STUDY ON IRON PROFILE IN CHILDREN WITH CYANOTIC CONGENITAL HEART DISEASE

A STUDY OF 50 CASES IN GOVERNMENT RAJAJI HOSPITAL

DISSERTATION SUBMITTED IN THE PARTIAL FULFILMENT

FOR THE DEGREE OF DOCTOR OF MEDICINE

PAEDIATRIC MEDICINE(BRANCH VII)

APRIL 2013

THE TAMILNADU

DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI, TAMILNADU

CERTIFICATE

This is to certify that the dissertation entitled **" STUDY ON IRON PROFILE IN CHILDREN WITH CYANOTIC CONGENITAL HEART DISEASE "– A STUDY OF 50 CASES IN GOVERNMENT RAJAJI HOSPITAL"** submitted by **DR.K.MEENALOSINI** to the faculty of paediatrics, The Tamil Nadu DR.M.G.R. Medical University, Chennai in partial fulfilment of the requirements for the award of M.D Degree Branch VII (Paediatrics) is a bonafide research work carried out by her under my direct supervision and guidance.

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DECLARATION

I **DR.K.MEENALOSINI** solemnly declare that the dissertation titled **"STUDY ON IRON PROFILE IN CHILDREN WITH CYANOTIC CONGENITAL HEART DISEASE – A STUDY OF 50 CASES IN GOVERNMENT RAJAJI HOSPITAL"** has been prepared by me.

This is submitted to the **Tamilnadu Dr.M.G.R.Medical University,** Chennai in partial fulfilment of the rules and regulations for the M.D. Degree Examination in Paediatrics.

PLACE: Madurai

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ACKNOWLEDGEMENT

I owe my thanks to **The Dean Dr.N.MOHAN M.S.,F.I.C.S** Madurai Medical College for allowing me to avail the facilities needed for my dissertation.

It is with immense pleasure and privilege that I express my heartful gratitude, admiration and sincere thanks to our **Director in Charge Prof.Dr.G.MATHEVAN** Professor and Head of the Department of Paediatrics, my guide and teacher for his guidance, supervision, encouragement and support during this study.

I would like to thank Prof.Dr.AmudhaRajeshwari, Prof.Dr.Chitra Ayyappan, Prof.Dr.R.A.SankaraSubramanian, Prof.Dr.S.Venkateswaran, Prof.Dr.M.Nagendran, Prof.Dr.S.Sambath, Prof.Dr.S.Balasankar, Prof.Dr.K.Mathiarasan who guided me during this study.

I would like to thank Prof.Dr.N.Sundari, Department of Radiology, Prof. Dr.Usharavikumar, Department of Pathology, Prof.Dr.R.A.Janarthanan, Department of cardiology for their valuable help and assistance throughout the study.

I would like to thank our Registar Dr.Mathivanan for his valuable suggestions for this study.

I gratefully acknowledge the help and guidance received from Assistant Professors DR.M.Kulandaivel, Dr.M.Balasubramanian, Dr.NandiniKuppusamy, Dr.P.Guna, and other Assistant professors for their constant support and suggestions during this study.

I also express my gratitude to all my fellow post graduates for their kind co operation in carrying out this study and for their critical analysis.

I thank the members of Ethical committee, Government Rajaji Hospital and Madurai Medical College, Madurai for permitting me to perform this study.

I thank all the parents and children who form the most integral part of the work without whom, this study would not have been possible.

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Paper ID	294088109
Paper title	Iron profile in cyanotic congenital heart disease in children
Assignment title	Medical
Author	Meenalosini 20103102 M.D. Paediatrics
E-mail	drlosini@yahoo.in
Submission time	24-Dec-2012 11:29PM
Total words	9667

First 100 words of your submission

INTRODUCTION: In the world, the most common cause of nutritional anemia is Iron deficiency anemia. Children during the phase of rapid growth such as preschool and adolescence are at higher risk of developing iron deficiency anemia . Rural areas and children from poor socioeconomic status show increased prevalence of iron deficiency .(1) Iron deficiency is an important problem in patients with cyanotic congenital heart disease .In CCHD , arterial oxygen saturation decreases and red blood cell count may reach to high level and hyperviscosity develops(2). In anemic patients especially those with microcytic iron deficiency anemia, permeability of microcyic erythrocytes decreases in comparison to...

INTRODUCTION

In the world, the most common cause of nutritional anemia is Iron deficiency anemia. Children during the phase of rapid growth such as preschool and adolescence are at higher risk of developing iron deficiency anemia . Rural areas and children from poor socioeconomic status show increased prevalence of iron deficiency .[1]

Iron deficiency is an important problem in patients with cyanotic congenital heart disease .In CCHD , arterial oxygen saturation decreases and red blood cell count may reach to high level and hyperviscosity develops[2]. In anemic patients especially those with microcytic iron deficiency anemia, permeability of microcyic erythrocytes decreases in comparison to normocytic cells, therefore thromboembolic and cardiovascular events are encountered more commonly.

As erythropoiesis and hence haemoglobin ,haematocrit and erythrocyte count increases in patients with cyanotic congenital heart disease, haemoglobin and haematocrit are not useful indicators of iron deficiency anaemia.

In children with cyanotic congenital heart disease, when there is decreasing arterial oxygen saturation, it causes compensatory increase in haemoglobin and haematocrit levels. Iron deficiency causes discrepant

values for arterial oxygen saturation and haemoglobin/haematocrit and that "normal" haemoglobin/haematocrit levels in such children may constitute anaemia[3]. In CCHD, normal hemoglobin represent relative anemia and may have disastrous effects. The normal postnatal fall in haemoglobin levels that occurs in neonates will not occur if arterial desaturation is marked from birth ,although relative anaemia in this situation develops by the third or fourth month of life[4].The bone marrow normally responds to hypoxia by increasing erythropoiesis, with an increase in red cell count, haemoglobin level and haematocrit. The consequences of iron deficiency anaemia in cyanotic heart disease are dire, either in infancy or later. Metabolic acidosis, and cyanotic attacks are exacerbated by the presence of iron deficiency anaemia . Similarly very high counts of red blood cells, blood viscosity is increased, and with it the tendency to cerebrovascular accidents. When the haematocrit is above 60%,further small increases produces large increments in viscosity[5]. At a haematocrit level of 70% , blood viscosity is so high that fluidity in small vessel becomes critical.Measurements of MCV, MCH and serum ferritin reveal the existence of iron deficiency anaemia. In this study we made an attempt to study the children with cyanotic congenital heart disease ,by simple tests like complete hemogram and red cell indices and ascertain if it be enough to diagnose iron deficiency than the more expensive diagnostic tests like serum iron, total iron binding capacity and serum ferritin levels.

AIMS AND OBJECTIVES

a) To study prevalence of iron deficiency anaemia in children having cyanotic congenital heart disease

b) To assess various biochemical and hematological parameters of iron status in children having cyanotic congenital heart disease

INCLUSION CRITERIA

1) All proven case of cyanotic congenital heart disease by history , examination and echocardiography

2) Age of children between 6 months and 12 years.

EXCLUSION CRITERIA

1) Children less than 6 months of age

2) Post operative patients

3) Patients with chronic infectious diseases

MATERIALS AND METHODS

50 Children aged between 6 months to 12 cyanotic congenital heart disease were studied .Written informed consent obtained from their parents. Infants less than 6 months were excluded ,so that measurements would not be influenced by haematological changes at birth and the nadir of physiological anaemia of infancy was avoided.

Details were recorded on a pre- designed proforma. Detailed Medical history was taken. Previous medical records were analyzed& recorded. Complete clinical examination was done with specific emphasis regarding complications of iron deficiency and cyanotic congenital heart disease like focal neurological deficits and brain abscess.

Radiological investigations including x ray chest, electrocardiogram, two dimensional echocardiography were done and evaluated by radiologist and cardiologist.

Blood samples collected for relevant investigations :

Haematological measurements included complete hemogram, peripheral smear,red cell indices. These tests were done by coulter machine.

Biochemical parameters included serum iron ,total binding capacity, transferrin saturation and serum ferritin. These tests were done using spectrophotometric measurements.

Transferrin saturation of < 16% categorized as iron deficient,and > 16% categorized as iron sufficient. The various parameters like age , sex, haemoglobin, haematocrit ,MCV,MCH,MCHC, RDW,Platelet count , serum iron ,total binding capacity, transferrin saturation and serum ferritin were compared between the iron deficient and iron sufficient group. Results were tabulated and statistical analysis was done.

REVIEW OF LITERATURE

Congenital heart defects are the most common type of birth defects, affecting 8 out of every 1000 newborns[6]. various studies on congenital heart disease both cyanotic and acyanotic and its correlation with iron status have been studied by various authors. A short literature is as follows.

Embryology:

There is a complex sequence of events that result in a well formed heart at birth and disruption of any portion may result in a defect[61]. Around day 15 of development, the cells that will become the heart exist in two horse shoe shaped bands of middle tissue layer(mesoderm) [62]. On day 19, a pair of vascular elements ,theendocardial tubes form,which fuse to form a ring of heart cells(myocytes) around it by day 21. On day 22, the heart begins to beat and by day 24, blood is circulating [63]. At day 22, the circulating system is bilaterally symmetrical with paired vessels on each side and heart consisting of a single tube. From day 23 -28, heart tube folds and twists, with the future ventricles moving left of center and atria moving towards the head. On day 28, the membranous septum septumprimum and muscular endocardial cushions, fuse to form the four heart chambers of the heart. After this, the neural crest divide the bulbous

cordis into two by the growth of spiralling septum and becomes the great vessels – the ascending segment of aorta and the pulmonary trunk. If separation is incomplete, the result is a persistent truncusarteriosus. The vessels may be reversed as in transposition of great vessels. A small vessel, the ductusarteriosus allows blood from pulmonary artery to pass to the aorta.[63]

GENETICS :

Most of the known causes of congenital heart disease are sporadic genetic changes, either focal mutations or deletion or addition of segments of DNA. ([64]) major chromosomal abnormalities such as trisomy 21,13 and 18 cause about 5 to 8 % of CHD, with trisomy 21 being the most common. Mutations of a heart muscle protein, a – myosin heavy chain(MYH6) are associated with atrial septal defects . mutations in NKX2-5 gene is associated with defects in the electrical conduction of the heart and TBX5 is related to holt-oram syndrome.T1BX, is involved in velo-cardio-facial syndrome and digeorge syndrome.

Mutations in jagged 1 are identified in cases of arterio hepatic dysplasia (Alagille syndrome). Mutations of cell regulatory mechanism , the Ras/MAPK pathway are responsible for noonan syndrome, leopard syndrome, Costello syndrome and cardio-facio-cutaneous syndrome. known antenatal risk factors include maternal infections(rubella), drugs

(alcohol, hydantoin,lithium and thalidomide) and maternal illness(diabetes mellitus,phenylketonuria and systemic lupus erythematosus).

Congenital heart disease are broadly classified as acyanotic and cyanotic congenital heart disease.cyanotic heart disease is further classified as conditions with decreased blood flow like tricuspid atresia, tetralogy of fallot, single ventricle with pulmonary stenosis ,isolated pulmonary stenosis and conditions with increased blood flow includes transposition of great vessels,total anomalous pulmonary venous connection,single ventricle and single atrium.

Cyanosis is a bluish discolouration of skin and mucous membrane resulting from an increased concentration of reduced hemoglobin to about 5 g/dl in the cutaneous veins. The level of reduced hemoglobin in cutaneous veins may result from either desaturation of arterial blood or increased extraction of oxygen from peripheral tissue in the presence of normal arterial saturation(circulatory shock, hypovolemia ,vasoconstriction from cold).Cyanosis associated with desaturation of arterial blood is called central cyanosis. Cyanosis with a normal arterial saturation is called peripheral cyanosis.

An arterial oxygen saturation of 90% or above does not rule out a cyanotic heart disease in a newborn infant. An arterial oxygen saturation of 90 % can be seen with po2 of 45 to 50 mm Hg in newborns because of a normally leftward oxygen hemoglobin dissociation curve .in older children and adults, a Po2 of 60 to 65 mm Hg is needed to have 90 % oxygen saturation.

CONSEQUENCES AND COMPLICATIONS OF CYANOSIS

POLYCYTHEMIA:

Low arterial oxygen content stimulates bone marrow through erythropoietin release from the kidneys and produces an increased number of red blood cells. Polycythemia , with a resulting increase in oxygen-carrying capacity , benefits cyanotic children . However, when the haematocrit reaches 65 % or higher, a sharp increase in the viscosity of blood occurs, and the polycythemic response becomes disadvantageous, particularly if the is congestive heart failure. Some cyanotic infants have a relative iron deficiency state,with a normal or lower than normal hemoglobin and hypochromia on peripheral smear. A normal hemoglobin in a cyanotic patient represents relative anaemic state. Although less cyanotic , these infants are usually more symptomatic and improve when iron therapy raises the hemoglobin.

CLUBBING:

Clubbing is caused by soft tissue growth under the nail bed as a consequence of central cyanosis. The mechanism of soft tissue growth is unclear. One hypothesis is that megakaryocytes present in systemic venous blood may be responsible for the change. In normal persons , platelets are formed from the cytoplasm of the megakaryocytes by fragmentation

during their normal passage through the pulmonary circulation. The cytoplasm of megakaryocytes contains growth factors (eg. Platelet derived growth factor and transforming growth factor ß) . In patients with right –to left shunts, megakaryocytes with their cytoplasm may enter the systemic circulation, become trapped in the capillaries of the digits, and release growth factors, which in turn cause clubbing. Clubbing usually does not occur until a child is 6 months or older, and it is seen first and is most pronounced in the thumb. In the early stage, it appears as shininess and redness of the finger tips. When it is fully developed , the fingers and toes become thick and wide and have convex nails.

CENTRAL NERVOUS SYSTEM COMPLICATIONS :

Either very high haematocrit level or iron-deficient red blood cells place individuals with cyanotic congenital heart defects at risk for disorders of the central nervous system, such as brain abscess and vascular stroke. In the past, cyanotic CHD s account for 5% to 10 % of all cases of brain abscesses. The predisposition for brain abscesses may partially result from the fact that right-to-left intra cardiac shunts may bypass the normally effective phagocytic filtering actions of the pulmonary capillary bed. This predisposition may also result from the fact that polycythemia and the consequent high viscosity of blood lead to tissue hypoxia and micro infarction of the brain, which are complicated by bacterial colonization

.The triad of symptoms of brain abscesses are fever, headache, and focal neurologic deficit. Vascular stroke caused by embolization arising from thrombus in the cardiac chamber or in the systemic veins may be associated with surgery or cardiac catheterization. Cerebral venous thrombosis may occur, often in infants younger than 2 years who have cyanosis and relative iron deficiency anemia. A possible explanation for these findings is that microcytosis further exacerbates hyperviscosity resulting from polycythemia.

BLEEDING DISORDERS :

Disturbances of haemostasis are frequently present in children with severe cyanosis and polycythemia . Most frequently noted are thrombocytopenia and defective platelet aggregation . Other abnormalities include prolonged prothrombin time and partial thromboplastin time and lower levels of fibrinogen and factors V and VIII . Clinical manifestations may include easy bruising, petechiaeof the skin and mucous membranes, epistaxis, and gingival bleeding.

HYPOXIC SPELLS AND SQUATTING :

Although most frequently seen in infants with Tetralogy of Fallot (TOF), hypoxic spells may occur in infants with other congenital heart defects.

DEPRESSED INTELLIGENT QUOTIENT:

Children with chronic hypoxia and cyanosis have a lower than expected intelligence quotient as well as poorer perceptual and gross motor functions than children with acyanotic congenital heart defects, even after surgical repair of cyanotic heart defects.

SCOLIOSIS :

Children with chronic cyanosis, particularly girls and in patients with TOF, often have scoliosis.

HYPERURICEMIA AND GOUT :

Hyperuricemia and gout tend to occur in older patients with uncorrected or inadequately repaired cyanotic heart defects.

HAEMATOLOGICAL MANIFESTATIONS OF CONGENITAL CYANOTIC HEART DISEASE:

Erythrocyte disturbances in children with cardiac disease must be understood in the context of compensatory states or the context of iron deficiency as a complication. In infants with cyanotic congenital heart disease have erythropoietin-induced compensatory polycythemia. aortic oxygen saturation higher than 80% is usually associated with low erythropoietin titres and haemoglobin levels that will not cause hyperviscosity. Even with moderate degrees of hypoxemia ,elevated erythropoietin levels are not seen;presumably modest elevation in haemoglobin levels provide adequate tissue oxygenation. [15-17]. Infants with cyanotic congenital heart disease have higher iron requirements because of greater haemoglobin mass.diminished iron stores are associated with more right-shifted oxyhemoglobin dissociation curve[21].Most children with cyanotic congenital heart disease have evidence of mild macrocytosis. A mean corpuscular volume greater than the 90th percentile for age and sex nearly eliminates the possibility of iron deficiency.[18]

Haematological adaptations to maintain transport of oxygen to metabolizing tissues include excessive erythropoiesis which may lead to polycythemia and hyperviscosity[5].There is high incidence of cerebrovascular accidents in children with cyanotic congenital heart

disease. Hyperviscosity alone may acount for some ischaemic infarctions. The possibility of neurologic defects after the onset of iron deficiency in the presence of polycythemia have been described[19,20].Card and Weintraub showed that altered deformability in the presence of increased blood viscosity could result in vascular ischaemia.[21]

Cyanotic heart disease may result in poor perfusion of the spleen and subsequent functional hyposplenism manifested by howell-jolly bodies in peripheral blood.[22]

Coagulation abnormalities :

Many investigations have suggested that a coagulopathy exists in some patients with cyanotic congenital heart disease. Thrombocytopenia,low plasma fibrinogen levels,defective clot retraction ,hypoprothrobinemia, factor V and VIII deficiency, and evidence of fibrin degradation products in serum have been reported.[23-27]

Dennis and associates [28] were the first to report five patients with cyanotic congenital heart disease associated with coagulation abnormalities that were correctable by heparin. The presence of coagulation abnormalities correlates with the extent of polycythemia.[29] The exact mechanism producing the coagulopathy is not known and could

be multifactorial in origin. Hyper viscosity may lead to tissue hypoxemia,which then triggers a consumptive process.[30]

Platelet abnormalities :

Quantitative and qualitative platelet abnormalities are commonly associated with cardiac disease. In one study,the mean platelet count in cyanotic patients with an arterial oxygen saturation of less than 60% was 1,85,000 cells/cu.mm as compared with 3,15,000 cells/cu.mm in patients with an arterial oxygen saturation greater than 60% [31,32]. Examination of bone marrow has failed to demonstrate any quantitative changes in megakaryocytes to account for these platelet differences[31].This, together with the finding of shortened platelet survival in many patients, has suggested that the mechanism of the thrombocytopenia is destructive.it should be noted that although iron deficiency is commonly associated with cyanotic heart disease ,the quantitative platelet abnormalities are not related to iron status.

