chi-square test or Fischer's exact test, as appropriate.

Explicit expressions that can be used to carry out various t-tests are given below. In each case, the formula for a test statistic that either exactly follows or closely approximates a t-distribution under the null hypothesis is given. Also, the appropriate degrees of freedom are given in each case. Each of these statistics can be used to carry out either a one-tailed test or a two-tailed test.

Once a t value is determined, a p-value can be found using a table of values from Student's t-distribution. If the calculated p-value is below the threshold chosen for statistical significance (usually the 0.10, the 0.05, or 0.01 level), then the null hypothesis is rejected in favor of the alternative hypothesis.

P-value ≤ 0.05 was considered for statistically significant.

RESULT & ANALYSIS

Table: Distribution of Group

Group	Frequency	Percent
Anemic	100	72.5
No Anemic	38	27.5
Total	138	100.0%

In our study, 100 (72.5%) patients had AnemicGroup and38 (27.5%) patients had Non-AnemicGroup.

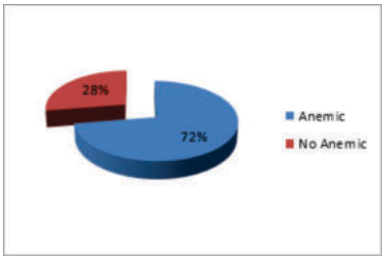


Table: Distribution of Age in Group

Age in group	Frequency	Percent
>70	49	49.0%
71-80	43	43.0%
≥81	8	8.0%
Total	100	100.0%

In our study, 49 (49.0%) patients were >70years of age, 43 (43.0%) patients were 71-80 years of age, and 8 (8.0%) patients were ≥81 years of age.

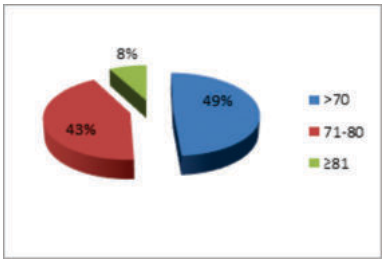


Table: Distribution of SEX

Sex	Frequency	Percent
Female	40	40.0%
Male	60	60.0%
Total	100	100.0%

In our study, 40 (40.0%) patients were Female and 60 (60.0%) patients were Male.

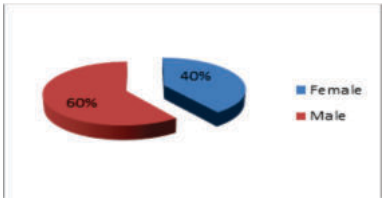


Table: Distribution of CAUSES OF IRON DEF ANEMIA

Causes Of Iron DEF Anemia	Frequency	Percent
Acute on chronic disease	55	55.0%
Dietary	17	17.0%
Hemorrhagic	18	18.0%
Hypothyroid	4	4.0%
Malignancy	6	6.0%
Total	100	100.0%

In our study, 55 (55.0%) patients had Acute on chronic disease, 17 (17.0%) patients had Dietary, 18 (55.0%) patients had Hemorrhagic, 4 (17.0%) patients had Hypothyroid, and 6 (55.0%) patients had Malignancy in Iron DEF Anemia.

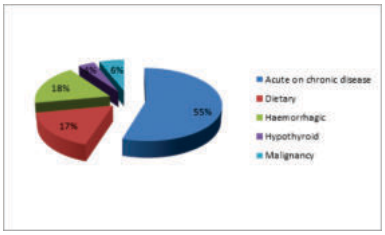


Table: Association between Age in group: CAUSES OF IRON DEF ANEMIA

Causes Of Iron DEF Anemia						
Age in group	Acute on chronic disease	Dietary	Hemor-rhagic	Hypo-thyroid	Malig-nancy	TOTAL
≤70 Row % Col %	26 53.1 47.3	8 16.3 47.1	10 20.4 55.6	3 6.1 75.0	2 4.1 33.3	49 100.0 49.0
71-80 Row % Col %	25 58.1 45.5	7 16.3 41.2	6 14.0 33.3	1 2.3 25.0	4 9.3 66.7	43 100.0 43.0
>81 Row % Col %	4 50.0 7.3	2 25.0 11.8	2 25.0 11.1	0 0.0 0.0	0 0.0 0.0	8 100.0 8.0
TOTAL Row % Col %	55 55.0 100.0	17 17.0 100.0	18 18.0 100.0	4 4.0 100.0	6 6.0 100.0	100 100.0 100.0

Chi-square value: 3.9764; p-value:0.8592

- In Acute on chronic disease, 26 (47.3%) patients were >70years of age, 25 (45.5%) patients were 71-80 years of age, and 4 (7.3%) patients were ≥81 years of age.
- In Dietary, 8 (47.1%) patients were >70years of age, 7 (41.2%) patients were 71-80 years of age, and 2 (11.8%) patients were ≥81 years of age.
- In Hemorrhagic, 10 (55.6%) patients were >70years of age, 6 (33.3%) patients were 71-80 years of age, and 2 (11.1%) patients were ≥81 years of age.
- In Hypothyroid, 3 (75.0%) patients were >70years of age and 1 (25.0%) patient was 71-80 years of age.
- In Malignancy, 2 (33.3%) patients were >70years of age and 4 (66.7%) patients were 71-80 years of age.

Association of Age in group with Causes of Iron DEF Anemiawas not statistically significant (p=0.8592).

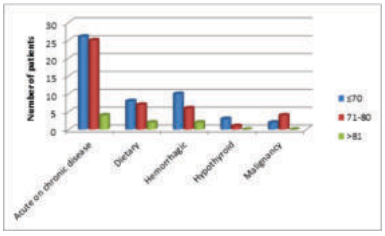


Table: Association between SEX: CAUSES OF IRON DEF ANEMIA

CAUSES OF IRON DEF ANEMIA						
SEX	Acute on chronic disease	Dietary	Haemor-rhagic	Hypo-thyroid	Malig-nancy	TOTAL
Female Row % Col %	21 52.5 38.2	2 5.0 11.8	12 30.0 66.7	1 2.5 25.0	4 10.0 66.7	40 100.0 40.0
Male Row % Col %	34 56.7 61.8	15 25.0 88.2	6 10.0 33.3	3 5.0 75.0	2 3.3 33.3	60 100.0 60.0
TOTAL Row % Col %	55 55.0 100.0	17 17.0 100.0	18 18.0 100.0	4 4.0 100.0	6 6.0 100.0	100 100.0 100.0

Chi-square value: 13.2089; p-value:0.0103

- In Acute on chronic disease, 21 (38.2%) patients were Female and 34 (61.8%) patients were Male.
- In Dietary, 2 (11.8%) patients were Female and 15 (88.2%) patients were Male.
- In Hemorrhagic, 12 (66.7%) patients were Female and 6 (33.3%) patients were Male.
- In Hypothyroid, 1 (25.0%) patient was Female and 3 (75.0%) patients were Male.
- In Malignancy, 4 (66.7%) patients were Female and 2 (33.3%) patients were Male.

Association of Sex with Causes of Iron DEF Anemia was statistically significant (p=0.0103).

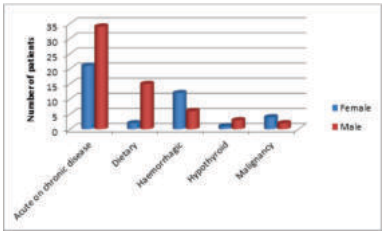


Table: Association between P SMEAR: CAUSES OF IRON DEF ANEMIA

CAUSES OF IRON DEF ANEMIA						
P SMEAR	Acute on chronic disease	Dietary	Haemor-rhagic	Hypo-thyroid	Malig-nancy	Total
DIMO-RPHIC ANAEMIA Row % Col %	1 100.0 1.8	0 0.0 0.0	0 0.0 0.0	0 0.0 0.0	0 0.0 0.0	1 100.0 1.0
MACRCY, THROMBO-CYTOPENIA Row % Col %	1 100.0 1.8	0 0.0 0.0	0 0.0 0.0	0 0.0 0.0	0 0.0 0.0	1 100.0 1.0
MACROCYTIC, NCHR Row % Col %	1 100.0 1.8	0 0.0 0.0	0 0.0 0.0	0 0.0 0.0	0 0.0 0.0	1 100.0 1.0
MCRCY, NCHR Row % Col %	1 33.3 1.8	0 0.0 0.0	0 0.0 0.0	2 66.7 50.0	0 0.0 0.0	3 100.0 3.0
MCY, HCHR Row % Col %	22 45.8 40.0	8 16.7 47.1	15 31.3 83.3	0 0.0 0.0	3 6.3 50.0	48 100.0 48.0
MCY, HCR Row % Col %	1 100.0 1.8	0 0.0 0.0	0 0.0 0.0	0 0.0 0.0	0 0.0 0.0	1 100.0 1.0
MCY,HCHR ,target & Polychromatic cells Row % Col %	0 0.0 0.0	0 0.0 0.0	0 0.0 0.0	0 0.0 0.0	1 100.0 16.7	1 100.0 1.0
MCY,HCHR, ANISO POIKILOCYTOSIS. Row % Col %	0 0.0 0.0	0 0.0 0.0	1 100.0 5.6	0 0.0 0.0	0 0.0 0.0	1 100.0 1.0
MCY,HCHR,ANISOCYTOSIS Row % Col %	1 100.0 1.8	0 0.0 0.0	0 0.0 0.0	0 0.0 0.0	0 0.0 0.0	1 100.0 1.0
NCY, NCHR Row % Col %	27 64.3 49.1	9 21.4 52.9	2 4.8 11.1	2 4.8 50.0	2 4.8 33.3	42 100.0 42.0
TOTAL Row % Col %	55 55.0 100.0	17 17.0 100.0	18 18.0 100.0	4 4.0 100.0	6 6.0 100.0	100 100.0 100.0

Chi-square value: 67.9960; p-value:0.0010

Association of P SMEAR with Causes of Iron DEF Anemia was statistically significant (p=0.0010).

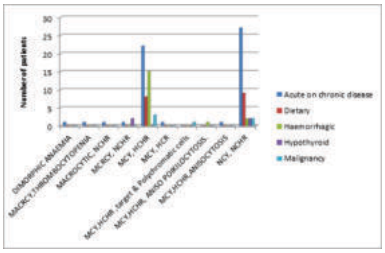


Table: Association between STOOL RE & OCLT BLD: CAUSES OF IRON DEF ANEMIA

CAUSES OF IRON DEF ANEMIA						
Stool RE & OCLT BLD	Acute on chronic disease	Dietary	Haemor-rhagic	Hypo-thyroid	Malig-nancy	TOTAL
Negative Row % Col %	51 64.6 92.7	17 21.5 100.0	1 1.3 5.6	4 5.1 100.0	6 7.6 100.0	79 100.0 79.0
Positive Row % Col %	4 19.0 7.3	0 0.0 0.0	17 81.0 94.4	0 0.0 0.0	0 0.0 0.0	21 100.0 21.0
TOTAL Row % Col %	55 55.0 100.0	17 17.0 100.0	18 18.0 100.0	4 4.0 100.0	6 6.0 100.0	100 100.0 100.0

Chi-square value: 71.9498; p-value:<0.0001

- In Acute on chronic disease, 4 (7.3%) patients had Stool RE & OCLT BLD.
- In Hemorrhagic, 17 (94.4%) patients had Stool RE & OCLT BLD.

Association of Stool RE & OCLT BLD with Causes of Iron DEF Anemiawas statistically significant (p<0.0001).

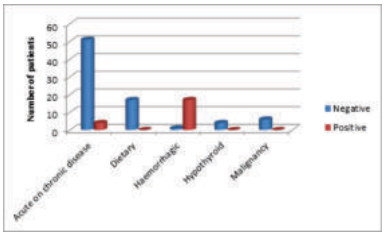


Table: Association between URINE RE & ME: CAUSES OF IRON DEF ANEMIA

CAUSES OF IRON DEF ANEMIA						
Urine RE & ME	Acute on chronic disease	Dietary	Hemor-rhagic	Hypo-thyroid	Malig-nancy	TOTAL
RBC - 10/HPF Row % Col %	0 0.0 0.0	0 0.0 0.0	0 0.0 0.0	0 0.0 0.0	1 100.0 16.7	1 100.0 1.0
WNL Row % Col %	55 55.6 100.0	17 17.2 100.0	18 18.2 100.0	4 4.0 100.0	5 5.1 83.3	99 100.0 99.0
TOTAL Row % Col %	55 55.0 100.0	17 17.0 100.0	18 18.0 100.0	4 4.0 100.0	6 6.0 100.0	100 100.0 100.0

Chi-square value: 15.8249; p-value:0.0033

- In Acute on chronic disease, 55 (100.0%) patients had WNL.
- In Dietary, 17 (100.0%) patients had WNL.
- In Hemorrhagic, 18 (100.0%) patients had WNL.
- In Hypothyroid, 4 (100.0%) patients had WNL.
- In Malignancy, 1 (16.7%) patient had RBC - 10/HPF and 4 (83.3%) patients had WNL.