Qualitative platelet defects associated with cyanotic congenital heart disease may include prolonged bleeding times and abnormal aggregation in response to adenosine diphosphate,epinephrine and collagen[33]. These platelet functional abnormalities appear to be due to a defective platelet release mechanism because diminished release of [^{14}C] serotonin occurs in response to adenosine diphosphate whereas uptake of [^{14}C] serotonin is

normal[34]. Platelet release abnormalities are common in patients older than 4 years, in those with platelet counts less than 1,75,000 cells/cu.mm. There is no correlation between abnormalities in platelet aggregation and release and abnormal bleeding time.

Iron is a critical element in the function of all cells, although the amount of iron required by individual tissues varies during development. This plebeian metal is vital to the function of critical enzymes, including catalases, aconitases, ribonucleotidereductase, peroxidases, and cytochromes, that exploit the flexible redox chemistry of iron to execute a number of chemical reactions essential for our survival[7].

IRON DEFICIENCY ANAEMIA :

Iron deficiency anaemia is defined as anaemia caused by inadequate availability of iron to sustain bone marrow erythropoiesis. Anaemia caused by iron deficiency is the most common hematologic disease of infancy and childhood .the body of the newborn infant contains 0.3 to 0.5 g of iron; the body of an adult contains upto 5 g. to make up the 4.5 g difference, an average net increase of 0.5 mg of iron must be absorbed each day during the first 15 years of life. In addition to this requirement for growth,a small amount of iron is necessary to balance the normal losses, estimated at 0.5 to 1 mg/day. To maintain a positive iron balance during childhood , 0.8 to

1.5 mg of iron must be absorbed each day from the diet. Because less than 10% of dietary iron is absorbed from the average mixed diet, 8 to 15 mg of iron daily is necessary for optimal nutrition.[8]

According to the fourth national health and nutrition examination survey (NHANES IV) , iron deficiency without anaemia exists in 7% of toddlers aged 1 to 2 years, 9% of adolescent girls, and 16% of women of child bearing age[9].

Socioeconomic factors are associated with iron deficiency anaemia in children. For example, infants and children of low-income and minority backgrounds have higher documented rates of iron deficiency anaemia.[10-12]

The prevalence of iron deficiency anaemia in developing countries like India has been found to be as high as 63% in 1-3 years group of 3-6 years as per study by ICMR in 1977.More recent report of the NFHS-3 shows that the prevalence has not much changed in 2005-2006 and is still 73% among children of 6-35 months of age.Iron deficiency anaemia is most common in the age group of 6 months to 3 years of age.Breast milk contains iron with high bioavailability and hence iron deficiency anaemia is uncommon in exclusively breast fed infants before 4-6 months of age.Whereas non introduction of iron containing of weaning foods or

continuation of only milk feeds,especially more than 16 oz.per day and that too with feeding bottle,leads to iron deficiency anaemia in infants.anaemia due to worms especially hook worms is very common in poor socioeconomic groups of patients.World Health Organization recommends use of palmar pallor as a screening measure for anaemia.In mild anaemia there may be no signs and symptoms but in severe deficiency, all the symptoms of anaemia like fatigue, breathlessness, irritability, anorexia, etc. may be seen. Pica usually is the manifestation of iron deficiency and relieved when condition is treated. Epithelial changes like koilonychias, platynachia, angularstomatitis, atropic gastritis, mucosal changes in the stomach leading to atropic gastritis and achlorhydria and small bowel leading to esophageal mucosal webs as seen in Plummer Wilson syndrome.These changes are rarely seen in children with iron deficiency and are more common in adolescent with nutritional anaemia.There is marked reduction in weight in iron deficient children,Though height seems to be unaffected.Work output falls significantly due to iron deficiency anaemia.Besidesanaemia, iron deficiency leads to many other systemic effects;Notable amongst them is its effect on growing brain where it has been shown to lead to cognitive dysfunction, which is sometime permanent.Thesebehavioural changes occur due to diminished activity of aldehyde oxidase, required for the serotonin catabolism,thus leading to increase level of serotonin and 6 –

hydroxyindolecompounds. Reduced attention span, irritability,decreased scholastic performance, poor academic achievement and conduct disorder occur in these iron deficient children. Humoral, cell mediated and nonspecific immunity and the activity of the cytokinesis which have an important role in various steps of immunogenic mechanisms are influenced by iron deficiency anaemia. This is simply avoidable by simple treatment with iron which is cost effective and easily available medicine, provided we realize the importance of treating infants with iron deficiency promptly and effectively.

IRON METABOLISM

There are two major sources of food iron: Heme iron and nonhemeiron. Heme iron is highly bio-available and is present in meat,fish, poultry as well as in blood products. nonheme iron is a more important source and is found to varying degrees in all foods of plant origin. Maximum amount of iron is absorbed from the duodenum. two steps are involved in the absorption of iron: entry of iron from the intestinal lumen into the mucosal cells and its passage from mucosal cell into the plasma. only a fraction of iron that enters the mucosal cells finds its way to the plasma the remainder being held in the cell as ferritin which is lost from the body as the mucosal cell is desquamated into the lumen at the end of its i.e 3-4 days. With increased iron stores there is increased

transferrin saturation and increased messenger iron in the mucosal cell.this messenger iron stimulates the production of apoferritin.Thus whenever there is increase in transferrin saturation,a large fraction of iron entering the mucosal cell is held back as ferritin and discorded,as the cell is desquamated.excessive accumulation of iron by absorption is thus prevented.

PHASES OF DEVELOPMENT OF IRON DEFICIENCY :

Most of the body`s iron is directed towards synthesis of haemoglobin, erythrocyte production is among the first casualities of iron deficiency to become clinically apparent in usual laboratory evaluations. iron deficiency progresses through three discernible phases:

1. Prelatent iron deficiency occurs when tissue stores are depleted, without a change in haematocrit or serum iron levels. This stage of iron deficiency may be detected by low serum ferritin measurements.
2. Latent iron deficiency occurs when the reticuloendothelial macrophage iron stores are depleted. The serum iron level drops and TIBC increases without a change in haematocrit. This stage may be detected by routine checking of fasting, early morning transferrin saturation .erythropoiesis begins to be limited by a lack of available

iron, and sTfR levels increase . the reticulocyte haemoglobin content (CHr) decreases because newly produced erythrocytes are iron deficient[13].The bulk of the erythrocyte population appears normal.

3. Frank iron deficiency anaemia is associated with erythrocyte microcytosis and hypochromia. It is detected when iron deficiency has persisted long enough that a large proportion of the circulating erythrocytes were produced after iron became limiting.[13,14]

STAGES IN THE DEVELOPMENT OF IRON DEFICIENCY ANEMIA

	Stage 1	Stage 2	Stage 3
	PRELATENT	LATENT	ANEMIA
Bone marrow iron	Reduced	Absent	Absent
Serum ferritin	Reduced	< 15 μg/l	>15 μg/l
Transferrin saturation	Normal	< 16 %	>16 %
Free erythrocyticprotoporphyrin	Normal	Increased	Increased
Seru transferrin receptor	Normal	Increased	Increased
Reticulocyte haemoglobin content	Normal	Decreased	Decreased
Haemoglobin	Normal	Normal	Reduced
MCV	Normal	Normal	Reduced
Symptoms	Fatigue, malaise	Fatigue, malaise	Pallor,pica, epithelial changes

LABORATORY ASSESSMENT OF BODY IRON STORES:

1. HAEMOGLOBIN :

Haemoglobin is the red , oxygen carrying pigment in the red blood cells. Haematocrit values are roughly equivalent to three times haemoglobin concentration. Child is said to be anaemic when these levels are two standard deviation below the mean for the particular age and sex.

2. RED CELL INDICES :[35]

a) MEAN CORPUSCULAR VOLUME

It is expressed as volume of red cells

MCV (fl) =Hct * 10/RBC (10^6/µl)

NORMAL RANGE: Normocytic 75-100 fl

In iron deficiency it is microcytic and < 72 in children.

b) MEAN CORPUSCULAR HEMOGLOBIN :

It is the amount of haemoglobin per red blood cell

MCH(pg) =Hb *10/ RBC (10^6/µl)

Normal range : 30 +/-4

In iron deficiency anaemia it is < 31 g/dl

C)MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION :

It is the average concentration of haemoglobin in the red cells

MCHC(g/dl) =Hb *100/ Hct

Normal range : 31-37 g/dl

In iron deficiency anaemia it is < 31 g/dl

d) RED CELL DISTRIBUTION WIDTH(RDW):

Red cell distribution width Is the variability in the red cell volume.

In iron deficiency anaemia it is > 14.5 %

It is more sensitive(90-100%) and less specific(50-70%).

3) SERUM IRON AND TOTAL IRON BINDING CAPACITY:

Serum iron level represents the amount of circulating iron bound to transferrin . The TIBC is an indirect measure of the circulating transferrin.

Normal range for serum iron : 50 – 150 µg/dl

Normal range for TIBC : 300 -360 µg/dl

4) TRANSFERRIN SATURATION :

Transferrin saturation = serum iron * 100/ TIBC

A Transferrin saturation of < 16% is considered iron deficient.

Age	Hb(g%) Mean (-2 SD)	Hct% Mean (- 2SD)	RBC(mill/cu.mm) Mean (-2 SD)	MCV(fl) Mean (-2SD)	MCH(pg) Mean (- 2SD)	MCHC(g/dl) Mean (-2 SD)
6 mon– 2 yr	12(11)	36(33)	4.5(3.7)	78(70)	27(23)	33(30)
2-6 yrs	12.5(11.5)	37(34)	4.6(3.9)	81(75)	27(24)	34(31)
6-12 yrs	13.5(11.5)	40(35)	4.6(4.0)	86(77)	29(25)	34(31)

5) SERUM FERRITIN :[36]

Iron is stored complexed to protein as ferritin within cells

To estimate iron stores,serum ferritin is an important laboratory investigation.

Serum ferritin levels <15 ng/dl are diagnostic of iron deficient anaemia and absent iron stores.

6) RED CELL PROTOPORPHYRIN LEVELS :

Red cell protoporphyrinlevels reflects an inadequate iron supply to erythroid precursors to support haemoglobin synthesis.

Normal values are <30 µg/dl .in iron deficiency it is > 100 µg/dl.

7) <u>TRANSFERRIN RECEPTOR PROTEIN LEVELS :</u>

Serum levels of transferrin receptor protein reflects the total erythroid marrow mass.It is determined by radioimmuno assay.

Normal values are 4 -9 µg/L , it is elevated in iron deficiency anaemia

8) <u>FREE ERYTHROCYTIC PROTOPORPHYRIN AND PROTOPORPHYRIN : HEME (P: H) RATIO :</u>

It indicates thesupply of iron to the red cells over a prolonged period .

Normal range of free erythrocyticprotoporphyrin 30- 40 µg/dl RBC and P:H ratio is 16 (=/- 5.3)

Free erythrocyticprotoporphyrin above 70 µg/dl RBC and P:H ratio above 32 is considered iron deficiency.

9) <u>RETICULOCYTE HEMOGLOBIN CONTENT :</u>

It is a new method for diagnosing iron deficiency anaemia, which is diagnosed by bone marrow examination.Response to therapy becomes the diagnostic measure. A positive response is defined as daily increment of 0.1 mg/dl of haemoglobin from fourth day onwards.

Several studies have been studied on cyanotic congenital heart disease in children , But few studies were done on Iron deficiency anaemia in children with cyanotic congenital heart disease. Here are some studies:

Onur CB[37] et al determined the incidence of iron deficiency in children with cyanotic congenital heart disease in 44 children with cyanotic congenital heart disease .The prevalence of iron deficiencyanemia was 63.6%. They concluded that MCV,MCH,RDW are useful indicators of iron deficiency than hemoglobin (Hb), hematocrit (Hct) and red blood cell (RBC) .

OlcayL[38] et al studied a group of 67 children with cyanotic congenital heart disease . Among the 67 patients with CCHD whose haematocrit was < 60 %, prevalence of iron deficiency was 52.2% .Out of this, iron treatment was given to 35 patients who had MCV < 60 by 6 mg/kg/day. Post treatment values were taken . Hemoglobin and haematocrit with RBC count had significant correlation. They concluded that Hemoglobin (Hb), hematocrit (Hct) , mean corpuscular haemoglobin and RBC count are useful measurements for diagnosis of iron deficiency anaemia in children with cyanotic congenital heart disease.

Drossos et al[39] determined Hemoglobin (Hb), hematocrit (Hct) and mean corpuscular hemoglobin concentration (MCHC),serum iron in

74 children (including both acyanotic and cyanotic) with congenital heart disease. Using serum iron as a predictor, incidence of anaemia was found as 37.5% for < 5 years and 12.5 % for 6-12 years. . Using MCHC as a predictor , incidence of anaemia was found as 44% for < 5 years and 23.8 % for 6-12 years. The data shows that the MCHC is a precise indicator of iron deficiency anaemia in children with cyanotic heart disease and that the incidence of hypochromic anaemia is high in cyanotic patients.

Lang'oMO[40] et al examined the haematological profile of children with cyanotic heart disease and to document the prevalence of abnormal coagulation and iron deficiency in these children. The prevalence of iron deficiency was found to be 16.9% , Prolonged APTT- 32.1%, prolonged PT-3.6%, low platelets-7.1%, and raised D-dimer - 60%

The sensitivity of Low MCV in detecting iron deficiency was 58.8% with a specificity of 51.2%. For Low MCH the sensitivity was 52.9% with a specificity of 50.6%. Findings of microcytic hypochromic anemia on peripheral blood film gave a sensitivity of 50% and specificity of 73.4%.

Braun SL Et al[41]studied that due to chronic hypoxia,to improve oxygen transportation, the adaptive response which occurs is erythrocytosis. Due to this the haematocrit levels increase leading to symptoms of hyperviscosity. Due to elevated haemoglobin concentration

iron deficiency is often underestimated. Hence for institution of accurate management and avoid errors, iron status must be accomplished by biochemical parameters.

Samia H. Osman, MD et al[42] stated that, Children with cyanotic heart disease have deficient oxygen transport to tissues that might be complicated by polycythaemia with the potential risk of brain injury and abnormal haemostatic mechanisms: thrombis or bleeding diathesis. Thirty-one patients were seen during the study, 19 were males and 12 were females. The complications were: iron deficiency in two-thirds of the cases, poylcythaemia in half of the cases, low serum ferritin and prolonged INR in one third of the cases. Thrombocytopenia and prolonged bleeding time was detected in 12% of the cases. Significant correlation between polycythemia and the oxygen saturation was detected (p= 0.03). No significant correlation was found between the prolonged INR and the age, duration of CCHD since diagnosis, type of CCHD and the oxygen saturation.

Haga P et al[3] between arterial oxygen saturation and haemoglobin and haematocrit. Measurements of MCV,MCH and serum ferritin reveal the existence of iron deficiency anaemia.

S. Roodepeyma et al[43] studied the red cell indicies in patients with cyanotic heart disease. 11 patients had MCV < 3 rd percentile for age and sex, MCH and MCHC < 3 rd percentile in 44.4% and 62% respectively. Hypochromia is more prevalent than microcytosis in patients.

David W.West et al[44] studied the risk of iron deficiency in children with CCHD < 6 years. In their study 50% of children had iron depletion 30% had iron deficiency with anaemia ,who were younger than those without iron deficiency.

Gaiha M et al [45]studied the hematological parameters of iron deficiency anaemia and its correlation with hyperviscosity symptoms in CCHD in 33 cases and to assess the response to low dose iron therapy. Results showed presence of IDA is18.2%,hyperviscosity symptoms is 30.3 % and low dose iron therapy was found effective in relieving the symptoms of hyperviscosity.

Otwin Linderkamp et al[46] studied 59 children with various cyanotic congenital heart disease for hyperviscosity and effect of microcytosis on the viscosity of blood.

The chance of developing hyperviscous blood and risk of cerebrovascular complications were higher due to iron deficient RBCs which were microcytic in morphology.

Charlie Phornphutkul et al[47] reviewed 30 cases of CCHD to determine the risk of cerebrovascular accidents. The overall incidence was 1.6 %. 90 % of cerebrovascular accidents occurred in patients with TOF and dextroposition of the great arteries. Cerebrovascular accidents in less than 4 years of age was associated with anaemia (low MCHC) and hypoxemia ,in contrast in older children it was associated with polycythemia and hypoxemia.

Robert R. Martelle et al[48] studied 50 patients with CCHD for Cerebrovascular accidents. They found that no correlation between haematocrit and occurrence of CVA.

Nassar Amash et al[49] studied 162 adults with CCHD for incidence of Cerebrovascular accidents .There was increased risk of CVA associated with microcytosis ($p < 0.01$). They concluded treating microcytosis is necessary.

D.W .Miligan et al[50] studied the impact of red cell indices on viscosity of blood in polycythemic patients. The results of this study was that reduction of MCH or MCV leads to increased viscosity of blood due to cell to cell interaction.