Association of URINE RE & ME with Causes Of Iron DEF Anemia was statistically significant (p=0.0033).

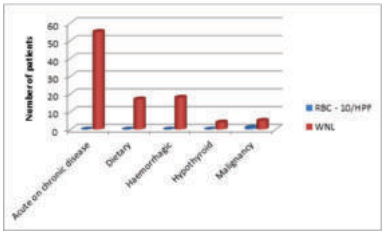


Table: Association between USG: CAUSES OF IRON DEF ANEMIA

	CAUSES OF IRON DEF ANEMIA					
USG	Acute on chronic disease	Dietary	Hemor-rhagic	Hypo-thyroid	Malig-nancy	TOTAL
NAD Row % Col %	55 55.0 100.0	17 17.0 100.0	18 18.0 100.0	4 4.0 100.0	6 6.0 100.0	100 100.0 100.0
TOTAL Row % Col %	55 55.0 100.0	17 17.0 100.0	18 18.0 100.0	4 4.0 100.0	6 6.0 100.0	100 100.0 100.0

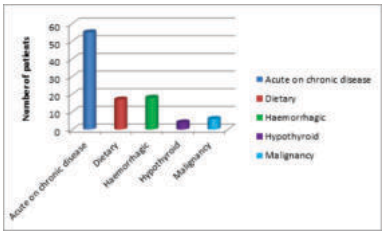


Table: Association between DIET: CAUSES OF IRON DEF ANEMIA

	CAUSES OF IRON DEF ANEMIA					
Diet	Acute on chronic disease	Dietary	Hemor-rhagic	Hypo-thyroid	Malig-nancy	TOTAL
Non-Veg Row % Col %	52 65.8 94.5	0 0.0 0.0	17 21.5 94.4	4 5.1 100.0	6 7.6 100.0	79 100.0 79.0
Veg Row % Col %	3 14.3 5.5	17 81.0 100.0	1 4.8 5.6	0 0.0 0.0	0 0.0 0.0	21 100.0 21.0
Total Row % Col %	55 55.0 100.0	17 17.0 100.0	18 18.0 100.0	4 4.0 100.0	6 6.0 100.0	100 100.0 100.0

Chi-square value: 77.2103; p-value:<0.0001

- In Acute on chronic disease, 52 (94.5%) patients had non-veg-diet and 3 (5.5%) patients had veg diet.
- In Dietary, 17 (100.0%) patients had veg-diet.
- In Hemorrhagic, 17 (94.4%) patients had non-veg-diet and 1 (5.6%) patient had veg diet.
- In Hypothyroid, 4 (100.0%) patients had non-veg-diet.
- In Malignancy, 6 (100.0%) patients had non-veg-diet.

Association of Diet in Years with Causes of Iron DEF Anemia was statistically significant (p<0.0001).

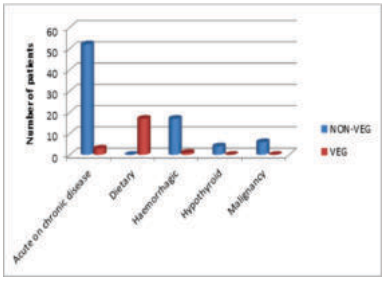


Table: Distribution of mean AGE: CAUSES OF IRON DEF ANEMIA

		Number	Mean	SD	Mini-mum	Maxi-mum	Median	p-value

AGE	Acute on chronic disease	55	72.1636	6.2026	65.0000	91.0000	71.0000	0.8826
	Dietary	17	72.2941	7.9511	65.0000	91.0000	72.0000	
	Hemorrhagic	18	71.8889	6.2862	65.0000	86.0000	70.0000	
	Hypothyroid	4	68.7500	7.5000	65.0000	80.0000	65.0000	
	Malignancy	6	71.1667	3.8687	65.0000	75.0000	72.5000	

- In Acute on chronic disease, the mean Age (mean± s.d.) of patients was 72.1636± 6.2026.
- In Dietary, the mean Age (mean± s.d.) of patients was 72.2941± 7.9511.
- In Hemorrhagic, the mean Age (mean± s.d.) of patients was 71.8889± 6.2862.
- In Hypothyroid, the mean Age (mean± s.d.) of patients was 68.7500± 7.5000.
- In Malignancy, the mean Age (mean± s.d.) of patients was 71.1667± 3.8687.

Distribution of mean Age with causes of iron DEF anemiawas not statistically significant (p=0.8826).

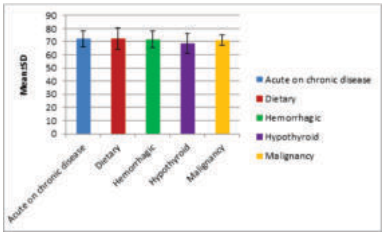


Table: Distribution of mean HB% (12-17 gm /dl): CAUSES OF IRON DEFANEMIA

		Number	Mean	SD	Minimum	Maximum	Median	p-value
HB% (12-17 gm /dl)	Acute on chronic disease	55	9.0982	1.5507	4.3000	11.7000	9.0000	0.1777
	Dietary	17	9.8059	1.3122	7.6000	11.6000	9.7000	
	Hemorrhagic	18	8.8056	2.0675	3.6000	11.4000	9.1500	
	Hypothyroid	4	10.5000	1.3115	8.6000	11.6000	10.9000	
	Malignancy	6	9.5667	1.6884	7.1000	11.5000	9.7000	

- In Acute on chronic disease, the mean HB% (12-17 gm /dl) (mean± s.d.) of patients was 9.0982± 1.5507.
- In Dietary, the mean HB% (12-17 gm /dl) (mean± s.d.) of patients was 9.8059± 1.3122.
- In Hemorrhagic, the mean HB% (12-17 gm /dl) (mean± s.d.) of patients was 8.8056± 2.0675.
- In Hypothyroid, the mean HB% (12-17 gm /dl) (mean± s.d.) of patients was 10.5000± 1.3115.
- In Malignancy, the mean HB% (12-17 gm /dl) (mean± s.d.) of patients was 9.5667± 1.6884.

Distribution of mean HB% (12-17 gm /dl) with causes of iron DEF anemiawas not statistically significant (p=0.1777).

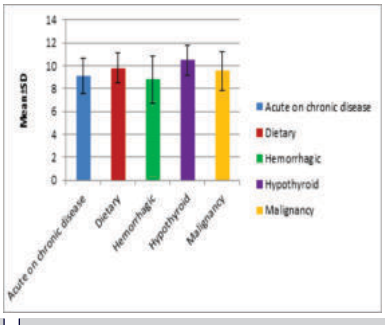


Table: Distribution of mean RBC TOTAL (3 ,8-5, 5) 10^6/ ml: CAUSES OF IRON DEFANEMIA

		Number	Mean	SD	Minimum	Maximum	Median	p-value
RBC Total (3, 8-5, 5) 10^6/ ml	Acute on chronic disease	55	2.8587	.5905	1.8000	3.8800	2.8000	0.5364
	Dietary	17	3.0612	.5673	2.1000	3.8200	2.9200	
	Hemorrhagic	18	2.9317	.6551	1.8600	3.8600	2.9500	
	Hypothyroid	4	3.2150	.7422	2.3800	3.8700	3.3050	
	Malignancy	6	3.1417	.6452	2.3600	3.9000	3.2050	

- In Acute on chronic disease, the mean RBC TOTAL (3, 8-5, 5) 10^6/ ml (mean± s.d.) of patients was 2.8587± .5905.
- In Dietary, the mean RBC TOTAL (3, 8-5, 5) 10^6/ ml (mean± s.d.) of patients was 3.0612± .5673.
- In Hemorrhagic, the mean RBC TOTAL (3, 8-5, 5) 10^6/ ml (mean± s.d.) of patients was 2.9317± .6551.
- In Hypothyroid, the mean RBC TOTAL (3, 8-5, 5) 10^6/ ml (mean± s.d.) of patients was 3.2150± .7422.
- In Malignancy, the mean RBC TOTAL (3, 8-5, 5) 10^6/ ml (mean± s.d.) of patients was 3.1417± .6452.

Distribution of mean RBC TOTAL (3, 8-5, 5) 10^6/ ml with causes of iron DEF anemiawas not statistically significant (p=0.5364).

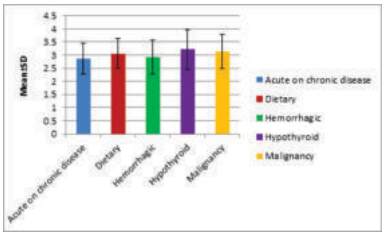


Table: Distribution of mean TLC 4000-10000/ Cumm): CAUSES OF IRON DEFANEMIA

		Number	Mean	SD	Minimum	Maximum	Median	p-value
TLC 4000-10000/ Cumm)	Acute on chronic disease	55	11798.1818	5934.7218	3300.0000	32800.0000	10400.0000	0.0012
	Dietary	17	6047.0588	1809.0459	3100.0000	9200.0000	6100.0000	
	Hemorrhagic	18	9150.0000	3730.9910	5300.0000	19500.0000	7850.0000	
	Hypothyroid	4	9225.0000	771.9024	8500.0000	10300.0000	9050.0000	
	Malignancy	6	9316.6667	2434.2692	6500.0000	12400.0000	9000.0000	

- In Acute on chronic disease, the mean TLC 4000-10000/ Cumm) (mean± s.d.) of patients was 11798.1818± 5934.7218.
- In Dietary, the mean TLC 4000-10000/ Cumm) (mean± s.d.) of patients was 6047.0588± 1809.0459.
- In Hemorrhagic, the mean TLC 4000-10000/ Cumm) (mean± s.d.) of patients was 9150.0000± 3730.9910.
- In Hypothyroid, the mean TLC 4000-10000/ Cumm) (mean± s.d.) of patients was 9225.0000± 771.9024.
- In Malignancy, the mean TLC 4000-10000/ Cumm) (mean± s.d.) of patients was 9316.6667± 2434.2692.

Distribution of mean TLC 4000-10000/ Cumm) with causes of iron DEF anemiawas statistically significant (p=0.0012).

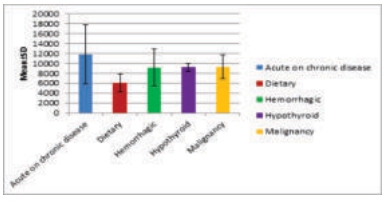


Table: Distribution of mean N (40-75)% : CAUSES OF IRON DEF ANEMIA

		Number	Mean	SD	Minimum	Maximum	Median	p-value
N (40-75) %	Acute on chronic disease	55	77.9273	12.2079	17.0000	93.0000	80.0000	0.0230
	Dietary	17	68.8235	13.0969	41.0000	85.0000	74.0000	
	Hemorrhagic	18	70.1667	9.3761	54.0000	87.0000	69.0000	
	Hypothyroid	4	78.5000	5.0000	72.0000	84.0000	79.0000	
	Malignancy	6	71.8333	11.7544	57.0000	87.0000	71.5000	

- In Acute on chronic disease, the mean N (40-75) %(mean± s.d.) of patients was 77.9273± 12.2079.
- In Dietary, the mean N (40-75) %(mean± s.d.) of patients was 68.8235± 13.0969.
- In Hemorrhagic, the mean N (40-75) %(mean± s.d.) of patients was 70.1667± 9.3761.
- In Hypothyroid, the mean N (40-75) %(mean± s.d.) of patients was 78.5000± 5.0000.
- In Malignancy, the mean N (40-75) %(mean± s.d.) of patients was 71.8333± 11.7544.

Distribution of mean N (40-75) %with causes of iron DEF anemia was statistically significant (p=0.0230).

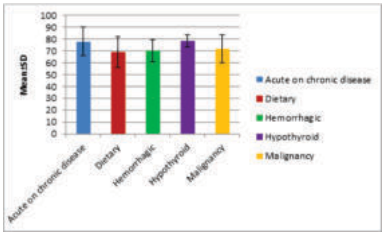


Table: Distribution of mean E (1-6) %: CAUSES OF IRON DEF ANEMIA

		Number	Mean	SD	Minimum	Maximum	Median	p-value
E (1-6) %	Acute on chronic disease	55	2.3091	1.5501	0.0000	9.0000	2.0000	0.1619
	Dietary	17	4.1176	4.8462	1.0000	18.0000	2.0000	
	Hemorrhagic	18	3.3333	3.7417	1.0000	17.0000	2.0000	
	Hypothyroid	4	2.0000	.8165	1.0000	3.0000	2.0000	
	Malignancy	6	2.1667	.7528	1.0000	3.0000	2.0000	

- In Acute on chronic disease, the mean E (1-6) %(mean± s.d.) of patients was 2.3091± 1.5501.
- In Dietary, the mean E (1-6) %(mean± s.d.) of patients was 4.1176± 4.8462.
- In Hemorrhagic, the mean E (1-6) %(mean± s.d.) of patients was 3.3333± 3.7417.
- In Hypothyroid, the mean E (1-6) %(mean± s.d.) of patients was 2.0000± .8165.
- In Malignancy, the mean E (1-6) %(mean± s.d.) of patients was 2.1667± .7528.

Distribution of mean E (1-6) %with causes of iron DEF anemiawas not statistically significant (p=0.1619).

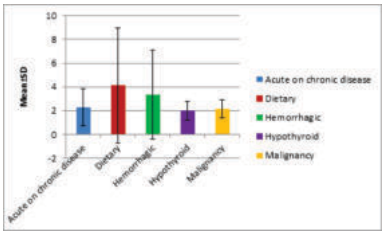


Table: Distribution of mean B (0-2) %: CAUSES OF IRON DEF ANEMIA

		Number	Mean	SD	Minimum	Maximum	Median	p-value
B (0-2) %	Acute on chronic disease	55	.0182	.1348	0.0000	1.0000	0.0000	0.2012
	Dietary	17	.1250	.3416	0.0000	1.0000	0.0000	
	Hemorrhagic	18	.0000	.0000	0.0000	0.0000	0.0000	
	Hypothyroid	4	.0000	.0000	0.0000	0.0000	0.0000	
	Malignancy	6	.0000	.0000	0.0000	0.0000	0.0000	

- In Acute on chronic disease, the mean B (0-2) % (mean± s.d.) of patients was .0182± .1348.
- In Dietary, the mean B (0-2) % (mean± s.d.) of patients was .1250± .3416.
- In Hemorrhagic, the mean B (0-2) % (mean± s.d.) of patients was .0000± .0000.
- In Hypothyroid, the mean B (0-2) % (mean± s.d.) of patients was .0000± .0000.
- In Malignancy, the mean B (0-2) %(mean± s.d.) of patients was .0000± .0000.

Distribution of mean B (0-2) %with causes of iron DEF anemia was not statistically significant (p=0.2012).

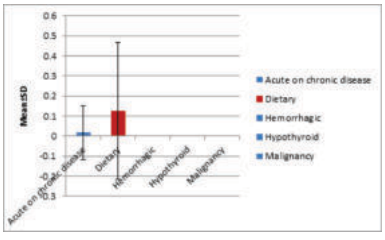


Table: Distribution of mean L (20-40) %: CAUSES OF IRON DEF ANEMIA

		Number	Mean	SD	Minimum	Maximum	Median	p-value
L (20-40) %	Acute on chronic disease	55	17.4727	12.1851	3.0000	82.0000	16.0000	0.0330
	Dietary	17	24.8235	10.5430	8.0000	43.0000	26.0000	
	Hemorrhagic	18	25.2222	7.8482	10.0000	39.0000	27.0000	
	Hypothyroid	4	17.5000	5.4467	12.0000	25.0000	16.5000	
	Malignancy	6	24.0000	11.0454	10.0000	40.0000	24.5000	

- In Acute on chronic disease, the mean L (20-40) %(mean± s.d.) of patients was 17.4727± 12.1851.
- In Dietary, the mean L (20-40) %(mean± s.d.) of patients was 24.8235± 10.5430.
- In Hemorrhagic, the mean L (20-40) %(mean± s.d.) of patients was 25.2222± 7.8482.
- In Hypothyroid, the mean L (20-40) %(mean± s.d.) of patients was 17.5000± 5.4467.
- In Malignancy, the mean L (20-40) %(mean± s.d.) of patients was 24.0000± 11.0454.

Distribution of mean L (20-40) %with causes of iron DEF anemiawas statistically significant (p=0.0330).

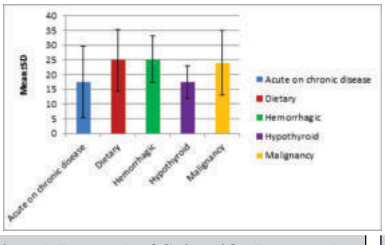


Table: Distribution of mean M (2-10)% : CAUSES OF IRON DEF ANEMIA

		Number	Mean	SD	Minimum	Maximum	Median	p-value
M (2-10) %	Acute on chronic disease	55	1.9636	1.4006	0.0000	6.0000	2.0000	0.1467
	Dietary	17	1.3529	.9315	0.0000	3.0000	1.0000	
	Hemorrhagic	18	1.2222	.6468	0.0000	2.0000	1.0000	
	Hypothyroid	4	2.0000	1.4142	1.0000	4.0000	1.5000	
	Malignancy	6	2.0000	1.6733	1.0000	5.0000	1.0000	

- In Acute on chronic disease, the mean M (2-10) %(mean± s.d.) of patients was 1.9636± 1.4006.
- In Dietary, the mean M (2-10) %(mean± s.d.) of patients was 1.3529± .9315.
- In Hemorrhagic, the mean M (2-10) %(mean± s.d.) of patients was 1.2222± .6468.
- In Hypothyroid, the mean M (2-10) %(mean± s.d.) of patients was 2.0000± 1.4142.
- In Malignancy, the mean M (2-10) %(mean± s.d.) of patients was 2.0000± 1.6733.

Distribution of mean M (2-10) %with causes of iron DEF anemia was not statistically significant (p=0.1467).

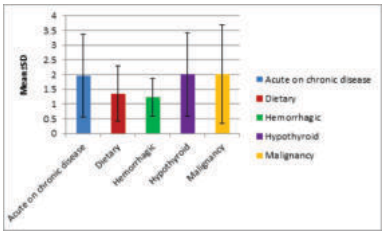


Table: Distribution of mean ESR: CAUSES OF IRON DEF ANEMIA

		Number	Mean	SD	Minimum	Maximum	Median	p-value
ESR	Acute on chronic disease	55	74.4907	38.5250	4.0000	180.0000	70.0000	0.0332
	Dietary	17	55.6471	20.2575	20.0000	86.0000	56.0000	
	Hemorrhagic	18	49.7222	33.0397	6.0000	128.0000	39.0000	
	Hypothyroid	4	44.7500	6.1847	36.0000	50.0000	46.5000	
	Malignancy	6	69.8333	19.1877	45.0000	98.0000	72.0000	

- In Acute on chronic disease, the meanESR(mean± s.d.) of patients was 74.4907± 38.5250.
- In Dietary, the mean ESR(mean± s.d.) of patients was 55.6471± 20.2575.
- In Hemorrhagic, the mean ESR(mean± s.d.) of patients was 49.7222± 33.0397.
- In Hypothyroid, the mean ESR(mean± s.d.) of patients was 44.7500± 6.1847.
- In Malignancy, the mean ESR(mean± s.d.) of patients was 69.8333± 19.1877.

Distribution of mean ESR with causes of iron DEF anemia was statistically significant (p=0.0332).

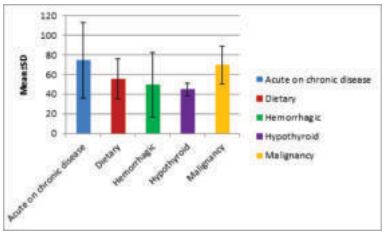
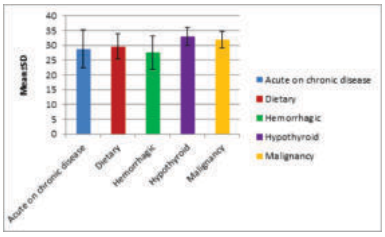


Table: Distribution of mean PCV (36-47) %: CAUSES OF IRON DEFANEMIA

		Number	Mean	SD	Minimum	Maximum	Median	p-value
PCV (36-47) %	Acute on chronic disease	55	28.8018	6.3230	2.0000	38.4000	29.0000	0.3071
	Dietary	17	29.5563	4.2716	22.0000	37.0000	30.0000	
	Hemorrhagic	18	27.5118	5.6587	12.9000	36.0000	28.0000	
	Hypothyroid	4	33.0000	3.1623	29.0000	36.0000	33.5000	
	Malignancy	6	31.8833	2.7644	28.0000	35.0000	31.6500	

- In Acute on chronic disease, the mean PCV (36-47) % (mean± s.d.) of patients was 28.8018± 6.3230.
- In Dietary, the mean PCV (36-47) % (mean± s.d.) of patients was 29.5563± 4.2716.
- In Hemorrhagic, the mean PCV (36-47) % (mean± s.d.) of patients was 27.5118± 5.6587.
- In Hypothyroid, the mean PCV (36-47) % (mean± s.d.) of patients was 33.0000± 3.1623.
- In Malignancy, the mean PCV (36-47) % (mean± s.d.) of patients was 31.8833± 2.7644.

Distribution of mean PCV (36-47) % with causes of iron DEF anemiawas not statistically significant (p=0.3071).



Distribution of mean MCV (83-101) %: CAUSES OF IRON DEF ANEMIA

		Number	Mean	SD	Minimum	Maximum	Median	p-value
MCV (83-101)	Acute on chronic disease	55	84.1545	8.0886	65.0000	107.0000	82.5000	0.0006
	Dietary	17	81.0294	13.8118	36.0000	102.0000	80.0000	
	Hemorrhagic	18	74.8667	5.2713	64.9000	89.0000	74.0000	
	Hypothyroid	4	94.5000	10.3441	82.0000	103.0000	96.5000	
	Malignancy	6	83.3333	11.2901	76.0000	106.0000	79.0000	

- In Acute on chronic disease, the mean MCV (83-101) (mean± s.d.) of patients was 84.1545± 8.0886.
- In Dietary, the mean MCV (83-101) (mean± s.d.) of patients was 81.0294± 13.8118.
- In Hemorrhagic, the mean MCV (83-101) (mean± s.d.) of patients was 27.5118± 5.2713.
- In Hypothyroid, the mean MCV (83-101) (mean± s.d.) of patients was 94.5000± 10.3441.
- In Malignancy, the mean MCV (83-101) (mean± s.d.) of patients was 83.3333± 11.2901.

Distribution of mean MCV (83-101) with causes of iron DEF anemia was statistically significant (p=0.0006).

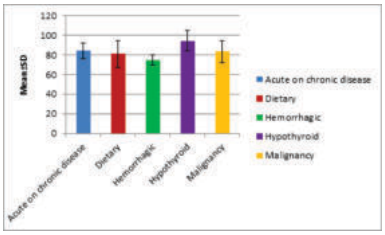


Table: Distribution of mean MCH (27-32) %: CAUSES OF IRON DEFANEMIA

		Number	Mean	SD	Mini-mum	Maxi-mum	Media n	p-value
MCH (27-32) %	Acute on chronic disease	55	27.2218	3.1461	18.6000	33.9000	27.0000	0.0023
	Dietary	17	27.5176	3.4109	23.7000	38.0000	26.0000	
	Hemor-rhagic	18	23.9889	2.1655	19.6000	28.4000	24.0000	
	Hypo-thyroid	4	27.9500	2.7923	26.0000	32.0000	26.9000	
	Malig-nancy	6	27.3667	3.5223	24.0000	34.2000	26.5000	

- In Acute on chronic disease, the mean MCH (27-32) % (mean± s.d.) of patients was 27.2218± 3.1461.
- In Dietary, the mean MCH (27-32) % (mean± s.d.) of patients was 27.5176± 3.4109.
- In Hemorrhagic, the mean MCH (27-32) % (mean± s.d.) of patients was 23.9889± 2.1655.
- In Hypothyroid, the mean MCH (27-32) % (mean± s.d.) of patients was 27.9500± 2.7923.
- In Malignancy, the mean MCH (27-32) % (mean± s.d.) of patients was 27.3667± 3.5223.

Distribution of mean MCH (27-32) % with causes of iron DEF anemiawas statistically significant (p=0.0023).

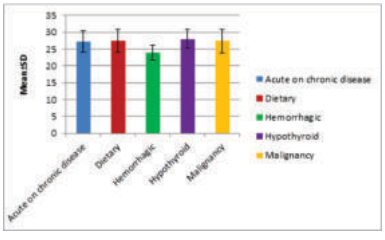


Table: Distribution of mean MCHC(31,5-34,5)% : CAUSES OF IRON DEFANEMIA

		Number	Mean	SD	Mini-mum	Maxi mum	Media n	p-value
MCHC (31,5-34,5) %	Acute on chronic disease	55	31.0036	1.9384	26.0000	36.0000	31.1000	0.3695
	Dietary	17	31.3176	1.9555	28.0000	36.0000	31.0000	
	Hemor-rhagic	18	30.2556	1.3993	27.9000	32.0000	30.0000	
	Hypo-thyroid	4	31.7250	1.2580	30.0000	33.0000	31.9500	
	Malig-nancy	6	30.5667	1.6021	28.0000	32.4000	30.5000	

- In Acute on chronic disease, the meanMCHC(31,5-34,5)% (mean± s.d.) of patients was 31.0036± 1.9384.
- In Dietary, the mean MCHC (31,5-34,5)% (mean± s.d.) of patients was 31.3176± 1.9555.
- In Hemorrhagic, the mean MCHC (31,5-34,5)% (mean± s.d.) of patients was 30.2556± 1.3993.
- In Hypothyroid, the mean MCHC (31,5-34,5)% (mean± s.d.) of patients was 31.7250± 2.7923.
- In Malignancy, the mean MCHC (31,5-34,5)% (mean± s.d.) of patients was 30.5667± 1.6021.