Milton H. Paul et al [51]studied about the association of thrombocytopenia with that of CCHD. They studied 200 patients and found that thrombocytopenia is rare unless haematocrit was > 65% or oxygen tension < 70%. Incidence of thrombocytopenia under 1 year of age was 10% whereas in more than 1 year ,it was 60 %.

Hitoshi Horigome et al[52] studied the role of platelets in the pathogenesis of abnormal coagulation in the patients with CCHD with polycythemia. Results showed production of microparticles correlated positively with haematocrit and markedly increased at haematocrit above 60% in patients with CCHD. Hence they concluded that circulating incompetent platelets as well as microparticles , might play a role in coagulation abnormalities.

Anne L.Wedemeyer et al[53] studied thecoagulation abnormalities in 33 CCHD with haematocrit > 45 %,age group 5 months -15 years and body weight >6 kgs .results showed that thrombocytopenia was common with haematocrit of 65% and rare when haematocrit was > 65%. The severity of thrombocytopenia was related to polycythemia. Patients with a haematocrit of 60 -70 % had higher incidence of thrombocytopenia , abnormal clot retraction, shortened euglobin clot lysis time and presence of fibrin degradation products.

Forest H .Adams[54], M.D. et al studied 18 children with moderate to severe cyanotic heart disease for the haemoglobin,the red blood cell count, the haematocrit ,the mean corpuscular diameter and the carbonic anhydrase content of the cells. their study showed that polycythemia in such children is not associated any changes in red cell size or carbonic anhydrase content.

ShelonitaS.Rose[55] et all studied cyanotic heart disease with symptomatic erythrocytosis and concluded that the treatment is volume replacement and low dose iron therapy. Because repeated phlebotomy causes iron deficiency with microcytic erythrocytes,which increases the viscosity of blood and increase the risk for cerebrovascular accident.

Edgar L W Tay[56] et al studied 25 adult with iron deficiency anaemia and gave oral ferrous fumarate. After 3 months of therapy in iron deficient patients, resulted in improvement in exercise tolerance and quality of life.

Michael H. Rosove[57] et al studied in adults with cyanotic congenital heart disease for decompensated erythrocytosis and iron supplementation was given for these patients.Despite the normal levels of iron and ferritin, microcytosis, hypochromia and raised erythrocyte protoporphyrin persisted ,representing failure of iron procurement and

delivery system in an extreme cases of erythroid hyperplasia induced by hypoxemia.

Jonathon L. Maguire [58]et al did a study from stroke registry and concluded that iron deficient children are 10 times more risk for stroke than iron sufficient children.

Andrea Sherriff [59]et al did study to define the normal ranges and investigated the associated factors for haemoglobin and ferritin concentration at 8, 12 and 18 months of age. According to their study a definition of anaemia based on the fifth centile gives a cut off point at 12 and 18 months of age of haemoglobin < 10 g/dl and for iron deficiency of ferritin < 16μg/L and < 12 μg respectively.

Samuel S.Gidding[60] et al from their study done in children memorial hospital , concluded that patients with high erythropoietin titres had lower $paO_{2,}$ lower aortic saturation and higher red cell 2,3 DPG. The relationship between aortic oxygen saturation and haemoglobin saturation was strong.

STUDY DESIGN

Prospective analytical study done in a tertiary care hospital for aperiod between October 2011 –November 2012

STATISTICAL ANALYSIS

The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using **Epidemiological Information Package (EPI 2010)** developed by Centre for Disease Control, Atlanta.

Using this software range, frequencies, percentages, means, standard deviations, chi square and 'p' values were calculated. Pearson chi-square test, Fisher exact test was used to test the significance of difference between quantitative variables. A 'p' value less than 0.05 is taken to denote significant relationship.

RESULTS

Totally 50 children were studied during the study period.

Table 1 :Distribution among cases -age (years) :