Distribution of mean MCHC (31,5-34,5)% with causes of iron DEF anemiawas not statistically significant (p=0.3695).

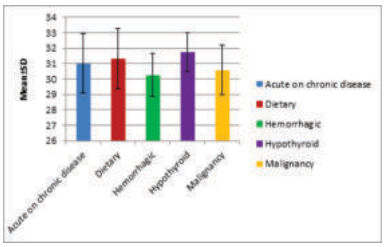


Table: Distribution of mean RETIC COUNT (0,5-2,5) %: CAUSES OF IRON DEFANEMIA

		Number	Mean	SD	Mini-mum	Maxi-mum	Media n	p-value
RETIC Count (0,5-2,5) %	Acute on chronic disease	55	1.3604	.4267	0.8000	2.5000	1.2000	<0.0001
	Dietary	17	1.5563	.5354	0.9000	2.6000	1.3500	
	Hemor-rhagic	18	2.2412	1.0989	1.2000	5.9000	2.1000	
	Hypo-thyroid	4	1.1250	.3594	0.8000	1.6000	1.0500	
	Malig-nancy	6	1.7833	.5776	1.1000	2.5000	1.9000	

- In Acute on chronic disease, the mean RETIC COUNT (0,5-2,5) % (mean± s.d.) of patients was 1.3604± .4267.
- In Dietary, the mean RETIC COUNT (0,5-2,5) % (mean± s.d.) of patients was 1.5563± .5354.
- In Hemorrhagic, the mean RETIC COUNT (0,5-2,5) % (mean± s.d.) of patients was 2.2412± 1.0989.
- In Hypothyroid, the mean RETIC COUNT (0,5-2,5) % (mean± s.d.) of patients was 1.1250± 2.7923.
- In Malignancy, the mean RETIC COUNT (0,5-2,5) % (mean± s.d.) of patients was 1.7833± .5776.

Distribution of mean RETIC COUNT (0,5-2,5) % with causes of iron DEF anemia was statistically significant (p<0.0001).

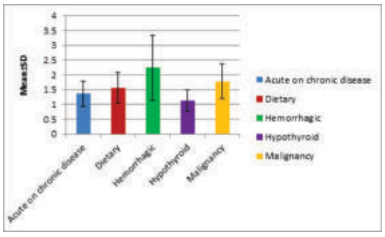


Table: Distribution of mean PLATELETS (150-450) X 10^3 microl: CAUSES OF IRON DEFANEMIA

		Number	Mean	SD	Mini-mum	Maxi-mum	Media n	p-value
Platelets (150-450) X 10^3 microl	Acute on chronic disease	55	219.7636	93.5355	60.0000	488.0000	195.0000	0.2286
	Dietary	17	183.6471	59.5965	100.0000	320.0000	170.0000	
	Hemor-rhagic	18	182.7222	66.7249	119.0000	400.0000	155.0000	
	Hypo-thyroid	4	156.5000	19.9750	140.0000	185.0000	150.5000	
	Malig-nancy	6	208.8333	68.3825	150.0000	310.0000	176.5000	

- In Acute on chronic disease, the mean PLATELETS (150-450) X 10^3 microl (mean± s.d.) of patients was 219.7636± 93.5355.
- In Dietary, the mean PLATELETS (150-450) X 10^3 microl (mean± s.d.) of patients was 183.6471± 59.5965.
- In Hemorrhagic, the mean PLATELETS (150-450) X 10^3 microl (mean± s.d.) of patients was 182.7222± 66.7249.
- In Hypothyroid, the mean PLATELETS (150-450) X 10^3 microl (mean± s.d.) of patients was 156.5000± 19.9750.
- In Malignancy, the mean PLATELETS (150-450) X 10^3 microl (mean± s.d.) of patients was 208.8333± 68.3825.

Distribution of mean PLATELETS (150-450) X 10^3 microl with causes of iron DEF anemiawas not statistically significant (p=0.2286).

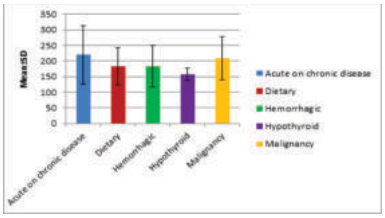


Table: Distribution of mean CRP: CAUSES OF IRON DEF ANEMIA

		Number	Mean	SD	Mini-mum	Maxi-mum	Median	p-value
CRP	Acute on chronic disease	55	62.3709	60.0772	8.0000	250.0000	34.0000	<0.0001
	Dietary	17	3.0882	1.4551	0.9000	5.1000	3.0000	
	Hemor-rhagic	18	5.1941	4.9913	2.0000	24.0000	4.1000	
	Hypo-thyroid	4	4.0250	.6652	3.5000	5.0000	3.8000	
	Malig-nancy	6	19.4167	9.8205	2.5000	32.0000	19.5000	

- InAcute on chronic disease, the mean CRP (mean± s.d.) of patients was 62.3709± 60.0772.
- In Dietary, the mean CRP (mean± s.d.) of patients was 3.0882± 1.4551.
- In Hemorrhagic, the mean CRP (mean± s.d.) of patients was 5.1941± 4.9913.
- In Hypothyroid, the mean CRP (mean± s.d.) of patients was 4.0250± .6652.
- In Malignancy, the mean CRP (mean± s.d.) of patients was 19.4167± 9.8205.

Distribution of mean CRP with causes of iron DEF anemia was statistically significant (p<0.0001).

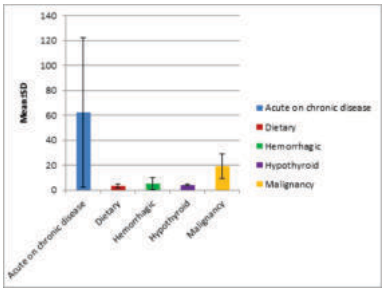


Table: Distribution of mean SR IRON (49-181) mcg/dl: Causes Of Iron DEF Anemia

		Number	Mean	SD	Mini-mum	Maxi-mum	Median	p-value
SR Iron (49-181) mcg/dl	Acute on chronic disease	55	45.0322	12.9027	9.1700	84.0000	45.0000	0.0076
	Dietary	17	46.4588	14.2436	21.8000	80.0000	45.0000	
	Hemor-rhagic	18	32.2167	16.1602	2.0000	57.0000	33.5000	
	Hypo-thyroid	4	41.2500	6.7020	36.0000	51.0000	39.0000	
	Malig-nancy	6	50.5000	17.8746	36.0000	84.0000	44.5000	

- InAcute on chronic disease, the mean SR IRON (49-181) mcg/dl (mean± s.d.) of patients was 45.0322± 12.9027.
- In Dietary, the mean SR IRON (49-181) mcg/dl (mean± s.d.) of patients was 46.4588± 14.2436.
- In Hemorrhagic , the mean SR IRON (49-181) mcg/dl (mean± s.d.) of patients was 34.0000± 10.4499.
- In Hypothyroid , the mean SR IRON (49-181) mcg/dl (mean± s.d.) of patients was 41.2500 ± 6.7020
- In Malignancy , the mean SR IRON (49-181) mcg/dl (mean± s.d.) of patients was 50.5000± 17.8746.

Distribution of mean SR IRON (49-181) mcg/dl with causes of iron DEF anemiawas statistically significant (p=0.0076).

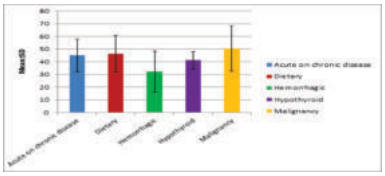


Table: Distribution of mean TIBC (250-450) mcg/dl: CAUSES OF IRON DEF ANEMIA

		Number	Mean	SD	Mini-mum	Maxi-mum	Median	p-value
TIBC (250-450) mcg/dl	Acute on chronic disease	55	326.4727	97.3451	104.0000	502.0000	317.0000	0.0470
	Dietary	17	341.1765	94.1310	104.0000	480.0000	318.0000	
	Hemor-rhagic	18	402.6111	73.1752	286.0000	510.0000	405.5000	
	Hypo-thyroid	4	375.2500	91.1533	317.0000	510.0000	337.0000	
	Malig-nancy	6	361.6667	70.7832	258.0000	417.0000	396.0000	

- InAcute on chronic disease, the mean TIBC (250-450) mcg/dl (mean± s.d.) of patients was 326.4727± 326.4727.
- In Dietary, the mean TIBC (250-450) mcg/dl(mean± s.d.) of patients was 341.1765± 94.1310.
- In Hemorrhagic, the mean TIBC (250-450) mcg/dl (mean± s.d.) of patients was 402.6111± 73.1752.
- In Hypothyroid, the mean TIBC (250-450) mcg/dl (mean± s.d.) of patients was 375.2500± 91.1533
- In Malignancy, the mean TIBC (250-450) mcg/dl (mean± s.d.) of patients was 361.6667± 70.7832.

Distribution of mean TIBC (250-450) mcg/dl with causes of iron DEF anemia was statistically significant (p=0.0470).

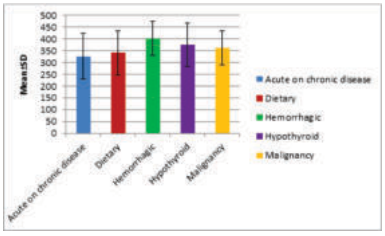


Table: Distribution of mean SR FERRITIN (10-291) ng/ml: CAUSES OF IRON DEF ANEMIA

		Number	Mean	SD	Mini-mum	Maxi-mum	Median	p-value
SR Ferritin (10-291) ng/ml	Acute on chronic disease	55	21.2673	24.1141	0.3500	86.7000	9.2500	0.9416
	Dietary	17	18.2506	15.1777	1.2000	48.0000	12.6000	
	Hemor-rhagic	18	20.1594	18.0090	4.7100	52.0000	9.4850	
	Hypo-thyroid	4	13.0150	5.5984	7.8000	20.0000	12.1300	
	Malig-nancy	6	21.6250	19.4562	1.2400	47.0000	19.4000	

- InAcute on chronic disease, the mean SR FERRITIN (10-291) ng/ml (mean± s.d.) of patients was 21.2673± 24.1141.
- In Dietary, the mean SR FERRITIN (10-291) ng/ml (mean± s.d.) of patients was 18.2506± 15.1777.
- In Hemorrhagic, the mean SR FERRITIN (10-291) ng/ml (mean± s.d.) of patients was 20.1594± 18.0090.
- In Hypothyroid, the mean SR FERRITIN (10-291) ng/ml (mean± s.d.) of patients was 13.0150 ± 5.5984
- In Malignancy, the mean SR FERRITIN (10-291) ng/ml (mean± s.d.) of patients was 21.6250± 19.4562.

Distribution of mean SR FERRITIN (10-291) ng/ml with causes of iron DEF anemiawas not statistically significant (p=0.9416).

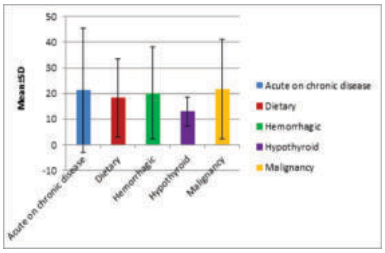


Table: Distribution of mean SR TRANSFERRIN SAT (15-50) (%): CAUSES OF IRON DEFANEMIA

		Number	Mean	SD	Minimum	Maximum	Median	p-value
SR Transferrin SAT (15-50) (%)	Acute on chronic disease	55	14.9182	6.0014	2.6000	25.1000	14.2000	0.0027
	Dietary	17	13.0765	5.5492	3.7000	21.1000	12.3000	
	Hemorrhagic	18	8.7244	4.9156	1.3400	17.3000	7.7000	
	Hypothyroid	4	10.9750	1.0112	9.5000	11.8000	11.3000	
	Malignancy	6	14.2667	4.8194	8.9000	20.1000	14.0500	

- InAcute on chronic disease, the mean SR TRANSFERRIN SAT (15-50) (%)(mean± s.d.)of patients was 14.9182± 6.0014.
- In Dietary, the mean SR TRANSFERRIN SAT (15-50) (%)(mean± s.d.)of patients was 13.0765± 5.5492.
- In Hemorrhagic, the mean SR TRANSFERRIN SAT (15-50) (%)(mean± s.d.)of patients was 8.7244± 4.9156.
- In Hypothyroid, the mean SR TRANSFERRIN SAT (15-50) (%)(mean± s.d.)of patients was 10.9750± 1.0112
- In Malignancy, the mean SR TRANSFERRIN SAT (15-50) (%)(mean± s.d.)of patients was 14.2667± 4.8194.