Age(years)	No.	Percentage
< 1	12	24%
1 to 6	24	48%
6 to 12	14	28%
Total	50	100%

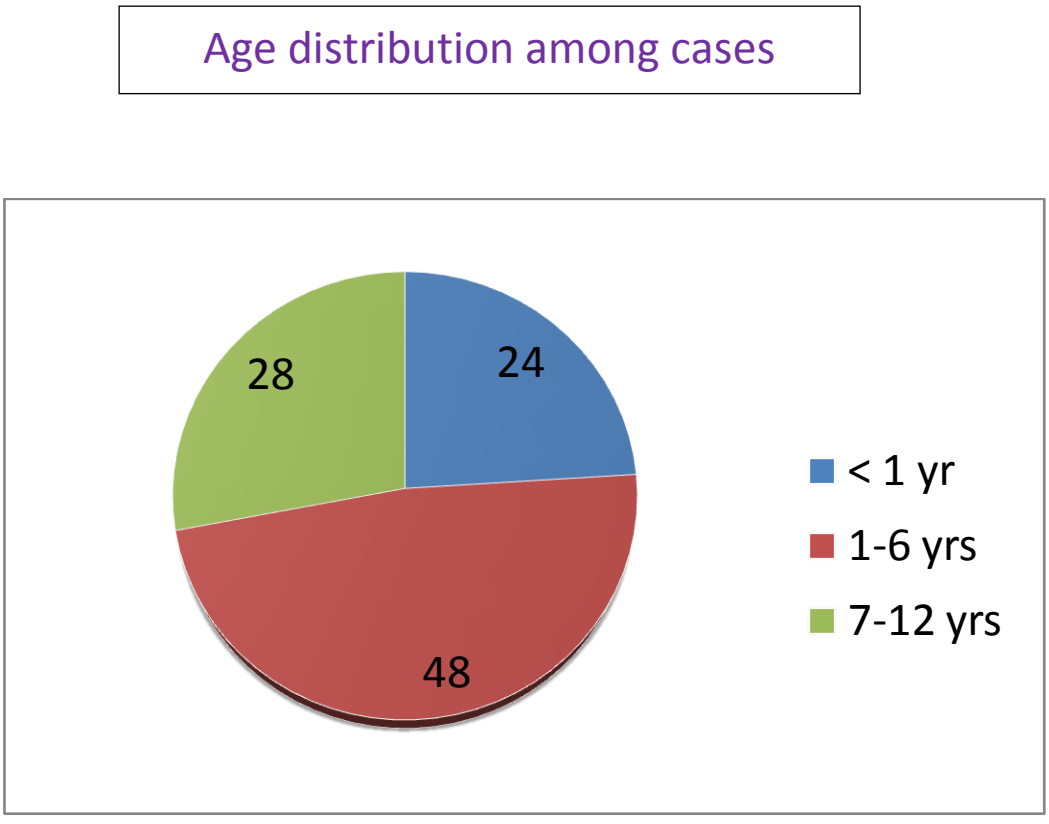

Table 2 : Distribution of cases among sex

Sex	No.	Percentage
Male	25	50%
Female	25	50%
Total	50	100%

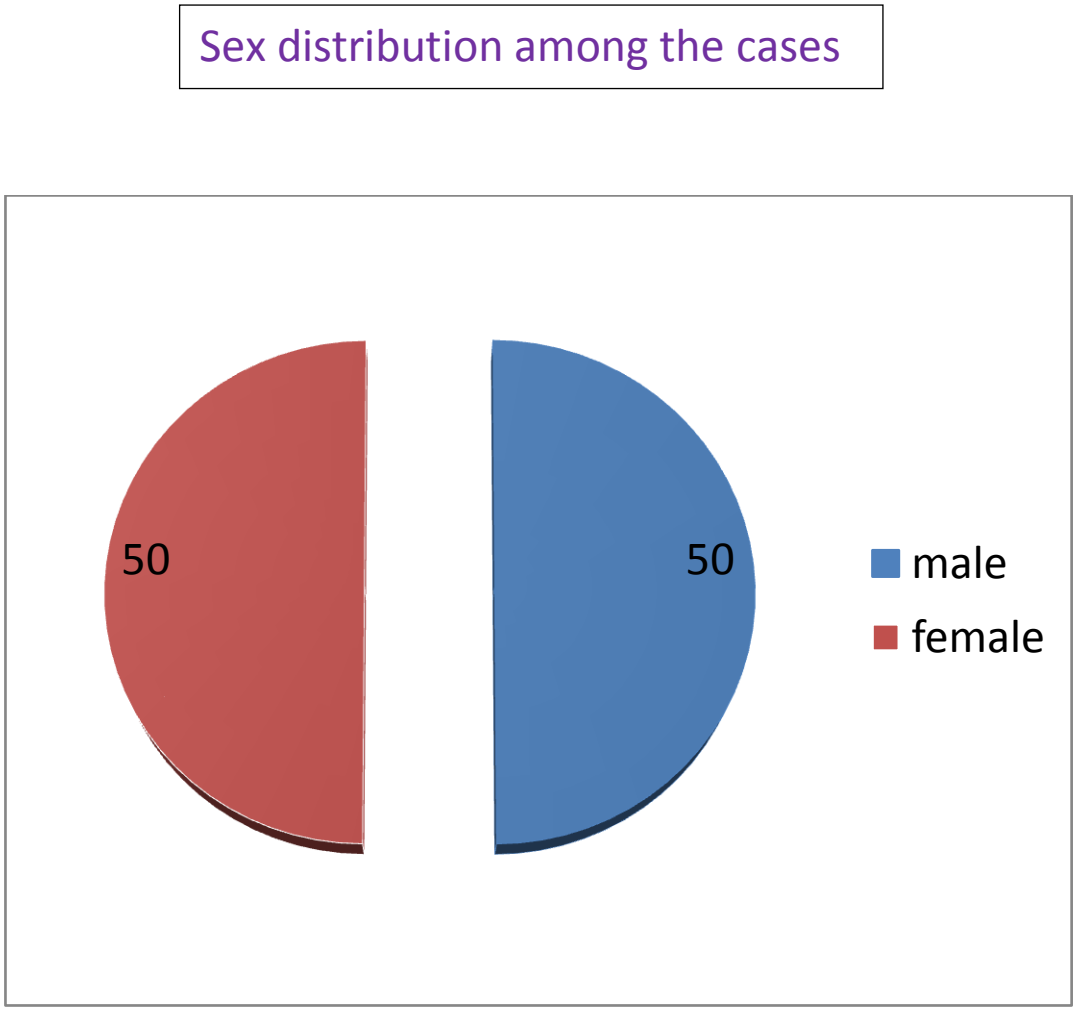

Table 3 : Distribution of cases - Cyanosis

Cyanosis	No.	Percentage
Present	40	80%
Absent	10	20%
Total	50	100%

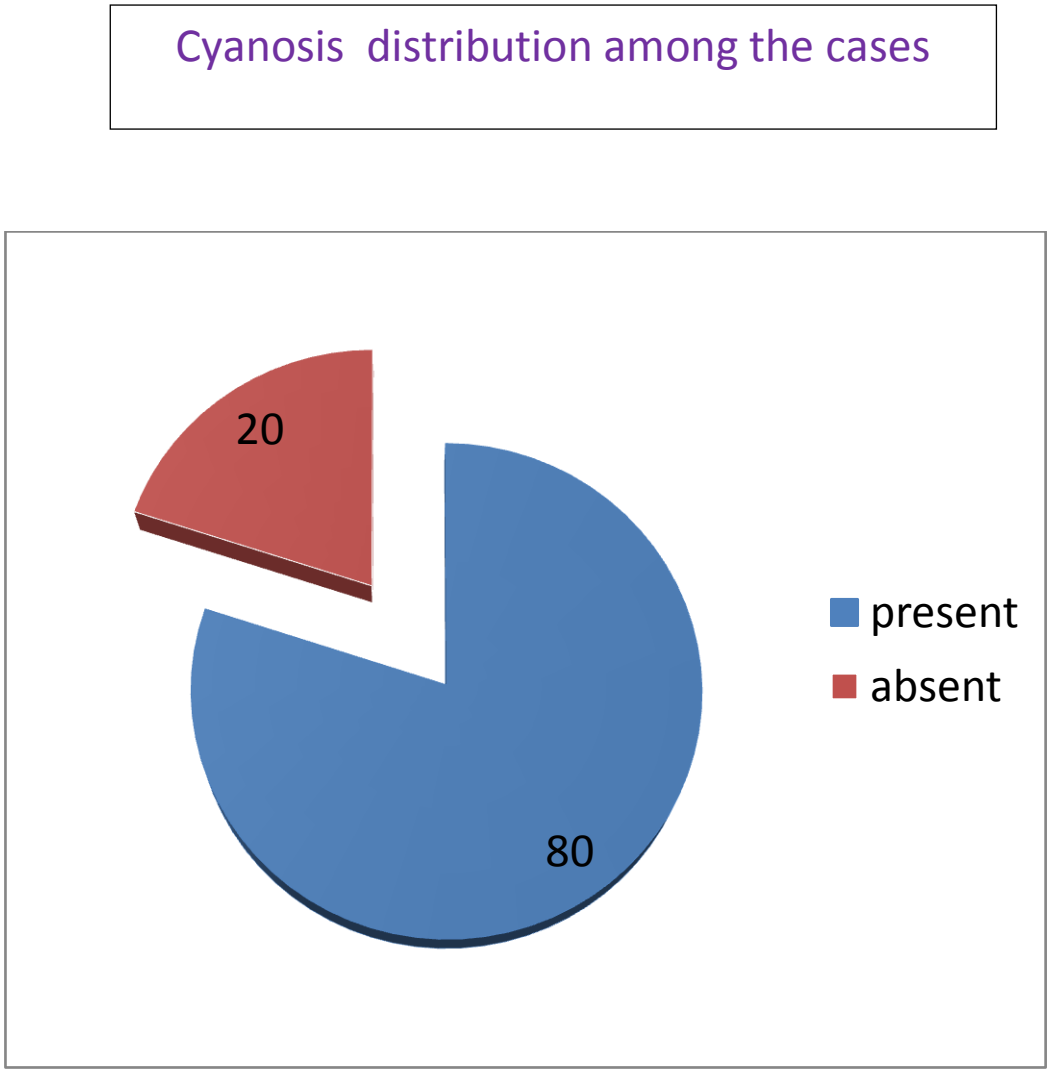

Table 4 : Distribution of cases - Clubbing

Clubbing	No.	Percentage
Present	35	70%
Absent	15	30%
Total	50	100%

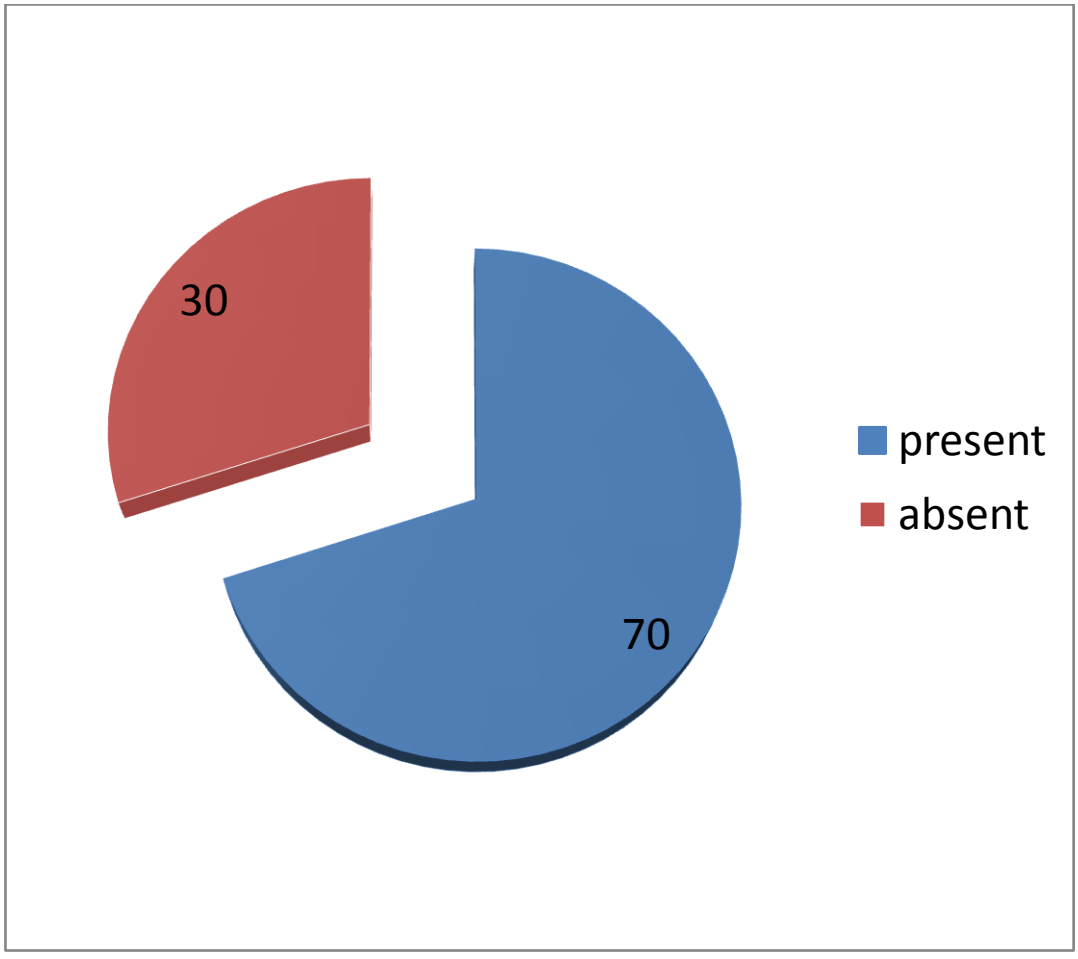

Table 5 :Distribution of cases among transferrin saturation

Transferrin saturation (%)	No.	Percentage
< 16	35	70%
>16	15	30%
Total	50	100%

Transferrin saturation of < 16% categorized as iron deficient

Transferrin saturation of > 16% categorized as iron sufficient

Prevalence of iron deficiency in children with cyanotic congenital heart disease

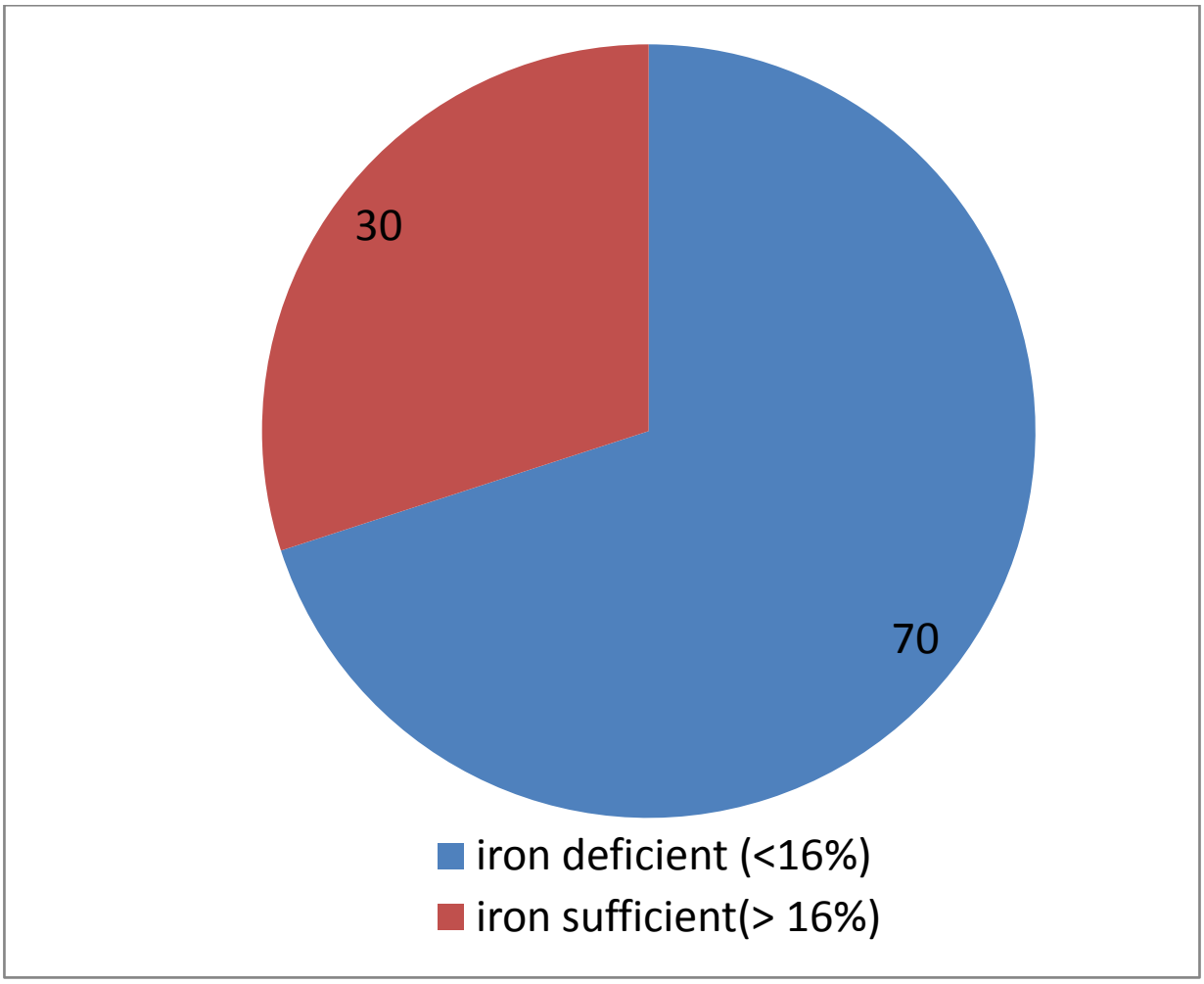

Table 6 :Association among cases between - age and iron status

Age (yrs)	Iron deficient		Iron sufficient		Total	
	No .	%	No.	%	No.	%
< 1	6	50	6	50	12	100
1 to 6	20	83.3	4	16.7	24	100
6 to 12	9	64.3	5	35.7	14	100
Total	35		15		50	

Chi-Square Tests			
	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	4.535[a]	2	.104
Likelihood Ratio	4.575	2	.102
Linear-by-Linear Association	.461	1	.497
N of Valid Cases	50		

Association among cases between - age and iron status

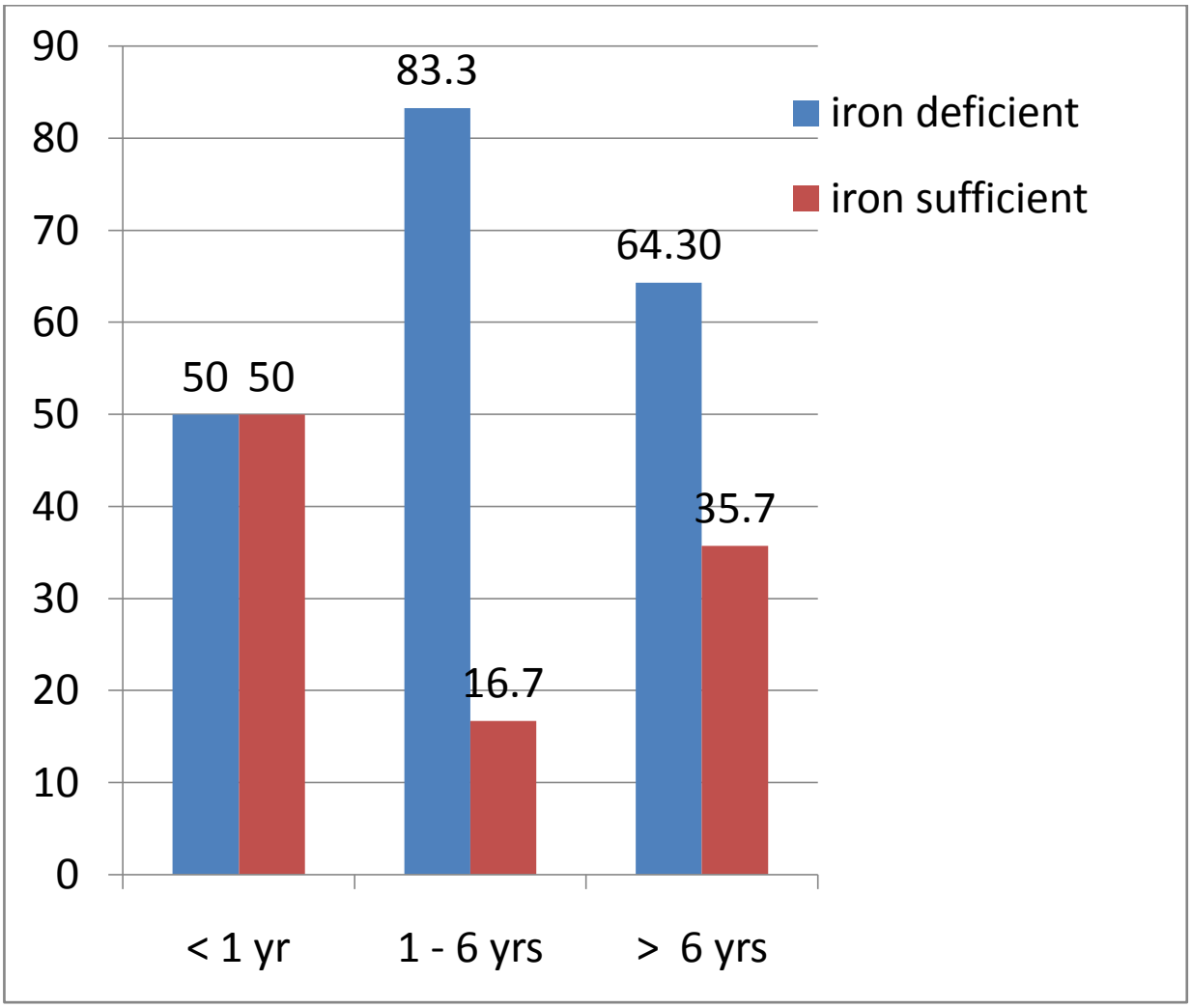

Table 7: Association among cases between - sex and iron status

Sex	Iron deficient		Iron sufficient		Total	
	No .	%	No.	%	No.	%
Male	16	64	9	36	25	100
Female	19	76	6	24	25	100
Total	35		15		50	

Chi-Square Tests					
	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.857[a]	1	.355		
Continuity Correction[b]	.381	1	.537		
Likelihood Ratio	.862	1	.353		
Fisher's Exact Test				.538	.269
Linear-by-Linear Association	.840	1	.359		
N of Valid Cases	50				

Association among cases between - sex and iron status

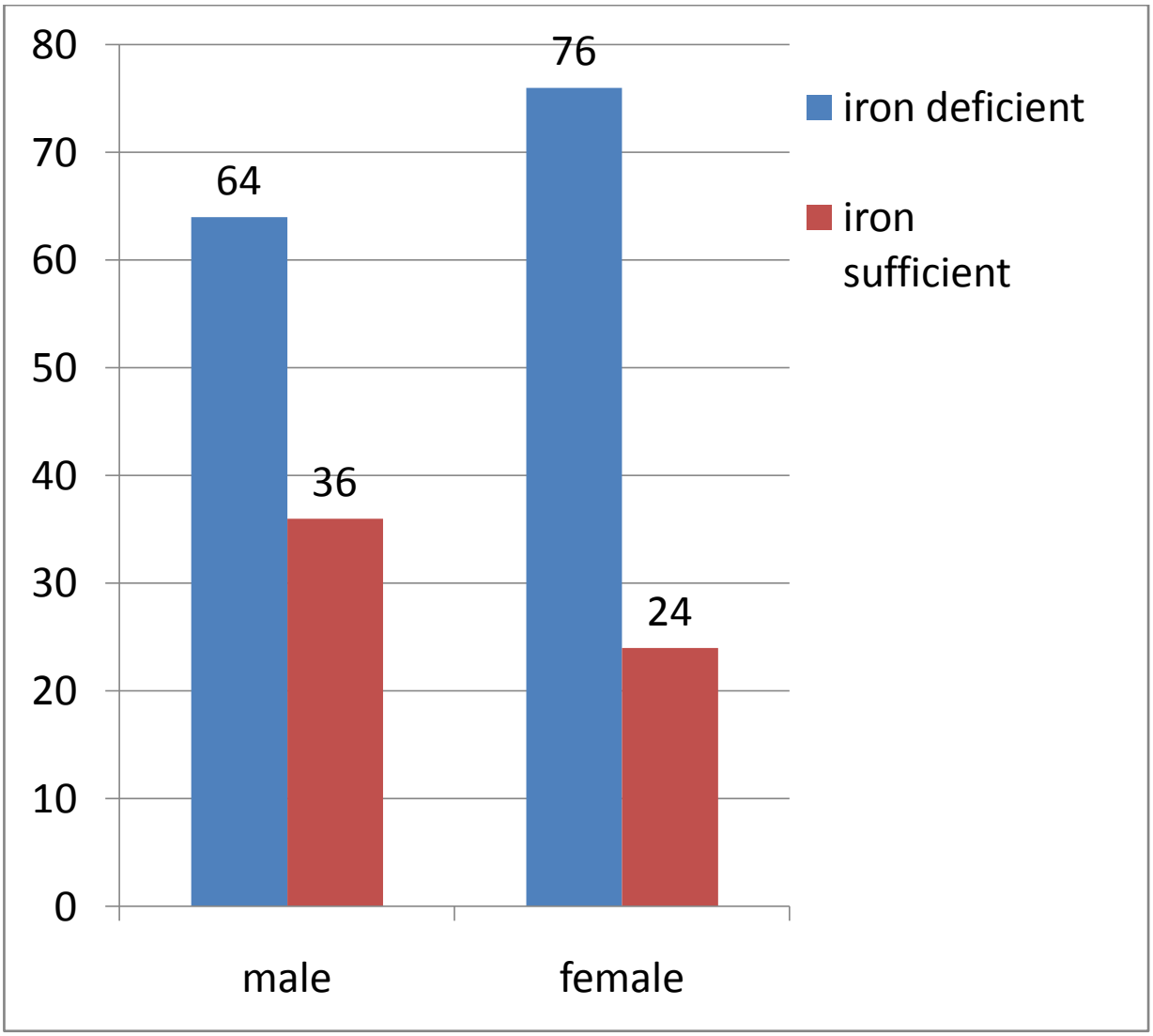

Table 8:Association between Hemoglobin(gm%) and iron status

Hemoglobin (gm%)	**Iron deficient**		**Iron sufficient**		**Total**	
	No .	**%**	**No.**	**%**	**No.**	**%**
< 11.5	4	50	4	50	8	100
11.5 – 14	11	64.7	6	35.3	17	100
>14	20	80	5	20	25	100
Total	35		15		50	

Chi-Square Tests			
	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	2.941[a]	2	.230
Likelihood Ratio	2.902	2	.234
Linear-by-Linear Association	2.882	1	.090
N of Valid Cases	50		

Association between Hemoglobin(gm%) and iron status

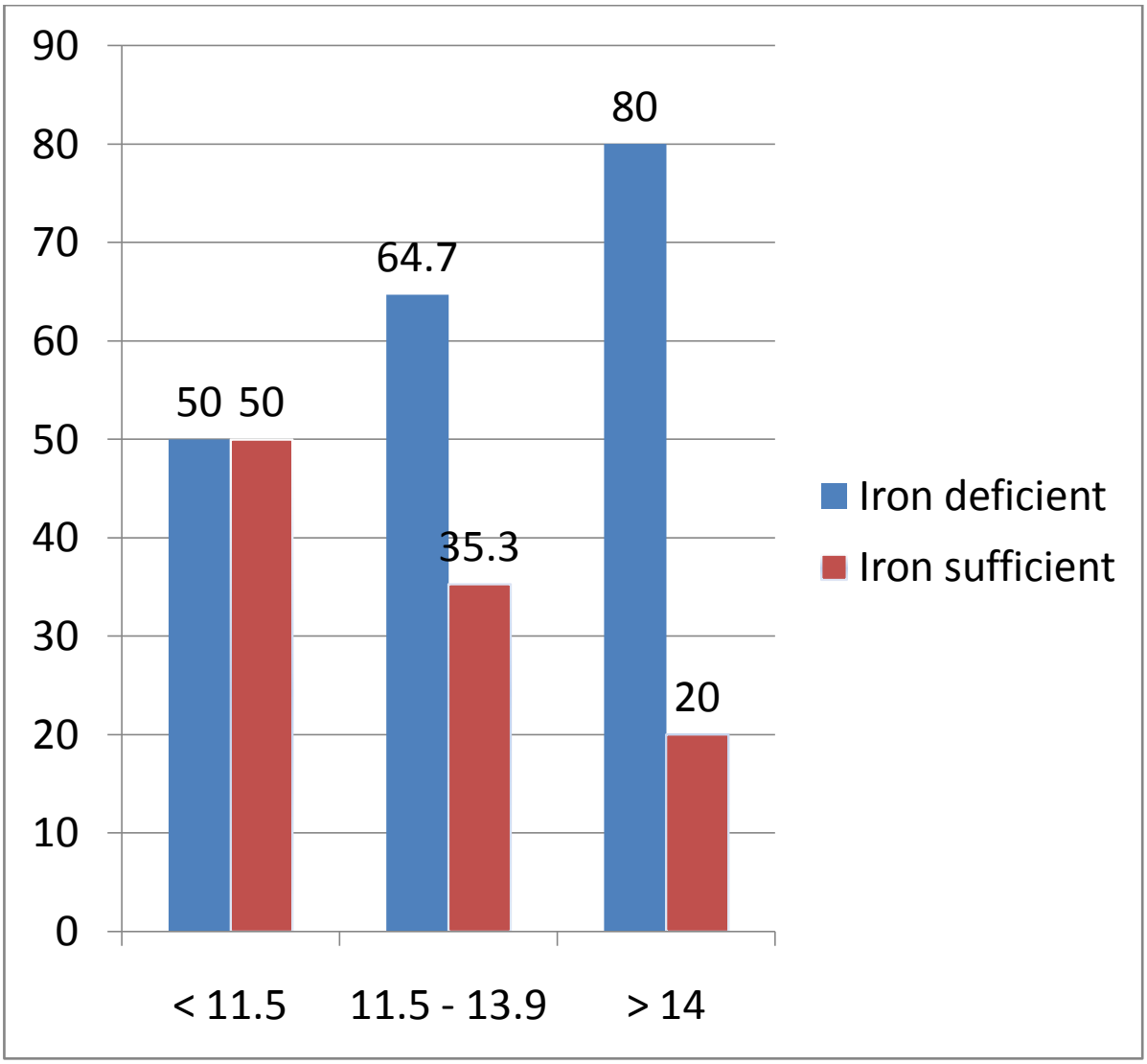

Table 9:Association among cases between - Hematocrit(%) and iron status

Hematocrit(%)	Iron deficient		Iron sufficient		Total	
	No .	%	No.	%	No.	%
< 34	2	50	2	50	4	100
34 -60	20	60.6	13	39.4	33	100
>60	13	100	0	0	13	100
Total	35		15		50	

Chi-Square Tests			
	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	7.720[a]	2	**.021**
Likelihood Ratio	11.290	2	**.004**
Linear-by-Linear Association	6.703	1	**.010**
N of Valid Cases	50		