Distribution of mean SR TRANSFERRIN SAT (15-50) (%) with causes of iron DEF anemia was statistically significant (p=0.0027).

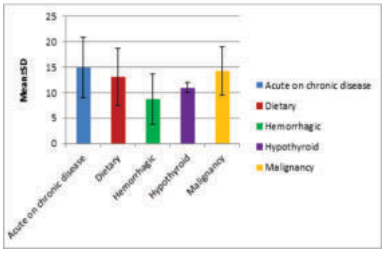


Table: Distribution of mean BIL TOTAL (0 2-1) mg/dl: CAUSES OF IRON DEFANEMIA

		Number	Mean	SD	Minimum	Maximum	Median	p-value
BIL Total (0 2-1) mg/dl	Acute on chronic disease	55	.9549	1.1242	0.2700	7.2700	0.5900	0.6306
	Dietary	17	.7129	.7756	0.2100	3.3100	0.4100	
	Hemorrhagic	18	.6156	.8035	0.2000	3.6800	0.4000	
	Hypothyroid	4	.5250	.3556	0.1700	0.9700	0.4800	
	Malignancy	6	1.0650	1.1435	0.2200	2.8900	0.3900	

- InAcute on chronic disease, the mean BIL TOTAL (0 2-1) mg/dl (mean± s.d.) of patients was .9549± 1.1242.
- In Dietary, the mean BIL TOTAL (0 2-1) mg/dl (mean± s.d.) of patients was .7129± .7756.
- In Hemorrhagic, the mean BIL TOTAL (0 2-1) mg/dl (mean± s.d.) of patients was .6156± .8035.
- In Hypothyroid, the mean BIL TOTAL (0 2-1) mg/dl (mean± s.d.) of patients was .5250± .3556
- In Malignancy, the mean BIL TOTAL (0 2-1) mg/dl (mean± s.d.) of patients was 1.0650± 1.1435.

Distribution of mean BIL TOTAL (0 2-1) mg/dl with causes of iron DEF anemiawas not statistically significant (p=0.6306).

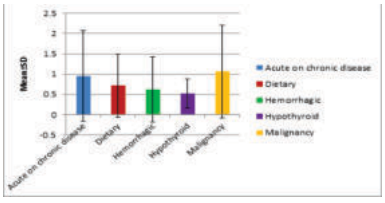


Table: Distribution of mean BIL DIRECT: CAUSES OF IRON DEFANEMIA

		Number	Mean	SD	Minimum	Maximum	Median	p-value
BIL Direct	Acute on chronic disease	55	.4591	.7853	0.1400	5.5900	0.2750	<0.0001
	Dietary	17	.3929	.3376	0.1600	1.5500	0.2900	
	Hemorrhagic	18	.4044	.5913	0.1200	2.6900	0.2400	
	Hypothyroid	4	18.2175	35.8554	0.1000	72.0000	0.3850	
	Malignancy	6	.6317	.6982	0.1400	1.8900	0.2550	

- In Acute on chronic disease, the mean BIL DIRECT (mean± s.d.) of patients was .4591± .7853.
- In Dietary, the mean BIL DIRECT (mean± s.d.) of patients was .3929± .3376.
- In Hemorrhagic, the mean BIL DIRECT (mean± s.d.) of patients was .4044± .5913.
- In Hypothyroid, the mean BIL DIRECT (mean± s.d.) of patients was 18.2175± 35.8554
- In Malignancy, the mean BIL DIRECT (mean± s.d.) of patients was .6317± .6982.

Distribution of mean BIL DIRECT with causes of iron DEF anemia was statistically significant (p<0.0001).

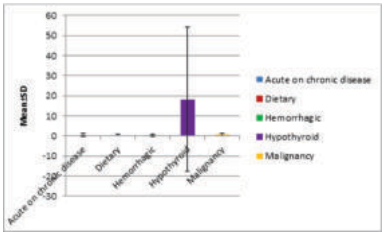


Table: Distribution of mean ALKP (46-116) U/L: CAUSES OF IRON DEFANEMIA

		Number	Mean	SD	Minimum	Maximum	Median	p-value
ALKP (46-116) U/L	Acute on chronic disease	55	94.3091	77.1208	14.0000	372.0000	78.0000	0.1005
	Dietary	17	71.2353	71.6594	20.0000	244.0000	34.0000	
	Hemorrhagic	18	75.8333	112.4624	13.0000	491.0000	36.0000	
	Hypothyroid	4	74.0000	61.4546	20.0000	153.0000	61.5000	
	Malignancy	6	191.6667	218.7562	29.0000	549.0000	75.0000	

- In Acute on chronic disease, the mean ALKP (46-116) U/L (mean± s.d.) of patients was 94.3091± 77.1208.
- In Dietary, the mean ALKP (46-116) U/L (mean± s.d.) of patients was 71.2353± 71.6594.
- In Hemorrhagic, the mean ALKP (46-116) U/L (mean± s.d.) of patients was 75.8333± 112.4624.
- In Hypothyroid, the mean ALKP (46-116) U/L (mean± s.d.) of patients was 74.0000± 61.4546
- In Malignancy, the mean ALKP (46-116) U/L (mean± s.d.) of patients was 191.6667± 218.7562.

Distribution of mean ALKP (46-116) U/L with causes of iron DEF anemiawas not statistically significant (p=0.1005).

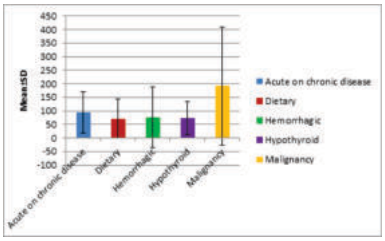


Table: Distribution of mean SGOT (15-37) U/L: CAUSES OF IRON DEFANEMIA

		Number	Mean	SD	Minimum	Maximum	Median	p-value
Age	Acute on chronic disease	55	75.2545	120.7988	14.0000	850.0000	43.0000	0.0457
	Dietary	17	66.4706	41.7090	13.0000	161.0000	50.0000	
	Hemorrhagic	18	51.9444	30.2178	15.0000	137.0000	43.5000	
	Hypothyroid	4	63.7500	42.2956	30.0000	125.0000	50.0000	
	Malignancy	6	196.0000	136.6031	55.0000	431.0000	143.0000	

- InAcute on chronic disease, the mean Age (mean± s.d.) of patients was 75.2545± 120.7988.
- In Dietary, the mean Age (mean± s.d.) of patients was 66.4706± 41.7090.
- In Hemorrhagic, the mean Age (mean± s.d.) of patients was 51.9444± 30.2178.
- In Hypothyroid, the mean Age (mean± s.d.) of patients was 63.7500± 42.2956
- In Malignancy, the mean Age (mean± s.d.) of patients was 196.0000± 136.6031.

Distribution of mean Age with causes of iron DEF anemia was statistically significant (p=0.0457).

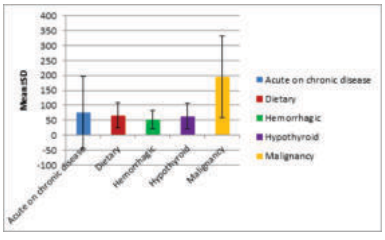


Table: Distribution of mean SGPT (16-63) U/L: CAUSES OF IRON DEFANEMIA

		Number	Mean	SD	Minimum	Maximum	Median	p-value
SGPT (16-63) U/L	Acute on chronic disease	55	72.7818	163.3616	14.0000	1238.0000	38.0000	0.9865
	Dietary	17	55.7059	44.1967	21.0000	167.0000	43.0000	
	Hemorrhagic	18	77.4444	82.1521	3.0000	271.0000	41.0000	
	Hypothyroid	4	81.7500	55.9188	38.0000	163.0000	63.0000	
	Malignancy	6	81.1667	91.8638	22.0000	262.0000	52.0000	

- In Acute on chronic disease, the mean SGPT (16-63) U/L (mean± s.d.) of patients was 72.7818± 163.3616.
- In Dietary, the mean SGPT (16-63) U/L (mean± s.d.) of patients was 55.7059± 44.1967.
- In Hemorrhagic, the mean SGPT (16-63) U/L (mean± s.d.) of patients was 77.4444± 82.1521.
- In Hypothyroid, the mean SGPT (16-63) U/L (mean± s.d.) of patients was 81.7500± 55.9188
- In Malignancy, the mean SGPT (16-63) U/L (mean± s.d.) of patients was 81.1667± 91.8638.

Distribution of mean SGPT (16-63) U/L with causes of iron DEF anemia was not statistically significant (p=0.9865).

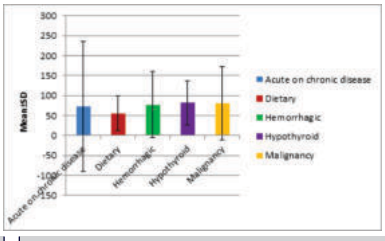


Table: Distribution of mean GGT (15-85) U/L: CAUSES OF IRON DEFANEMIA

		Number	Mean	SD	Minimum	Maximum	Median	p-value
GGT (15-85) U/L	Acute on chronic disease	55	70.4000	97.9897	12.0000	568.0000	39.0000	0.0056
	Dietary	17	67.64	60.7062	15.0000	271.0000	51.0000	
	Hemorrhagic	18	64.4444	140.6804	15.0000	624.0000	24.5000	
	Hypothyroid	4	43.7500	26.9985	25.0000	83.0000	33.5000	
	Malignancy	6	661.0000	1495.1675	21.0000	3712.0000	40.0000	

- In Acute on chronic disease, the mean GGT (15-85) U/L (mean± s.d.) of patients was 70.4000± 97.9897.
- In Dietary, the mean GGT (15-85) U/L (mean± s.d.) of patients was 67.6471± 60.7062.
- In Hemorrhagic, the mean GGT (15-85) U/L (mean± s.d.) of patients was 64.4444± 140.6804.
- In Hypothyroid, the mean GGT (15-85) U/L (mean± s.d.) of patients was 43.7500± 26.9985
- In Malignancy, the mean GGT (15-85) U/L (mean± s.d.) of patients was 661.0000± 1495.1675.

Distribution of mean GGT (15-85) U/L with causes of iron DEF anemia was statistically significant (p=0.0056).

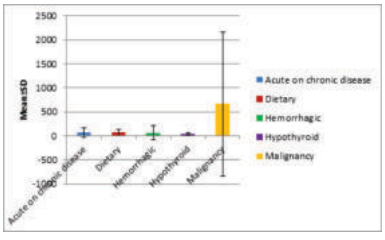


Table: Distribution of mean SR PROTEIN TOTAL (6 3-8 2) gm/dl : CAUSES OF IRON DEFANEMIA

		Number	Mean	SD	Minimum	Maximum	Median	p-value
SR Protein Total (6 3-8 2) gm/dl	Acute on chronic disease	55	6.2548	1.0998	3.5000	8.6400	6.3200	0.5243
	Dietary	17	6.2341	.6752	5.2300	7.8300	6.2300	
	Hemorrhagic	18	6.6183	.3886	6.1800	7.9200	6.5000	
	Hypothyroid	4	6.7700	.5938	6.3000	7.6400	6.5700	
	Malignancy	6	6.3233	.9159	5.4000	8.0700	6.0600	

- InAcute on chronic disease, the mean SR PROTEIN TOTAL (6 3-8 2) gm/dl (mean± s.d.) of patients was 6.2548± 1.0998.
- In Dietary, the mean SR PROTEIN TOTAL (6 3-8 2) gm/dl (mean± s.d.) of patients was 6.2341± .6752.
- In Hemorrhagic, the mean SR PROTEIN TOTAL (6 3-8 2) gm/dl (mean± s.d.) of patients was 6.6183± .3886.
- In Hypothyroid, the mean SR PROTEIN TOTAL (6 3-8 2) gm/dl (mean± s.d.) of patients was 6.7700± .5938
- In Malignancy, the mean SR PROTEIN TOTAL (6 3-8 2) gm/dl (mean± s.d.) of patients was 6.3233± .9159.

Distribution of mean SR PROTEIN TOTAL (6 3-8 2) gm/dl with causes of iron DEF anemiawas not statistically significant (p=0.5243).

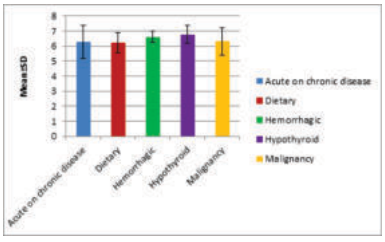


Table: Distribution of mean ALB (3 5-5) gm/dl: CAUSES OF IRON DEF ANEMIA

		Number	Mean	SD	Mini-mum	Maxi-mum	Media n	p-value
ALB (3 5-5) gm/dl	Acute on chronic disease	55	3.2519	.7994	1.3500	5.0000	3.2600	0.0573
	Dietary	17	3.2918	.6957	2.0500	4.4300	3.4500	
	Hemor-rhagic	18	3.5889	.3647	2.6100	4.2000	3.5850	
	Hypo-thyroid	4	4.0900	.7410	3.5400	5.1600	3.8300	
	Malig-nancy	6	3.7550	.3354	3.4000	4.1600	3.6750	

- InAcute on chronic disease, the mean ALB (3 5-5) gm/dl (mean± s.d.) of patients was 3.2519±.7994.
- In Dietary, the mean ALB (3 5-5) gm/dl (mean± s.d.) of patients was 3.2918± .6957.
- In Hemorrhagic, the mean ALB (3 5-5) gm/dl (mean± s.d.) of patients was 3.5889± .3647.
- In Hypothyroid, the mean ALB (3 5-5) gm/dl (mean± s.d.) of patients was 4.0900± .7410
- In Malignancy, the mean ALB (3 5-5) gm/dl (mean± s.d.) of patients was 3.7550± .3354.

Distribution of mean ALB (3 5-5) gm/dl with causes of iron DEF anemia was not statistically significant (p=0.0573).

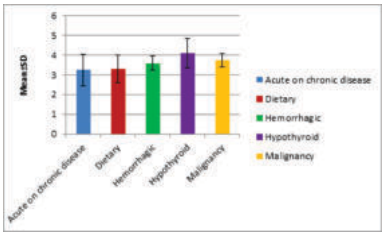


Table: Distribution of mean GLB (1 80-3 60) gm/dl : CAUSES OF IRON DEF ANEMIA

		Number	Mean	SD	Mini-mum	Maxi-mum	Media n	p-value
GLB (1 80-3 60) gm/dl	Acute on chronic disease	55	3.0033	.7518	1.7000	5.6800	2.9500	0.8855
	Dietary	17	2.9547	.5366	2.0000	3.9800	2.9800	
	Hemor-rhagic	18	3.0283	.4325	2.4200	4.3500	3.0000	
	Hypo-thyroid	4	2.7325	.2571	2.4800	3.0300	2.7100	
	Malig-nancy	6	2.8150	.6504	1.9000	3.9100	2.7900	

- InAcute on chronic disease, the mean GLB (1 80-3 60) gm/dl (mean± s.d.) of patients was 3.0033± .7518.
- In Dietary, the mean GLB (1 80-3 60) gm/dl (mean± s.d.) of patients was 2.9547± .5366.
- In Hemorrhagic, the mean GLB (1 80-3 60) gm/dl (mean± s.d.) of patients was 3.0283± .4325.
- In Hypothyroid, the mean GLB (1 80-3 60) gm/dl (mean± s.d.) of patients was 2.7325± .2571
- In Malignancy, the mean GLB (1 80-3 60) gm/dl (mean± s.d.) of patients was 2.8150± .6504.

Distribution of mean GLB (1 80-3 60) gm/dl with causes of iron DEF anemia was not statistically significant (p=0.8855).

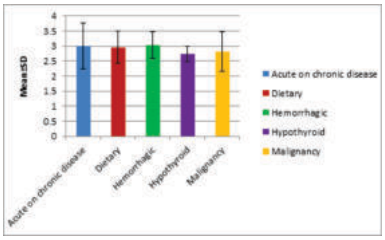


Table: Distribution of mean AG (0-0): CAUSES OF IRON DEF ANEMIA

		Number	Mean	SD	Mini-mum	Maxi-mum	Media n	p-value
AG (0-0)	Acute on chronic disease	55	1.1415	.4380	0.5200	2.4700	1.0900	0.0357
	Dietary	17	1.1647	.3976	0.5500	2.2100	1.1500	
	Hemor-rhagic	18	1.1459	.3145	0.5000	1.6500	1.1600	
	Hypo-thyroid	4	1.5375	.4121	1.1700	2.0800	1.4500	
	Malig-nancy	6	1.6583	.6621	1.0600	2.9000	1.4250	

- InAcute on chronic disease, the mean AG (0-0) (mean± s.d.) of patients was 1.1415± .4380.
- In Dietary, the mean AG (0-0) (mean± s.d.) of patients was 1.1647± .3976.
- In Hemorrhagic, the mean AG (0-0) (mean± s.d.) of patients was 1.1459± .3145.
- In Hypothyroid, the mean AG (0-0) (mean± s.d.) of patients was 1.5375± .4121
- In Malignancy, the mean AG (0-0) (mean± s.d.) of patients was 1.6583± .6621.

Distribution of mean AG (0-0) with causes of iron DEF anemiawas statistically significant (p=0.0357).

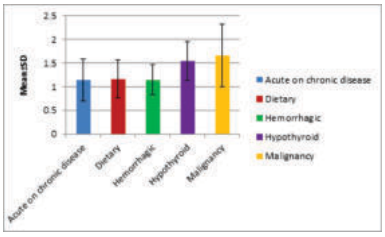


Table: Distribution of mean UREA (19-43) mg/dl : CAUSES OF IRON DEF ANEMIA

		Number	Mean	SD	Mini-mum	Maxi-mum	Media n	p-value
UREA (19-43) mg/dl	Acute on chronic disease	55	48.2636	38.5786	8.4000	174.0000	32.0000	0.1348
	Dietary	17	29.9059	5.9940	20.4000	42.0000	29.8000	
	Hemor-rhagic	18	33.0556	7.7723	14.0000	44.0000	33.5000	
	Hypo-thyroid	4	49.9000	54.0164	11.7000	130.0000	28.9500	
	Malig-nancy	6	31.9000	9.6073	18.2000	45.0000	33.8000	

- InAcute on chronic disease, the mean UREA (19-43) mg/dl(mean± s.d.) of patients was 48.2636± 38.5786.
- In Dietary, the mean UREA (19-43) mg/dl (mean± s.d.) of patients was 29.9059± 5.9940.
- In Hemorrhagic, the mean UREA (19-43) mg/dl (mean± s.d.) of patients was 33.0556± 7.7723.
- In Hypothyroid, the mean UREA (19-43) mg/dl (mean± s.d.) of patients was 49.9000± 54.0164
- In Malignancy, the mean UREA (19-43) mg/dl(mean± s.d.) of patients was 31.9000± 9.6073.

Distribution of mean UREA (19-43) mg/dl with causes of iron DEF anemiawas not statistically significant (p=0.1348).

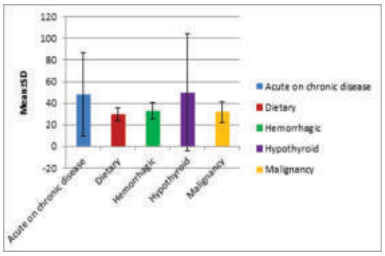


Table: Distribution of mean CREAT (0 7-1 25) mg/dl: CAUSES OF IRON DEF ANEMIA

		Number	Mean	SD	Minimum	Maximum	Median	p-value
CREAT (0 7-1 25) mg/dl	Acute on chronic disease	55	1.5511	1.6602	0.4000	7.2800	0.9100	0.1468
	Dietary	17	.8582	.1434	0.5900	1.2900	0.8600	
	Hemorrhagic	18	.8756	.1591	0.5900	1.1000	0.9100	
	Hypothyroid	4	.9650	.1584	0.8400	1.1900	0.9150	
	Malignancy	6	.9483	.0496	0.8700	1.0100	0.9550	

- InAcute on chronic disease, the mean CREAT (0 7-1 25) mg/dl (mean± s.d.) of patients was 1.5511± 1.6602.
- In Dietary, the mean CREAT (0 7-1 25) mg/dl (mean± s.d.) of patients was .8582± .1434.
- In Hemorrhagic, the mean CREAT (0 7-1 25) mg/dl (mean± s.d.) of patients was .8756± .1591.
- In Hypothyroid, the mean CREAT (0 7-1 25) mg/dl (mean± s.d.) of patients was .9650± .1584
- In Malignancy, the mean CREAT (0 7-1 25) mg/dl (mean± s.d.) of patients was .9483± .0496.

Distribution of mean CREAT (0 7-1 25) mg/dl with causes of iron DEF anemia was not statistically significant (p=0.1468).

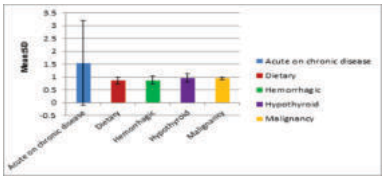


Table: Distribution of mean VIT B12 (211-911) pg/ml: CAUSES OF IRON DEF ANEMIA

		Number	Mean	SD	Minimum	Maximum	Median	p-value
VIT B12 (211-911) pg/ml	Acute on chronic disease	55	320.7273	125.7707	175.0000	897.0000	278.0000	0.0031
	Dietary	17	203.6875	59.3731	105.0000	356.0000	207.5000	
	Hemorrhagic	18	360.8889	204.9680	215.0000	900.0000	280.0000	
	Hypothyroid	4	184.0000	53.6718	105.0000	220.0000	205.5000	
	Malignancy	6	265.0000	40.4920	216.0000	324.0000	258.0000	

- InAcute on chronic disease, the mean VIT B12 (211-911) pg/ml (mean± s.d.) of patients was 320.7273± 125.7707.
- In Dietary, the mean VIT B12 (211-911) pg/ml (mean± s.d.) of patients was 203.6875± 59.3731.
- In Hemorrhagic, the mean VIT B12 (211-911) pg/ml (mean± s.d.) of patients was 360.8889± 204.9680.
- In Hypothyroid, the mean VIT B12 (211-911) pg/ml (mean± s.d.) of patients was 184.0000± 53.6718
- In Malignancy, the mean VIT B12 (211-911) pg/ml (mean± s.d.) of patients was 265.0000± 40.4920.

Distribution of mean VIT B12 (211-911) pg/ml with causes of iron DEF anemiawas statistically significant (p=0.0031).

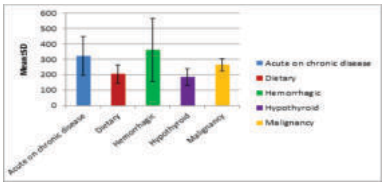


Table: Distribution of mean FOLIC ACID (2 76-20) ng/ml: CAUSES OF IRON DEF ANEMIA

		Number	Mean	SD	Minimum	Maximum	Median	p-value
Folic Acid (2 76-20) ng/ml	Acute on chronic disease	55	9.2142	3.3100	4.0000	18.5000	8.7000	0.1390
	Dietary	17	7.0471	4.0492	1.2000	20.0000	6.1000	
	Hemorrhagic	18	8.2367	3.1143	2.4000	17.0000	8.3000	
	Hypothyroid	4	7.3100	4.6606	3.1600	12.7000	6.6900	
	Malignancy	6	10.1167	2.8520	6.9000	13.9000	9.5000	

- InAcute on chronic disease, the mean FOLIC ACID (2 76-20) ng/ml (mean± s.d.) of patients was 9.2142± 3.3100.
- In Dietary, the mean FOLIC ACID (2 76-20) ng/ml (mean± s.d.) of patients was 7.0471± 4.0492.
- In Hemorrhagic, the mean FOLIC ACID (2 76-20) ng/ml (mean± s.d.) of patients was 8.2367± 3.1143.
- In Hypothyroid, the mean FOLIC ACID (2 76-20) ng/ml (mean± s.d.) of patients was 7.3100± 4.6606
- In Malignancy, the mean FOLIC ACID (2 76-20) ng/ml (mean± s.d.) of patients was 10.1167± 2.8520.

Distribution of mean FOLIC ACID (2 76-20) ng/ml with causes of iron DEF anemia was not statistically significant (p=0.1390).

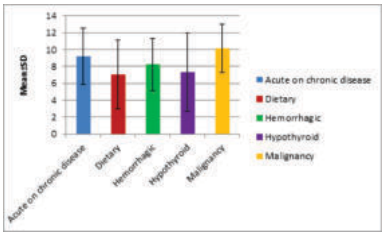


Table: Distribution of mean INTAKE OF PROTEIN (gm)/ day: CAUSES OF IRON DEF ANEMIA

		Number	Mean	SD	Minimum	Maximum	Median	p-value
Intake of protein (gm)/ day	Acute on chronic disease	55	64.3776	5.5689	46.0000	75.0000	64.0000	0.0001
	Dietary	17	54.5388	7.1821	44.2700	72.8500	54.6100	
	Hemorrhagic	18	61.6156	10.2163	33.9600	78.0000	61.7000	
	Hypothyroid	4	66.9525	7.1126	60.0000	74.9100	66.4500	
	Malignancy	6	64.6000	7.3007	58.0000	78.6000	63.0000	

- In Acute on chronic disease, the mean Intake of protein (gm)/ day (mean± s.d.) of patients was 64.3776± 5.5689.
- In Dietary, the mean Intake of protein (gm)/ day (mean± s.d.) of patients was 54.5388± 7.1821.
- In Hemorrhagic, the mean Intake of protein (gm)/ day (mean± s.d.) of patients was 61.6156± 10.2163.
- In Hypothyroid, the mean Intake of protein (gm)/ day (mean± s.d.) of patients was 66.9525± 7.1126
- In Malignancy, the mean Intake of protein (gm)/ day (mean± s.d.) of patients was 64.6000± 7.3007.