Association among cases between – Hematocrit(%) and iron status

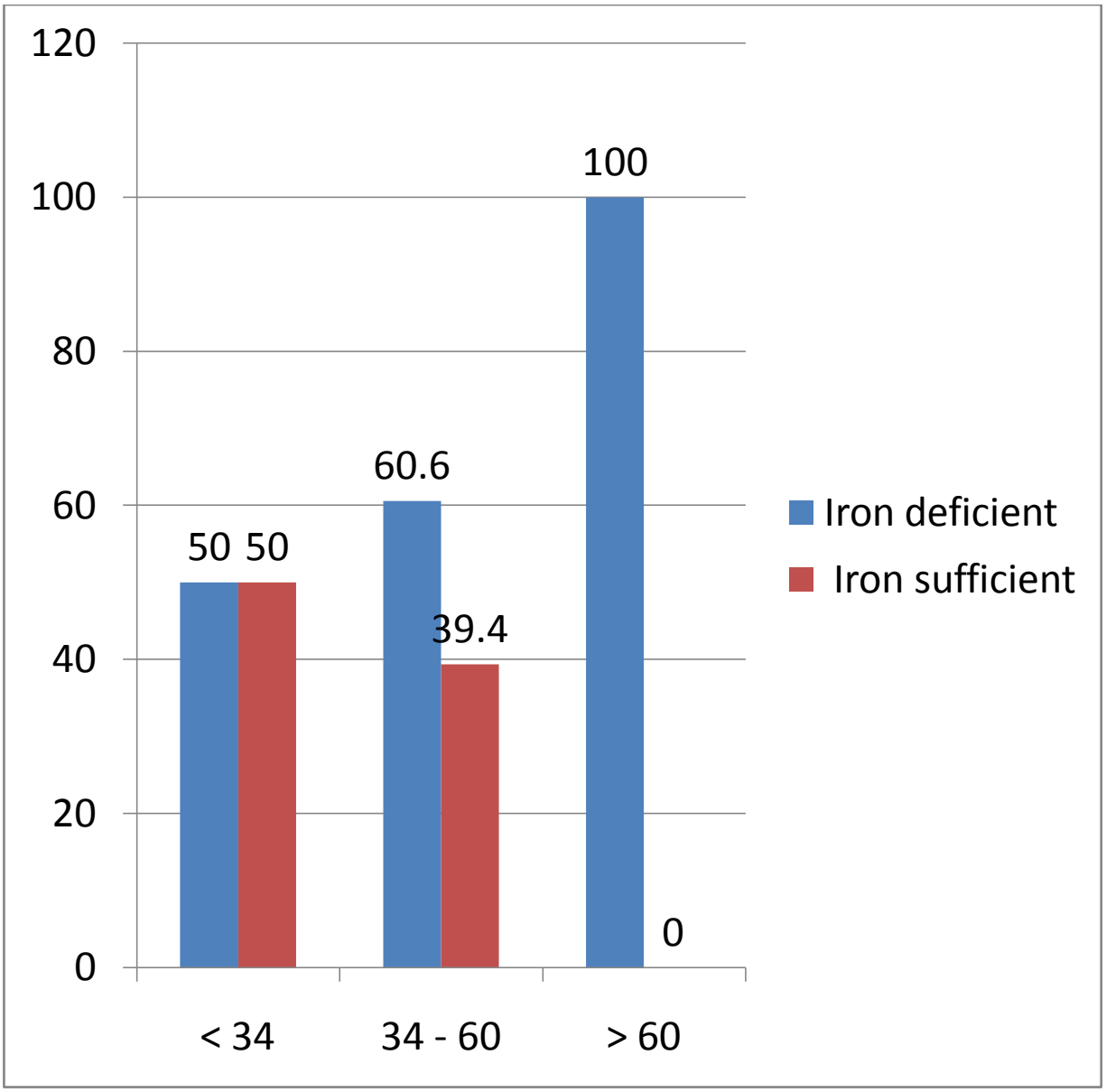

Table 10:Association among cases between - MCV (fl) and iron status

MCV(fl)	Iron deficient		Iron sufficient		Total	
	No .	%	No.	%	No.	%
< 75	29	82.9	6	17.1	35	100
75 -100	6	42.9	8	57.1	14	100
>100	0	0	1	100	1	100
Total	35		15		50	

Chi- square test	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	10.000[a]	2	**.007**
Likelihood Ratio	9.895	2	**.007**
Linear-by-Linear Association	9.797	1	**.002**
N of Valid Cases	50		

Association among cases between - MCV (fl) and iron status

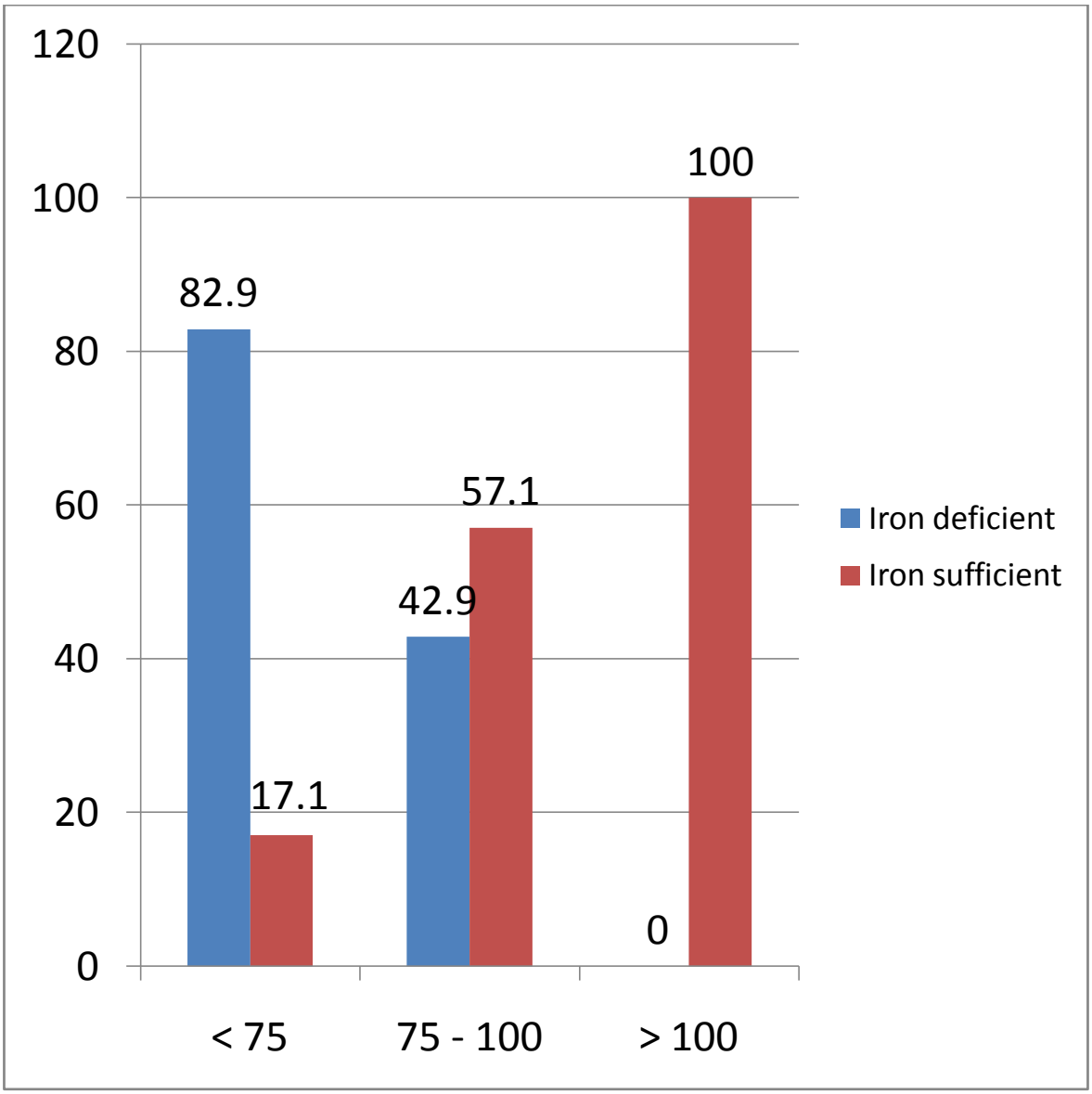

Table 11 :Associationamong cases between - MCH(pg) and iron status

MCH(pg)	Iron deficient		Iron sufficient		Total	
	No .	%	No.	%	No.	%
<24	27	73	10	27	37	100
>24	8	61.5	5	38.5	13	100
Total	35		15		50	

Chi-Square Tests					
	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.599[a]	1	.439		
Continuity Correction[b]	.178	1	.673		
Likelihood Ratio	.582	1	.445		
Fisher's Exact Test				.493	.330
Linear-by-Linear Association	.587	1	.444		
N of Valid Cases	50				

Association among cases between - MCH(pg) and iron status

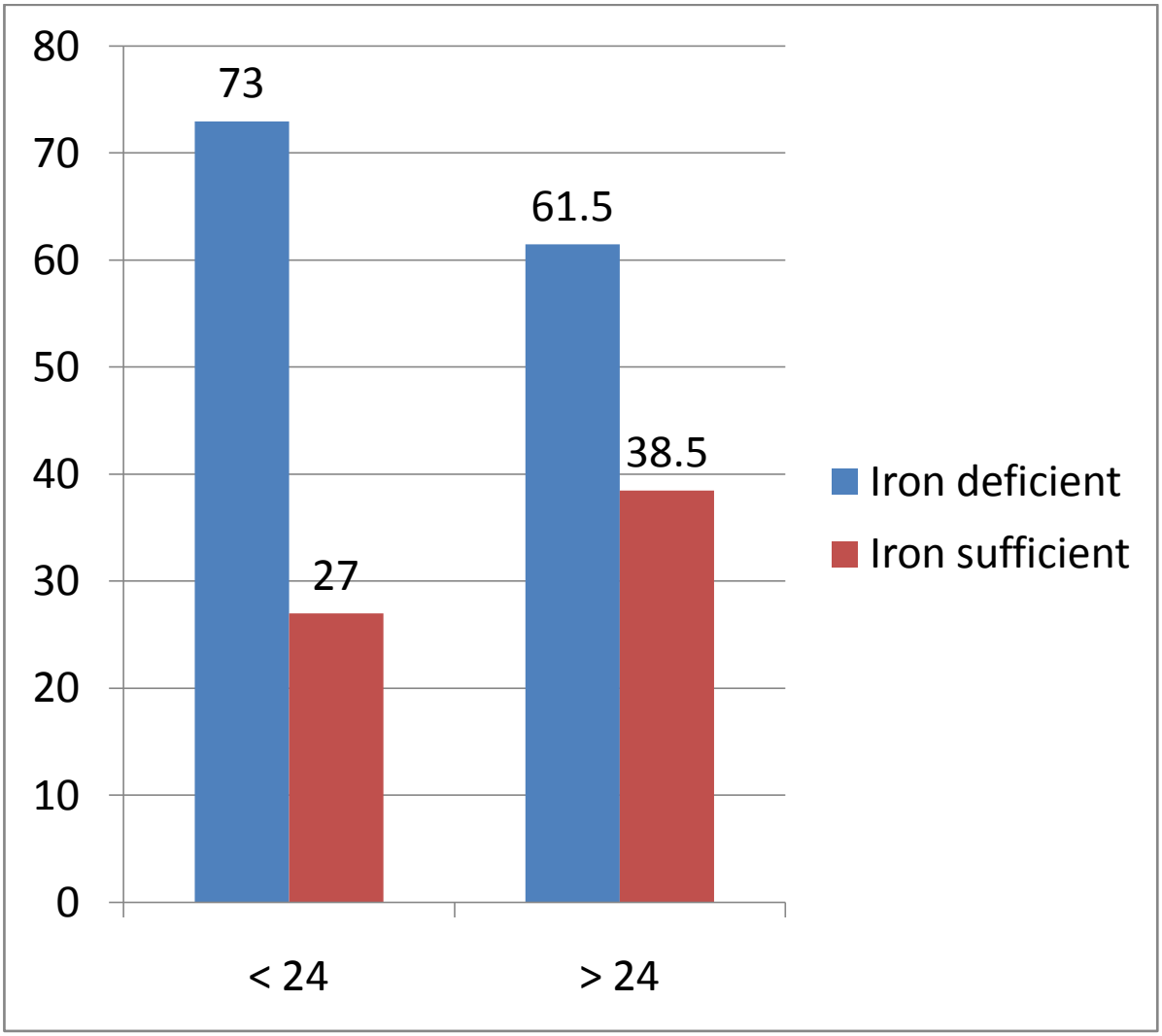

Table 12 :Association between MCHC(g/dl) and iron status

MCHC(g/dl)	Iron deficient		Iron sufficient		Total	
	No .	%	No.	%	No.	%
<31	19	70.4	8	29.6	27	100
>31	16	69.6	7	30.4	23	100
Total	35		15		50	

Chi-Square Tests					
	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.004[a]	1	.951		
Continuity Correction[b]	.000	1	1.000		
Likelihood Ratio	.004	1	.951		
Fisher's Exact Test				1.000	.596
Linear-by-Linear Association	.004	1	.951		
N of Valid Cases	50				

Association between MCHC(g/dl) and iron status

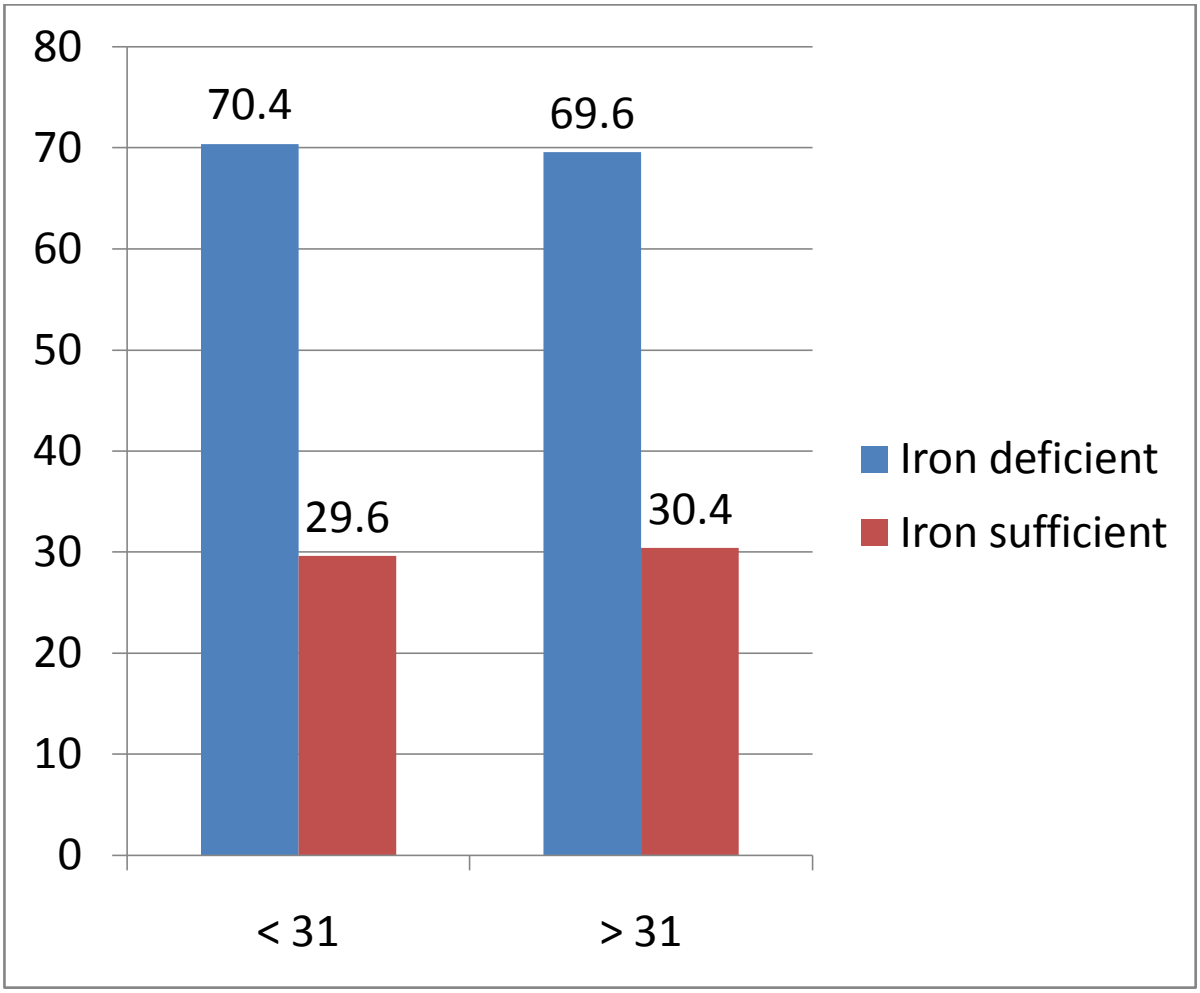

Table 13.Association between - serum iron (mg%) and iron status

Serum iron	Iron deficient		Iron sufficient		Total	
	No .	%	No.	%	No.	%
< 50	20	100	0	0	20	100
50 -100	15	65.2	8	34.8	23	100
>100	0	0	7	100	7	100
Total	35		15		50	

Chi-Square Tests			
	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	25.155[a]	2	**.000**
Likelihood Ratio	31.366	2	**.000**
Linear-by-Linear Association	23.474	1	**.000**
N of Valid Cases	50		

Association between - serum iron (mg%) and iron status

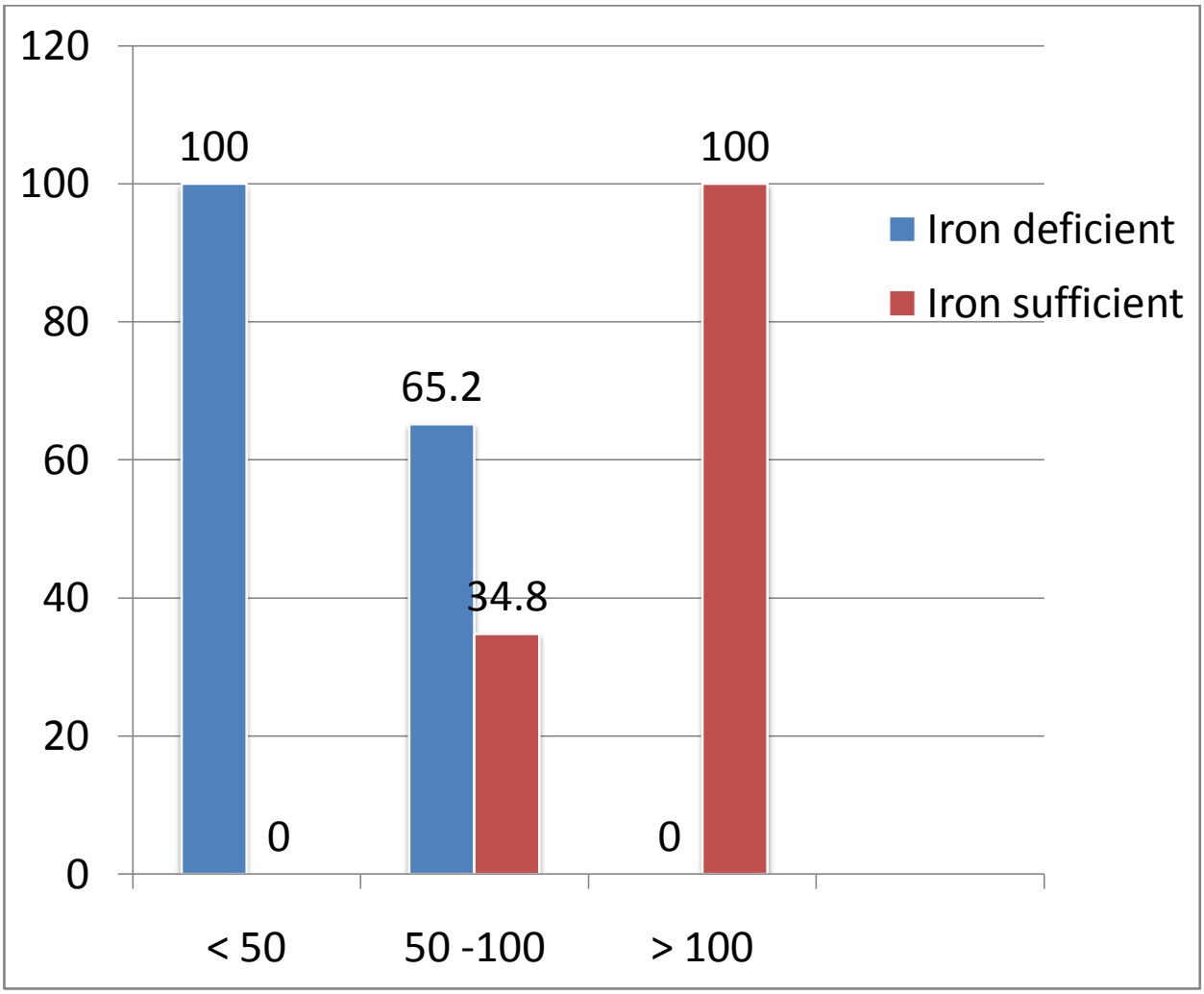

Table 14:Association between TIBC and iron status

TIBC	Iron deficient		Iron sufficient		Total	
	No .	%	No.	%	No.	%
< 250	1	100	0	0	1	100
250 -450	19		11		30	100
>450	15	78.9	4	21.1	19	100
Total	35		15		50	

Chi-Square Tests					
	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	1.168[a]	1	.280		
Continuity Correction[b]	.582	1	.445		
Likelihood Ratio	1.205	1	.272		
Fisher's Exact Test				.351	.225
Linear-by-Linear Association	1.145	1	.285		
N of Valid Cases	50				

Association between TIBC (µg%) and iron status

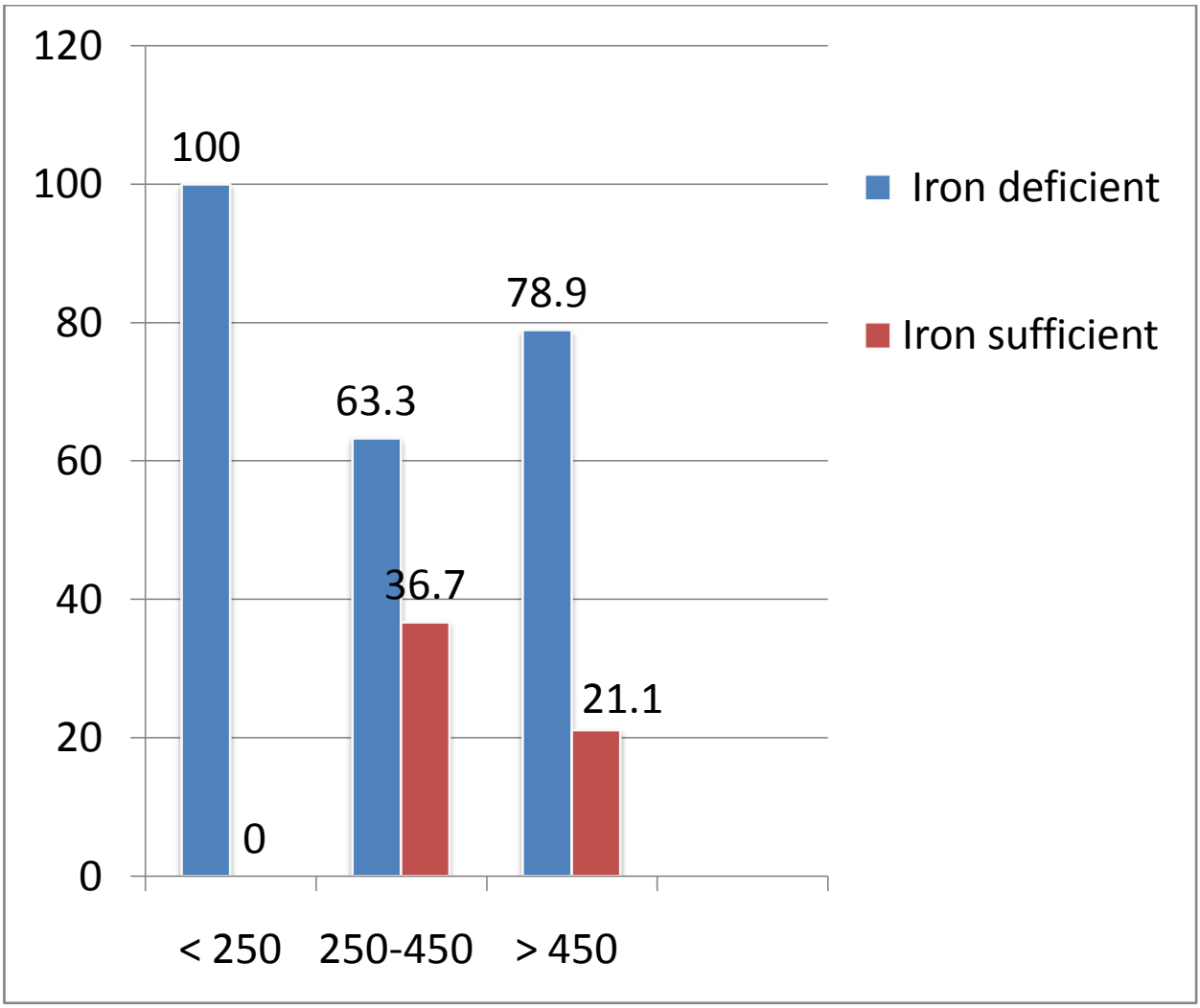

Table 15 :Association between RDW(%) and iron status

RDW	Iron deficient		Iron sufficient		Total	
	No .	%	No.	%	No.	%
11.5-14.5	7	87.5	1	12.5	8	100
>14.5	28	66.7	15	33.3	42	100
Total	35		15		50	

Chi-Square Tests					
	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	1.389[a]	1	.239		
Continuity Correction[b]	.574	1	.449		
Likelihood Ratio	1.591	1	.207		
Fisher's Exact Test				.407	.232
Linear-by-Linear Association	1.361	1	.243		
N of Valid Cases	50				

Association between RDW(%) and iron status

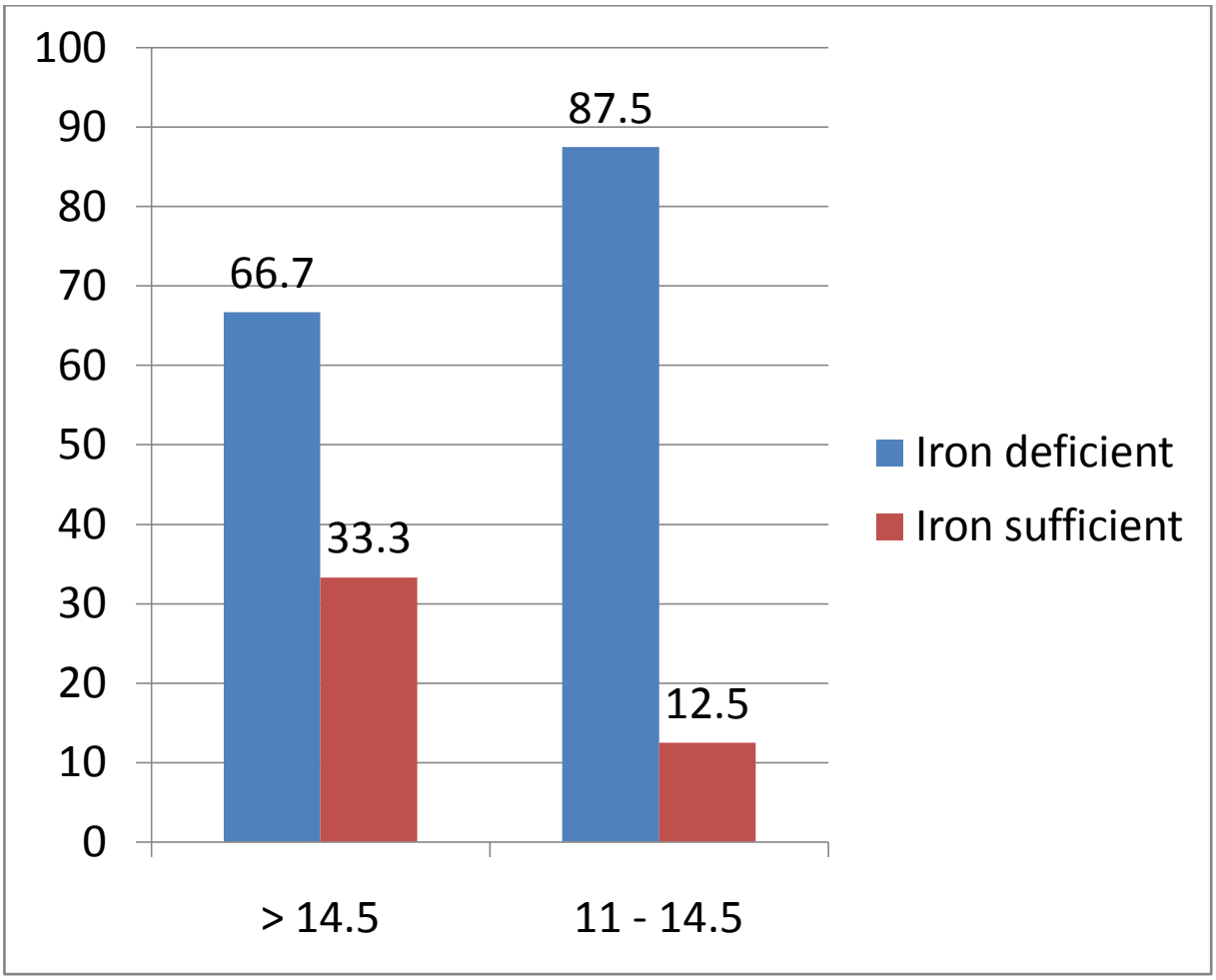

Table 16 :Association between Sr.ferritin and iron status

Serum ferritin	Iron deficient		Iron sufficient		Total	
	No .	%	No.	%	No.	%
< 15	27	100	0	0	27	100
>15	8	34.8	15	65.2	23	100
Total	35		15		50	

Chi-Square Tests					
	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	25.155[a]	1	**.000**		
Continuity Correction[b]	22.146	1	**.000**		
Likelihood Ratio	31.366	1	**.000**		
Fisher's Exact Test				.000	.000
Linear-by-Linear Association	24.652	1	**.000**		
N of Valid Cases	50				

Association between Sr.ferritin (ng/dl) and iron status

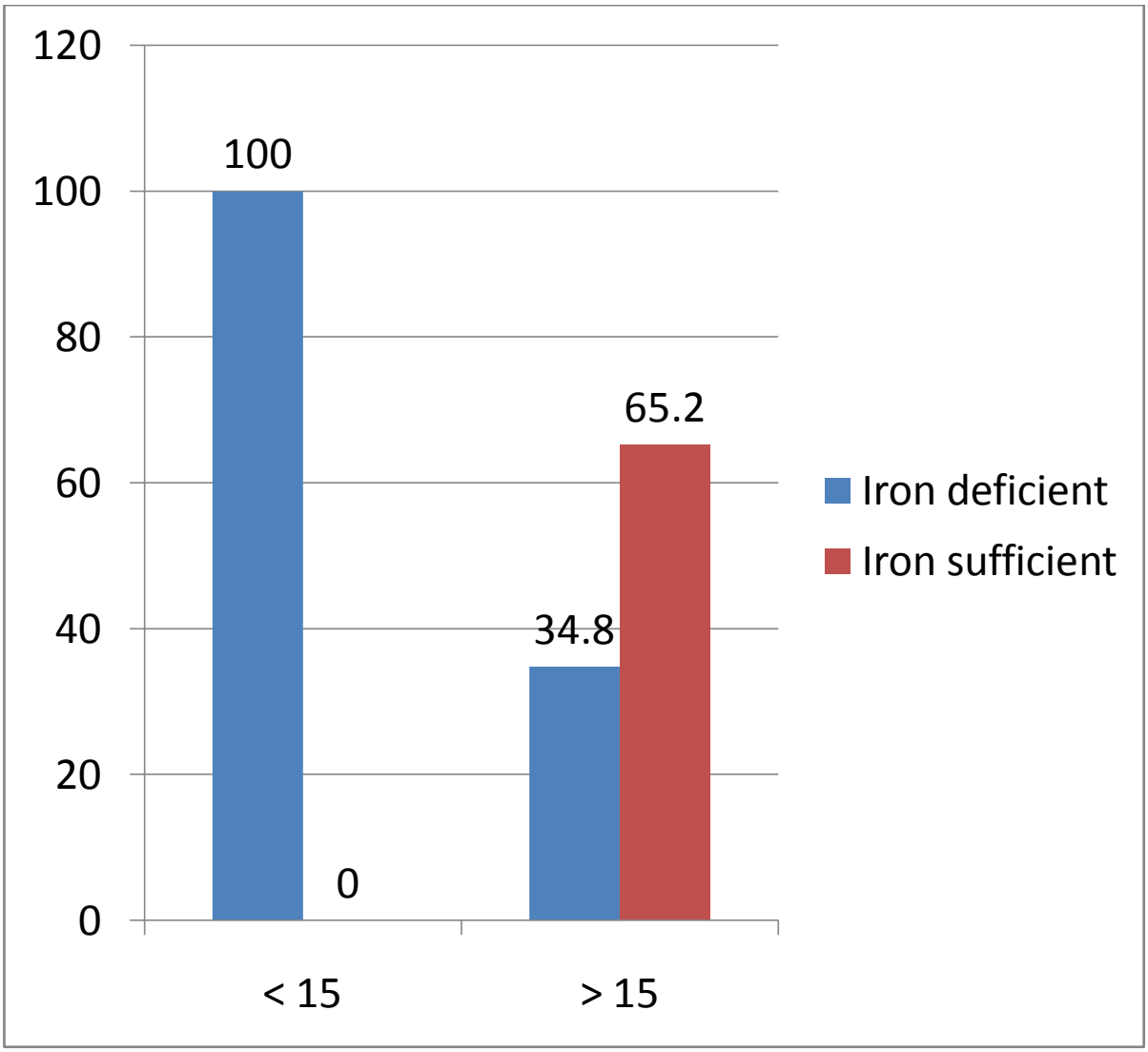

Table 17:Association between RBC Count (million/cu.mm) and iron status

RBC count	Iron deficient		Iron sufficient		Total	
	No .	%	No.	%	No.	%
< 4.5	11	55	9	45	20	100
>4.5	24	80	6	20	30	100
Total	35		15		50	

Chi-Square Tests					
	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	3.571[a]	1	.059		
Continuity Correction[b]	2.480	1	.115		
Likelihood Ratio	3.537	1	.060		
Fisher's Exact Test				.114	.058
Linear-by-Linear Association	3.500	1	.061		
N of Valid Cases	50				