Distribution of mean Intake of protein (gm)/ day with causes of iron DEF anemia was statistically significant (p=0.0001).

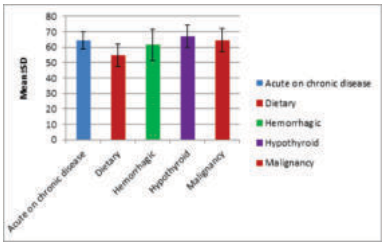


Table: Distribution of mean INTAKE OF IRON (mg)/ day: CAUSES OF IRON DEF ANEMIA

		Number	Mean	SD	Minimum	Maximum	Median	p-value

Intake of iron (mg)/ day	Acute on chronic disease	55	18.3273	6.7265	8.6400	60.2500	18.2700	0.3690
	Dietary	17	15.0053	6.1188	8.3800	26.5200	12.9500	
	Hemorrhagic	18	18.0044	4.6185	11.6000	29.6500	16.8800	
	Hypothyroid	4	19.1475	3.9080	15.4600	24.2100	18.4600	
	Malignancy	6	18.5400	2.6971	15.6300	22.8200	17.6300	

- In Acute on chronic disease, the mean Intake of iron (mg)/ day (mean± s.d.) of patients was 18.3273± 6.7265.
- In Dietary, the mean Intake of iron (mg)/ day (mean± s.d.) of patients was 15.0053± 6.1188.
- In Hemorrhagic, the mean Intake of iron (mg)/ day (mean± s.d.) of patients was 18.0044± 4.6185.
- In Hypothyroid, the mean Intake of iron (mg)/ day (mean± s.d.) of patients was 19.1475± 3.9080
- In Malignancy, the mean Intake of iron (mg)/ day (mean± s.d.) of patients was 18.5400± 2.6971.

Distribution of mean Intake of iron (mg)/ day with causes of iron DEF anemiawas not statistically significant (p=0.3690).

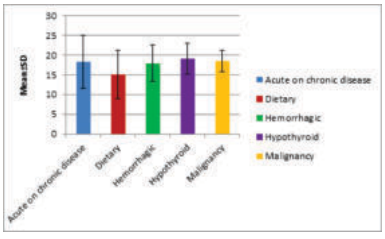


Table: Distribution of mean INTAKE OF VIT C (mcg)/ day: CAUSES OF IRON DEFANEMIA

		Number	Mean	SD	Mini-mum	Maxi-mum	Media n	p-value
Intak of VIT C (mcg)/ day	Acute on chronic disease	55	64.9462	19.9725	0.6800	94.0600	70.2800	0.8826
	Dietary	17	49.5571	24.4632	7.0500	97.0000	46.2800	
	Hemor-rhagic	18	60.8078	10.5570	36.4200	78.6000	58.6500	
	Hypo-thyroid	4	62.2800	8.5470	52.2500	70.0000	63.4350	
	Malig-nancy	6	62.8500	10.2732	47.9000	78.1100	63.2600	

- In Acute on chronic disease, the mean Intake of VIT C (mcg)/ day (mean± s.d.) of patients was 64.9462± 19.9725.
- In Dietary, the mean Intake of VIT C (mcg)/ day (mean± s.d.) of patients was 49.5571± 24.4632.
- In Hemorrhagic, the mean Intake of VIT C (mcg)/ day (mean± s.d.) of patients was 60.8078± 10.5570.
- In Hypothyroid, the mean Intake of VIT C (mcg)/ day (mean± s.d.) of patients was 62.2800± 8.5470
- In Malignancy, the mean Intake of VIT C (mcg)/ day (mean± s.d.) of patients was 62.8500± 10.2732.

Distribution of mean Intake of VIT C (mcg)/ day with causes of iron DEF anemia was not statistically significant (p=0.8826).

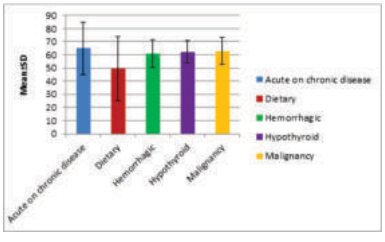


Table: Distribution of mean INTAKE OF VIT B12 (mcg)/ day: CAUSES OF IRON DEFANEMIA

		Number	Mean	SD	Mini-mum	Maxi-mum	Media n	p-value
Intake of VIT B12 (mcg)/ day	Acute on chronic disease	55	.9183	.4332	0.0000	2.1000	0.9360	0.4214
	Dietary	17	.6979	.6775	0.0000	2.1000	0.5800	
	Hemor-rhagic	18	1.0728	.7960	0.0000	3.8400	0.9925	
	Hypo-thyroid	4	.8025	.5441	0.0000	1.2000	1.0050	
	Malig-nancy	6	.9602	.6787	0.1200	2.1400	0.9550	

- In Acute on chronic disease, the mean Intake of VIT B12 (mcg)/ day (mean± s.d.) of patients was .9183± .4332.
- In Dietary, the mean Intake of VIT B12 (mcg)/ day (mean± s.d.) of patients was .6979± .6775.
- In Hemorrhagic, the mean Intake of VIT B12 (mcg)/ day (mean± s.d.) of patients was 1.0728± .7960.
- In Hypothyroid, the mean Intake of VIT B12 (mcg)/ day (mean± s.d.) of patients was .8025± .5441
- In Malignancy, the mean Intake of VIT B12 (mcg)/ day (mean± s.d.) of patients was .9602± .6787.

Distribution of mean Intake of VIT B12 (mcg)/ day with causes of iron DEF anemia was not statistically significant (p=0.4214).

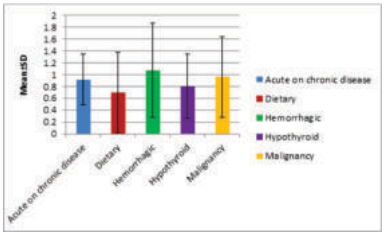
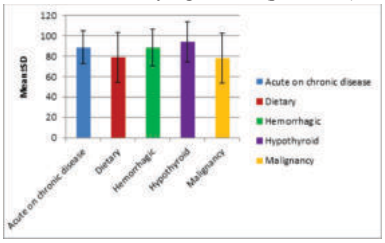


Table: Distribution of mean INTAKE OF FOLIC ACID (mcg)/ day : CAUSES OF IRON DEFANEMIA

		Number	Mean	SD	Mini-mum	Maxi-mum	Media n	p-value
Intake of folic acid (mcg) / day	Acute on chronic disease	54	88.6674	16.1602	48.6500	131.4000	90.7150	0.2467
	Dietary	17	78.8641	24.4335	42.1400	127.3600	75.4700	
	Hemor-rhagic	18	88.2183	18.0699	46.4200	128.4800	88.7450	
	Hypo-thyroid	4	94.1325	19.3346	74.1200	113.1200	94.6450	
	Malig-nancy	6	78.2100	24.5753	43.1200	108.1000	83.2800	

- In Acute on chronic disease, the mean Intake of folic acid (mcg)/ day (mean± s.d.) of patients was 88.6674± 16.1602.
- In Dietary, the mean Intake of folic acid (mcg)/ day (mean± s.d.) of patients was 78.8641± 24.4335.
- In Hemorrhagic, the mean Intake of folic acid (mcg)/ day (mean± s.d.) of patients was 88.2183± 18.0699.
- In Hypothyroid, the mean Intake of folic acid (mcg)/ day (mean± s.d.) of patients was 94.1325± 19.3346
- In Malignancy, the mean Intake of folic acid (mcg)/ day (mean± s.d.) of patients was 78.2100± 24.5753.

Distribution of mean Intake of folic acid (mcg)/ day with causes of iron DEF anemiawas not statistically significant (p=0.2467).



DISCUSSION

This Study was conducted from August 2006 and July 2008 at rheumatology clinic of The Tertiary Care Hospital in South Kolkata. Total 100 patients were included in this study.

Pivetta G et al 1(2022) found thatiron-deficiency anemia in the elderly may be due to numerous gastrointestinal conditions.DM histopathology was significantly inversely correlated with age group, with prevalences of 27%, 20%, 12.5%, 10%, and 2.5%, in the <40–50, 51–60, 61–70, 71–80, and >80-year age groups, respectively (p = 0.0010).

Contreras-Manzano A et al 22(2015) found thatto describe de prevalence of iron deficiency (ID) and anemia in a sample of Mexican elderly population from the National Health and Nutrition Survey (Ensanut) 2012.The prevalence of anemia was high in the elderly, however the prevalence of ID was low; there is a need to further investigate the causes of anemia in this age group.

Bach V et al 23(2014) found thatanemia in later life is associated with increased morbidity and mortality. The prevalence of anemia was significantly correlated with advanced age (r=0.21; P<0.001) and male sex (P<0.001).

In our study, out of 100 patients most of the patients were ≤70 years old [49 (49.0%)].

26 (47.3%) patients were ≤70 years of age in Acute on chronic diseaseGroup and 25 (45.5%) patients were 71-80 years of age in Acute on chronic diseaseGroup but this was not statistically significant (p=0.8592). In our study, Age was higher in Dietary [72.2941± 7.9511] compared to Hypothyroid [68.7500± 7.5000] but this was not statistically significant (p=0.8826).In our study, Age was less in Haemorrhagic [51.9444±30.2178] compared to Malignancy [196.0000± 136.6031] but this was statistically significant (p=0.0457).

Prakash KG et al 27(2015) found thatit is easy to overlook anemia in the elderly, since such symptoms as fatigue, weakness, or shortness of breath may be attributed to the aging process itself.64% patients were male and the mean age of patients was 66.65 years.

We found that, male [60 (60.0%)] population was higher than the female population [40 (40.0%)]. In Acute on chronic diseaseGroup was significantly more observed in male patients [34 (61.8%)] compared to female patients [21 (38.2%)]. (p=0.0103).

Present study showed that, P SMEAR with Causes of Iron DEF Anaemia was statistically significant (p=0.0010).

We examined that, 17 (94.4%) patients had Stool RE & OCLT BLD in Haemorrhagic groupand was statistically significant (p<0.0001).

In our study, 55 (100.0%) patients had WNL in Acute on chronic disease group compared to Dietary group [17 (100.0%)] but was statistically significant (p=0.0033).

In Acute on chronic diseaseGroup was significantly more observed in Non-Veg Diet patients [52 (94.5%)] compared to Veg Diet patients [3 (5.5%)]. (p<0.0001).

Prakash KG et al 27(2015) found thatit is easy to overlook anemia in the elderly, since such symptoms as fatigue, weakness, or shortness of breath may be attributed to the aging process itself.50 patients aged 60years and above with Hb% <13gms% in males, and< 12 gms% in females were enrolled into the study

We found that, HB% (12-17 gm /dl) was less in Acute on chronic disease [9.0982± 1.5507] compared to Hypothyroid [10.5000± 1.3115] but this was not statistically significant (p=0.1777).

Chatterjee P et al 10(2018) found thatblood is a fluid inside living creatures that carries vital substances to the cells and transports metabolic waste products away from the same cells.The function of the RBC is to deliver oxygen from the lungs to the tissues and carbon dioxide from the tissues to the lungs.

Our study showed that, RBC Total (3, 8-5, 5) 10^6/ ml was more in Hypothyroid [3.2150± .7422] compared to Acute on chronic disease [2.8587± .5905] but this was not statistically significant (p=0.5364).

We observed that, TLC 4000-10000/ Cumm) was lower in Hemorrhagic [9150.0000± 3730.9910] compared to Acute on chronic disease [11798.1818± 5934.7218] but this was statistically significant (p=0.0012).

It was found that, N (40-75) % was most in Hypothyroid [78.5000± 5.0000] compared to Dietary [68.8235± 13.0969] but this was statistically significant (p=0.0230).

We examined that, E (1-6) % was higher in Dietary [4.1176± 4.8462] compared to Hypothyroid [2.0000± .8165] but this was not statistically significant (p=0.1619).

In our study, B (0-2) % was less in Dietary [.1250±.3416] compared to Acute on chronic disease [.0182± .1348] but this was not statistically significant (p=0.2012).

We found that, significantly L (20-40) % was reduced in Acute on chronic disease [17.4727± 12.1851] compared to Haemorrhagic [25.2222±7.8482]. (p=0.0330).

Our study showed that, M (2-10) % was lower in Haemorrhagic [1.2222± .6468] compared to Malignancy [2.0000±1.6733] but this was not statistically significant (p=0.1467).