Association between RBC Count (million/cu.mm) and iron status

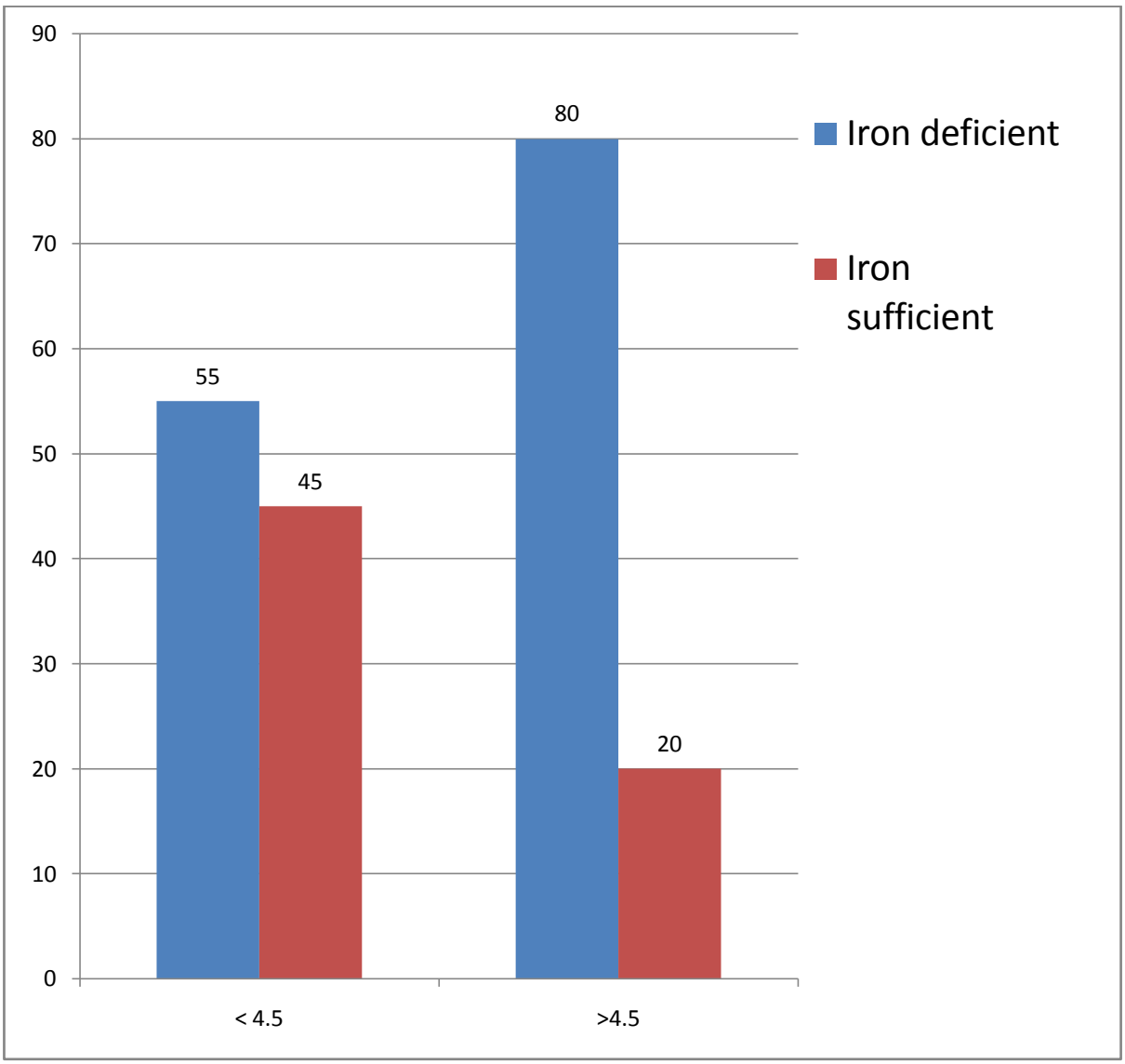

Table 18: Association between Platelet Count (lakhs/cu.mm) and iron status

Platelet count	Iron deficient		Iron sufficient		Total	
	No .	%	No.	%	No.	%
< 1.5	3	42.9	4	57.1	7	100
1.5 -4.0	30	76.9	9	23.1	39	100
>4	2	50	2	50	4	100
Total	35		15		50	

Chi-Square Tests			
	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	4.108[a]	2	.128
Likelihood Ratio	3.845	2	.146
Linear-by-Linear Association	.522	1	.470
N of Valid Cases	50		

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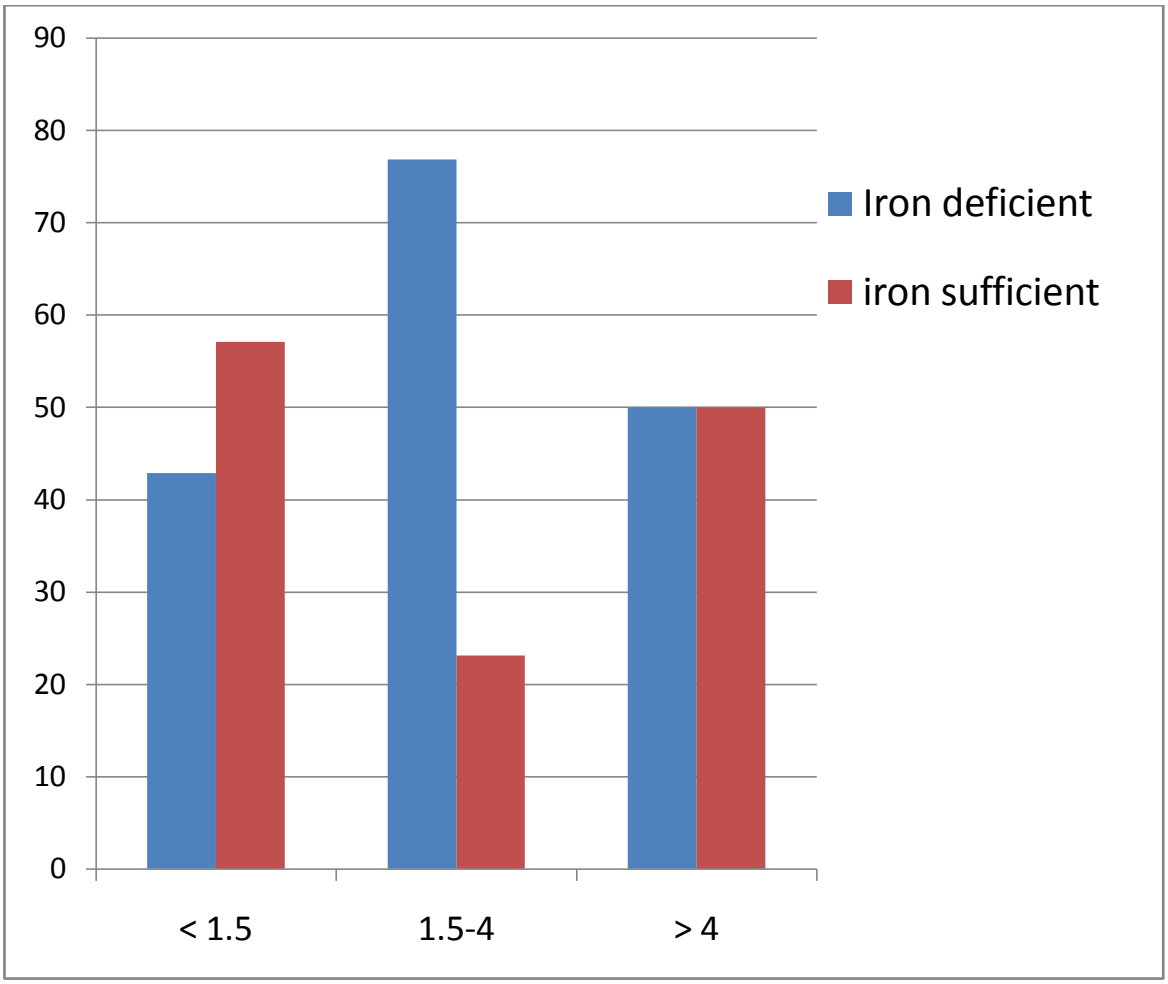
Association between Platelet Count (lakhs/cu.mm) and iron status



DISCUSSION

In this study ,50 children with congenital cyanotic heart disease were included and investigated by doing complete blood count and iron profile . Based on the transferrin saturation ,they were categorised into two groups as iron deficient and iron sufficient. Children who had a transferrin saturation level < 16% were grouped as iron deficient and those who had a transferrin saturation level >16% were grouped as iron sufficient.

In table 1(age distribution among cases), out of 50 children included in this study, 12 (24%) were less than 1 year ,24(48%) children were between 1 to 6 years and 14 (28%) children were in between 6 to 12 years. Most of the children were in the age group of 1 to 6 years.

In table 2 (sex distribution among cases) ,Among the total of 50 patients,25(50%)were male and 25(50%) were female.

In table 3: Distribution of cyanosis among cases :

40 cases(80%) have cyanosis, 10 cases(20%) donot have cyanosis.

In table 4 :Distribution of clubbing among cases :

35 cases(70%) have clubbing, 15 cases(30%) donot have clubbing.

In table 5,Based on transferrin saturation, children were grouped into iron deficient and iron sufficient.35(70%) children who had transferrin saturation of < 16% and 15(30%) children had transferrin saturation of >16%. In this study **the prevalence of iron deficiency among children with cyanotic congenital heart disease was 70%.** In other similar studies, cemilebanu et al[37]the prevalence of iron deficiency anemia was 63.6% and Lango et al[40] the prevalence was 16.9%.

In table 6, we see that, out of 12 children who were <1 year of age, 6(50%) were iron deficient and 6(50%) were iron sufficient, and out of 24 patients between 1-6 years age group,20 (83.3%) were iron deficient and 4 (16.7%) were iron sufficient. Among 14 children between 6-12 years age group,9(64.3%) were iron deficient and 5(35.7%) were iron sufficient. This shows that iron deficiency is more common between 1-6 years. This finding is consistent with the finding of NFHS 3, where maximum number of children with iron deficiency anaemia lie between 6 -35 months of age. But there was no statistical association between age of the patient and iron status (p=0.0104.).

From table 7,among 25 male children,16 (64%) were iron deficient and 9(36%) were iron sufficient .Out of 25 female children 19(76%) were iron deficient and 7 (24%) were iron sufficient. He we observed females

are more prone to develop iron deficiency anaemia. But there was no statistical association between sex of the patient and iron status (p=0.355)

Table 8 shows, out of 8 children who had Hemoglobin of < 11.5 gm%,4 (50%) were iron deficient and 4(50%) were iron sufficient.Out of 17 children who had haemoglobin between 11.5-14gm%,11(64.7%) were iron deficient and 6(35.3%) were iron sufficient. Out of 25 children who had haemoglobin >14gm%,20(80%) were iron deficient and 5(20%) were iron sufficient. Even in children with a haemoglobin of > 14 , 80 % were iron deficient . There is no statistical association between haemoglobin and iron status (p= 0.230). Hence haemoglobin was not a significant parameter to determine iron deficiency. This finding is consistent with the study done by cemilebanu et al(37) and david et al[44] where haemoglobin was not considered as a significant parameter in the diagnosis of iron deficiency anaemia.

Table 9 shows ,out of 4 children who had haematocrit of < 34,2 (50%) were iron deficient and 2(50%) were iron sufficient. out of 33 children who had haematocrit between 34-60,20(60.6%) were iron deficient and 13(39.4%) were iron sufficient. out of 13 children who had haematocrit of >60%,13 all (100%) were iron deficient . Here we observe that in patients with a haematocrit of > 60%, 100 % are iron deficient. There is statistical association between haematocrit and iron status

(p=0.021). This is similar to olcay et al[38] where haematocrit was a significant parameter in determining iron status.(p <0.05)

Table 10 shows ,Out of 35 children who had MCV level of < 75,29 (82.9%) were iron deficient and 6(17.1%) were iron sufficient. Out of 14 children who had MCV level between 75-100 ,6(42.9%) were iron deficient and 8(57.1%) were iron sufficient,and only one child (100%) had MCV level of >100 fl. there was statistical significant association between MCV level of the patient and iron status (p=0.07) . This is comparable to study done by L.Olcay et al[38] and cemilebanu et al[37] where they concluded MCV is an important indicator of iron deficiency in CCHD.

Table 11 shows ,Out of 37 children who had MCH of < 24,27 (73%) were iron deficient and 10(27%) were iron sufficient. out of 13 children who had MCH of >24,8(61.5%) were iron deficient and 5(38.5%) were iron sufficient.There was no statistical significant association between MCH level of the patient and iron status (p= 0.439). This was similar to study done by L.Olcay et al[38] and M Gaiha et al[45].

Table 12 shows, out of 27 children who had MCHC of < 31,19 (70.4%) were iron deficient and 8(29.6%) were iron sufficient. out of 23 children who had MCHC of >31,16(69.6%) were iron deficient and 7(30.4%) were iron sufficient. There was no statistical significant association between MCHC level of the patient and iron status (p=0.951) .

This was in contrast to Drossos[39] et al where they concluded MCHC is a precise indicator for diagnosis of iron deficiency anaemia in children with cyanotic heart disease.

Table 13 shows, out of 20 children who had serum iron level of < 50 mg%, All (100%) were iron deficient. out of 23 children who had serum ironlevel between 50-100 ,15(65.2%) were iron deficient and 8(34.8%) were iron sufficient, and 7 children (100%) had serum iron level of >100 mg%. There was statistical significant association between serum iron level of the patient and iron status (p=0.000). This is comparable to study done by Gaiha et al [45]and Cemilebanu et al[38] where they concluded serum iron is an important indicator of iron deficiency in CCHD.

Table 14 shows, only one(100%) child had TIBC of < 250 ,who was iron deficient. out of 30 children who had TIBC between 250-450,19(63.3%) were iron deficient and 11(36.7%) were iron sufficient. out of 19 children who had TIBC of >450,15(78.9%) were iron deficient and 4(21.1%) was iron sufficient. There was no statistical significant association between serum iron level of the patient and iron status (p=0.280).

Table 15 shows, out of 8 children who had RDW between 11.5-14.5, 7(87.5%) were iron deficient and 1(12.5%) were iron sufficient. out of 42 children who had RDW of >14.5, 28(66.7%) were iron deficient

and 14(33.3%) were iron sufficient. There was no statistical significant association between RDW of the patient and iron status (p=0.239).

Table 16 shows, out of 27 children who had serum ferritin between < 15, 27 all (100%) were iron deficient .Out of 23 children who had Serum ferritin of >15,8(34.8%) were iron deficient and 15(65.2%) were iron sufficient. There was statistical significant association between serum ferritin of the patient and iron status (p=0.000).

Table 17shows, out of 20 children who had RBC count < 4.5 ,11(55 %) were iron deficient and 9(45%) were iron sufficient. out of 30 children who had RBC count of >4.5,24(80%) were iron deficient and 15(65.2%) were iron sufficient. There was no statistical significant association between RBC count of the patient and iron status (p=0.059).

Table 18 shows ,Out of 7 children who had platelet count of < 1.5,3 (42.9%) were iron deficient and 4(57.1%) were iron sufficient. out of 39 children who had platelet count of 1.5-4,30(76.9%) were iron deficient and 9(23.1%) were iron sufficient. Out of 4 children who had a platelet count of >4,2(50%) were iron sufficient and 2(50%) were iron sufficient. There was no statistical significant association between platelet count of the patient and iron status (p=0.128).

CONCLUSION

1. Prevalence of iron deficiency anaemia in children with cyanotic congenital heart disease was 70%
2. Iron deficiency anaemia was more common among 1 – 6 yrs(83.3%) and female children(76%), though there was no statistical association.
3. 82.9 % of cases with iron deficiency anaemia had MCV < 72 fl. There was statistical significant association between MCV level of the patient and iron status (p=0.07).
4. Raised HCT> 60% was found in 100 %(13 cases) of children with iron deficiency anaemia in children with cyanotic congenital heart disease . There was statistical significant association between HCT of the patient and iron status (p=0.021).
5. 20(100%) of children who had sr .iron < 50 mg/dl were iron deficient . There was statistical significant association between iron status of the patient and iron status (p=0.00).
6. 27(100%) of children who had sr .ferritin < 15 ng/dl were iron deficient . There was statistical significant association between serum ferritin status of the patient and iron status (p=0.00).

SUMMARY

Iron deficiency anaemia is a common nutritional anaemia, which is often underestimated in children with cyanotic congenital heart disease. Presence of microcytic anaemia in cyanotic congenital heart disease can lead to hyperviscosity which can further lead to cerebrovascular accidents and death. Hence prevention of anaemia in cyanotic congenital heart disease patients is extremely important.

Definitive tests like serum ferritin, serum iron,TIBC are expensive and not freely available. Hence we made an attempt to study simple tests to determine the iron status in children with cyanotic congenital heart disease.

50 children with cyanotic congenital heart disease were enrolled in the study. Based on the transferrin saturation level, a cut off value of < 16% was taken to be iron deficient. This was compared against age ,sex ,haemoglobin, haematocrit, RDW, MCV, MCH, MCHC, Platelet count ,serum iron, TIBC and serum ferritin.

Age distribution among cases :

12 (24%) - < 1 year ,24(48%) -1 to 6 years and 14 (28%) - 6 to 12 years.

Sex distribution among cases :

25(50%)- male and 25(50%) -female .

Based on transferrinsaturation:

Iron deficient - 35(70%) and iron sufficient- 15(30%)

Distribution of cyanosis among cases :

40 cases(80%) have cyanosis, 10 cases(20%) donot have cyanosis.

Distribution of clubbing among cases :

35 cases(70%) have clubbing, 15 cases(30%) donot have clubbing.

Hemoglobin and iron status :

Hb% < 11.5 gm%-4(50%) were iron deficient and 4(50%) were iron sufficient, Between 11.5-14gm% - 11(64.7%) were iron deficient and 6(35.3%) were iron sufficient ,>14gm% ,20(80%) were iron deficient and 5(20%) were iron sufficient.

Haematocrit and iron status :

HCT < 34,2 (50%) were iron deficient and 2(50%) were iron sufficient, between 34-60,20(60.6%) were iron deficient and 13(39.4%) were iron sufficient,>60%,13(100%) were iron deficient .

MCV level and iron status:

MCV< 75,29 (82.9%) were iron deficient and 6(17.1%) were iron sufficient, between 75-100 ,6(42.9%) were iron deficient and 8(57.1%) were iron sufficient,and>100 fl - 1 child (100%).

MCH level and iron status :

MCH < 24,27 (73%) were iron deficient and 10(27%) were iron sufficient. >24, 8(61.5%) were iron deficient and 5(38.5%) were iron sufficient.

MCHC and iron status:

MCHC < 31,19 (70.4%) were iron deficient and 8(29.6%) were iron sufficient, >31,16(69.6%) were iron deficient and 7(30.4%) were iron sufficient.

Serum iron level and iron status:

serum iron < 50 mg%, 20,all (100%) were iron deficient, between 50-100, 15(65.2%) were iron deficient and 8(34.8%) were iron sufficient, and 7 (100%) had serum iron level of >100 mg%.

TIBC and iron status:

TIBC <250 ,1 child(100%) iron deficient, Between 250-450,19(63.3%) were iron deficient and 11(36.7%) were iron sufficient,>450,15(78.94%) were iron deficient and 4(21.1%) was iron sufficient.

RDW and iron status:

RDW between 11.5-14.5 ,7(87.5%) were iron deficient and 1(12.5%) were iron sufficient, >14.5,28(66.7%) were iron deficient and 14(33.3%) were iron sufficient.

Serum ferritin and iron status:

Serum ferritin < 15,27(100%) were iron deficient, >15,8(34.8%) were iron deficient and 15(65.2%) were iron sufficient.

RBC COUNT and iron status:

RBC Count <4.5 ,11(55%) were iron deficient and 9(45%) were iron sufficient, >4.5,24(80%) were iron deficient and 6(20%) were iron sufficient.

PLATELET COUNT and iron status:

Platelet count <1.5 ,3 (42.9%) iron deficient and 4(57.1) were iron sufficient, Between 1.5-4,30(76.9%) were iron deficient and 9(23.1%) were iron sufficient, >4,2(50%) were iron deficient and 2(50%) was iron sufficient.

Only haematocrit, MCV,serumiron,serum ferritin had significant correlation with iron deficiency in children with cyanotic congenital heart disease.

Hence we concluded **Haematocrit And MCV** levels can used to determine iron deficiency in children with cyanotic congenital heart disease in resource poor settings.

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PROFORMA

- NAME: CARDIOLOGY NO:
- AGE:
- SEX:
- ADDRESS:
- CONSANGUINITY:
- **GENERAL EXAMINATION :**

	yes	no
CYANOSIS:	☐	☐
CLUBBING:	☐	☐

- **INVESTIGATIONS:**

HAEMOGLOBIN:	
RBC COUNT:	
PLATELET COUNT:	
HAEMATOCRIT:	
MEAN CORPUSCULAR VOLUME:	

MEAN CORPUSCULAR HAEMOGLOBIN CONCENTRATION:	
RED CELL DISTRIBUTION WIDTH:	
SERUM IRON:	
TOTAL IRON BINDING CAPACITY:	
SERUM FERRITIN:	
TRANSFERRIN SATURATION:	
PERIPHERAL SMEAR:	

ELECTROCARDIOGRAPHY:

2D-ECHO:

CHEST X-RAY:

TREATMENT TAKING:

COMPLICATION:

ABBREVIATIONS

CCHD	**-**	**CYANOTIC CONGENITAL HEART DISEASE**
Hb	**-**	**HAEMOGLOBIN**
HCT	**-**	**HAEMATOCRIT**
RBC	**-**	**RED BLOOD CELL**
MCV	**-**	**MEAN CORPUSCULAR VOLUME**
MCH	**-**	**MEAN CORPUSCULAR HAEMOGLOBIN**
MCHC	**-**	**MEAN CORPUSCULAR HAEMOGLOBIN CONCENTRATION**
TIBC	**-**	**TOTAL IRON BINDING CAPACITY**
RDW	**-**	**RED CELL DISTRIBUTION WIDTH**
sTFR	**-**	**TRANSFERRIN SATURATION**

Ref. No. 14290 /E4/3/2012

Govt. Rajaji Hospital,
Madurai.20. Dated: . 12.2012

Institutional Review Board / Independent Ethics Committee.
Dr. N. Mohan, M.S., F.I.C.S., F.A.I.S.,
Dean, Madurai Medical College &
Govt Rajaji Hospital, Madurai- 625020.
Convenor
grhethicssecy @gmail.com.

Sub: Establishment-Govt. Rajaji Hospital, Madurai-20-
Ethics committee Meeting- approval -regarding.

The Ethics Committee meeting of the Govt. Rajaji Hospital, Madurai was held at 10.00 am to 12.30.Pm on 10.12.2012 at the Surgery Seminar Hall, Govt. Rajaji Hospital, Madurai. The following members of the committee have attended the meeting.

1.Dr. V. Nagarajan, M.D., D.M (Neuro) Ph: 0452-2629629 Cell.No 9843052029	Professor of Neurology (Retired) D.No.72, Vakkil New Street, Simmakkal, Madurai -1	Chairman
2. Dr.Mohan Prasad , M.S M.Ch Cell.No.9843050822 (Oncology)	Professor & H.O.D of Medical Oncology(Retired) D.No.72, West Avani Moola Street, Madurai -1	Member Secretary
3. Dr.L. Santhana Lakshmi.MD Cell.No 9842593412	Associate Professor of Physiology/V.P Madurai Medical College	Member
4. Dr. Parameswari M.D (Pharmacology) Cell.No.9994026056	Director of Pharmacology Madurai Medical College	Member
5. Dr.Moses K.Daniel MD(Gen.Medicine) Cell.No 09842156066	Professor & H.O.D of Medicine Madurai Medical College	Member
6. Dr.D. Soundara Rajan,MS(Gen.Surgery) Cell.No 9842120127	Professor & H.O.D of Surgery Madurai Medical College	Member
7. Dr.Angayarkanni MD(O&G) Cell.No 9443567724	Professor & H.O.D of O&G Madurai Medical College	Member
8. Dr.P.V. Pugalenthi M.S, (Ortho) Cell.No 9443725840	Professor & H.O.D Ortho Madurai Medical College	Member
9. Dr. M. Sundarajan M.S., Mch Cell.No 9994924369 (Neuro Surgery)	Professor (Neuro Surgery) Madurai Medical College	Member
10 Thiru..Pala. .Ramasamy , BA.,B.L., Cell.No 9842165127	Advocate, D.No.72.Palam Station Road, Sellur, Madurai -2	Member
11. Thiru. P.K.M. Chelliah ,B.A Cell.No 9894349599	Businessman, 21 Jawahar Street, Gandhi Nagar, Madurai-20.	Member

The following Project was approved by the committee

Name of P.G.	Course	Name of the Project	Remarks
K. Meenalosini	PG in M.D. Pediatrics Govt. Rajaji Hospital Madurai.-20	Iron profile in Children with congenital cyanotic heart disease.	Approved

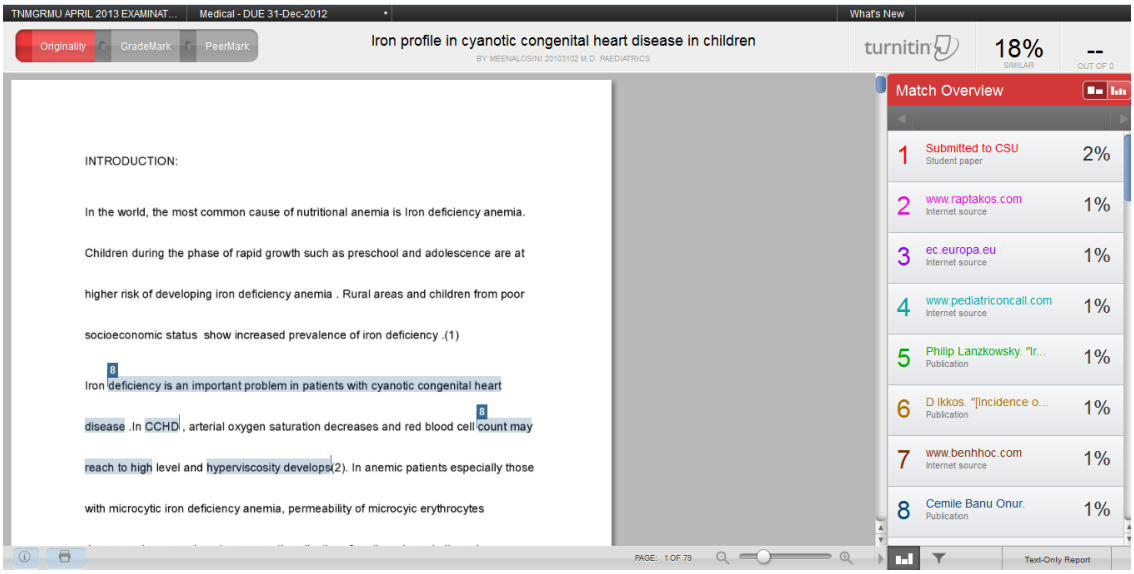
Please note that the investigator should adhere the following: She/He should get a detailed informed consent from the patients/participants and maintain Confidentially.

1. She/He should carry out the work without detrimental to regular activities as well as without extra expenditure to the institution to Government.
2. She/He should inform the institution Ethical Committee in case of any change of study procedure site and investigation or guide.
3. She/He should not deviate for the area of the work for which applied for Ethical clearance. She/He should inform the IEC immediately, in case of any adverse events pr Serious adverse reactions.
4. She/he should abide to the rules and regulations of the institution.
5. She/He should complete the work within the specific period and apply for if any Extension of time is required She should apply for permission again and do the work.
6. She/He should submit the summary of the work to the Ethical Committee on Completion of the work.
7. She/He should not claim any funds from the institution while doing the word or on completion.
8. She/He should understand that the members of IEC have the right to monitor the work with prior intimation.

Member Secretary **Chairman**

DEAN/Convenor
Govt. Rajaji Hospital.
Madurai- 20.

To
The above PG student - thro' Head of the Department concerned.


TNMGRMU APRIL 2013 EXAMINAT...
Medical - DUE 31-Dec-2012
What's New
Originality
GradeMark
PeerMark
Iron profile in cyanotic congenital heart disease in children
BY MEENALOSINI 20103102 M.D. PAEDIATRICS
turnitin
18%
SIMILAR
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OUT OF 0
INTRODUCTION:
In the world, the most common cause of nutritional anemia is Iron deficiency anemia.
Children during the phase of rapid growth such as preschool and adolescence are at
higher risk of developing iron deficiency anemia . Rural areas and children from poor
socioeconomic status show increased prevalence of iron deficiency .(1)
Iron deficiency is an important problem in patients with cyanotic congenital heart
disease .In CCHD , arterial oxygen saturation decreases and red blood cell count may
reach to high level and hyperviscosity develops(2). In anemic patients especially those
with microcytic iron deficiency anemia, permeability of microcyic erythrocytes
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MASTER CHART

S.NO	Name	Age	Sex	Diagnosis	cyanosis 0- Absent , 1- Present	clubbing 0- Absent , 1- Present	hb %	rbc count	platelets	HCT	MCV	MCH	MCHC	RDW	sr.iron	TIBC	transferrin saturation	sr.ferritin
1	Iyyammal	9	F	TOF/dextrocardia	1	1	21.3	10.06	0.55	82.7	82.2	21.2	25.8	25.9	72	375	15	11.6
2	Dinesh Kumar	7	M	TOF	1	1	10.1	5.73	3.66	39.7	69.3	17.6	25.4	21.5	18	557	3	10.7
3	vaishnavi	5	F	pul.atresia/LVSD/MAPCOS SCA	1	1	21.3	9.37	0.8	77.6	82.8	22.7	27.4	26.2	68	477.4	14	13
4	Santha Kumar	6	M	DORV,VSD,PS	1	1	15.6	4.33	4.4	45.04	86	29.8	34.7	18.6	48	356	13.8	8.8
5	Muthumuneeswaran	10	M	TOF	1	1	20.4	5.67	2.67	60.21	66	22.2	33.9	25	56	428	13.08	8.4
6	Aarthy	2	F	DORV,TGA	0	0	22.1	10.26	1.82	79	77	21.5	28	28.5	10	768	1	5.2
7	Pandeeshwari	4.5	F	TOF	1	1	15.4	6.05	2.86	48.32	71.22	18.6	31.87	16	46.6	354	12.99	11.5
8	Shrishanth Vijay	2	M	Single ventricle,PS	1	1	13.6	4.7	3.28	38	85.1	16.4	30.6	22.4	32	322	10	32.5
9	Uma Maheshwari	8	F	TOF	1	1	10.2	3	2.9	36	68	16.2	28.3	19.2	15	535	3	18.8
10	Vignesh kumar	3	M	TOF	1	1	17.9	9.8	1.77	68.6	70	18.3	26.1	12.2	14	420	3	10
11	Divya	3	F	TOF	1	1	13.2	3.8	2.36	38	59	14.2	35.78	20	28	426	6.57	12
12	Nandagopal	7.5	M	DORV	1	1	10.6	2.7	3.28	34.2	76.2	16.4	33.9	22.4	62.22	364.46	17	134
13	RamKumar	2	M	TOF	1	0	11.8	3.25	3.24	35	71	23	31.42	16	29.41	250	11.5	9
14	Dhanasree	5	Fch	TA &PS	1	1	14.2	3.92	1.88	42.34	60.2	18.2	33.5	13.6	28.2	628	4.49	52
15	Ajay	10m	mch	DORV,VSD,PA,MAPCA	0	0	11.7	2.86	4.18	36.1	54.6	17.7	32.4	17.7	116.27	382.92	30.36	215
16	Ganesan	10	mch	d-TGA,VSD,PS	1	1	14.6	5.7	1.41	53.44	90	24.7	27.4	18.8	96.96	321.96	30.2	158
17	Muniyasamy	4	mch	TOF	1	1	22.99	8.86	0.78	74.31	63	25.5	30.9	12.2	62.86	482.36	13.86	15.7
18	Karthikeyan	12	mch	TOF	1	1	21	9.01	1.6	73	68.5	22.4	28.5	12.8	52.36	492	10.64	11.1
19	Saravanan	8	mch	VSD/ PS	1	1	18.4	7.56	1.9	65.7	69.75	19.53	28.01	60	56.39	428	13.86	13.5
20	Dharaneshwaran	3	mch	TOF,MAPCA	1	1	12.6	3.75	4.48	56	68	26	22.5	16	39.36	410.3	9.59	8.4

21	Rajesh	1.5	mch	LASD,VSD,PS	0	0	11.6	3.62	1.96	32.76	56	22.3	29.9	18.6	28	327.34	8.55	9.5
22	Dhanalakshmi	7	fch	TOF	1	1	13.6	4.75	2.36	38	59	14.2	35.78	20	28	426	6.57	6
23	Logavarshini	2.5	fch	LVSD,PS	1	1	11.8	3.86	3.24	35	71	23	31.42	16	29.41	255.51	11.51	12
24	Thanga	6.5	fch	SA TAPVC,PAH	1	1	12.6	3.45	2.84	38	82	28	37	21	104	506	24	84
25	Sujithra	1	fch	TAPVC	0	0	13.7	2.8	0.98	32	51	20	30.68	17	59	356	28	68
26	Vaishnavi	5	fch	PDA/VSD/MAPCOS	1	1	16.2	5.2	3.27	56.4	82	21.5	23.86	16	54	596	30.6	92
27	Boomika	1.5	fch	SV PS,OS VSD	0	0	9.8	2.14	2.37	35.8	64	18.6	27.3	20.7	171	536	31.95	112
28	Sivapandi	11	mch	TGV	1	1	11.7	3.56	4.18	36	78.6	17.7	32.4	17.7	116.27	382.92	30.36	150
29	Dhanalakshmi	2	fch	TA/LVSD/	0	0	21	7.5	2.6	73	68.5	22.4	28.5	22.8	52.36	492	10.64	13.4
30	Arunkumar	7	mch	Dextrocardia/DORV	1	1	17.5	6.82	2.5	58.9	87	25.6	29.6	18.7	97.22	389.26	24.07	88
31	Abinayashree	7 m	fch	VSD/PA,PDA,MAPCOS	0	0	11.5	3.1	2.6	49.1	71.8	22	23.46	18	13.45	328	3.99	7.5
32	Rathinakumar	2.5	mch	TA-Type 2b	1	1	13.6	4	2.36	38	59	14.2	35.78	20	28	426	6.57	9.4
33	Ruba	7 m	fch	LVSD,PA	0	0	9.8	2.5	2.37	35.4	64	18.6	27.3	20.7	171.05	387.72	44.11	76
34	Hariharan	8m	mch	TOF	0	0	10.62	3.02	0.9	31.4	52.6	16.5	30.9	24.2	62	472	19	102
35	Akalya	8 m	fch	TOF,PA,MAPCOS	0	0	17.9	8.65	1.05	56	117	20.6	34.6	16	161.76	387.76	41.73	64
36	Vuppili	6 m	mch	PA/VSD/MAPCOS	0	0	12.2	4.2	3.03	38	69	19.2	32.1	19.6	56.6	462.34	12.24	13.58
37	Sarika	8 m	fch	C CHD/DORV/VSD	1	0	15.2	5.4	2.11	45.68	71	18.98	33.27	13.6	42	386	10.88	17.42
38	Sudansrinivasan	8 m	mch	TOF	1	0	14.8	4.8	3.22	43	68	24.4	34.41	22	48.2	380	12.68	30.46
39	Iyyanar	9m	mch	TA/LVSD/PS	1	0	11.6	3.33	3.28	34.2	58.2	16.4	33.9	22.4	62.22	364.46	17.07	54
40	kannan	1	m	TOF	1	1	16.6	5.82	2.3	50	84	24.8	30	17.2	84	351	18.23	81
41	muthu mahalakshmi	8	f	TOF	1	1	23.8	9.2	3.12	77	66	24.7	32.6	13.4	60.25	472	12.52	14.82
42	kanagarani	10 m	f	TGV / MOD, PS	1	1	14.3	4.6	3.42	52	72	21.8	36.4	18	58	416	13.5	13.74
43	chinnaponnu	8 m	f	TOF	1	1	19.2	7.25	2.67	72	24	26.4	38.2	12.8	54	342	14.8	16.7
44	eswari	4	f	DORV/ PS	1	1	22.5	11.2	1.8	84	86.5	23.4	26.2	27	84	412	16	56
45	karrupusamy	1.5	m	TGA /PS	1	1	11.4	6.34	2.82	35.6	68.7	18.2	24.6	23	23	587	9	10.4
46	hemalini	2.5	f	D TGA	1	1	12.6	7.4	2.9	38	66	25.2	32	19	52	502	12	10.85
47	mareeswaran	10 m	m	RPA Stenosis/vsd	1	1	13.2	7.8	1.9	42	88	26.4	32.8	14	102	315	18	94
48	vijay	3.5	m	TOF	1	1	16.5	8.6	2	60	70	22.5	27	18	54	462	13	11.72
49	rohini	5	f	TOF	1	1	10.9	6	2.26	33	64	21	25.6	17	48	512	12.5	12
50	saradha	10	f	TOF	1	1	18.4	9.1	3.2	65	58	26.5	29.3	26	59	457	14	12.38