We observed that, significantly ESR was more in Acute on chronic disease [74.4907±38.5250] compared to Haemorrhagic [49.7222± 33.0397]. (p=0.0332).

We found that, PCV (36-47) % was less in Haemorrhagic [27.5118± 5.6587] compared to Hypothyroid [33.0000± 3.1623] but this was not statistically significant (p=0.3071).

Karoopongse E et al 25(2013) found thatanemia is one of the most common health problems in the elderly in low- and middle-income countries.Data from subjects aged ≥ 60 years from the Fourth Thai National Health Examination Survey were analyzed. Comorbidity and hematologic indexes including MCV were obtained.

Our study showed that, significantly MCV (83-101) was more in Hypothyroid [94.5000± 10.3441] compared to Haemorrhagic [27.5118± 5.2713]. (p=0.0006).

We observed that, MCH (27-32) % was lower in Haemorrhagic [23.9889± 2.1655] compared to Hypothyroid [27.9500±2.7923] but this was statistically significant (p=0.0023).

It was found that, MCHC (31,5-34,5) % was most in Hypothyroid [31.7250± 2.7923] compared to Haemorrhagic [30.2556±1.3993] but this was not statistically significant (p=0.3695).

We examined that, significantly RETIC Count (0,5-2,5) % was higher in Haemorrhagic [2.2412± 1.0989] compared to Hypothyroid [1.1250±2.7923]. (p<0.0001).

In our study, Platelets (150-450) X 10^3 microl was less in Hypothyroid [156.5000± 19.9750] compared to Acute on chronic disease [219.7636± 93.5355] but this was not statistically significant (p=0.2286).

Contreras-Manzano A et al 22(2015) found thatto describe de prevalence of iron deficiency (ID) and anemia in a sample of Mexican elderly population from the National Health and Nutrition Survey (Ensanut) 2012. 1 920 subjects ≥60 years of age were included. Hemoglobin, serum concentrations of ferritin and CRP were measured.

We found that, significantly CRP was reduced in Hypothyroid [4.0250± .6652] compared to Acute on chronic disease [62.3709± 60.0772]. (p<0.0001).

Our study showed that, SR Iron (49-181) mcg/dl was lower in Haemorrhagic [34.0000± 10.4499] compared to Malignancy [50.5000± 17.8746] but this was statistically significant (p=0.0076).

We observed that, significantly TIBC (250-450) mcg/dl was more in Haemorrhagic [74.4907±38.5250] compared to Acute on chronic disease [326.4727± 326.4727]. (p=0.0470).

Babaei M et al 20(2017) found thatdiagnosis and treatment of iron deficiency anemia in older subjects improves their quality of life.

Serum ferritin as a marker of iron stores is an acute phase protein. In older subjects who usually have many concomitant chronic medical conditions, serum ferritin may increase in response to inflammatory processes irrespective of iron stores. This study was performed to determine the diagnostic properties of serum ferritin in the diagnosis of iron deficiency anemia in older subjects.

We found that, SR Ferritin (10-291) ng/ml was less in Hypothyroid [13.0150 ± 5.5984] compared to Malignancy [21.6250± 19.4562] but this was not statistically significant (p=0.9416).

Our study showed that, significantly SR Transferrin SAT (15-50) (%) was more in Acute on chronic disease [14.9182±6.0014] compared to Haemorrhagic [8.7244± 4.9156]. (p=0.0027).

We observed that, BIL Total (0 2-1) mg/dl was lower in Hypothyroid [.5250± .3556] compared to Malignancy [1.0650± 1.1435] but this was not statistically significant (p=0.6306).

It was found that, BIL Direct was most in Hypothyroid [18.2175± 35.8554] compared to Dietary [.3929± .3376] but this was statistically significant (p<0.0001).

We examined that, ALKP (46-116) U/L was higher in Malignancy [191.6667± 218.7562] compared to Dietary [71.2353± 71.6594] but this was not statistically significant (p=0.1005).

We found that, SGPT (16-63) U/L was reduced in Dietary [55.7059± 44.1967] compared to Hypothyroid [81.7500± 55.9188]. It was not statistically significant (p=0.9865).

Our study showed that, significantly GGT (15-85) U/L was lower in Hypothyroid [43.7500± 26.9985] compared to Malignancy [661.0000± 1495.1675]. (p=0.0056).

We observed that, SR Protein Total (6 3-8 2) gm/dl was more in Hypothyroid [6.7700±.5938] compared to Dietary [6.2341± .6752] which was not statistically significant (p=0.5243).

We found that, ALB (3 5-5) gm/dl was less in Acute on chronic disease [3.2519±.7994] compared to Hypothyroid [4.0900±.7410] but this was not statistically significant (p=0.0573).

Our study showed that, GLB (1 80-3 60) gm/dl was more in Haemorrhagic [94.5000± 10.3441] compared to Hypothyroid [2.7325± .2571]. It was not statistically significant (p=0.8855).

We observed that, AG (0-0) was lower in Acute on chronic disease [1.1415±.4380] compared to Malignancy [1.6583± .6621] which was statistically significant (p=0.0357).

It was found that, UREA (19-43) mg/dl was most in Hypothyroid [49.9000± 54.0164] compared to Dietary [29.9059± 5.9940] but this was not statistically significant (p=0.1348).

We examined that, CREAT (0 7-1 25) mg/dl was higher in Acute on chronic disease [1.5511± 1.6602] compared to Dietary [.8582± .1434].

In our study, VIT B12 (211-911) pg/ml was less in Hypothyroid [184.0000± 53.6718] compared to Haemorrhagic [360.8889± 204.9680.] but this was statistically significant (p=0.0031).

Vadakattu SS et al 6(2019) found thatthe data on the prevalence of nutritional anemia among the urban elderly population in India was limited.Plasma Folic acid and vitamin B12 levels were estimated by RIA and homocysteine and ferritin levels were estimated by ELISA methods.

Barbind SR et al 8(2020) found thatanemia is one of the most common health problems in India.There were 26% of subjects in their study who were anemic, and majority of them were females Although, Iron deficiency anemia revealed as a most common type of anemia, vitamin B12 and folic acid deficiencies also contributed significantly to nutritional anemias.

We found that, Folic Acid (2 76-20) ng/ml was reduced in Dietary [7.0471± 4.0492] compared to Malignancy [10.1167±2.8520]. It was not statistically significant (p=0.1390).

Our study showed that, significantly Intake of protein (gm)/ day was lower in Dietary [54.5388± 7.1821] compared to Hypothyroid [66.9525± 7.1126] (p=0.0001)

We observed that, Intake of iron (mg)/ day was more in Hypothyroid [19.1475± 3.9080] compared to Dietary [15.0053± 6.1188]. It was not statistically significant (p=0.3690).

We observed that, significantly Intake of VIT C (mcg)/ day was more in Dietary[49.5571± 24.4632] compared to Acute on chronic disease [64.9462±19.9725]. It was not statistically significant (p=0.8826).

We found that, Intake of VIT B12 (mcg)/ day was less in Dietary[.6979± .6775] compared to Haemorrhagic[1.0728± .7960] but this was not statistically significant (p=0.4214).

Our study showed that, Intake of folic acid (mcg)/ day was more in Hypothyroid[94.1325±19.3346] compared to Malignancy[78.2100± 24.5753]. It was not statistically significant (p=0.2467).

SUMMARY AND CONCLUSION

- In our study, out of 100 patients most of the patients were ≤70 years of age in Acute on chronic disease Group and patients were 71-80 years of age in Acute on chronic disease Group but this was not statistically significant. In our study, Age was higher in Dietary compared to Hypothyroid but this was not statistically significant.
- We found that, male population was higher than the female population. In Acute on chronic disease Group was significantly more observed in male patients compared to female patients.
- Present study showed that, P SMEAR with Causes of Iron DEF Anaemia was statistically significant.
- We examined that,patients had Stool RE & OCCULT BLOOD in Haemorrhagic group and was statistically significant.
- In our study, patients had WNL in Acute on chronic disease group compared to Dietary group but was statistically significant.
- In Acute on chronic disease Group was significantly more observed in Non-Veg Diet patients compared to Vegetarian Diet patients.
- We found that, HB% (12-17 gm /dl) was less in Acute on chronic disease compared to Hypothyroid but this was not statistically significant.
- Our study showed that, RBC Total (3, 8-5, 5) 10^6/ ml was more in Hypothyroid compared to Acute on chronic disease but this was not statistically significant.
- We observed that, TLC 4000-10000/ Cumm) was lower in Haemorrhagic compared to Acute on chronic disease but this was statistically significant.
- It was found that, N (40-75) % was most in Hypothyroid compared to Dietary but this was statistically significant.
- We examined that, E (1-6) % was higher in Dietary compared to Hypothyroid but this was not statistically significant.
- In our study, B (0-2) % was less in Dietary compared to Acute on chronic disease but this was not statistically significant.
- We found that, significantly L (20-40) % was reduced in Acute on chronic disease compared to Haemorrhagic.
- Our study showed that, M (2-10) % was lower in Haemorrhagic compared to Malignancy but this was not statistically significant.
- We observed that, significantly ESR was more in Acute on chronic disease compared to Haemorrhagic.
- We found that, PCV (36-47) % was less in Haemorrhagic compared to Hypothyroid but this was not statistically significant.
- Our study showed that, significantly MCV (83-101) was more in Hypothyroid compared to Haemorrhagic.
- We observed that, MCH (27-32) % was lower in Haemorrhagic compared to Hypothyroid but this was statistically significant.
- It was found that, MCHC (31,5-34,5) % was most in Hypothyroid compared to Haemorrhagic but this was not statistically significant.
- We examined that, significantly RETIC Count (0,5-2,5) % was higher in Haemorrhagic compared to Hypothyroid.
- In our study, Platelets (150-450) X 10^3 microl was less in Hypothyroid compared to Acute on chronic disease but this was not statistically significant.
- We found that, significantly CRP was reduced in Hypothyroid compared to Acute on chronic disease.
- Our study showed that, SR Iron (49-181) mcg/dl was lower in Haemorrhagic compared to Malignancy but this was statistically significant.

- We observed that, significantly TIBC (250-450) mcg/dl was more in Haemorrhagic compared to Acute on chronic disease.We found that, SR Ferritin (10-291) ng/ml was less in Hypothyroid compared to Malignancy but this was not statistically significant.
- Our study showed that, significantly SR Transferrin SAT (15-50) (%) was more in Acute on chronic disease compared to Haemorrhagic.
- We observed that, BIL Total (0 2-1) mg/dl was lower in Hypothyroid compared to Malignancy but this was not statistically significant.
- It was found that, BIL Direct was most in Hypothyroid compared to Dietary but this was statistically significant.
- We examined that, ALKP (46-116) U/L was higher in Malignancy compared to Dietary but this was not statistically significant.
- In our study, Age was less in Haemorrhagic compared to Malignancy but this was statistically significant.
- We found that, SGPT (16-63) U/L was reduced in Dietary compared to Hypothyroid. It was not statistically significant.
- Our study showed that, significantly GGT (15-85) U/L was lower in Hypothyroid compared to Malignancy.
- We observed that, SR Protein Total (6 3-8 2) gm/dl was more in Hypothyroid compared to Dietary which was not statistically significant.We found that, ALB (3 5-5) gm/dl was less in Acute on chronic disease compared to Hypothyroid but this was not statistically significant.
- Our study showed that, GLB (1 80-3 60) gm/dl was more in Haemorrhagic compared to Hypothyroid. It was not statistically significant.
- We observed that, AG (0-0) was lower in Acute on chronic disease compared to Malignancy which was statistically significant.
- It was found that, UREA (19-43) mg/dl was most in Hypothyroid compared to Dietary but this was not statistically significant.
- We examined that, CREAT (0 7-1 25) mg/dl was higher in Acute on chronic disease compared to Dietary.
- In our study, VITAMIN B12 (211-911) pg/ml was less in Hypothyroid compared to Haemorrhagic but this was statistically significant.
- We found that, Folic Acid (2 76-20) ng/ml was reduced in Dietary compared to Malignancy. It was not statistically significant.
- Our study showed that, significantly Intake of protein (gm)/ day was lower in Dietary compared to Hypothyroid.
- We observed that, Intake of iron (mg)/ day was more in Hypothyroid compared to Dietary. It was not statistically significant.
- We observed that, significantly Intake of VIT C (mcg)/ day was more in Dietary compared to Acute on chronic disease. It was not statistically significant.
- We found that, Intake of VITAMIN B12 (mcg)/ day was less in Dietary compared to Haemorrhagic but this was not statistically significant.
- Our study showed that, Intake of folic acid (mcg)/ day was more in Hypothyroid compared to Malignancy. It was not statistically significant.

Limitations Of The Study

In spite of every sincere effort my study has lacunae.
The notable short comings of this study are:

1. The sample size was small. Only 100 cases are not sufficient for this kind of study.
2. The study has been done in a single centre.
3. The study was carried out in a tertiary care hospital, so hospital bias cannot be ruled out.

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